VE Vet eBooks
VET-EBOOKS.COM

FREE · NO SIGN-UP

BASIC SCIENCES · CARDIOVASCULAR PHYSIOLOGY · STUDY GUIDE

BLOOD PRESSURE CONTROL

How Blood Pressure Is Controlled in Animals: The Baroreflex, RAAS and ADH Explained (PDF)

Blood pressure regulation in animals keeps tissue perfusion constant against a moving target — the animal stands, bleeds, exercises, sleeps. Inside: Nine labelled figures, one reference table and a 15-question self-test.

Contents

The 12 sections of this guide, in order.

IN SHORT

Blood pressure regulation in animals keeps **tissue perfusion** constant against a moving target — the animal stands, bleeds, exercises, sleeps. The circulation defends **mean arterial pressure (MAP)**, defined by **MAP = CO × TPR**, on three time-scales that all layer together. In *seconds*, the **baroreceptor reflex** from the **carotid sinus** and **aortic arch** pulls heart rate and vessel tone up or down through the medulla. In *minutes to hours*, the **renin–angiotensin–aldosterone system (RAAS)** and **ADH (vasopressin)** add water, salt and vasoconstriction. Over *hours to days*, the kidney enforces the long-term set-point through **pressure natriuresis**. When these systems fail chronically — a CKD cat, a hyperthyroid cat, a Cushing's dog — you get **hypertension in dogs and cats**, and the drug logic (ACE inhibitor, telmisartan, amlodipine) falls out of the physiology.

KEY POINTS

1. **Blood pressure regulation in animals** is the coordinated defence of **mean arterial pressure (MAP)** so every capillary bed can perfuse its tissue. The clinical target is not blood pressure itself but the tissue behind it — brain, kidney, retina, gut — which fails at low perfusion and is damaged over years by high pressure.
2. The whole system is built around one equation: **MAP = CO × TPR**. Cardiac output (CO) comes from the heart (see the cardiac cycle) and total peripheral resistance (TPR) from arteriolar tone. Move either and MAP moves with it, unless a compensating reflex intervenes.
3. The circulation has three time-scales of control that layer together, not compete. **Seconds**: the **baroreceptor reflex** corrects rapid changes. **Minutes to hours**: the **renin–angiotensin–aldosterone system (RAAS)** and **ADH (vasopressin)** add vasoconstriction, salt and water. **Hours to days**: the kidney enforces the long-term set-point through **pressure natriuresis**. Cunningham puts this framework in Ch25 Table 25.1 (p273).
4. The **baroreflex** is the fastest and most familiar: stretch receptors in the **carotid sinus** and **aortic arch** send afferents in CN IX (glossopharyngeal) and CN X (vagus) to the medullary **nucleus tractus solitarii (NTS)**, which reciprocally adjusts sympathetic and parasympathetic outflow to heart and vessels. Rise in BP → more firing → parasympathetic dominance → HR down, TPR down. Fall in BP → less firing → sympathetic dominance → HR up, contractility up, venoconstriction up, TPR up.
5. **RAAS** is the minutes-to-hours axis. Renal juxtaglomerular (JG) cells sense ↓ renal perfusion, ↓ NaCl at the macula densa or sympathetic drive, and release **renin**. Renin cleaves liver-made **angiotensinogen** to **angiotensin I**; pulmonary **angiotensin-converting enzyme (ACE)** converts it to **angiotensin II**, which does four things at once: potent vasoconstriction (40× more potent than noradrenaline), stimulation of **aldosterone** release from the adrenal cortex, stimulation of thirst, and release of ADH.
6. **ADH** (vasopressin) is the water lever. It is released from the posterior pituitary in response to hypothalamic osmoreceptors and atrial low-pressure volume receptors. At the kidney it acts on **V2 receptors** to insert aquaporin-2 water channels in the collecting duct (water reabsorption); at high dose it acts on vascular **V1 receptors** to vasoconstrict. In practice ADH is the reason a dehydrated animal makes concentrated urine.

