

## Blood pressure – why bother? (A medic’s perspective)

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**Abstract:** Hypertension may result in significant organ dysfunction: blindness (retinal detachment, hyphaema)<sup>11,12</sup>; sudden onset neurological signs (vestibular, cerebellar / midbrain localization most common, owing to haemorrhagic strokes); heart murmur, gallop rhythm, arrhythmias and rarely congestive heart failure owing to left ventricular myocardial hypertrophy; or worsening renal disease. Rarely, it may result in epistaxis. Signs of hypotension are vague: prolonged CRT, weak pulses, cold extremities and lethargy, rarely inappetence and pu/pd. Yet prolonged hypotension can have significant and profound effect on organ function especially renal function (including acute renal failure). Unless you measure blood pressure, you will not pick up changes until severe end-organ damage has occurred.

Diseases where hypertension is prevalent <sup>5,13</sup> :	Diseases where hypotension is prevalent <sup>5</sup>
<ul style="list-style-type: none"> <li>- <b>Chronic renal failure (CRF)</b></li> <li>- Hyperthyroidism (5-22%)<sup>13</sup></li> <li>- <b>Hyperadrenocorticism (Cushing’s disease)</b></li> <li>- Diabetes mellitus</li> <li>- Acromegaly (in theory)</li> <li>- <b>Hyperaldosteronism (Conn’s disease)</b></li> <li>- <b>Phaeochromocytoma</b></li> <li>- Hyperviscosity (gammopathy, polycythemia)</li> <li>- Obesity in dogs<sup>1</sup></li> <li>- Anaemia</li> <li>- Fever</li> <li>- Pain</li> <li>- arteriovenous fistula</li> </ul> <p>Idiopathic / primary hypertension</p>	<ul style="list-style-type: none"> <li>- Congestive heart failure</li> <li>- Cardiac tamponade</li> <li>- Shock</li> <li>- Bradyarrhythmia</li> <li>- Hypovolaemia (eg Addison’s, diuresis, GIT disease)</li> <li>- Hypothyroidism (myxoedema)</li> <li>- Drugs (<math>\alpha</math>2 agonists, beta blockers, anything causing histamine release)</li> <li>- anaphylaxis</li> <li>- Sepsis</li> <li>- Haemorrhage</li> <li>- Pneumothorax</li> <li>- Thrombosis</li> <li>- Under GA: hypoxia, hypercapnoea</li> </ul>

Animals with the diseases in bold often initially present because of the effect of the associated hypertension on end organs. Note that obesity has a relatively small effect on BP (< 5 mmHg for systolic, diastolic and mean BP)<sup>1</sup>

### Physiology – what controls blood pressure?

Blood pressure is regulated so that delivery of oxygen and nutrients to all body tissues is maintained and waste products are effectively removed. Local autoregulation controls blood pressure in essential vascular beds (eg glomeruli, brain) through the range of normal blood pressures, constraining pressure in these vascular beds to a narrow range. Extremely high or low pressures (< 60 or > 160 mmHg MAP<sup>5</sup>) or local disease (eg pre-existing renal disease, brain trauma) override autoregulation and can cause / exacerbate organ dysfunction.

Blood pressure is affected by

- the heart (anything that affects cardiac output ie heart rate or force of contraction of the myocardium)
- blood volume (preload)
- blood vessel tone (afterload, elasticity of the wall)
- blood viscosity

Blood pressure is closely regulated:

- immediate control (< 1 minute)- by baroreceptor and chemoreceptors: Baroreceptors are found in the heart and the great vessels close to the heart and respond directly to a decrease in blood pressure by increasing sympathetic tone. Chemoreceptors are located in the aortic sinus and carotid arch and respond to decreases in oxygen and increases in CO<sub>2</sub> in the same way - increasing circulating catecholamines cause vasoconstriction, tachycardia, increased inotropy and splanchnic vasoconstriction, thus raising blood pressure and perfusion to vital organs.

- medium term regulation- by the renin-angiotensin-aldosterone system (RAAS - resulting in retention of sodium and water as well as vasoconstriction). The RAAS is counter-regulated by prostaglandins (PGI<sub>2</sub>) which cause vasodilation and natriuresis.
- long term control measures: in general, these are focused around adjusting blood volume and activating counter-regulatory hormones. ADH release is stimulated by angiotensin II and hyperosmolarity. ADH stimulates thirst, retention of sodium free water and causes further vasoconstriction. The natriuretic peptides (ANP and BNP) and adrenomedullin antagonize the effects of the RAAS.

### How can I determine blood pressure?

There are 2 main indirect means of determining blood pressure: oscillometric and Doppler techniques.

