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Guidelines for the Identification, Evaluation, and Management of Systemic Hypertension in Dogs and Cats

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Key words: Blood pressure; Hypertension; Proteinuria.

Section 1: Generation of the Guidelines

In the past 15 years, recognition of the importance of systemic hypertension in dogs and cats has led to fundamental changes in our understanding of the pathophysiology and management of several diseases. Accordingly, in 2000, the ACVIM organized an expert panel on systemic hypertension in dogs and cats. The panel presented its initial findings as an oral Consensus Statement at the ACVIM Forum in 2001. We continued our efforts at the ACVIM Forum in 2002, where we presented a revised position statement. Following the recent publications of *The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure* by the National High Blood Pressure Education program (National Heart, Lung, and Blood Institute, National Institutes of Health)[2] and the *2003 European Society of Hypertension-European Society of Cardiology Guidelines for the Management of Arterial Hypertension*,[3] the panel met at the ACVIM Forum in 2004 to expand the

consensus statement and to develop similar comprehensive guidelines for dogs and cats. The guidelines were further revised in 2005–2006, and this update was presented at the ACVIM Forum in 2006. Our collective goal was to utilize the best available evidence in making our recommendations and to provide the veterinary practitioner with practical information, to facilitate informed decisions. We do not intend these guidelines to be prescriptive, and we are aware the rapid pace of discovery of knowledge in this area ensures that our guidelines will be outdated soon after publication. It is our hope that these guidelines will form the basis for educating veterinarians on the clinical importance of hypertension and measurements of blood pressure (BP), and will facilitate the development of a rational approach to the diagnosis and management of hypertension.

Section 2: Measurement of BP

Diagnosis and management of hypertension in the clinical patient is based on measurement of the patient's BP. The BP can be measured directly by an intra-arterial means[5–8] or indirectly by devices that incorporate a compressive cuff. Arterial puncture by needle has been used for clinical patients in veterinary medicine,[9] and radiotelemetric implants have been employed in dogs[10–13] and cats[14–19] and may yet prove to be clinically useful as direct devices in veterinary medicine. However, at the present time, indirect devices are generally more clinically acceptable and in much wider use.[1,4,11,20–65] A new technology, high definition oscillometry, has been evaluated in dogs and cats under anesthesia. Preliminary results are promising but further supportive data in conscious animals are needed.

Ideally, BP should be measured with devices that have been validated in the species of interest and under

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Table 1. Examples of indirect blood pressure measurement devices in use in dogs and cats.

Device	Manufacturer	Device type
Cardell Model 9401, 2, 3	Sharn Veterinary Inc. http://www.sharnvet.com	Oscillometry
Dinamap Model 8300	No longer available	Oscillometry
Jorgensen Model J5373	Jorgensen Labs http://www.jorvet.com	Doppler ultrasonography
Memoprint, Memodiagnostic	No longer available	Oscillometry
Parks Model 811-B	Parks Medical Electronics http://www.parksmed.com	Doppler ultrasonography
Vet-Dop	Vmed Technology Inc http://www.vmedtech.com	Doppler ultrasonography
	VetSpecs Medical Systems http://www.vetspecs.com	Pressure plethysmography
MemoDiagnostic	S+B medVET http://www.submedvet.com	High Definition Oscillometry
MDpro, MD15, MD90	Distributed through HESKA (USA/CAN) MDscientific http://www.heska.com	High Definition Oscillometry

the circumstances in which the patient is being tested. Thus, for the diagnosis of systemic hypertension in dogs and cats, the indirect device used should be one that is commonly utilized or designed for veterinary use and that has been previously validated in conscious animals of the species of interest. Standards for the validation of indirect BP measuring devices in people have been published.[66,67] Unfortunately, at the present time, no indirect device has met these validation criteria for use in conscious dogs or cats. The original validation criteria were established for use in humans, in whom the methods for BP measurement are more standardized and much easier to achieve. Recognizing this limitation, the panel advocates different criteria for the validation of devices in animals (Appendix).

Until proper validation studies are conducted, BP measurements in veterinary patients will continue to be obtained with the use of currently available indirect devices. We recognize that validation is ongoing for indirect devices in current use and that many of these devices are reliable (Table 1). We anticipate that some of these devices will meet our criteria and that more reliable devices may be required as our knowledge advances and thresholds for intervention are further refined. We also anticipate that subsequent revision of our guidelines and the acquisition of new knowledge will lead to refinement of our validation criteria.

To obtain reliable values in the measurement of BP, it is important to follow a standard protocol (Table 2). The individual making the measurements should be patient and skilled in the handling of animals, clients, and equipment. Although it is critical for the veterinarian to appreciate fully the subtleties of BP measurement, it is generally preferred to have these measurements obtained by a skilled animal health technician who has been suitably trained in obtaining BP. Acquiring expertise in the use of indirect BP measurement devices requires hours of training; experienced operators enhance the reliability of indirect measurement. In the panel's experience, a leading cause of indirect device failure is technical error associated with personnel inexperience.

BP may be affected by stress or anxiety associated with the measurement process[16,32,33,45] and these changes may result in a false diagnosis of hypertension.[44] This anxiety-induced, artifactual increase in BP is often referred to as white-coat hypertension, a reference to the white coat of the medical professional measuring the BP. Thus, it is important for the measurement room to be quiet and for 5–10 minutes to elapse for the patient to acclimate to the room. This accommodation reduces the mean anxiety-induced artifact (so-called white-coat effect) to <20 mm Hg in cats.[16] Similar results may be expected in dogs. Although many causes of measurement error lead to an erroneously high value for BP, this is not always the case. The minute-to-minute variability of BP, inconsistency of BP measuring devices, technical errors, transient dehydration, and parasympathetic overactivity can all produce falsely low BP values.

The position of the patient and cuff should be well-tolerated, with the cuff at or close to the level of the right atrium. The first measurement should be discarded and the average of 3 to 7 consecutive, consistent indirect measurements should be obtained. A standard form for recording the results of the BP measurements should be developed. To allow for reliable comparison of serial measurements, each facility should follow carefully a standard protocol. This requires that the animal position and attitude, cuff size and site, and cuff site circumference (cm) be carefully considered (ideally these parameters would also be noted in the animal's record). All of the values obtained, the rationale for excluding certain values, the final (mean) result, and the interpretation of the final result by the veterinarian should be noted.

Table 2. Protocol for a blood pressure (BP) measurement session.

- The accuracy (calibration) of the BP device should be tested semi-annually (see Appendix).
- The procedure must be standardized.
- The environment should be isolated, quiet, away from other animals, and generally with the owner present. The patient should not be sedated and should be allowed to remain quietly in the measurement room for 5–10 minutes before attempting BP measurement.
- The animal should be gently restrained in a comfortable position, ideally in ventral or lateral recumbency to limit the distance from the heart base to the cuff (if more than 10 cm, a correction factor of +0.8 mm Hg/cm below the heart base can be applied).
- The cuff should be approximately 40% of circumference of the cuff site in dogs[1] and 30–40% in cats.[4] The cuff size should be noted in the medical record for future reference.
- The cuff may be placed on a limb or the tail, and will vary with animal conformation and user preference. The site for cuff placement should be recorded in the medical record.
- The same individual (preferably a technician) should perform all blood pressure measurements following this standard protocol. Training of this individual is essential.
- The patient should be calm and motionless.
- The first measurement should be discarded. At least 3, and preferably 5–7, consecutive, consistent (<20% variability in systolic values) values should be recorded.
- Repeat as necessary, changing cuff placement as needed to obtain consistent values.
- Average all values to obtain the BP measurement.
- If in doubt, repeat the measurement subsequently.
- Written records should be kept on a standard form and include cuff size and site, values obtained, rationale for excluding any values, the final (mean) result, and interpretation of the results by a veterinarian.