7. Over days, the kidney sets the long-term BP mean through **pressure natriuresis** (Guyton's principle): higher arterial pressure at the renal artery drives more sodium and water excretion, which lowers blood volume, which lowers BP. This is why chronic kidney disease and salt handling are so central to **hypertension in dogs and cats**.
8. In veterinary practice, **idiopathic hypertension** is rare (unlike in humans). Almost every hypertensive animal has a secondary cause: in cats it is usually **chronic kidney disease (CKD)** or **hyperthyroidism**; in dogs, CKD, **hyperadrenocorticism (Cushing's)**, diabetes, phaeochromocytoma, or an adrenal tumour (tumor). The ACVIM 2018 consensus defines feline systolic > 160 mmHg as borderline and > 180 as hypertensive, with treatment usually starting at persistent > 160 with end-organ damage.
9. Drug logic falls straight out of the physiology. **ACE inhibitors** (enalapril, benazepril) block angiotensin II synthesis. **Angiotensin receptor blockers (ARBs)** such as telmisartan block AT1 receptors and are especially useful in feline proteinuric CKD. **Calcium-channel blockers** (amlodipine) are potent arteriolar vasodilators and the drug of choice for cats with hypertension. **β-blockers** (atenolol) treat hyperthyroid cats with tachycardia. Never start α-blockers or diuretics as first-line without addressing the underlying cause.

01 Why blood pressure control matters in animals

Every clinical animal you meet is quietly defending one number. Not its heart rate, not its haematocrit, not its blood glucose — but its **mean arterial pressure**, the pressure that pushes blood through every capillary bed in every organ. The frame is **blood pressure regulation in animals**: the coordinated defence of tissue perfusion in the face of everything that would disturb it — the animal standing up from lateral recumbency, the dog haemorrhaging after a road traffic accident, the racehorse in the last furlong, the cat asleep on the radiator. The circulation is not obsessed with pressure for its own sake. It is obsessed with getting blood to the brain, kidney, retina and gut, and pressure is only the tool it uses to do that.

That is why every organ in the body has a set of receptors, hormones and reflexes wired into its wall to defend perfusion. The **baroreceptor reflex** from the carotid sinus and aortic arch corrects rapid changes in seconds. The **renin–angiotensin–aldosterone system (RAAS)** and **ADH (vasopressin)** refine the response over minutes to hours. The kidney, through **pressure natriuresis**, enforces the long-term set-point over days. Miss any one of these and you cannot explain what happens when a Cavalier decompensates in left heart failure, when a Cushing's dog develops hypertension, when a CKD cat goes suddenly blind, or when a phaeochromocytoma presents with intermittent collapse. This article walks the whole system from the ground up: the equation MAP = CO × TPR, the three time-scales, the baroreflex, the chemoreceptor and CNS ischaemic responses, RAAS, ADH, pressure natriuresis, a worked haemorrhage case, hypertension in dogs and cats, and the drug logic that falls out of the physiology. It links closely to our companion pieces on the cardiac cycle (where cardiac output is generated) and on oedema and the Starling forces (where a failure of BP control drives raised capillary pressure and oedema).

02 What blood pressure actually is: MAP = CO × TPR

Arterial blood pressure is the pressure exerted by the flowing blood on the walls of the systemic arteries. It rises and falls with each heartbeat: it peaks at *systolic* pressure (SBP) during ventricular ejection and falls to a low of *diastolic* pressure (DBP) during ventricular relaxation. In a resting healthy dog you would expect systolic ~120 mmHg and diastolic ~80 mmHg, though there is a wide healthy range (roughly 100–160 systolic). Cats sit slightly higher on average (~120–160 mmHg systolic), horses slightly lower (~90–140 mmHg systolic at rest), cows (~100–180 mmHg). The precise numbers matter but the framework matters more.

What actually drives tissue perfusion is not systolic or diastolic in isolation but the **mean arterial pressure (MAP)** — the average arterial pressure over one complete cardiac cycle. At physiological heart rates MAP is closer to diastolic than systolic (because diastole is longer than systole), and the working rule of thumb is **MAP ≈ DBP + ⅓(SBP − DBP)**. So in the resting dog above, MAP ≈ 80 + ⅓(40) = ~93 mmHg. MAP is the number that keeps the brain, kidney, retina and gut perfused; it is the number the reflexes and hormones defend.

Every mechanism that raises or lowers arterial pressure does so by moving one of only two things. This is the central equation of the whole article and worth memorising:

> **MAP = CO × TPR**
>
> . Mean arterial pressure equals cardiac output (CO, the pump: HR × SV) multiplied by total peripheral resistance (TPR, the pipe: arteriolar tone).