Oscillometric	Doppler
Measures systolic and diastolic pressure Machine detects oscillations of the arterial wall No clipping or gel required	Measures systolic pressure only Operator detects sound of blood flow with Doppler probe Need to clip coat to apply the probe
Cardell monitors sold by Kyron 011 6181544 Cost: +/- R21000 (exchange rate dependent)	Cat Doppler kit from Thames Medical (can be used on dogs) 0044 1903 522911 Cost: 800 pounds sterling shipped to SA

Cuff width: 40-60% of the circumference of the limb / tail. A wide cuff will underestimate and a narrow one overestimate the BP. Hold the leg so the cuff is level with the heart (approximately)<sup>13</sup>

Get the environment right: everyone calm, quiet part of the practice with no through traffic, minimize noise, allow patient to acclimatize, turn the Doppler sound down or wear ear phones for stressy patients, don't mess with the foot (most animals have tickly feet).<sup>13</sup>

You need at least 6 readings within 10 mmHg of each other to trust the reading. It is normal for blood pressure to vary by 10-15 mmHg from heart beat to heart beat<sup>5</sup>. If there is a downward trend in the reading, chat a little longer with the owner and continue measuring.

Alternatively, direct blood pressure measurements can be determined after placing an arterial or central venous catheter. These are more fiddly (and painful) to apply and are usually only used in severely ill or anaesthetized patients.

### What is normal?

Blood pressures vary between species, between breeds and between techniques – thus serial measurements in the same individual over time using the same instrument are most sensitive.

Cat	Systolic	Diastolic
Doppler <sup>5</sup>	118 mmHg	
Oscillometric <sup>5</sup>	110-140 mmHg	75-100 mmHg
Dog (Oscillometric)	Systolic	Diastolic
Irish Wolfhound <sup>2</sup>	116 mmHg	70 mmHg
Retrievers (Labrador, Golden) <sup>5</sup>	110-135 mmHg	60-80mmHg
GSD, Bullterrier, Toy Pom <sup>5</sup>	120-150 mmHg	65-90mmHg
Greyhound, Saluki, Pointer <sup>5</sup>	130-165mmHg	70-100mmHg
Deerhound <sup>1</sup>	140-165 mmHg	80-95 mmHg

### False elevations

- stress<sup>5</sup>: anything causing fear or aggression will release catecholamines and elevated blood pressure eg door slamming, something dropping to the floor, loud voices, barking dog, telephone ringing, rough handling, stressed owner
- white coat effect<sup>5</sup>: cat's BP usually normalized within 10 minutes of reaching the veterinary practice. Taking rectal temperature or examining the mouth increased BP by about 30 mmHg

### Cardiac changes consistent with hypertension:

*Clinical signs:* gallop rhythm, arrhythmia, mitral /aortic systolic murmur that may increase in volume at higher heart rates.

*What happens:* Increased afterload results in concentric left ventricular hypertrophy. Muscle fibres and connective tissue hypertrophy. Hypertrophied muscle is more sensitive to catecholamines and to ischaemic injury, both predisposing to arrhythmias (13% in a study of 30 hypertensive cats)<sup>6</sup>. Myocyte hypoxia stimulates further fibrosis so the proportion of myocyte hypertrophy and myocardial fibrosis varies between patients. Fibrosis manifests as a decrease in diastolic compliance and may result in a gallop rhythm (27% of hypertensive cats)<sup>6</sup>. The diastolic dysfunction contributes to left atrial enlargement which may in turn lead to mitral insufficiency (40-54%)<sup>6,12</sup>. It is unusual for the cardiac changes to progress to congestive heart failure<sup>5,12</sup>

*ECG:* P mitrale, tall R waves, left anterior fascicular block<sup>5</sup>

*Echocardiographic changes:*<sup>3</sup>: NB generally mild

- mild left atrial enlargement
- mitral insufficiency on colour flow and continuous wave Doppler
- **mild, usually symmetrical** thickening of left ventricular (LV) free wall and septum
- normal fractional shortening
- decreased relaxation on mitral inflow patterns
- **LV internal diameter in diastole is normal** (decreased in cats with hypertrophic cardiomyopathy - HCM)
- Remember to r/o (sub)aortic stenosis – especially in dogs

**Ocular changes:** see lecture

**When do I treat?**

HYPERTENSION	Low risk	Moderate risk	High risk
cat	150-160 mmHg systolic	160-180 mmHg systolic	> 180 mmHg systolic
dog	150-160 mmHg systolic	160-180 mmHg systolic	> 180 mmHg systolic
Breed specific reference range	10-20 mmHg above	20-40 mmHg above	> 40 mmHg above

Treat if you have<sup>5</sup>

- convincing signs of end-organ disease – even if BP is only mildly elevated
- if BP is > 180 mmHg and you've excluded stress
- if there is a persistent upward trend in BP readings despite treating the underlying disease
- if BP is persistently in the low risk range (on multiple readings) and treating the underlying disease does not normalize the BP within 4 weeks
- if BP is persistently in the moderate risk range (on multiple readings over several days) and treatment of the primary disease does not improve readings within 10-14 days

HYPOTENSION	Mild	moderate	severe
Dog and cat, awake	< 100/60 mmHg	< 90/50 mmHg	< 70/50 mmHg
Dog and cat, anaesthetised	< 90/60 mmHg	< 80/50 mmHg	< 60/40 mmHg