Table 3. Arterial blood pressure (mm Hg) values obtained from normal dogs and cats.

Measurement Method	N	Systolic	Mean	Diastolic
Dogs				
Intra-arterial				
Anderson et al[163]	28	144 ± 156	104 ± 13	81 ± 9
Cowgill et al[164]	21	148 ± 16	102 ± 9	87 ± 8
Chalifoux et al[165]	12	154 ± 31	115 ± 16	96 ± 12
Stepien et al[64]	27	154 ± 20	107 ± 11	84 ± 9
Oscillometry				
Bodey and Michell[42]	1267	131 ± 20	97 ± 16	74 ± 15
Coulter et al[43]	51	144 ± 27	110 ± 21	91 ± 20
Kallet et al[44]	14	137 ± 15	102 ± 12	82 ± 14
Stepien et al[64]	28	150 ± 20	108 ± 15	71 ± 18
Meurs et al[41]	22	136 ± 16	101 ± 11	81 ± 9
Doppler ultrasonography				
Chalifoux et al[165]	12	145 ± 23		
Stepien et al[64]	28	151 ± 27		
Remillard et al[33]	5	150 ± 16		
Cats				
Intra-arterial				
Brown et al, 1997[166]	6	125 ± 11	105 ± 10	89 ± 9
Belew et al, 1999[16]	6	126 ± 9	106 ± 10	91 ± 11
Oscillometry				
Bodey et al, 1998[59]	104	139 ± 27	99 ± 27	77 ± 25
Mishina et al, 1998[60]	60	115 ± 10	96 ± 12	74 ± 11
Doppler ultrasonography				
Klevans et al[58]	4	139 ± 8		
Kobayashi et al[69]	33	118 ± 11		
Sparkes et al[4]	50	162 ± 19		
Lin et al[167]	53	134 ± 16		

Section 3: Normal Values for Canine and Feline BP

Various studies have reported the BP values for normal dogs and cats (Table 3). These values vary, reflecting differences in subject populations, measurement techniques, and animal handling. This variability emphasizes the importance of standardization of the technique in veterinary practice. Additional factors affect what is considered to be the normal BP. In people, age-related increases in systolic BP and pulse pressure have been well-characterized.[68] The effects of age are less clear in dogs and cats. A small increase in BP of 1–3 mm Hg/year has been noted with aging in dogs,[42,50] although a similar effect of age has not been observed in all studies of dogs.[33,41] In cats, BP was found to increases with age in a heterogeneous population of animals.[59] However, there was no age effect in 2 studies of normal cats,[4,69] and a comparatively small increase of 1.5 mm Hg/year was noted for mean BP in a third study of apparently healthy cats.[60]

There is a reported effect of sex in dogs, with males having higher and intact females lower BP values, the difference being <10 mm Hg.[42] Most cats evaluated by veterinarians are neutered and there is no apparent effect of sex on the BP values in these cats.[59,60]

There are substantial interbreed differences in the BP of dogs, most notably with sighthounds (eg, Greyhounds and Deerhounds), in which BP is higher than in mongrels[70] by approximately 10–20 mm Hg.[42,70,71] The BP values of other breeds vary by 7–10 mm Hg[42] and the panel recognizes that breed-specific ranges for normal and abnormal BP may need to be developed. In cats, there is no apparent effect of breed on BP.[59]

Obesity is associated with increases in BP in a variety of species.[72–77] This effect has been studied experimentally in dogs;[72,78] a small (<5 mm Hg) increase in BP was noted in obese dogs by oscillometry[42] but not by

Table 4. Diseases associated with secondary hypertension in dogs and cats.

Disease or Condition	Prevalence of Hypertension (%)	Reference(s)
Dogs		
Chronic kidney disease	93	163
	60	164
	80	164
	79	168
	9	42
	62	101
	19	169
Acute kidney disease	87	170
Hyperadrenocorticism		
Native	73	81,168
Iatrogenic	80	
Diabetes mellitus	46	171
	24	168
Obesity	small effect	33,42,72,78
Primary hyperaldo-steronism	rare disease	172
Pheochromocytoma	43%	173
	86%	174
Hypothyroidism	uncommon	175
Cats		
Chronic kidney disease	46	69
	65	105
	19	176
Diabetes mellitus	0	55
	no increase of BP	
Hyperthyroidism	87	65
	23	69
	5 pre-treatment and	105
	25 post-treatment	82
Obesity	hypertension uncommon	59
Primary hyperaldo-steronism	50%–100% uncommon disease	89,177–183
Pheochromocytoma	up to 100% unusual disease	184, 185

Variabilities in inclusion criteria, measurement techniques, and definition of hypertension make direct comparisons of prevalence data difficult.

Doppler ultrasonography.[33] In cats, there was no effect of obesity on BP observed by oscillometry.[59]

Section 4: Hypertension Definitions

Systemic hypertension, which is synonymous with sustained increases in BP, can generally be categorized into 1 of 3 types. It may be caused by measurement artifact (ie, stress-induced or white-coat hypertension), occur in association with other disease processes that may increase BP (ie, secondary hypertension) or occur in the absence of other potentially causative disease processes (ie, idiopathic hypertension). Accordingly, we suggest the following definitions and criteria.

White-Coat Hypertension

High BP caused by an artifactual increase in BP that occurs as a consequence of the measurement process,

generally due to autonomic nervous system alterations from the effects of excitement or anxiety on higher centers of the central nervous system. This type of hypertension resolves under conditions that reduce or eliminate the artifact (eg, altering measurement conditions to reduce the animal's anxiety or measuring BP at the animal's home). Although there is some evidence that the presence of white-coat hypertension in an otherwise normotensive person is a risk factor for subsequent hypertensive damage,[79] there is presently no justification for treating white-coat hypertension in dogs or cats. Anxiety-induced increases in BP can lead to a false diagnosis of systemic hypertension.[33,44] Unfortunately, the effects of anxiety on BP are not predictable, as some animals exhibit a dramatic increase in BP whereas others do not, and some animals may even exhibit a decrease in BP as a result of the measurement process.[16] The latter effect presumably is due to parasympathetic nervous system overactivity.

