

Am Vet Med Assoc. Author manuscript; available in PMC 2014 October 07.

Published in final edited form as:

J Am Vet Med Assoc. 2011 April 1; 238(7): 915–921. doi:10.2460/javma.238.7.915.

Ocular lesions associated with systemic hypertension in dogs: 65 cases (2005–2007)

Nicole L. LeBlanc, DVM, Rebecca L. Stepien, DVM, MS, DACVIM, and Ellison Bentley, DVM, DACVO

Departments of Medical Sciences (LeBlanc, Stepien) and Surgical Sciences (Bentley), School of Veterinary Medicine, University of Wisconsin, Madison, WI 53706

Abstract

Objective—To characterize ocular findings in hypertensive dogs, determine prevalence of hypertension in dogs with ocular disease suggestive of hypertension, and examine possible relationships between degree of hypertension and ocular disease.

Design—Retrospective case series.

Animals—65 dogs initially referred for blood pressure measurement (n = 22), ophthalmic examination (25), or both (18).

Procedures—Medical records were reviewed to identify dogs examined at the teaching hospital that underwent a complete ophthalmic examination and blood pressure measurement within a 24-hour period between January 1, 2005, and December 31, 2007. Signalment, history, blood pressure measurements, ophthalmic examination findings, and any vasoactive drug treatments were recorded. Ocular lesions considered likely to be associated with systemic hypertension included retinal hemorrhage, retinal detachment, hyphema, tortuous vessels, and subretinal edema.

Results—Of the 65 dogs, 42 were hypertensive (systolic blood pressure 160 mm Hg) and 23 were normotensive. Sixty-two percent (26/42) of hypertensive dogs had 1 type of ocular lesion identified. Retinal hemorrhage was the most common ocular lesion in hypertensive dogs (17/42 [40%]). The presence of 1 type of ocular lesion had moderate sensitivity and specificity of 62% and 61%, respectively, for identification of hypertension. Fifteen of the 25 (60%) dogs referred for blood pressure measurement after initial ophthalmic examination were found to be hypertensive.

Conclusions and Clinical Relevance—Ocular lesions are common in dogs with systemic hypertension. Dogs with hypertension or diseases associated with hypertension should be monitored carefully for evidence of ocular target organ damage, and hypertension should be systematically ruled out in dogs with characteristic ocular lesions.

Systemic hypertension is defined as persistently high arterial blood pressure. Systemic hypertension is an increasingly recognized source of morbidity and, in some instances, death in human and veterinary patients with certain systemic diseases. ^{1–6} Overt and sometimes devastating damage caused by hypertension is typically found in the eyes, ^{4,7} CNS, ^{8,9}

heart, ^{6,10–12} and kidneys. ^{5,7} Injury related to hypertension in these organ systems is often collectively referred to as TOD and is generally considered an indication for treatment with antihypertensive drugs. Hypertension in people can occur as a primary disease process (ie, essential hypertension) or, less commonly, secondary to renal, cardiovascular, neural, or endocrine diseases. ¹³ In contrast, naturally occurring primary (idiopathic) hypertension has been found in dogs, but appears to be rare. ^{14–18} Systemic hypertension in dogs is most frequently diagnosed in association with systemic diseases, including acute or chronic renal failure, protein-losing nephropathies, hyperadrenocorticism, diabetes mellitus, pheochromocytoma, and other less common diseases. ^{19–21}

The pathogenesis of ocular lesions in response to hypertension involves an initial period of arteriolar vasoconstriction, presumably due to autoregulatory mechanisms. ²² If the hypertension persists, possible occlusion and ischemic necrosis of vasculature follow with subsequent increased vascular permeability. ²² Vascular changes within the choroid may result in subretinal fluid and potential RD. ²³ Differential diagnoses for these ocular lesions other than hypertension include coagulopathies, plasma cell myeloma and associated hyperviscosity syndrome, infectious disease manifestations, lymphosarcoma, polycythemia, and systemic lupus erythematosus. ^{22,24,25}

Visual disturbance is often the initial sign of systemic hypertension reported for both dogs and cats. A, 26–28 Acute onset of blindness due to intraocular hemorrhage, RD, or secondary glaucoma may be the initial clinical sign for both species. Acute oset of blindness due to intraocular hemorrhage, RD, or secondary glaucoma may be the initial clinical sign for both species. Acute oset, the most commonly reported ocular lesions include hyphema, intraretinal edema and SRE, RH, retinal TV, and RD. A, 26, 27, 29 In people, ocular TOD in response to hypertension is manifest as a triad of independent clinical syndromes, including hypertensive retinopathy (eg, RH and TV), hypertensive choroidopathy (eg, serous RD), and optic neuropathy (eg, papilledema and optic nerve atrophy in chronic disease). Ocular lesions described anecdotally for hypertensive dogs include the following: retinal TV and edema, perivasculitis, preretinal hemorrhage and RH, RD, papilledema, intraocular hemorrhage, and secondary glaucoma. A, 16, 17, 28, 31

To our knowledge, there are no published studies reporting either the prevalence of ocular disease in hypertensive dogs or the prevalence of hypertension in dogs evaluated because of ocular lesions suggestive of hypertension. The purposes of the study presented here were to determine the distribution of ocular lesions in hypertensive dogs and prevalence of hypertension in dogs with ocular lesions suggestive of hypertension and to document any correlation between the presence of suggestive ocular lesions and severity of hypertension.