**How do I treat hypertension?**

- Reduce obesity and increase exercise (expected decrease is around 5 mmHg for each)<sup>1</sup>
- Treat primary disease. (In a small case series BP of dogs with pituitary dependent Cushing's rarely returned to normal after stabilization with mitotane<sup>7</sup>)
- Avoid medication that could cause hypertension
- ACE – inhibitors: these usually decrease BP by 5-15 mmHg<sup>3,8</sup>, but may be specifically indicated by the primary disease (proteinuric renal disease)<sup>3</sup>
- Calcium channel blockers (amlodipine): cause more profound decreases in BP (30-60mmHg in cats<sup>10</sup>)
- Other:
  - beta blockers (propranolol – mixed, atenolol b1 selective): these are first line treatment for hypertensive hyperthyroid cats (though a 2<sup>nd</sup> antihypertensive may be needed)<sup>9</sup>
  - mixed alpha blockers (phenoxybenzamine) or alpha 1 receptor blockers (prazosin): these are first line treatments for phaeochromocytoma

- diuretics

*Treatment of ocular manifestations of hypertension:*

- Retinal detachment: Amlodipine at 0.625-1.25 mg/cat once daily. In one case series, 4/30 eyes with detached retinas regained vision after treatment<sup>12</sup>. This was transient in 2 eyes.
- Hyphaema: add topical corticosteroids (eg 0.1% dexamethasone 3-4x daily) and 1% atropine. Monitor intra-ocular pressure for glaucoma and discuss treatment with veterinary ophthalmologist if it develops.

IF you've managed to resolve the cause of the hypertension (eg removed the thyroids) wean off medication over 4 weeks, while monitoring blood pressure. Sudden withdrawal may cause rebound hypertension, especially if you were using beta / alpha blockers<sup>5</sup>

Echocardiographic changes that are consequence of hypertension may resolve if blood pressure is controlled<sup>5,14</sup>, but this does not happen in all cases.

**When do I treat hypotension?**

- If asymptomatic, treat primary disease
- If clinical signs of hypotension / shock (CRT > 2 sec, weak pulses, cold extremities, lethargy/collapse, syncope, pale mucosae<sup>5</sup>) treat primary disease and do something about the hypotension.

**How do I treat hypotension?**

- treat underlying disease (eg rapid acting corticosteroid and adrenalin for anaphylaxis, broad spectrum iv antibiotics and get rid of septic focus for sepsis)
- volume replacement
  - crystalloids at 80 ml/kg/hr for dogs and 55 ml/kg/hr for cats
  - colloids (10-20 ml/kg/day; test dose of 5 ml/kg as a bolus; reduce daily crystalloid volume by 1/3)
  - rate and amount depend on the underlying cause for hypotension ie
    - If CHF, decide: do you need fluid into or out of the patient. If there is pulmonary oedema, DO NOT GIVE MORE FLUID. Raise blood pressure by other means.
    - If sepsis or anaphylaxis, remember vascular permeability is increased so pulmonary oedema is a possible complication
- cause vasoconstriction<sup>4</sup>: dopamine CRI 2-10 µg/kg/min
- use inotrope: dobutamine CRI 2-20 µg/kg/min, pimobendan
- do NOT warm patient till it is rehydrated – warming will cause peripheral vasodilation and can drop BP further
- consider supplemental oxygen

	dose	receptor	effect	SE
Dopamine	< 3 µg/kg/min	D <sub>1</sub>	Dilation of renal and mesenteric bv, thus increased GFR	- Pulmonary hypertension
	3-5 µg/kg/min	β <sub>1</sub>	Positive inotrope, mildly increased HR	- Tissue necrosis if gets outside vein
	5-10 µg/kg/min	α <sub>1</sub>	vasoconstriction	
Dobutamine	5-10 µg/kg/min	β <sub>1</sub> , slight β <sub>2</sub>	Positive inotrope, mildly increased HR Decreased pulmonary pressures	Cat dose 2.5-5 µg/kg/min
Adrenalin	0.5 µg/kg/min	β <sub>1</sub> , β <sub>2</sub>	Positive inotrope, peripheral vasodilation, increased HR, relieves bronchospasm, decreases histamine release	- increased myocardial oxygen demand
	> 1 µg/kg/min	α <sub>1</sub>	Peripheral vasoconstriction, increased blood pressure	Can decrease renal perfusion

**What evidence is there that controlling blood pressure improves survival?**

- In a study of 60 dogs with a variety of diseases, severe hypertension (> 180 mmHg systolic) was most common amongst the group with CRF. Dogs with hypertension and CRF had shorter survival times<sup>15</sup>.

- In a study of 141 hypertensive cats with CRF (52 with concurrent hyperthyroidism), degree of blood pressure control did not have a direct effect on survival. Good control decreased the urine protein:creatinine ratio. A urine protein:creatinine ratio < 0.2 was associated with longer survival times<sup>10</sup>

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