Secondary Hypertension

This type of hypertension involves high BP concurrent with clinical disease or a condition known to cause hypertension (Table 4) or hypertension associated with the administration of therapeutic agents that are known to cause an elevation of BP, such as glucocorticoids, mineralocorticoids, erythropoietin, sodium chloride, phenylpropanolamine, and nonsteroidal anti-inflammatory drugs. Hypertension may persist despite effective treatment of the primary condition[80–82] and the BP may increase after therapy is initiated.[83] The presence of a condition known to cause secondary hypertension, even if effectively resolved by therapeutic intervention, should prompt serial follow-up evaluations.

Idiopathic Hypertension

The terms primary or essential hypertension have often been used in people to describe hypertension in the absence of any identifiable predisposing causes, and essential hypertension has been reported in dogs.[84–88] Subclinical kidney disease is present frequently in people and animals with hypertension, making it difficult to establish a valid diagnosis of primary or essential hypertension. Furthermore, the presence of chronically increased BP suggests that one or both of the neurohumoral and renal systems responsible for regulating BP is abnormal. Thus, the panel recommends the use of the term idiopathic in place of essential for animals in which high BP occurs in the absence of an overt, clinically apparent disease that is known to cause secondary hypertension.

A diagnosis of idiopathic hypertension is established when reliable BP measurements demonstrate a sustained increase in BP concurrent with normal CBC, serum biochemistry, and urinalysis. Unfortunately, increased BP may induce polyuria (so-called pressure diuresis) and thus, the presence of low urine specific gravity (<1.030) in a patient with high BP does not establish that kidney disease is present. On the other hand, the presence of concentrated urine (>1.030) makes kidney disease less likely.

Table 5. Evidence of target organ damage (TOD).

Tissue	Hypertensive injury (TOD)	Clinical Findings Indicative of TOD	Diagnostic Test(s)
Kidney	Progression of chronic kidney disease	Serial increases in SCr or decrease in GFR Proteinuria, microalbuminuria	SCr and BUN Urinalysis with quantitative assessment of proteinuria and/or albuminuria GFR measurement
Eye	Retinopathy/choroidopathy	Acute onset blindness Exudative retinal detachment Retinal hemorrhage/edema Retinal vessel tortuosity or perivascular edema Papilledema Vitreal hemorrhage Hyphema Secondary glaucoma Retinal degeneration	Ophthalmic evaluation including a funduscopic examination
Brain	Encephalopathy Stroke	Centrally localizing neurological signs (brain or spinal cord)	Neurological exam Magnetic resonance or other imaging
Heart and Vessels	Left ventricular hypertrophy Cardiac failure	Left ventricular hypertrophy Gallop rhythm Arrhythmias Systolic murmur Evidence of cardiac failure Hemorrhage (eg, epistaxis, stroke)	Auscultation Thoracic radiography Cardiac ultrasound Electrocardiogram

SCr, serum creatinine concentration; GFR, glomerular filtration rate; BUN, blood urea nitrogen concentration.

Since subclinical kidney disease or other condition known to cause secondary hypertension may be present in animals with idiopathic hypertension, the panel recommends that diagnostic tests in addition to CBC, serum biochemistry, and urinalysis be considered in affected animals. Depending on the clinical findings, these tests may include renal ultrasound examination (dog, cat), measurement of glomerular filtration rate (dog, cat), quantitative assessment of proteinuria (dog, cat), thyroid hormone determination (cat), and blood cortisol profile (dog). Furthermore, additional tests to consider in individual patients include serum and urine aldosterone and catecholamine concentrations and adrenal ultrasound examination. Although secondary hypertension remains the most common category of high BP in dogs and cats, idiopathic hypertension is more common than previously recognized, accounting for approximately 18–20% of cases in cats.[89,90]

Isolated Systolic or Diastolic Hypertension

These terms refer to the occurrence of an increase in systolic only or diastolic only pressure. Such a finding may be artifactually produced by underestimation or overestimation of the peak or trough of the blood pressure curve by an indirect device. The presence of this type of artifact should be considered whenever a very small (<20 mm Hg) or large (>60 mm Hg) pulse pressure is reported by an indirect device. True isolated hypertension of either type may represent white-coat, secondary or idiopathic hypertension. Currently, there is an emphasis on the diagnosis of systolic hypertension in veterinary medicine. This emphasis is largely due to reliance on measurement techniques to provide systolic readings only, as Doppler ultrasonography generally is used to obtain only systolic BP, particularly in cats, and recent evidence suggests that systolic BP is the more important determinant of hypertensive tissue damage in other species.[91,92] However, isolated systolic and diastolic hypertension can and do occur in dogs and cats and when properly diagnosed, warrant classification and management, as outlined below.

Section 5: Target Organ Damage

Systemic hypertension is problematic only because chronically sustained increases in BP cause injury to tissues; the rationale for treatment of hypertension is the prevention of this injury. Damage that results from the presence of sustained high BP is commonly referred to as end-organ or target-organ damage (TOD; Table 5), and the presence of TOD is generally a strong indication for antihypertensive therapy.

In the kidney, TOD is generally manifested as an enhanced rate of decrease in renal function, early renal death, and proteinuria or some combination of these findings. Furthermore, microalbuminuria is a marker of hypertensive TOD in the human heart and kidney,[93,94] and the severity of albuminuria has been directly related to the degree of increase in BP in an experimental study of chronic kidney disease (CKD) in cats.[19] Proteinuria has been directly related to the extent of increase in BP and to the rate of decrease in GFR in an experimental study in dogs.[95] The magnitude of proteinuria is a negative prognostic factor in spontaneous feline CKD.[96–98] Reduction of proteinuria is perhaps the most reliable evidence of benefit in an animal treated with antihypertensive agents, particularly in cats. With co-

existent renal azotemia, TOD to the kidney is more likely to occur at systolic BP >160 mm Hg in dogs and cats,[17,78,95,99] although a linear relation may exist between hypertensive injury and BP in dogs with CKD.[95] Hypertension may be present at any stage of CKD, and the serum creatinine concentration is not directly related to BP.[69,100] Hypertensive cats and dogs with CKD may have minimal or no azotemia.[101]

Ocular lesions are observed in many cats with hypertension, and although prevalence rates for ocular injury vary, it has been reported to be as high as 100%.[61,86,89,90,102–107] Ocular lesions also are common in hypertensive dogs.[85,86,99,108] This syndrome[109] is commonly termed hypertensive retinopathy or choroidopathy and has been frequently reported in dogs[85,86,99] and cats.[9,89,90,104,105,110–112] Exudative retinal detachment is the most commonly observed finding. Other lesions include retinal hemorrhage, multifocal retinal edema, retinal vessel tortuosity, retinal perivascular edema, papilledema, vitreal hemorrhage, hyphema, secondary glaucoma, and retinal degeneration (late sequela). Acute onset of blindness from complete, bilateral exudative retinal detachment may be a presenting complaint in both species.[9,86,110] Effective antihypertensive treatment can lead to retinal reattachment but restoration of vision[107] generally occurs in only a minority[89] of patients. Hypertensive ocular injury has been reported at systolic BP values as low as 168 mm Hg,[106] and there is a substantially increased risk of occurrence when the systolic BP exceeds 180 mm Hg.[86,102,110,111]

Hypertensive encephalopathy[113] has been reported in dogs[99] and cats,[9,28,89,114] and it occurs in people as a well-described entity that is characterized by white matter edema and vascular lesions.[115,116] Neurologic signs have been reported in 29%[89] and 46% of hypertensive cats.[9] Hypertensive encephalopathy also occurs after renal transplantation in people[117] and is a cause of otherwise unexplained death in this setting in cats.[114,118,119] In its early phases, this syndrome is responsive to antihypertensive therapy.[19,114] Hypertensive encephalopathy is more likely to occur in cats with a sudden increase in BP, a systolic BP that exceeds 180 mm Hg, or both.[19,28] Observed clinical signs are typical of intracranial disease and include lethargy, seizures, acute onset of altered mentation, altered behavior, disorientation, balance disturbances (eg, vestibular signs, head tilt and nystagmus), and focal neurologic defects due to stroke-associated ischemia. Other central nervous system abnormalities, including hemorrhage and infarction, which accompany chronic hypertension in people,[120] are also observed in dogs and cats.

