

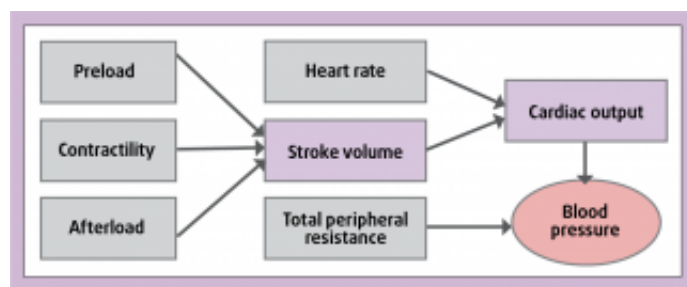
# Management and treatment of hypotension and hypertension

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Categories : [Companion animal](#), [Vets](#)

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Measurement of systemic blood pressure (BP) is becoming increasingly common in veterinary patients. Initially driven by the diagnosis of hypertension in cats, we are now recognising blood pressure issues are also common in dogs.



**Figure 1.** Cardiac and vascular factors influencing blood pressure.

We care what an animal's BP is as it is a surrogate marker of tissue perfusion, as well as describing the peak wall stress on blood vessels in the body. BP can be described in terms of systolic, diastolic and mean arterial pressure.

The American College of Veterinary Internal Medicine has produced guidelines for the identification, evaluation and management of systemic hypertension in dogs and cats (2006) that includes suggested reference intervals for BP (**Table 1**), but acknowledges BP measured is also dependent on:

- operator
- measurement system
- breed
- physiologic state of the patient (white coat effect)

Arterial blood pressure (ABP) is affected by cardiac output and vascular resistance (VR), and these factors are inter-related (**Figure 1**).

$$\text{ABP} = \text{cardiac output (CO)} \times \text{VR}$$

$$\text{CO} = \text{heart rate (HR)} \times \text{stroke volume (SV)}$$

$$? ABP = HR \times SV \times \text{total peripheral resistance (TPR)}$$

## Hypotension

Risk category	Systolic blood pressure (mmHg)	Diastolic blood pressure (mmHg)	Risk of target organ damage
I	<150	<95	Minimal
II	150-159	95-99	Mild
III	160-179	100-119	Moderate
IV	≥180	≥120	Severe

**Table 1.** American College of Veterinary Internal Medicine consensus statement on canine blood pressure.

While a general tendency exists to focus on hypertension when measuring BP, hypotension can have much more immediately profound and life-threatening consequences to the patient. Clinical signs of hypotension are listed in **Table 2**.

Hypotension is most commonly encountered in primary care practices in anaesthetised patients. Any disease process that leads to a fall in stroke volume, HR or peripheral resistance can lead to hypotension depending on the ability of the patient to compensate.

## Management

The best approach to the management of hypotension is to treat the underlying cause (**Table 3**), but in patients where the hypotension is severe, the initial goal is simply to raise the BP usually by fluid therapy. Hypotension is defined as a systolic arterial pressure (SAP) of less than 90mmHg, or a mean arterial pressure (MAP) of less than 60mmHg.

Renal autoregulation can normally maintain glomerular filtration pressure with systolic BP between 80mmHg and 200mmHg. Therefore, a systolic BP lower than 80 requires urgent intervention to prevent acute kidney injury (AKI).

Table 2. Clinical signs of hypotension	
Non-septic	Septic/systemic inflammatory response syndrome
Tachycardia	Tachycardia
Bounding → weak pulses	Mucosa injected → red
Pale mucous membranes	Rapid CRT
Slow capillary refill time (CRT)	
Mental dullness	
Weakness	
Hyperventilation	
Hypothermia and cold extremities	
↓ urine output	

**Table 2.** Clinical signs of hypotension.

The ability of renal autoregulation can be severely compromised by the use of angiotensin-converting enzyme (ACE) inhibitors and NSAIDs – especially in combination (**Figure 2**).