Move **CO** and MAP moves with it. Move **TPR** and MAP moves with it. Move both in the same direction and MAP moves further; in opposite directions and MAP can be defended even as both individual factors change. Cunningham (Ch25 p273) opens the chapter with exactly this framing, because every reflex, hormone and drug you will meet in this article is doing one of three things: touching CO (heart rate, contractility, stroke volume), touching TPR (arteriolar vasoconstriction or vasodilation), or both. A β-blocker lowers CO. Amlodipine lowers

TPR. An ACE inhibitor lowers TPR and, indirectly, blood volume. Furosemide lowers preload and thus SV. Angiotensin II raises TPR and, over hours via aldosterone, blood volume and SV. Once you have the equation in your head, the drug logic is inevitable.

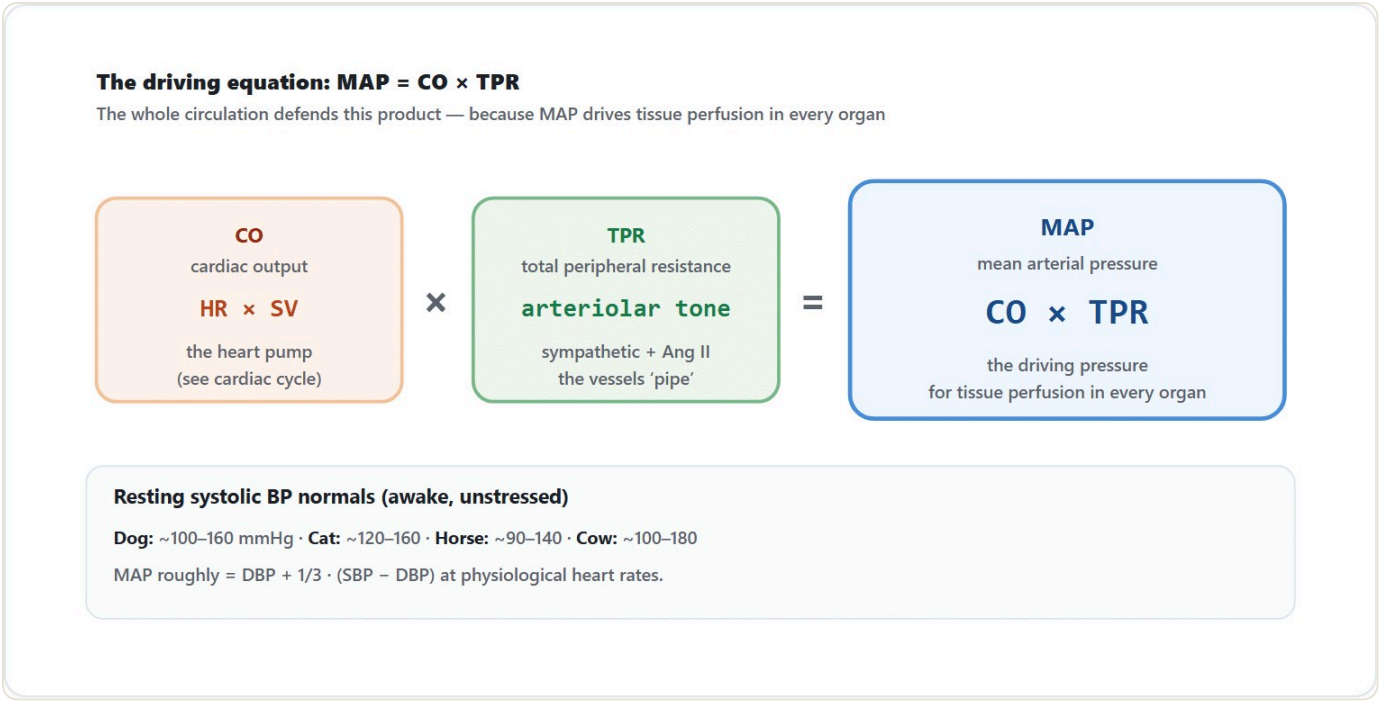


Fig 1 — The driving equation. MAP is defended by two dials: the heart pump (CO = HR x SV) and the arteriolar tone (TPR). Species normals for resting systolic BP: dog ~100–160 mmHg, cat ~120–160, horse ~90–140. (Cunningham 6e Ch25 p273.)

03 The three time-scales of blood pressure regulation

Before we walk through each mechanism in turn it helps to see the whole map. The circulation defends MAP on three time-scales, and every mechanism you will meet in the rest of the article slots into one of them (Cunningham Ch25 Table 25.1, p273). They do not compete; they layer.