Cardiac changes in hypertensive dogs include systolic murmurs, cardiac gallops,[85,121] and left ventricular hypertrophy.[121,122] Cardiac abnormalities are frequent, occurring in 4 out of 5 hypertensive cats.[89,90,102] When affected, the heart is a target organ and increased cardiac output rarely is the primary cause of hypertension in animals.[89] Clinical signs observed in affected cats include systolic murmur, gallop rhythm,[89,90,123] and cardiomegaly (generally left ventricular hypertrophy [LVH] but echocardiographic findings are vari-

able).[9,55,89,110,122,124] Although LVH may not be a risk factor for decreased survival time,[102] effective antihypertensive therapy may decrease the prevalence of LVH in affected cats.[125] Cardiac failure and other serious complications are infrequent but may occur.[9,89,126] Cats with previously undiagnosed hypertension may unexpectedly develop signs of congestive heart failure after receiving fluid therapy. Furthermore, cats with secondary hypertension due to other causes (eg, CKD) may die of cardiovascular complications,[90] as is frequently the case in hypertensive people.[127,128] Epistaxis, presumably due to hypertension-induced vascular abnormalities, has been associated with systemic hypertension.

Section 6: Prevalence and Selection of Patients for Hypertension Screening

There are at least 2 clear indications for evaluating BP in a patient. First, BP should be measured in patients with clinical abnormalities consistent with hypertensive TOD (Table 5); the presence of otherwise unexplained clinical findings associated with systemic hypertension should lead to BP measurement at the time of diagnosis. These include signs of hypertensive choroidopathy or retinopathy, hyphema, intracranial neurologic signs (eg, seizures, altered mentation, and focal neurological deficits), renal abnormalities (eg, proteinuria, microalbuminuria, azotemia), and cardiovascular abnormalities (ie, LVH, gallop rhythm, arrhythmia, systolic murmur, and epistaxis). In the absence of these clinical findings, a high index of suspicion must be maintained to diagnose systemic hypertension in animals. Clinical signs evident to the owner or the veterinarian may be subtle and attributable to aging or underlying clinical conditions that may as yet be undiagnosed. In people, early signs of hypertension are subjective and include morning headaches, facial flushing, and feelings of anxiety. Such clinical signs are difficult to recognize in dogs and cats. In cats, some nonspecific clinical signs have been associated with hypertension in the laboratory setting, including inactivity, lethargy, light sensitivity with frequent blinking, and altered (increased or decreased) appetite (S. Brown, unpublished data, 2004).

A second indication for the measurement of BP is the presence of diseases or conditions that are causally associated with secondary hypertension (Table 4), as well as those being treated with pharmacological agents that may increase BP (see Section 4). A thorough physical examination, including fuduscopic evaluation, cardiac auscultation, and neurologic examination, should be performed concurrently in these at-risk populations to assess TOD. Although the correlation between advancing age and prevalence of systemic hypertension is not as clear in animals as in humans, the conditions that cause secondary hypertension are more frequently observed in geriatric pets and it is prudent to screen routinely for the presence of these conditions (eg, CKD and hyperthyroidism).

The prevalence of hypertension in dogs and cats is not well known. Surveys of apparently healthy dogs have identified hypertension in 0.5% of 400 dogs,[129] 0.9% of

Table 6. Classification of blood pressure (BP in mmHg) in dogs and cats based on risk for future target-organ damage (TOD)[1,2,3]

Risk Category	Systolic BP		Diastolic BP	Risk of future TOD
I	<150	and[3]	<95	Minimal
II	150–159		95–99	Mild
III	160–179		100–119	Moderate
IV	≥180		≥120	Severe

[1] BP measurements should always be interpreted in light of the condition of the animal. Factors to consider include those that may alter cardiovascular control mechanisms (eg, excitement, anxiety, pharmacological agents) as well as those that may affect cardiovascular function directly (eg, dehydration, pharmacological agents).

[2] Breed, sex, and age variations should be considered in evaluating BP measurements. Known effects generally are small, except for the increased BP commonly observed in sighthounds (approximately 10–20 mm Hg).

[3] When reliable measurements (see text) lead to different categories based on separate consideration of the systolic and diastolic BP, the risk category of the patient should be taken as the higher risk.

1000 young dogs,[130] 2% of 215 dogs,[131] and 10% of 102 dogs.[33] Others have provided evidence that dogs are resistant to the development of hypertension.[42,100,132] The prevalence of hypertension may be comparable in cats. In one study of 104 apparently healthy cats, 2% had systolic BP >170 mm Hg.[59] However, as noted elsewhere in these guidelines, the lack of uniform measurement techniques, variable inclusion criteria, and inconsistent thresholds for establishing a diagnosis of hypertension in veterinary medicine make it difficult to interpret the prevalence data. Nonetheless, hypertension seems to be rare in young, otherwise healthy animals. Based on the available data, it is still unclear whether routine screening of healthy animals is a reliable method of detecting hypertension in the population, and the risk of false diagnosis is high with widespread screening. Furthermore, there is limited evidence to support the diagnosis and treatment of hypertension as an isolated problem. Therefore, the panel does not recommend routine screening of all dogs and cats for the presence of systemic hypertension. One approach, which is endorsed by some members of the panel, is to employ a screening program that achieves baseline values in animals by measuring BP at 2–3 years of age, again at 4–6 years of age, and a third time at 7–9 years of age, in order to develop reference values for that particular animal (not to identify hypertension). Some conditions (eg, hyperthyroidism, hyperadrenocorticism, and CKD) that cause secondary hypertension are more prevalent in older animals, yet they may be occult. Thus, it is reasonable to institute screening of cats and dogs that are ≥10 years of age. Still, high BP results in otherwise healthy, particularly young, animals should be assumed to reflect white-coat hypertension until proven otherwise.