Materials and Methods

Case selection

The electronic medical database at the University of Wisconsin, Veterinary Medical Teaching Hospital was reviewed to identify dogs that underwent a complete ophthalmic examination and blood pressure measurement within a 24-hour period between January 1, 2005, and December 31, 2007. Patients were excluded if they were diagnosed with giant retinal tears and vitreal degeneration,³² multiple myeloma, or uveitis attributable to another

cause (eg, systemic fungal infection, progressive uveitis in Golden Retrievers, or recent cataract surgery). Patients without complete fundic examinations were excluded, unless the fundic examination was impaired because of the presence of hyphema. These patients were included because of the historical association between hypertension and hyphema. ²²

Medical records review

Data extracted from medical records included signalment, clinical history, reasons for initial evaluation at the hospital, method of blood pressure measurement (ie, Doppler ultrasonographic, oscillometric, or direct measurement), blood pressure measurements, complete ophthalmic examination findings, etiology of hypertension if known, and any administration of vasoactive drugs. In patients with several examination occasions, the earliest pair of eligible data points was recorded for the purpose of this study.

Measurements of SBP were obtained by use of indirect (Doppler ultrasonographic^a or oscillometric^b) or direct^c, d methods as performed by personnel of the veterinary medical teaching hospital cardiology department. Blood pressure measurements were routinely performed by a certified veterinary technician, in accordance with the current recommendations for diagnosis and management of hypertension in companion animals.³³ The mean of a series of 5 SBP readings was used as a representative value for the session.^{34,35} A measurement of SBP 160 mm Hg was considered hypertensive for the purposes of this study.³³ Medical records of hypertensive dogs were further scrutinized for comments that reflected clinical suspicion of situational hypertension on the basis of the notation of either pain or substantial signs of anxiety at the time of blood pressure measurement.³³

Complete ophthalmic examinations consisted of slit-lamp biomicroscopy, indirect ophthalmoscopy, Schirmer tear test, and applanation tonometry. Based on available data for people and cats, 5 major lesions associated with hypertension were identified as consistent with systemic hypertension: RH, RD, hyphema, TV, and SRE. 4,26,27,29,30 The presence of these lesions in our patient population was noted. Although both the severity and distribution (ie, unilateral vs bilateral) of ocular lesions were noted in the original data collection phase, these characteristics were not analyzed statistically. Ocular lesions recorded were considered on a per patient basis, rather than counted as independent lesions per eye. In other words, if a patient had multifocal areas of RH in both eyes with concurrent RD in only 1 eye, these were considered to be 2 lesions for study purposes (ie, 1 RH and 1 RD). Additional ocular lesions noted during data collection included uveitis (16 dogs) and secondary glaucoma (3 dogs). Further scrutiny of medical records indicated that secondary glaucoma was evident in these 3 dogs as a sequela to hyphema or RD. Fifteen of 16 dogs with uveitis had concurrent hyphema, RD, RH or some combination of these. The single dog that had uveitis as the sole finding was normotensive (SBP, 117 mm Hg) and had a history

^aParks model 811-BL, Parks Medical Electronics, Aloha, Ore.

^bCardell model 9401, Midmark Inc, Versailles, Ohio.

^cGE Marquette Eagle 3000 Patient Monitor, Milwaukee, Wis.

^dBecton Dickinson Transducer DTX Plus DT-4812, Sandy, Utah.

of ocular trauma. Therefore, both secondary glaucoma and uveitis were considered secondary to major ocular lesions and were not considered for further analysis.

The presence of ongoing treatments with vasoactive drugs was noted, including angiotensin-converting enzyme inhibitors, amlodipine, β blockers, furosemide, hydralazine, spironolactone, phenylpropanolamine, or combinations of these drugs. With the exception of phenylpropanolamine, these vasoactive medications were considered likely to decrease blood pressure. Therefore, dogs receiving phenylpropanolamine were excluded from statistical analysis when comparing the presence or absence of treatment with antihypertensive drugs. There was no systematic attempt to correlate individual drug treatments with the presence of ocular disease or degree of severity.

Statistical analysis

Descriptive statistics were performed with a commercially available statistical software package. Nonparametric unpaired data analysis methods were used. The Mann-Whitney U test was used to compare median SBP in selected groups, and the Fisher exact test was used to compare prevalence of selected findings in groups. Data are presented as median values and ranges where appropriate. Values of P < 0.05 were considered significant.