### Key decisions

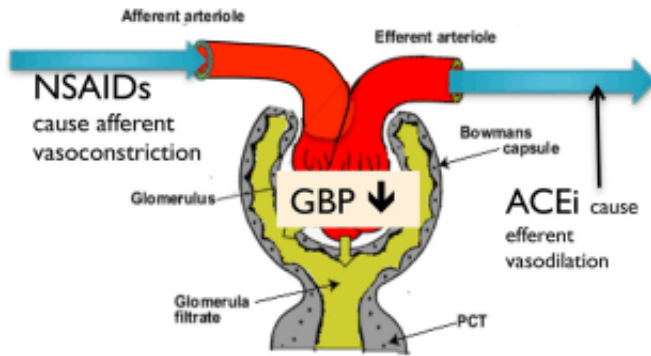
Is it decreased preload (for example, hypovolaemia) versus cardiac dysfunction versus vasodilation? Differentiating cardiac dysfunction from hypovolaemia is a key decision as, for many patients, the mainstay of treatment will be the use of fluids. Clearly, if significant cardiac dysfunction exists (which is rare in the absence of cardiac-related signs – murmur or arrhythmia), high fluid rates could cause potentially fatal pulmonary oedema.

### Treatment

For decreased preload and/or vasodilation, consider:

- volume loading – shock rates indicated
  - isotonic crystalloid versus colloids versus hypertonic saline versus blood products
- use of pressor agents

A guideline for a dose of bolus fluids includes 20ml/kg to 30ml/kg of crystalloids, 5ml/kg to 20ml/kg of synthetic colloids and 2ml/kg to 4ml/kg of hypertonic saline. Synthetic colloids have been largely withdrawn from the market due to poorer outcomes compared to crystalloids under certain circumstances in people.



**Figure 2.** Effects of angiotensin-converting enzyme inhibitors and NSAIDs on glomerular blood pressure.

Low-molecular-weight, gelatin-based compounds are still available. Hydroxyethyl starches, such as pentastarch and hetastarch, may become available again in the future. Blood products are generally indicated if there is marked blood loss.

With regards to cardiac dysfunction, manage underlying cardiac issues, including:

- positive inotropes
- anti-arrhythmic drugs
- vasoactive drugs

### Vasopressor drugs

Dobutamine (2.5micrograms/kg/min to 10micrograms/kg/min), a  $\beta_1$  agonist, increases cardiac output by enhancing myocardial contraction.

Dopamine in dogs enhances renal and mesenteric perfusion at a dose of 2.5mcg/kg/min. At higher doses (5mcg/kg/min to 10mcg/kg/min), dopamine exerts its effects on  $\beta_1$  and  $\alpha_1$ -receptors, resulting in vasoconstriction and increased myocardial contractility. At doses of 10micrograms/kg/min to 20micrograms/kg/min, dopamine can cause constriction of the renal arteries, thereby reducing kidney perfusion and causing potential irreversible renal damage.

Norepinephrine is a potent  $\alpha_1$ -mediated vasoconstrictor and is usually reserved for severe, unresponsive hypotension at doses of 0.05mcg/kg/min to 1mcg/kg/min.

Decreased preload		Decreased cardiac function	Decreased vascular tone
Hypovolaemia	Decreased venous return		
Haemorrhage	Pericardial tamponade	Cardiomyopathy	Sepsis/systemic inflammatory response syndrome
Trauma	Restrictive pericarditis	Valvular disease	Anaphylaxis
Gastrointestinal losses	Severe pneumothorax	Bradyarrhythmias	Neurogenic
Polyuria	Positive pressure ventilation	Tachyarrhythmias	Drug-induced
Hypoadrenocorticism	Gastric dilatation and volvulus	Electrolyte abnormalities	Anaesthetic agent
Effusions or third spacing of fluid	Endoscopy	Acid-base disturbance	Vasodilators
Burns	Heartworm disease (caval syndrome)	Severe hypoxia	$\beta$ -blockers
Heatstroke		Sepsis/systemic inflammatory response syndrome	Calcium channel blockers
			Electrolyte abnormalities
			Acid-base disturbances
			Severe hypoxia

**Table 3.** Causes of hypotension.

Ephedrine is a non-catecholamine sympathomimetic that stimulates both  $\alpha$  and  $\beta$ -receptors directly and indirectly via norepinephrine and is being increasingly used to manage intraoperative hypotension at a dose rate of 0.05mg/kg to 0.2mg/kg IV, repeated as necessary until the desired BP is achieved; a constant rate infusion at 5mcg/kg/min to 10mcg/kg/min can be used if continued BP is required.

Where there is less experience in managing this drug, giving small doses of 0.03mg/kg every 30 to 40 seconds is the safest way to achieve an acceptable BP. Duration of action is short (5 to 15 minutes), so further support to BP should be given, but the ephedrine may need to be repeated.

High doses of ephedrine can cause sufficient vasoconstriction to lead to reduced renal perfusion and potentially AKI. Ephedrine is appropriate for patients with American Society of Anesthesiologists scores of I or II.