Section 7: Diagnosis of Hypertension

A decision to use antihypertensive therapy should always be based on reliable measurements of BP. Diagnosis should rarely be established on the basis of a single measurement session. Multiple measurements, as well as a thorough search for TOD (Table 5) and conditions that may cause secondary hypertension (Table 4) should be considered before establishing a diagnosis. In patients with TOD consistent with
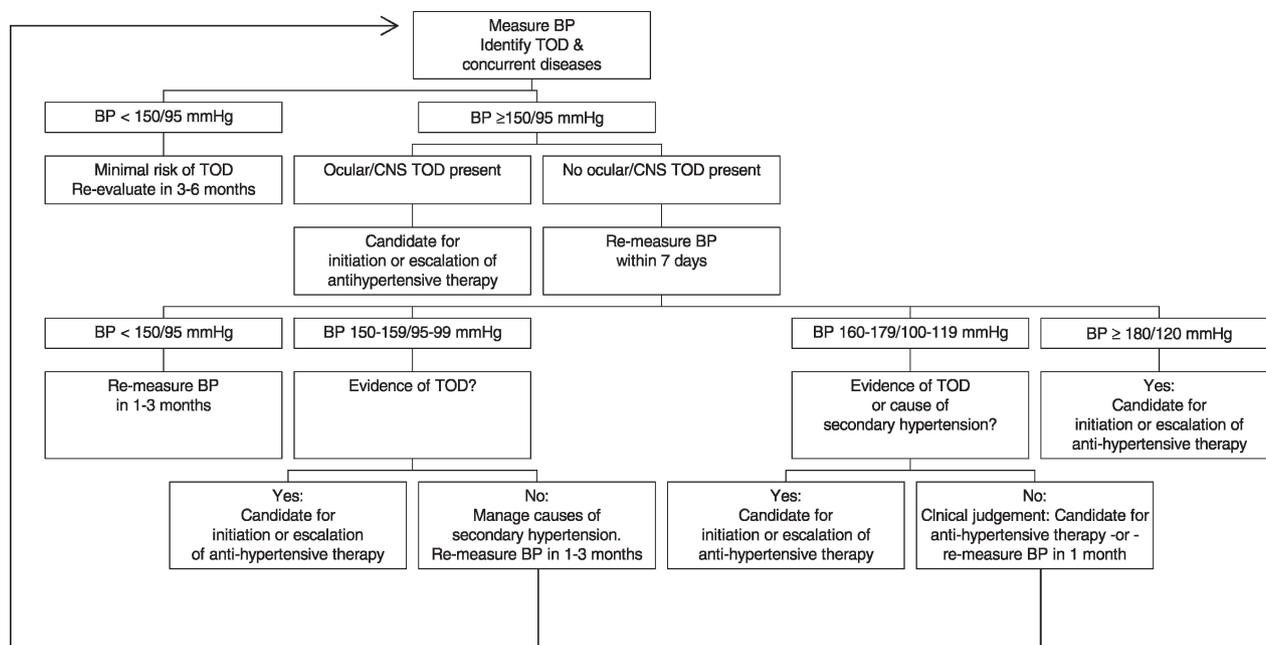

Fig 1. The recommended approach to patient evaluation is based upon reliable measurements of blood pressure (BP). However, the identification and characterization of pre-existing target organ damage (TOD), the recognition of concurrent conditions (especially those causing secondary hypertension), and the categorization of risk for further TOD form the basis for treatment decisions.

hypertension that may rapidly progress (eg, hypertensive retinopathy/choroidopathy or encephalopathy), a single measurement of BP can be used to establish temporarily a rationale for antihypertensive therapy. Although there are breed-related differences in BP in dogs, only the mean difference (10–20 mm Hg higher values for each category) for sighthounds mandates separate categorization at this time.[42,71] Publication of data for specific breeds of dogs and cats may justify further modification of this recommendation in the future.

We recommend the categorization of BP on the basis of risk of developing subsequent TOD (Table 6). In humans, any reduction of BP that does not produce overt hypotension lowers the risk of TOD. Admittedly, this latter finding remains to be confirmed in dogs and cats.

Minimal Risk of TOD (BP <150/95 mm Hg)

BP below 150/95 mm Hg should be interpreted as representing minimal risk for TOD, and antihypertensive therapy is not recommended (Fig 1). This BP also represents the treatment goal for antihypertensive therapy. The panel uses the term minimal because certain predisposing causes (eg, CKD) can make an animal more susceptible to additional TOD (eg, progressive renal injury). Furthermore, there is a continuous relationship between BP and risk of TOD in people as BP increases above 120/80 mm Hg.[2,3] However, comparable data relating risk to BP <150/95 mm Hg in dogs and cats are not available. The risk of TOD in these animals is in part related to the fact that minute-to-minute variability of BP, inconsistency of BP measuring devices, technical errors, transient dehydration, negative white-coat effects or some combination of these factors could lead to a falsely low measurement of BP, and periodic re-evaluation should be employed in a patient with TOD and a BP <150/95 mm Hg.

Mild Risk of TOD (BP 150–159/95–99 mm Hg)

Currently, there is a paucity of data to support intervention when the BP is <160/100 mm Hg, and we do not recommend routine antihypertensive therapy in this situation (Fig 1). Some surveys of normal dogs and cats have reported values within or near this range (Table 3). Furthermore, it is likely that some animals in this category are exhibiting white-coat hypertension. We do not know if the presence of white-coat hypertension is a risk factor for TOD in dogs and cats as it is in people[79] but we do not recommend treatment in the absence of knowledge in this regard.

Moderate Risk of TOD (BP 160–179/ 100–119 mm Hg)

The rationale for the treatment of patients with this category of risk is to limit subsequent TOD, particularly in those animals with pre-existent TOD. Some changes, such as LVH[125] and hypertensive encephalopathy,[114] may partially or wholly resolve with therapy. Antihy-

pertensive therapy may reduce the incidence or delay the development of other abnormalities, such as hypertensive encephalopathy or choroidopathy.[19,114,133] Antihypertensive therapy may slow disease progression in animals with CKD.[134,135] Most animals in this category, particularly those with TOD or secondary hypertension, are candidates for antihypertensive therapy (Fig 1). Other animals without TOD, particularly those with BP at the lower end of this range or those in which white-coat hypertension cannot be ruled out as the sole cause of the increased BP, generally will not be treated. The data remain incomplete for dogs and cats, and this decision requires the integration of all factors related to the patient and sound clinical judgment.

Severe Risk of TOD (BP ≥ 180/120 mm Hg)

The rationale for treating patients with this category of risk is to limit the degree of TOD, for which the risk is high. Although only extreme technical error or dramatic white-coat hypertension would be expected to produce such a large increase in BP in a normal dog or cat, we recommend at least 2 measurement sessions to confirm the degree of risk. The sole exception would be a patient in which a potentially rapidly progressive TOD, such as hypertensive choroidopathy or encephalopathy, is already present. Animals in this risk category are candidates for antihypertensive therapy (Fig 1) and appropriate, disease-specific management of any conditions that might be causing secondary hypertension.