Results

Ninety-eight medical records matched the initial search criteria. Patients diagnosed with uveitis attributable to another cause (12 dogs), giant retinal tears and vitreal degeneration (2 dogs), or multiple myeloma (2 dogs) were excluded from the study. Nine dogs without complete fundic examinations were excluded. Eight dogs were excluded on the basis of suspicion of situational hypertension as noted in the medical record. Ultimately, 65 dogs met eligibility criteria for inclusion in the study and comprehensive record review.

Of 65 eligible dogs, there were 4 mixed-breed dogs and 61 purebred dogs according to hospital records. Thirty breeds were represented, the most common being the Labrador Retriever (n = 7), Beagle (5), English Springer Spaniel (5), and Shih Tzu (5). Other breeds included the Bassett Hound (n = 2), Bichon Frise (2), Cairn Terrier (2), Cocker Spaniel (3), Dachshund (2), Dalmatian (3), Golden Retriever (3), Jack Russell Terrier (2), Lhasa Apso (2), Miniature Poodle (2), and Shetland Sheepdog (2). The following breeds had 1 representative dog: Australian Shepherd, Bernese Mountain Dog, Border Terrier, Collie, German Shepherd Dog, Greyhound, Miniature Pinscher, Pit Bull Terrier, Pomeranian, Standard Schnauzer, Shiba Inu, Siberian Husky, Welsh Terrier, and Yorkshire Terrier. Breed prevalence within the study population mirrored the pedigree distribution in the immediate geographic area. Thirty-five dogs were male (4 sexually intact), and 30 were female (1 sexually intact). The median age was 10.6 years (range, 1.1 to 16.9 years).

Twenty-two of 65 (34%) dogs initially had their blood pressure measured (median SBP, 192 mm Hg [range, 105 to 253 mm Hg]) and subsequently were referred for ophthalmic examination; 18 of these 22 (82%) dogs were hypertensive. Twenty-five of 65 (38%) dogs

eGraphpad Prism, version 5.01 for Windows, Graphpad Software Inc, San Diego, Calif.

initially underwent an ophthalmic examination and were later referred for blood pressure measurement (median SBP, 160 mm Hg [range, 121 to 265 mm Hg]); 15 of these 25 (60%) dogs were hypertensive. Lastly, 18 of 65 (28%) dogs were referred to both services concurrently for secondary referral evaluation (median SBP, 162 mm Hg [range, 117 to 263 mm Hg]); 9 of these 18 dogs were hypertensive. Twelve of 65 dogs had had SBP recorded by use of a Doppler ultrasonographic technique, and 52 dogs had had SBP recorded by use of oscillometric methods. One dog had a direct measurement via arterial puncture.

Normotensive dogs

Twenty-three of 65 (35%) dogs were considered normotensive according to study criteria (median SBP, 144 mm Hg [range, 105 to 159 mm Hg]). Fourteen of 23 (61%) normotensive dogs had no ocular lesions; of these 14 dogs, 7 were receiving antihypertensive drugs. The remaining 7 normotensive dogs without ocular lesions were not receiving any antihypertensive drugs. Nine of 23 (39%) normotensive dogs had 1 of the 5 major type of ocular lesions identified (RH, RD, hyphema, SRE, or TV); 4 of these 9 dogs were receiving antihypertensive treatment for a previous diagnosis of hypertension. Of these 4 normotensive dogs with ocular lesions suggestive of hypertension that were receiving antihypertensive drugs, 1 dog had RH, 1 dog had RD, and 2 dogs had RD with concurrent hyphema. The remaining 5 dogs with lesions had alternate etiologies identified at the time of the examination for their ocular disease. There was no significant (P = 1.0) difference in the prevalence of ocular disease in normotensive dogs based on the presence or absence of treatment with antihypertensive drugs.

Hypertensive dogs

Forty-two dogs were considered hypertensive according to study criteria (median SBP, 192 mm Hg [range, 165 to 265 mm Hg]). Hypertensive dogs were frequently diagnosed with various concurrent clinical conditions such as renal disease (11 dogs), diabetes mellitus (n = 5), iatrogenic (phenylpropanolamine-induced) hypertension (5), hyperadrenocorticism (3), pheochromocytoma (2), congestive heart failure (1), neoplasia (1), or combinations of several existing conditions (4). In 10 dogs, a definitive diagnosis of underlying disease was not recorded, either as a result of an incomplete diagnostic workup or inconclusive findings following a thorough clinical evaluation.

Of 42 hypertensive dogs, 26 (62%) had $\, 1$ of the major ocular lesions identified, whereas 16 (38%) dogs did not have any evidence of ocular disease. The median SBP of hypertensive dogs with $\, 1$ major ocular lesion was 197 mm Hg (range, 165 to 265 mm Hg); the median SBP of hypertensive dogs without any major ocular lesions was 189 mm Hg (range, 165 to 210 mm Hg). Although hypertensive dogs with $\, 1$ major ocular lesion had a higher median SBP, the median SBP of hypertensive dogs with ocular lesions was not significantly (P = 0.23) different from that of hypertensive dogs without ocular disease.