Time-scale 1 — seconds. Stretch-sensitive baroreceptors in the **carotid sinus** and **aortic arch** fire faster when arterial pressure rises and slower when it falls. Their afferents feed the medullary NTS, which sends reciprocal signals up sympathetic and parasympathetic outflows. Rise in BP → sympathetic down + parasympathetic up → HR down, contractility down, TPR down. Fall in BP → sympathetic up + parasympathetic down → HR up, contractility up, venoconstriction, TPR up. Peripheral **chemoreceptors** in the carotid and aortic bodies (O_2/CO_2/pH sensors) contribute in emergencies, and the **CNS ischaemic response** is a last-ditch reflex at MAP under about 50 mmHg.

Time-scale 2 — minutes to hours. Two circulating systems now do their work. The **renin-angiotensin-aldosterone system (RAAS)** raises TPR (via angiotensin II vasoconstriction) and, over hours, blood volume (via aldosterone-driven sodium and water retention). **ADH (vasopressin)**, released from the posterior pituitary, drives water reabsorption at the kidney (V2 receptors, aquaporin-2) and, at high dose, adds vascular tone (V1). Both raise MAP but through different levers (RAAS → TPR + volume; ADH → volume + a little TPR at high dose).

Time-scale 3 — hours to days. The kidney owns the long-term BP set-point via **pressure natriuresis** (Guyton). Any sustained rise in arterial pressure at the renal artery drives increased sodium and water excretion; the resulting fall in blood volume drops CO and MAP back toward the set-point. Because pressure natriuresis operates over hours to days and does not habituate, no other mechanism can override it for long — the kidney gets the final vote. This is why the diseases that damage the renal set-point (CKD, hyperaldosteronism) drive chronic hypertension.