Section 8: Management of the Hypertensive Patient

The Hypertensive Patient: Evaluation and Decision to Treat

The goals of this assessment (Fig 1) include recognizing conditions that may be contributing to increased BP, identifying and characterizing TOD, and determining if there are any seemingly unrelated concurrent conditions that may complicate antihypertensive therapy. Underlying diseases that may be causing secondary hypertension should be identified and treated while continuing to monitor BP. With the exception of advanced hypertensive choroidopathy or encephalopathy, antihypertensive therapy generally is not an emergency intervention (see "The Hypertensive Patient: Emergency Management" below). Since hypertension is often a silent, slowly progressive condition that requires vigilance and life-long therapy, it is important to be absolutely certain about the diagnosis; a high BP measurement may represent idiopathic, secondary or white-coat hypertension. A decision to treat (Fig 1) is made on the basis of categorization of risk (Table 6) for further TOD, characterization of concurrent conditions, and existing TOD. Appropriate management of selected patients may require referral, so as to utilize the complementary skills or second opinions of outside experts.

The client should be educated thoroughly at the outset and involved in all decision-making processes. The client
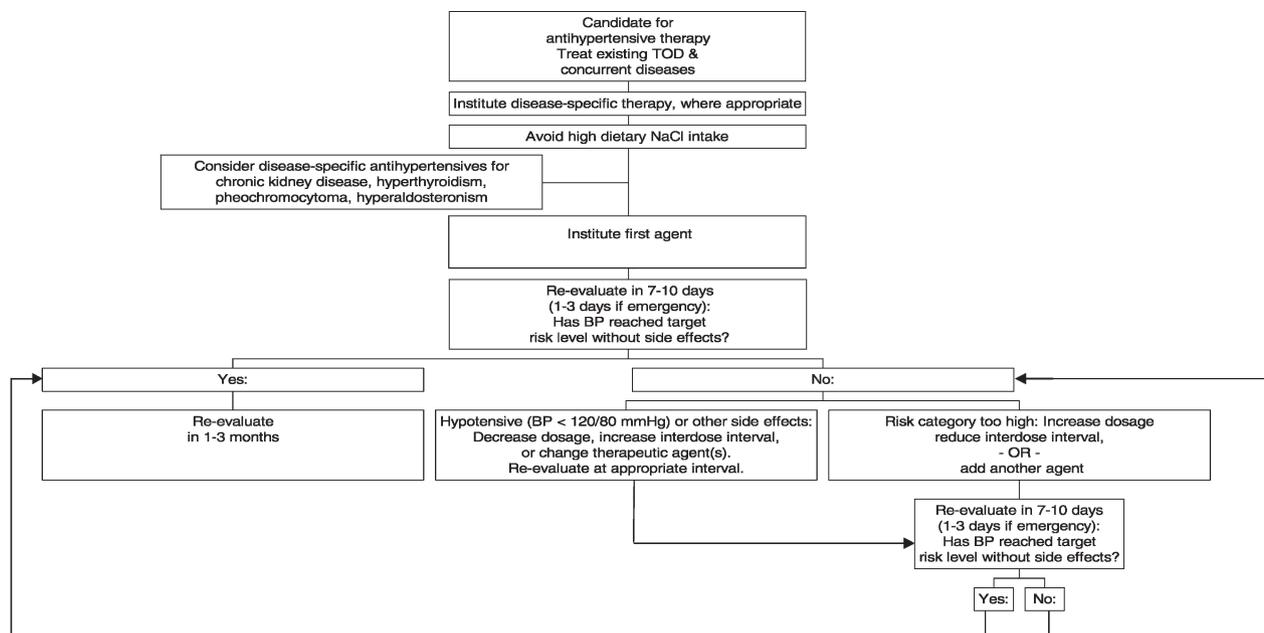
Fig 2. Management of hypertension involves a stepwise approach, including consideration of therapy for TOD and concurrent conditions, and staged institution of dietary and pharmacological regimens. Thorough patient evaluation, repeated BP measurements, and scheduled reassessment of treatment decisions are essential. At each evaluation, therapeutic intervention or escalation of therapy should be based on BP measurements and evidence of TOD.

should understand what is meant by the term systemic hypertension, that treatment will often be lifelong and generally prevent adverse consequences rather than immediately improving the quality of life of their pet, that the disease is silent, and that control does not mean cure. The veterinarian should provide the client with blood pressure readings, a working knowledge of complications (of hypertension and the drugs used to manage it) and their identification, and a clear outline of the goals of treatment. The owner of a hypertensive pet should never leave the office without a clear plan for re-evaluation, preferably after scheduling the next appointment.

Hypertensive Patient: Treatment

As hypertension in dogs and cats is most often secondary ($\geq$80% based on current data), antihypertensive drug therapy by itself is often not sufficient. Initial considerations (Fig 2) should always include the identification and management of conditions likely to be causing secondary hypertension and the identification and treatment of TOD. If possible, these considerations should be addressed with specific, targeted diagnostic and therapeutic regimens. Effective management of a condition that causes secondary hypertension will lead to complete or partial resolution of high BP in some, but not all, cases.[69,81,99] Decisions as to the use of antihypertensive drugs should be based on the integration of all clinically available information, and a decision to treat (which may effectively mandate lifelong drug therapy) warrants periodic, judicious re-evaluation.

The therapy must be tailored to the patient and its concurrent conditions. Once-daily treatment is ideal; fewer treatments are always preferred. A gradual,

persistent reduction of BP is the therapeutic goal. Generally, acute and severe decreases in BP should be avoided. If the antihypertensive agent of choice is only partially effective, the usual approach is to consider increasing the dosage or adding an additional drug. Although not ideal, management of highly resistant hypertension in people often requires 4 to 5 agents, and most veterinary patients with clinically relevant hypertension will require more than 1 agent. It is often helpful to discuss the variable nature of responses to antihypertensive medications with the owner when the first medication is prescribed.

Although frequently recommended as an initial step in the pharmacological management of high BP (Fig 2), dietary salt restriction is controversial,[18,104,136] and the available evidence suggests that substantial sodium restriction alone generally does not reduce BP.[18,136,137] In fact, sodium restriction activates the renin-angiotensin-aldosterone axis[18,85] and may actually increase BP in certain settings.[136,137] These effects may lead to progression of undesirable vascular, renal, and cardiac changes[138–142] and necessitate utilization of antihypertensive agents that interfere with this hormonal system (eg, angiotensin-converting enzyme inhibitors [ACEI], angiotensin receptor blockers [ARB], and aldosterone receptor blockers). However, this approach is controversial, as high salt intake may produce adverse consequences in some settings,[143] particularly in animals with CKD.[104] Currently, there is no clear rationale for this approach and the panel recommends avoiding high dietary sodium chloride intake in hypertensive animals but does not recommend that a specific effort be made solely to restrict dietary sodium chloride intake. Until

Table 7. Oral antihypertensive agents.