The distribution of lesion type among hypertensive dogs included 17 of 42 (40%) dogs with RH, 10 (24%) with RD, 8 (19%) with hyphema, 5 (12%) with SRE, and 2 (5%) with TV. Thirteen of 26 (50%) hypertensive dogs with ocular lesions had 2 major ocular lesions noted. Hypertensive dogs with RH had a significantly (P = 0.003) higher SBP (median SBP,

220 mm Hg [range, 190 to 263 mm Hg]), compared with that of hypertensive dogs without RH (median SBP, 187 mm Hg [range, 165 to 265 mm Hg]). However, when hypertensive dogs with RD or hyphema were compared with hypertensive dogs without ocular lesions, the median SBP did not differ significantly between these groups.

Data from the 20 hypertensive dogs receiving vasoactive medications were analyzed in more detail. This group included 5 dogs receiving phenylpropanolamine at the time of blood pressure measurement and ophthalmic examination. Of the 5 hypertensive dogs that received phenylpropanolamine, 2 dogs (SBP, 175 and 201 mm Hg, respectively) had no major ocular lesions and 2 dogs (SBP, 180 and 253 mm Hg, respectively) had RH. One dog with an SBP of 193 mm Hg had received an overdose of phenylpropanolamine 24 hours earlier and subsequently was found to have RH, RD, and hyphema. Because phenylpropanolamine is a known vasoconstrictive agent with hypertensive effects at therapeutic doses, dogs receiving phenylpropanolamine were excluded from analysis when investigating the relevance of treatment with antihypertensive drugs in our patient population.

There was no significant (P = 0.78) difference in SBP in hypertensive dogs on the basis of whether they received antihypertensive drugs. Of 22 hypertensive dogs not receiving medications, 9 had no ocular lesions and 13 had -1 major lesion. Of 15 hypertensive dogs receiving antihypertensive drugs, 5 had no major lesions whereas 10 had -1 lesion. The number of lesion types present in hypertensive dogs did not differ significantly despite the presence or absence of treatment with antihypertensive drugs (Table 1).

Comparative data for normotensive and hypertensive dogs

Normotensive dogs and hypertensive dogs were compared on the basis of the number of lesions by type (Table 2). The presence of 1 type of ocular lesion had a moderate sensitivity and specificity of 62% and 61%, respectively, for identification of dogs with hypertension. A greater percentage of normotensive dogs (14/23 [61%]) had no ocular lesions, compared the percentage of hypertensive dogs (16/42 [38%]) that had no ocular lesions, but this difference was not significant (P = 0.12). Also, a greater percentage of hypertensive dogs (13/42 [31%]) had 2 types of ocular lesion, compared with the percentage of normotensive dogs (3/23 [13%]) that had 2 types of ocular lesions, but this difference was not significant (P = 0.14).

The distribution of lesion type in normotensive and hypertensive dogs was evaluated (Table 3). Many dogs (n = 16) had more than 1 major ocular finding noted. The sensitivity of any single ocular lesion type to predict hypertension was variable but generally low (range, 5% to 41%). However, because SRE and TV were found only in hypertensive dogs, the presence of these findings was specific for hypertension in our patient population. Although RH and RD appeared to be more prevalent in hypertensive dogs, there were no significant differences in the distribution of lesion type between hypertensive and normotensive dogs.

Discussion

In this study, more dogs admitted for blood pressure evaluation were hypertensive, compared with the dogs admitted for ophthalmic examination. This seems logical, as dogs brought to the hospital for blood pressure measurement would consequently be referred for ophthalmic examination only if hypertensive. Conversely, dogs with ocular disease investigated in this study may have had ocular lesions as a result of various etiologies, and hypertension is often routinely ruled out in these patients during the course of their clinical evaluation.

Most of the normotensive dogs (14/23 [61%]) in this study had none of the major ocular lesions investigated, while the remaining normotensive dogs (9/23 [39%]) had 1 of the 5 major lesions noted. The presence of 4 normotensive dogs with ocular lesions suggestive of hypertension while receiving antihypertensive drugs may indicate the failure of antihypertensive drugs to relieve or prevent signs of ocular TOD. Alternatively, these lesions in normotensive dogs may reflect previous TOD that had not yet resolved or indicate that these ocular lesions may result from TOD occurring below the SBP threshold of 160 mm Hg. Because noninvasive blood pressure measurement methods may underestimate true blood pressure, some of these patients may have been hypertensive in actuality. 36,37 Because of the lack of standardization in this retrospective study, appropriateness of drug choice, dosage information, owner compliance, and duration of treatment were not considered for patients receiving antihypertensive drugs. Furthermore, alternate etiologies may be responsible, as they may not have been systematically ruled out in affected dogs with a preexisting diagnosis of hypertension. Lastly, there may be variables other than the degree of hypertension that play an integral role in the development of ocular disease in dogs; these may include factors such as capillary fragility and increased vascular permeability. Ultimately, these findings suggest that vigilance regarding ocular lesions is necessary in the successful treatment of hypertensive dogs. Because most of the normotensive dogs receiving antihypertensive drugs in this study had no ocular lesions, our data suggest the potential for successful use of pharmacotherapy in treating hypertension and resulting ocular TOD.