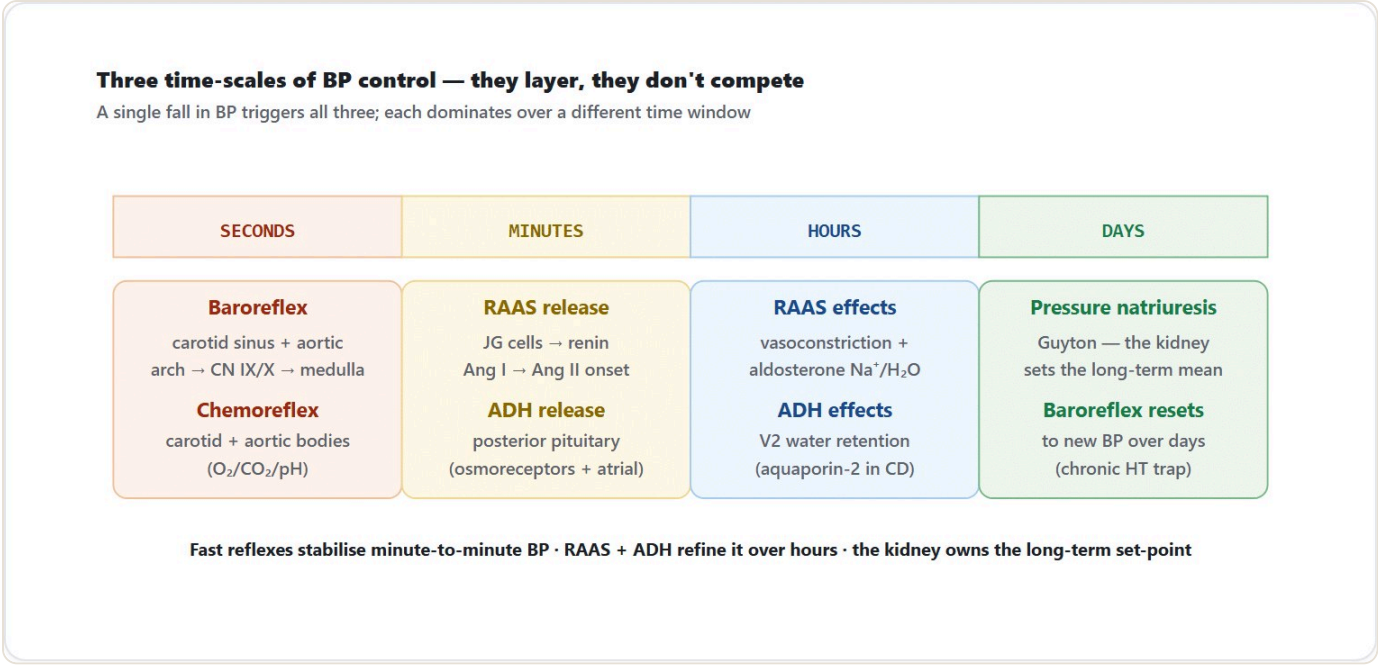


Fig 2 — The three time-scales of blood pressure regulation in animals. Seconds: baroreflex + chemoreflex. Minutes-hours: RAAS + ADH. Days: pressure natriuresis (and baroreflex resetting). All three layer together after any BP disturbance. (After Cunningham 6e Ch25 Table 25.1.)

The three time-scales, side by side. They do not compete — they layer, each taking over as the one before it runs out of authority.

TIME-SCALE	MECHANISM	SENSOR	WHAT IT CHANGES
Seconds	Baroreceptor reflex	Stretch receptors in the carotid sinus and aortic arch	Heart rate, contractility, venoconstriction and TPR, through reciprocal sympathetic and parasympathetic outflow
Seconds (emergency)	Chemoreceptor and CNS ischaemic responses	Peripheral chemoreceptors; the medulla itself when its own perfusion fails	A powerful sympathetic surge — reserved for extremes, not everyday control
Minutes to hours	RAAS	Juxtaglomerular cells — perfusion pressure, macula densa NaCl, and β_1 sympathetic drive	Vasoconstriction through angiotensin II; sodium and water retention through aldosterone
Minutes to hours	ADH (vasopressin)	Hypothalamic osmoreceptors, sensitive to a few mOsm/kg around a set-point near 280 mOsm/kg	Water reabsorption, and vasoconstriction at higher concentrations
Days to years	Pressure natriuresis (Guyton)	The kidney itself, responding to arterial pressure	Sodium and water excretion, which sets the long-term pressure set-point

04 The baroreceptor reflex — the seconds-scale controller

The **baroreceptor reflex** (baroreflex) is the fastest and most familiar mechanism in blood pressure regulation in animals. It is the reason a dog does not faint every time it stands up, and the reason a cat can go from asleep on the sofa to sprinting after a mouse without collapsing halfway. Its job is to hold MAP steady over seconds, and it does so by adjusting heart rate, cardiac contractility and vascular tone through the autonomic nervous system.

The receptors. The baroreceptors are stretch-sensitive nerve endings in the walls of two arterial sites: the **carotid sinus**, a slight dilation of the internal carotid artery just past its origin from the common carotid, and the **aortic arch**. Both sites are richly innervated with endings that fire more rapidly as the arterial wall stretches with rising pressure and less rapidly as it recoils with falling pressure. The carotid sinus baroreceptors dominate the reflex output in most species, but both contribute.

The afferents. Signals from the carotid sinus travel to the brainstem in **CN IX (glossopharyngeal)**. Signals from the aortic arch travel in the **aortic depressor nerve**, a branch of **CN X (vagus)**. Both converge on the **nucleus tractus solitarii (NTS)** in the medulla — the integration centre for cardiovascular reflex afferents. The NTS in turn projects to two output nuclei: the **rostral ventrolateral medulla (RVLM)**, from which

sympathetic outflow is set, and the **nucleus ambiguus** and **dorsal motor nucleus of the vagus**, from which parasympathetic outflow is set. NTS activation excites the parasympathetic output nuclei and inhibits the RVLM — the reflex is reciprocal by design.

The effectors. Sympathetic outflow leaves the spinal cord at thoracic and upper lumbar segments and reaches the heart (through the cardiac sympathetic nerves) and every systemic arteriole and vein. It raises heart rate, myocardial contractility, venoconstriction (increasing preload) and arteriolar tone (increasing TPR). Parasympathetic outflow from the vagus reaches the SA node and slows the heart directly (through muscarinic M2 receptors and increased I-K,ACh); it has little effect on vessels themselves. So on a rise in BP the baroreflex withdraws sympathetic drive (HR↓, contractility↓, TPR↓) and adds parasympathetic drive (HR↓ further); on a fall in BP it does the reverse.