Class	Drug (Trade Names)	Usual Oral Dosage
Angiotensin-converting enzyme inhibitor	Benazepril (Lotensin, Fortekor)	D: 0.5 mg/kg q12–24h C: 0.5 mg/kg q12h
	Enalapril (Vasotec, Enacard)	D: 0.5 mg/kg q12–24h C: 0.5 mg/kg q24h
Calcium channel blocker	Amlodipine (Norvasc)	D/C: 0.1–0.25 mg/kg q24h (up to 0.5 mg/kg in cats)
α_1 blocker	Prazosin (Minipress)	D: 0.5–2 mg/kg q8–12h C: 0.25–0.5 mg/cat q24h
	Phenoxybenazime (Dibenzyline)	D: 0.25 mg/kg q8–12h or 0.5 mg/kg q24h C: 2.5 mg per cat q8–12h or 0.5 mg/cat q24h
	Acepromazine (PromAce)	D/C: 0.5–2 mg/kg q8h
Direct vasodilator	Hydralazine (Apresoline)	D: 0.5–2 mg/kg q12h (start at low end of range) C: 2.5 mg/cat q12-24h
Aldosterone antagonist	Spironolactone (Aldactone)	D/C: 1.0–2.0 mg/kg q12h
β Blocker	Propranolol (Inderal)	D: 0.2–1.0 mg/kg q8h (titrate to effect) C: 2.5–5 mg/cat q8h
	Atenolol (Tenormin)	D: 0.25–1.0 mg/kg q12h C: 6.25–12.5 mg/cat q12h
Thiazide diuretic	Hydrochlorothiazide (HydroDiuril)	D/C: 2–4 mg/kg q12-24h
Loop diuretic	Furosemide (Lasix)	D/C: 1–4 mg/kg q 8-24h

D, dog; C, cat.

more data are available, the selection of appropriate diet should be based on other patient-specific factors, such as underlying or concurrent diseases and palatability.

Once a decision is made to treat an animal with high BP, therapeutic intervention will generally be carried out with a pharmacological agent. Furthermore, certain disease conditions may be best addressed with specific classes of agents, such as beta-blockers for hypertension associated with hyperthyroidism, alpha- and beta-blockers or surgical excision for pheochromocytomas, aldosterone receptor blockers or surgical excision of adrenal tumors in animals with hypertension associated with hyperaldosteronism, and some combination of ACEI, ARB, and aldosterone receptor blockers for hypertension associated with CKD in dogs.[19,134,135,144,145]

ACEI and calcium channel blockers (CCB) are the most widely used antihypertensive agents in veterinary medicine. In dogs, ACEI are usually recommended as the initial agent of choice. Although there has been some concern regarding acute exacerbation of azotemia with these agents, this is an unusual complication of ACEI therapy.[97,146–148] Since ACEI preferentially dilate the efferent arteriole, they lower the intraglomerular pressure[135,146] and frequently decrease the magnitude of proteinuria.[97,134,135,149] The magnitude of severity of proteinuria is a negative prognostic factor in feline CKD,[96–98] and decreased proteinuria is associated with prolonged survival. Furthermore, the adverse cardiac and renal consequences of angiotensin II and aldosterone[138–142] may be attenuated by this class of agent. However, a secondary consequence of efferent arteriolar dilatation is a theoretical tendency for the glomerular filtration rate (GFR) to decline. Single-nephron studies indicate that this is not necessarily the case in dogs[135] and cats.[146] In dogs and cats with CKD that is not

complicated by cardiovascular disease, the administration of ACEI commonly produces only very modest increases in serum creatinine concentration (<0.5 mg/dL; <50 µmol/L) and this degree of change is usually tolerable. ACEI have been shown to have no adverse effects on renal function, based on serum creatinine concentrations, when administered chronically to older dogs with heart disease but not heart failure.[150] However, ACEI should not be used in dehydrated patients in which the GFR might drop precipitously. These patients should be carefully rehydrated and then re-evaluated before instituting antihypertensive therapy. Nonetheless, a BP <120/60 mm Hg combined with clinical findings of weakness, syncope, or tachycardia indicate systemic hypotension and therapy should be adjusted accordingly (Fig 2).

In cats, although the renin-angiotensin-aldosterone axis may play a role in the genesis or maintenance of systemic hypertension,[60,151–155] CCB are often the first choice for antihypertensive therapy due to established efficacy.[19,90,125,156,157] A mean decline in systolic BP of 40–55 mm Hg is typically observed in cats with moderate-to-high risk of TOD.[90,156,157] Despite significant antihypertensive efficacy, CCB have not been shown to increase survival time in treated cats[90] and their use may activate the systemic or intrarenal renin-angiotensin system. A key predictive factor is the effect on proteinuria; a renoprotective effect is predicted if the antihypertensive regimen used decreases proteinuria. In this setting, the coadministration of ACEI and CCB is a possibility.[144,158,159] Combination therapy may be especially useful in cats, in which there is some evidence for a beneficial effect of ACEI in CKD[97,149] and for an established antihypertensive efficacy of CCB. This approach is being evaluated[160] but is in need of further study.

There are several other agents with BP-lowering efficacy (Table 7) and these may be used as appropriate in patients in which adequate risk reduction is not achieved with ACEI, CCB or a combination of these drugs (Fig 2). Although diuretics are frequently administered to hypertensive people, these agents are not first-choice drugs for veterinary patients with CKD, in which dehydration and volume depletion may prove problematic. However, diuretic and other classes of antihypertensive agents may prove useful in individual patients. Diuretics should be considered in hypertensive animals in which volume expansion is apparent (eg, those with edema).

The goal of antihypertensive therapy is to decrease the magnitude, severity, and likelihood of TOD. Hypertension generally is not an emergency situation, and rapid reductions of BP usually should not be sought aggressively. Studies in people indicate that reduction of risk for TOD using antihypertensive therapy is a continuum and that the lower the BP, the lower the risk for TOD. Analysis of the results of a recent laboratory study in dogs[95] suggests that BP is a continuous risk marker for progression of kidney disease, which may partly justify a similar approach in veterinary patients. The panel feels that regardless of the initial magnitude of BP, the goal of therapy should be to decrease maximally the risk of TOD (SBP <150 mm Hg and DBP <95 mm Hg), and that antihypertensive therapy should be adjusted on re-evaluation if the BP is ≥150/95 mm Hg or the systolic BP is too low (<120 mm Hg). Certainly, a minimal goal of therapy is to achieve a reduction in the category of risk for TOD (Table 6).

The Hypertensive Patient: Follow-Up

Follow-up evaluations should include measurements of BP, serum creatinine concentration, urinalysis, funduscopic examination, and other specific assessments depending on the individual circumstances (eg, TOD, causes of secondary hypertension, concurrent conditions). Since signs of progression to TOD can be subtle, BP should be closely monitored over time in patients receiving antihypertensive therapy, even when the hypertension is seemingly well-controlled. The frequency and nature of re-evaluations will vary depending on BP category, BP stability, other aspects of the health of the patient, and the frequency of dosage adjustment of the antihypertensive therapy. The assessment of any associated conditions or intercurrent diseases at intervals is dictated by standard management practices for those conditions. Animals with serious or rapidly progressive lesions (eg, ocular or neurological signs) constitute a special category, regardless of the magnitude of BP, and should be re-evaluated in 1–3 days. In other animals, we recommend re-evaluation 7–10 days after changes in therapy and at 1–4-month intervals depending on stability (more frequent if BP or other conditions are unstable) and the magnitude of the hypertension (more frequent if BP remains >180 mm Hg). Hospitalized patients, particularly those receiving fluid therapy or pharmacological agents with cardiovascular effects, should be assessed daily.