There was no significant difference in the number of lesions or the prevalence of RH, RD, hyphema, SRE, or TV between hypertensive and normotensive dogs. A greater proportion of normotensive dogs had no lesions, compared with the proportion of hypertensive dogs, and more hypertensive dogs had 2 ocular lesions, compared with that of normotensive dogs. In hypertensive dogs, RH was significantly associated with higher blood pressures, compared with that of hypertensive dogs without RH, but similar findings were not identified with RD or hyphema. In agreement with these findings, the presence of RH was the most sensitive indicator of hypertension in this population, although the sensitivity was only moderate. Therefore, identification of RH should be considered an indication for blood pressure measurement in the affected patient, although scrutiny of ocular lesion type does not generally facilitate a diagnosis of hypertension, nor does the lack of specific types of lesions rule out hypertension. In this study, the median SBP of hypertensive dogs with ocular lesions did not differ significantly from that of hypertensive dogs without ocular disease, but dogs with ocular lesions suggestive of hypertension had a higher median SBP than did dogs without ocular lesions.

There was no significant difference in SBP of hypertensive dogs when the presence or absence of treatment with antihypertensive drugs was compared. This suggests that some of these dogs remained hypertensive despite treatment and some had ocular lesions. Considering both normotensive and hypertensive dogs had ocular lesions suggestive of hypertension despite ongoing treatment with antihypertensive drugs suggests that patients receiving antihypertensive drugs, whether effective or not, are not uniformly spared from ocular TOD. Lastly, our finding that all 5 dogs receiving phenylpropanolamine, either chronically or acutely, were hypertensive and most had ocular disease supports previous anecdotal evidence that even therapeutic doses of phenylpropanolamine may incite hypertension and result in ocular TOD.

Based on the pathophysiologic mechanism of arterial hypertension in other species, the ocular lesions noted in the present study likely reflect a more extensive choroidal disease in affected dogs. In an angiographic study of dogs with hypertension, choroidal ischemia was noted in all examined eyes. These results suggest that the subretinal fluid and edema noted in dogs of the study reported here may be due to hypertension-related choroidal ischemia.

Ocular lesions are the most commonly observed complication of hypertension in cats, ^{27,39} although the prevalence rate varies amongst studies from 50% to 100%. ^{4,27,33,40} The prevalence of ocular lesions in hypertensive dogs in this study is comparable at 62%. Ophthalmic changes secondary to hypertension in cats include intraocular hemorrhage and fundic changes. Intraocular hemorrhage, RD, and RH are the most common findings in hypertensive cats, ^{4,26,27} which are similar to our findings in which RD and RH were the most common findings in hypertensive dogs with ocular lesions. In another study, ¹⁰ the mean SBP of hypertensive cats with ocular lesions was 262 mm Hg, compared with a significantly lower mean SBP of 221 mm Hg in hypertensive cats without ocular disease. In our study, the median SBP of hypertensive dogs without any major ocular lesions was lower (189 mm Hg), compared with the median SBP of hypertensive dogs with 1 major ocular lesion (197 mm Hg); however, this difference was not significant (*P* = 0.23).

One limitation of this retrospective study is that dogs with ocular lesions suggestive of hypertension were sent for blood pressure measurement, whereas only dogs with documented hypertension were likely referred for ocular examination after blood pressure measurement. Therefore, we lack sufficient numbers of patients that were both normotensive and without ocular lesions. Also, at the veterinary teaching hospital, multiple ophthalmologists and cardiologists examined study patients during the 2-year study period. Although the diagnostic threshold for hypertension did not change, the limitations of blood pressure measurement and subsequent interpretation are multifold. Research suggests indirect blood pressure measurements tend to vary in accuracy, particularly in patients with hypertension, leading to an underestimation of SBP in conscious dogs. 35,36,41 So-called white-coat hypertension may also falsely increase SBP, admittedly even under the best clinical circumstances, because anxiety can cause neurogenic stimulation followed by a subsequent false diagnosis of hypertension. 42–45 Although dogs were excluded from this study on the basis of suspicion of situational hypertension, dogs considered hypertensive may have been normotensive but anxious or painful during blood pressure measurement.

Furthermore, diurnal variability, momentary lability, and transient dehydration can all lead to erroneous blood pressure readings.³³ Lastly, the high prevalence of patients receiving antihypertensive drugs potentially confounds our results, especially in light of the fact that most patients receiving antihypertensive drugs were neither normotensive nor spared from characteristic ocular disease. Because we could not assume correct dosage or administration in our study, we cannot discern whether patients were treated inadequately or whether antihypertensive drugs as a sole means of treatment may be inadequate if the primary disease process is not managed accordingly.