Arginine vasopressin (AVP), or antidiuretic hormone, is produced in the pituitary gland and released in response to hypovolaemia. AVP has been found in both humans and veterinary patients to be decreased in cases of septic shock (vasoplegia), and may be the vasopressors of choice when treating dogs with septic shock-related hypotension.

One veterinary study (Silverstein et al, 2007) evaluated the use of vasopressin to treat sepsis-related hypotension in five dogs with dopamine-resistant hypotension. MAP significantly increased in all five dogs, with a mean dose administered of 2.1 micro-units/kg/min.

### **Intraoperative hypotension**

With regards to intraoperative hypotension, the following can be carried out:

- checking the tube
- reducing anaesthetic depth
- maintaining body temperature

- hypothermia reduces effectiveness of anticholinergic drugs
- managing arrhythmias
  - bradycardia – use glycopyrrolate or atropine if HR less than 60 in large breed dogs and less than 80 in medium to small breed dogs
- increasing vascular volume
  - IV bolus of fluid 5ml/kg to 10ml/kg crystalloids
- use of vasopressors (see aforementioned)
- what’s happening to end-tidal CO2 or blood gas analysis

## Systemic hypertension

Table 4. Differential diagnosis of hypertension	
Primary “essential” hypertension	
Secondary hypertension	
Common causes	Rare causes
Renal disease	Primary hyperaldosteronism
Diabetes mellitus	Acromegaly
Hyperadrenocorticism	Hyperparathyroidism
Hyperthyroidism	Phaeochromocytoma
Stress response	Polycythaemia
Use of hypertension-inducing medication	Anaemia
Phenylpropanolamine, corticosteroids	
? Ageing	
? Obesity	

**Table 4.** Differential diagnosis of hypertension.

Despite a lot of conference presentations on hypertension, comparatively few published studies exist on prevalence, diagnosis and treatment of hypertension in dogs. This, in part, reflects the difficulties in accurate, reliable and repeatable measurement of systolic BP in dogs.

The complexities of accurate BP measurement is demonstrated by the [National Institute for Health and Care Excellence guidelines in man](#), but also give a number of pointers of how BP monitoring in veterinary practice can be made more accurate and reproducible.

Hypertension can be divided into primary disease or the hypertension can be secondary to another underlying cause (**Table 4**). As in man, in the majority of cases, hypertension is a silent disease or causes low-grade, non-specific signs – typically, dullness, lethargy, depression and reduced exercise tolerance, often dismissed as signs of ageing.

Some patients present with a hypertensive prior crisis, such as:

- ocular signs – haemorrhage (**Figure 3**), acute onset blindness, glaucoma
- epistaxis
- intracranial neurologic or vascular disease
- heart failure
- chronic kidney disease

## Further investigation

To investigate further, the following should be considered:

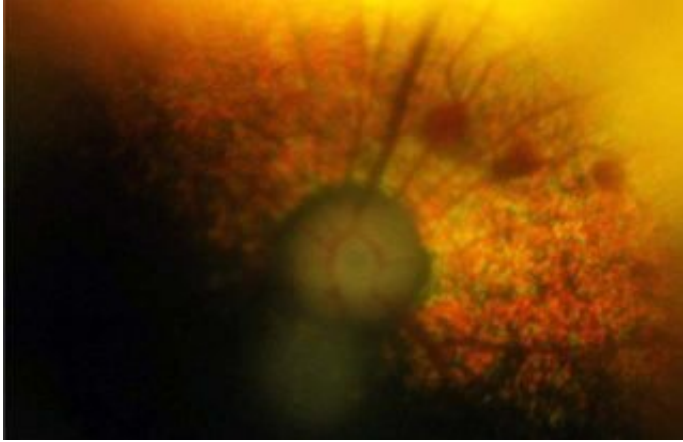
- primary tests
  - fundoscopic examination
  - biochemistry, including electrolytes, cholesterol and triglycerides
  - haematology
  - urine protein:creatinine ratio
  - lungworm screening if evidence of haemorrhage
  - secondary tests
    - n screening for hyperadrenocorticism
    - abdominal ultrasound
    - echocardiography
- tertiary tests
  - thyroid screening
- renin:aldosterone ratio
- urine/plasma metanephrines

## Consequences

Consequences of hypertension include:

- ocular
- myocardial
  - hypertrophy and, ultimately, failure
- renal
  - major cause of hypertension, but subsequent hypertension will also exacerbate the renal disease
  - results in hyperfiltration and glomerulosclerosis
- neurologic
  - ataxia, seizures, dementia, coma

## Management



**Figure 3.** Retinal haemorrhage in a hypertensive dog.

Generally, it is appropriate to treat hypertension if the mean systolic BP is greater than 160mmHg with clinical signs present – for example, renal disease, ocular haemorrhage or if the mean systolic greater than 180mmHg to 200mmHg in the absence of other supporting evidence.