For animals with TOD, organ-specific markers should be monitored. For example, proteinuria and microalbuminuria are adverse consequences of systemic hypertension in animals with coexistent CKD. With respect to the benefits of decreasing BP in animals with CKD, decreasing the magnitude of proteinuria is perhaps the most effective treatment goal,[161] particularly in cats.

The Hypertensive Patient: Emergency Management

The magnitude of increase in BP or TOD present may dictate that some patients require immediate and more aggressive attention. With acute onset of TOD that may be rapidly progressive (eg, severe hypertensive choroidopathy and encephalopathy), it is appropriate to consider treatment if the BP is ≥180/120 mm Hg (Severe Risk Category) for only 1 measurement session, although subsequent re-evaluations of the decision to treat the patient are mandated. Although the panel does not recommend that a patient with high BP but no TOD be treated as an emergency, it is prudent to institute therapy and re-evaluations rapidly in animals with severe risk for TOD.

For emergency management, agents with a rapid onset of action are indicated. Appropriate agents include parenteral agents, such as hydralazine (0.2 mg/kg IV or IM, repeated q2h as needed), enalaprilat (0.2 mg/kg IV, repeated q1–2h as needed), labetolol (0.25 mg/kg IV over 2 minutes, repeated up to a total dose of 3.75 mg/kg, followed by a constant rate infusion of 25 μg/kg/min), and esmolol (50–75 μg/kg/min constant rate infusion),[162] as well as those with rapid onset of efficacy when administered orally (eg, amlodipine besylate at 0.1–0.25 mg/kg q24h; dosages up to 0.5 mg/kg q24h may be employed with caution). If parenteral medications are used, continuous BP monitoring by arterial catheterization is strongly recommended. Many clinicians prefer oral CCB, particularly in cats, as they generally decrease BP in severely hypertensive animals regardless of primary disease, and these agents have limited risk of causing hypotension.

The BP and TOD should be assessed frequently (every 1–3 days) and the drug dosage or interdose interval adjusted accordingly. Once the BP or TOD is stabilized at an appropriate level, the patient should be managed on the basis of our other recommendations. These patients should be carefully re-evaluated before instituting chronic antihypertensive therapy.

Section 9: Conclusions

This panel recognizes that additional lines of evidence are required to advance this important area of veterinary medicine. We specifically call for large multicenter clinical studies to establish normal values based on breed, age, sex, and measurement technique. In addition, the proper clinical approach to the measurement of BP should be more precisely defined. For example, should we screen all dogs and cats over a certain age, measure BP only in animals with associated clinical signs

or disease conditions, and make different recommendations for dogs and cats? Furthermore, the link between increased BP and adverse events, the magnitude of increase in BP for which intervention is warranted, and the benefits and adverse effects of various antihypertensive therapies should all be determined in future studies.

At the ACVIM meeting in Dallas in 2002, our ACVIM Consensus Study Panel and an international group with interest in the field of veterinary hypertension (the International Blood Pressure Forum) formed the joint Veterinary Blood Pressure Society (VBPS). The members of VPBS have been involved in the preparation of these guidelines and it is anticipated that VPBS will revise and publish new BP guidelines as further advances are made in this field.

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Appendix

Validation of BP Measurement Devices according to ACVIM Hypertension Consensus Panel and Veterinary Blood Pressure Society Recommendations (AHCP-VBPS Validation) Rationale

Recommended standards for the validation of BP measuring devices in people have been published by the Association for the Advancement of Medical Instrumentation.[66,67] For pressure measurements to be comparable among the devices in veterinary use, it will be important to establish universal veterinary standards for the validation of BP measurement devices. Such standards would foster the development of standard measurement protocols and reliable reference ranges for dogs and cats. The current recommendations for BP validation of the Association for the Advancement of Medical Instrumentation have been met by one BP measurement device according to one study,[56] although the device failed a validation test in a second study.[22] Both of these studies utilized anesthetized cats, in which pressure measurements are more readily obtained, are more homogeneous, and are probably not directly applicable to screening conscious patients. The panel recognizes that obtaining accurate and precise BP measurements is substantially more difficult in dogs and cats than in people. Nonetheless, we recognize the utility of the existing guidelines and recommend that modified guidelines be applied to indirect devices used in veterinary medicine for validation purposes. Until validated devices become widely available, the approach endorsed by this panel is to utilize receiver-operator curve characteristics.[35]

Calibration

Even if validated, the calibration of a device can drift over time, producing an unrecognized artifactual bias that can lead to false diagnoses. An indirect BP measuring device used in clinical practice should be tested against a standard to assure accuracy at least twice annually. There are routine methods and standards for testing the accuracy of these devices,[66] and the instruments should have a test mode and instructions for achieving this calibration check. Veterinary practices probably will not have a mercury manometer or national pressure standard readily available. A suitable alternative is to compare the device against a second pressure-measuring instrument. In brief, the static pressure output of the device should be compared to a static pressure-measuring device (eg, an aneroid manometer) at 10–20 mm Hg increments across the clinically useful range of 30–300 mm Hg, to ensure agreement within ± 3 mm Hg across this range. Aneroid manometers used in conjunction with an indirect device, such as a Doppler ultrasonographic instrument, should similarly be assessed for accuracy by comparison to a second device. Failure to meet these accuracy criteria should lead to action to replace or repair the indirect device.

Validation

- Our recommendations are based on the guidelines of the Association for the Advancement of Medical Instrumentation (AAMI) for validation of blood pressure (BP) measurement devices intended for use in people. However, this panel recognizes that: (i) BP measurement is substantially more difficult with indirect devices in dogs and cats than in people; (ii) the available data in the literature demonstrate substantial heterogeneity of indirect devices; (iii) no device presently in use is likely to meet the published AAMI guidelines when applied to conscious dogs and cats; (iv) the adoption of a standardized approach to the evaluation of indirect BP devices is valuable in veterinary medicine; and (v) the intervention points in dog and cats are substantially higher than those currently used in people.
- The tested indirect device should be compared to a direct, intra-arterial pressure measurement device or an indirect device for which validation has been previously published in a refereed journal. A device is validated for only the species and conditions in which the validation test is conducted. Thus, a device validated for use in anesthetized cats is not validated for use in anesthetized dogs or conscious cats. A device may be validated for systolic measurements, diastolic measurements or (preferably) both.
- In general, the criteria and recommendations of the AAMI must be followed. These include recommendations for patient selection, pressure range, number of observers, blinding of observations, and reporting of study findings.
- System efficacy is validated if the following conditions are met:
  - The mean difference of paired measurements for systolic and diastolic pressures treated separately is ±10 mm Hg or less, with a standard deviation of 15 mm Hg or less;
  - the correlation between paired measures for systolic and diastolic pressures treated separately is ≥0.9 across the range of measured values of BP;
  - 50% of all measurements for systolic and diastolic pressures treated separately lie within 10 mm Hg of the reference method;
  - 80% of all measurements for systolic and diastolic pressures treated separately lie within 20 mm Hg of the reference method;
  - the study results have been accepted for publication in a referred journal; and
  - the subject database contains no fewer than 8 animals for comparison with an intra-arterial method or 25 animals for comparison with a previously validated indirect device.