The inherent ophthalmic limitations should also be recognized. Ocular lesions associated with TOD and hypertension were chosen a priori on the basis of findings in hypertensive people and cats; other possible related lesions were not systematically recorded. The presence of hyphema may have obscured fundic disease in some dogs, which may have led to underestimation of the prevalence of retinal lesions. Additionally, the ocular findings noted were dependent on the ophthalmologist recording the examination. Within the field of ophthalmoscopy, fundic examination with a funduscope is undoubtedly subjective, with data suggesting that significant interobserver and intraobserver variation exists. ⁴⁶ With the advent of computerized digital imaging, objective quantitative imaging is performed more routinely in patients with hypertensive retinopathy. ⁴⁷

In conclusion, systemic hypertension should be considered as a possible etiology for RH, RD, hyphema, SRE, or TV in dogs. The high prevalence of major ocular lesions in hypertensive dogs suggests that dogs with known hypertension should be routinely screened for ocular TOD, and the high prevalence of hypertension in dogs with suspicious ocular lesions suggests that these patients should be evaluated for hypertension. In addition, treatment with antihypertensive drugs should not unquestioningly be considered effective in preventing ocular TOD even when the medicated patient is normotensive, and administration does not obviate the need for continued vigilance regarding ocular TOD in affected patients. Patients receiving antihypertensive drugs should be monitored for evidence of blood pressure response to treatment as well as for the presence of ocular TOD. Results of blood pressure measurements and ophthalmic findings should be interpreted collaboratively with clinicopathologic indices, physical examination findings, and pertinent historical data.

Abbreviations

RD Retinal detachment
RH Retinal hemorrhage
SBP Systolic blood pressure
SRE Subretinal edema
TOD Target organ damage
TV Tortuous vessels

References

 He J, Whelton PK. Elevated systolic blood pressure and risk of cardiovascular and renal disease: overview of evidence from observational epidemiologic studies and randomized controlled trials. Am Heart J. 1999; 138:211–219. [PubMed: 10467215]

- Luke RG. Hypertensive nephrosclerosis: pathogenesis and prevalence. Essential hypertension is an important cause of end-stage renal disease. Nephrol Dial Transplant. 1999; 14:2271–2278.
 [PubMed: 10528641]
- 3. Wehner A, Hartmann K, Hirschberger J. Associations between proteinuria, systemic hypertension and glomerular filtration rate in dogs with renal and non-renal diseases. Vet Rec. 2008; 162:141–147. [PubMed: 18245745]
- Maggio F, DeFrancesco TC, Atkins CE, et al. Ocular lesions associated with systemic hypertension in cats: 69 cases (1985–1998). J Am Vet Med Assoc. 2000; 217:695–702. [PubMed: 10976302]
- 5. Finco DR. Association of systemic hypertension with renal injury in dogs with induced renal failure. J Vet Intern Med. 2004; 18:289–294. [PubMed: 15188813]
- Cortadellas O, del Palacio MJ, Bayon A, et al. Systemic hypertension in dogs with leishmaniasis: prevalence and clinical consequences. J Vet Intern Med. 2006; 20:941–947. [PubMed: 16955820]
- Crispin SM, Mould JR. Systemic hypertensive disease and the feline fundus. Vet Ophthalmol. 2001;
 4:131–140. [PubMed: 11422995]
- 8. Brown CA, Munday JS, Mathur S, et al. Hypertensive encephalopathy in cats with reduced renal function. Vet Pathol. 2005; 42:642–649. [PubMed: 16145210]
- 9. Jacob F, Polzin DJ, Osborne CA, et al. Association between initial systolic blood pressure and risk of developing a uremic crisis or of dying in dogs with chronic renal failure. J Am Vet Med Assoc. 2003; 222:322–329. [PubMed: 12564594]
- Chetboul V, Lefebvre HP, Pinhas C, et al. Spontaneous feline hypertension: clinical and echocardiographic abnormalities, and survival rate. J Vet Intern Med. 2003; 17:89–95. [PubMed: 12564732]
- 11. Snyder PS, Sadek D, Jones GL. Effect of amlodipine on echocardiographic variables in cats with systemic hypertension. J Vet Intern Med. 2001; 15:52–56. [PubMed: 11215913]
- Henik RA, Stepien RL, Bortnowski HB. Spectrum of M-mode echocardiographic abnormalities in 75 cats with systemic hypertension. J Am Anim Hosp Assoc. 2004; 40:359–363. [PubMed: 15347614]
- 13. Rushmer, RF. Cardiovascular dynamics. 4th ed. WB Saunders Co; Philadelphia: 1976.
- 14. Bovée KC, Littman MP, Saleh F, et al. Essential hereditary hypertension in dogs: a new animal model. J Hypertens Suppl. 1986; 4:S172–S171. [PubMed: 3471902]
- 15. Tippett FE, Padgett GA, Eyster G, et al. Primary hypertension in a colony of dogs. Hypertension. 1987; 9:49–58. [PubMed: 3793200]
- 16. Littman MP, Robertson JL, Bovée KC. Spontaneous systemic hypertension in dogs: five cases (1981–1983). J Am Vet Med Assoc. 1988; 193:486–494. [PubMed: 3170325]
- 17. Bovée KC, Littman MP, Crabtree BJ, et al. Essential hypertension in a dog. J Am Vet Med Assoc. 1989; 195:81–86. [PubMed: 2759900]
- 18. Slaughter JB, Padgett GA, Blanchard G, et al. Canine essential hypertension: probable mode of inheritance. J Hypertens Suppl. 1986; 4:S170–S171. [PubMed: 3471901]
- 19. Howard EB, Nielsen SW. Pheochromocytomas associated with hypertensive lesions in dogs. J Am Vet Med Assoc. 1965; 147:245–252. [PubMed: 14325458]
- 20. Dimski DS, Hawkins EC. Canine systemic hypertension. Com-pend Contin Educ Vet Pract. 1988; 10:1152–1155.
- 21. Ortega TM, Feldman EC, Nelson RW, et al. Systemic arterial blood pressure and urine protein/creatinine ratio in dogs with hyperadrenocorticism. J Am Vet Med Assoc. 1996; 209:1724–1729. [PubMed: 8921029]
- Cullen, CL.; Webb, AA. Ocular manifestations of systemic diseases. In: Gelatt, KN., editor.
 Veterinary ophthalmology. 4th ed. Blackwell Publishing; Ames, Iowa: 2007. p. 1477-1481.