It is important to remember a single measurement series giving a mean systolic BP more than 180mmHg does not necessarily make a patient hypertensive. In such cases, depending on the patient, temperament hospitalisation for the rest of the day and repeated measurement may show quite dramatic falls in BP. In some patients, however, arranging to measure their BP in their home environment may be a better solution and more accurately reflect their true BP.

Treatment of hypertension, to some extent, depends on severity and underlying cause. Where the cause is defined, drug choice or method of control is directed by the condition (**Table 5**). Where the cause of the hypertension is unknown or untreatable then any potentially hypertensive drugs should be stopped if possible – for example, phenylpropanolamine, millophyline and terbutaline – and the BP reassessed.

If the patient is not receiving hypertensive drugs or their withdrawal is not possible/has not resulted in a sufficient fall in BP then amlodipine 0.05mg/kg to 0.1mg/kg by mouth every 24 hours initially is the drug of choice. Amlodipine takes around five to six days to reach full efficacy, at which point BP should be rechecked. If still high, the dose rate increased to a maximum of 0.25mg/kg by mouth every 12 hours.

Amlodipine undergoes hepatic metabolism, hence dose rates should be reduced if there is significant hepatic dysfunction.

Side effects are rare, but include lethargy, inappetence and hypotension. Other reported side effects include peripheral oedema and gingival hyperplasia. Target systolic BP in clinic/at home is 120mmHg to 150mmHg; generally, the author tends to go towards the higher end of this range.



<b>Underlying disease process</b>	<b>Initial therapy</b>
Phaeochromocytoma	$\alpha$ and $\beta$ -adrenergic blockers
Hyperaldosteronism	Spironolactone inhibitors
Diabetes	Improving diabetic control
Proteinuria	Angiotensin-converting enzyme inhibitor – benazepril 0.5mg-1/kg every 12 to 24 hours usually results on a fall in BP of around 10 per cent.

**Table 5.** Treatment options for secondary hypertension.

As signs of hypertension are subtle or non-existent, some ongoing monitoring is required to assess the response to treatment and ensure the patient has not become hypotensive. Ideally, urea and creatinine should be estimated before and during treatment.

In those cases with pre-existing renal disease, depending on the severity and associated clinical signs, ACE inhibitors can be a preferred initial choice, but, often, the effect is modest and further intervention required. Once the BP is stable, it should be measured every two to three months, extending the interval gradually if stability is maintained.

### **What to do if BP is not falling**

The general consensus is hypertension in dogs appears to be more refractory and harder to treat than in cats. Prior to deciding on changes in therapy, it is important to consider the following questions:

- What is the state of end organ damage?
- Is the medication being reliably taken?
- Is there an underlying cause I can treat/requiring a different approach?

If the answer to these questions is no then:

- maximise the amlodipine dose
- use a second anti-hypertensive agent

No studies have been published on the next best choice. Drugs to consider would be beta-blockers (such as atenolol or propranolol), alpha-blockers (such as prazosin, hydralazine or phenoxybenzamine) and diuretics (such as spironolactone, furosemide or ACE-inhibitors).

Once a drug has been chosen, assuming it is having some effect, the dose should be increased to the maximum recommended or tolerated dose, whichever is lower. Some cases can require a

combination of three different classes of drugs to achieve effective control of BP.

## Hypertensive emergencies

Where evidence shows severe and progressive end-organ damage, a decision has to be made balancing the benefit of controlling BP versus consequence of hypotension. Severely hypertensive cases may require hospitalisation for treatment.

IV nitroprusside sodium is tricky to manage, difficult to obtain and expensive. Use requires constant BP monitoring. Sodium nitroprusside is given as a constant rate infusion, beginning at 0.5mcg/kg/min and titrated upwards every five minutes, with an aim to reduce BP by 25% over a four-hour period. Oral hydralazine is more straightforward to use, but has a slower onset of action, given orally at 1mg/kg to 2mg/kg by mouth every 8 hours, with a similar target to decrease BP by 25%.

## Conclusion

BP monitoring is simple, safe and cost effective, and can be used as part of preventive medicine programme. Failure to appreciate significant hypotension or hypertension can have disastrous and irreparable consequences.

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