23. Keyes JE. Experimental hypertension. IV. Clinical and pathological studies of the eyes: a preliminary report. Arch Ophthamol. 1937; 17:1040–1054.

- 24. Green, RA. Bleeding disorders. In: Ettinger, SJ., editor. Textbook of veterinary internal medicine. 2nd ed. WB Saunders Co; Philadelphia: 1983. p. 2076-2098.
- 25. Keyes JE, Goldblatt H. Experimental hypertension. VIII. Vascular changes in the eyes. Arch Ophthalmol. 1938; 20:812–828.
- Sansom J, Barnett KC, Dunn KA, et al. Ocular disease associated with hypertension in 16 cats. J Small Anim Pract. 1994; 35:604–611.
- 27. Littman MP. Spontaneous systemic hypertension in 24 cats. J Vet Intern Med. 1994; 8:79–86. [PubMed: 8046680]
- 28. Sansom J, Bodey A. Ocular signs in four dogs with hypertension. Vet Rec. 1997; 140:593–598. [PubMed: 9203316]
- 29. Morgan RV. Systemic hypertension in four cats: ocular and medical findings. J Am Anim Hosp Assoc. 1986; 22:615–621.
- 30. Hayreh SS. Hypertensive retinopathy. Ophthalmol Clin North Am. 1998; 11:535–558.
- 31. Gwin RW, Gelatt KN, Terrell TG, et al. Hypertensive retinopathy associated with hypothyroidism, hypercholesterolemia, and renal failure in the dog. J Am Anim Hosp Assoc. 1978; 14:200–209.
- 32. Vainisi, SJ.; Wolfer, JC.; Smith, PJ. Surgery of the canine posterior segment. In: Gelatt, KN., editor. Veterinary ophthalmology. 4th ed. Vol. 1027. Blackwell Publishing; Ames, Iowa: 2007.
- 33. Brown S, Atkins C, Bagley R, et al. Guidelines for the identification, evaluation, and management of systemic hypertension in dogs and cats. J Vet Intern Med. 2007; 21:542–558. [PubMed: 17552466]
- 34. Henik RA, Dolson MK, Wenholz LJ. How to obtain a blood pressure measurement. Clin Tech Small Anim Pract. 2005; 20:144–150. [PubMed: 16180396]
- 35. Bodey AR, Michell AR, Bovee KC, et al. Comparison of direct and indirect (oscillometric) measurements of arterial blood pressure in conscious dogs. Res Vet Sci. 1996; 61:17–21. [PubMed: 8819188]
- 36. Stepien RL, Rapoport GS. Clinical comparison of three methods to measure blood pressure in nonsedated dogs. J Am Vet Med Assoc. 1999; 215:1623–1628. [PubMed: 14567424]
- 37. Haberman CE, Kang CW, Morgan JD, et al. Evaluation of oscillo-metric and Doppler ultrasonic methods of indirect blood pressure estimation in conscious dogs. Can J Vet Res. 2006; 70:211–217. [PubMed: 16850944]
- 38. Villagrasa M, Cascales MJ. Arterial hypertension: angiographic aspects of the ocular fundus in dogs. Eur J Comp Anim Pract. 2000; 10:177–190.
- 39. Snyder PS. Amlodipine: a randomized, blinded clinical trial in 9 cats with systemic hypertension. J Vet Intern Med. 1998; 12:157–162. [PubMed: 9595376]
- 40. Sansom J, Rogers K, Wood JL. Blood pressure assessment in healthy cats and cats with hypertensive retinopathy. Am J Vet Res. 2004; 65:245–252. [PubMed: 14974583]
- 41. Chalifoux A, Dallaire A, Blais D, et al. Evaluation of the arterial blood pressure of dogs by two noninvasive methods. Can J Comp Med. 1985; 49:419–423. [PubMed: 4075242]
- 42. Remillard RL, Ross JN, Eddy JB. Variance of indirect blood pressure measurements and prevalence of hypertension in clinically normal dogs. Am J Vet Res. 1991; 52:561–565. [PubMed: 2053724]
- 43. Kallet AJ, Cowgill LD, Kass PH. Comparison of blood pressure measurements obtained in dogs by use of indirect oscillometry in a veterinary clinic versus at home. J Am Vet Med Assoc. 1997; 210:651–654. [PubMed: 9054993]
- 44. Vincent IC, Michell AR, Leahy RA. Non-invasive measurement of arterial blood pressure in dogs: a potential indicator for the identification of stress. Res Vet Sci. 1993; 54:195–201. [PubMed: 8460259]
- 45. Vincent IC. Michell AR. Relationship between blood pressure and stress-prone temperament in dogs. Physiol Behav. 1996; 60:135–138. [PubMed: 8804653]
- 46. Dimmitt SB, West JN, Eames SM, et al. Usefulness of ophthalmos-copy in mild to moderate hypertension. Lancet. 1989; 1:1103–1106. [PubMed: 2566051]

47. DellaCroce JT, Vitale AT. Hypertension and the eye. Curr Opin Ophthalmol. 2008; 19:493–498. [PubMed: 18854694]

Table 1

Comparison of median (range) SBP measurements (mm Hg) and number of ocular lesions in 37 hypertensive dogs* receiving and not receiving drugs to treat hypertension

			No lesions		1 lesion		2 lesions	
Variable	No. of dogs	SBP	No. of dogs (%)	SBP	No. of dogs (%)	SBP	No. of dogs (%)	SBP
AHTD	15	190 (166–236)	5/15 (33)	185 (173–202)	4/15 (27)	210 (166–253)	6/15 (40)	190 (174–263)
No AHTD	22	201 (165–265)	9/22 (41)	191 (165–210)	7/22 (32)	219 (165–263)	6/22 (27)	193 (168–265)
P value †	NA	0.78	0.75	0.84	0.73	0.93	0.49	0.57

AHTD = Antihypertensive drugs. NA = Not applicable.

^{*} Of the 42 hypertensive dogs in this study, 5 that received phenylpropanolamine were excluded from this analysis.

 $^{^{\}dagger}$ Neither median SBP nor the number of ocular lesions was significantly (P < 0.05) associated with treatment status.

Table 2

Comparison of 23 normotensive dogs with 42 hypertensive dogs on the basis of number of ocular lesions and median (range) SBP measurements (mm Hg) without regard to medication status

			No lesions		1 lesion		2 lesions	
Variable	No. of dogs	SBP	No. of dogs (%)	SBP	No. of dogs (%)	SBP	No. of dogs (%)	SBP
Normo	23	44 (105–159)	14/23 (61)	49 (105–159)	6/23 (26)	40 (121–147)	3/23 (13)	142 (125–155)
Hyper	42	92 (165–265)	16/42 (38)	89 (165–210)	13/42 (31)	219 (165–263)	13/42 (31)	190 (168–265)
P value*	NA	NA	0.12	NA	0.78	NA	0.14	NA
Sensitivity (%) [†]	NA		NA		62 [§]		31	
Specificity (%) [†]	NA		NA		62 [§]		87	

NA = Not applicable. Normo = Normotensive dogs. Hyper = Hypertensive dogs.

^{*} The percentage of dogs with 0,1, or > 2 ocular lesions was not significantly (P < 0.05) different between normotensive dogs and hypertensive dogs.

 $^{^{\}dagger}$ Sensitivity and specificity of the number of ocular lesions by type in the prediction of hypertension.

Values of 1 lesion.

Table 3

Comparison of distribution of lesion type in 23 normotensive dogs and 42 hypertensive dogs on the basis of ocular lesions by type (eg, RD, RH, hyphema, SRE, or TV) without regard to medication status

	No. of dogs (%) by lesion type							
Variable	RH RD		Hyphema	SRE	TV			
Normotensive dogs*	5/23 (22)	3/23 (13)	4/23 (17)	0	0			
Hypertensive dogs*	17/42 (40)	10/42 (24)	8/42 (19)	5/42 (12)	2/42 (5)			
P value †	0.17	0.35	1.0	0.15	0.54			
Sensitivity (%)‡	41	29	19	12	5			
Specificity (%) [‡]	78	87	83	100	100			

^{*} Three normotensive and 13 hypertensive dogs had > 1 type of lesion observed.

 $^{^{\}dagger}$ The percentage of dogs by lesion type was not significantly (P < 0.05) different between normotensive dogs and hypertensive dogs.

 $^{^{\}ddagger}$ Sensitivity and specificity of ocular lesion type in the prediction of hypertension.