A worked example. A young greyhound stands up quickly from lateral recumbency. Gravity pools blood in the abdomen and hindlimbs, venous return falls, stroke volume drops (Frank-Starling), and MAP would drop by 20–30 mmHg if left uncorrected. Baroreceptor firing falls, the NTS senses it, the RVLM fires the sympathetic outflow harder, and within a beat or two heart rate has risen from ~90 to ~130 bpm, contractility has risen, veins have constricted (raising venous return again) and TPR has risen by ~15%. MAP is defended within about two seconds and the greyhound walks out of the ward unaware of the physiology it just performed.

Callout — the baroreflex resets over days. Chronic hypertension does not scream forever. Sustained BP elevation resets the baroreflex threshold upward: the receptors continue to fire, but their 'normal' is now the higher pressure. This is why a chronically hypertensive cat with CKD does not have persistent bradycardia despite systolic pressures of 200 mmHg — the alarm has moved. It is also why we screen BP proactively in older cats: they will not tell you they are hypertensive; you have to measure.

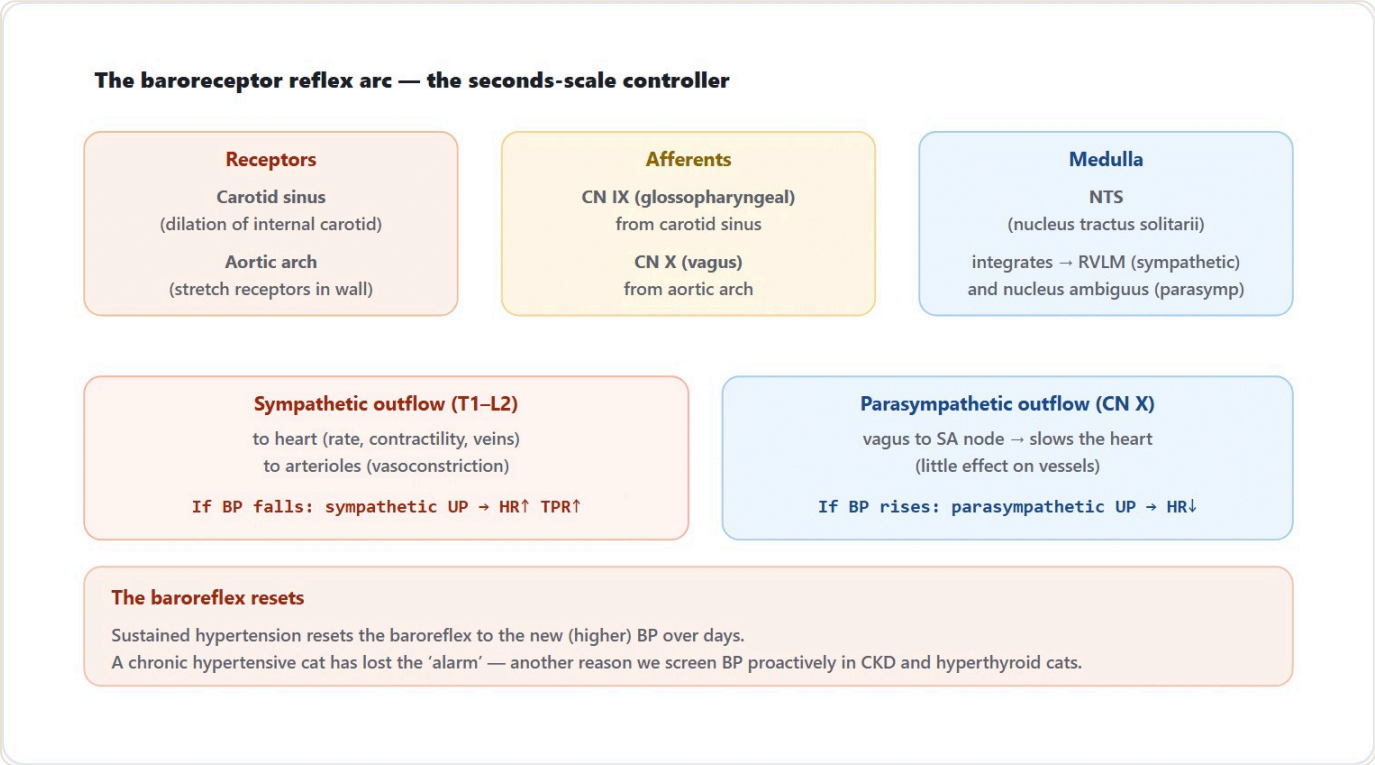
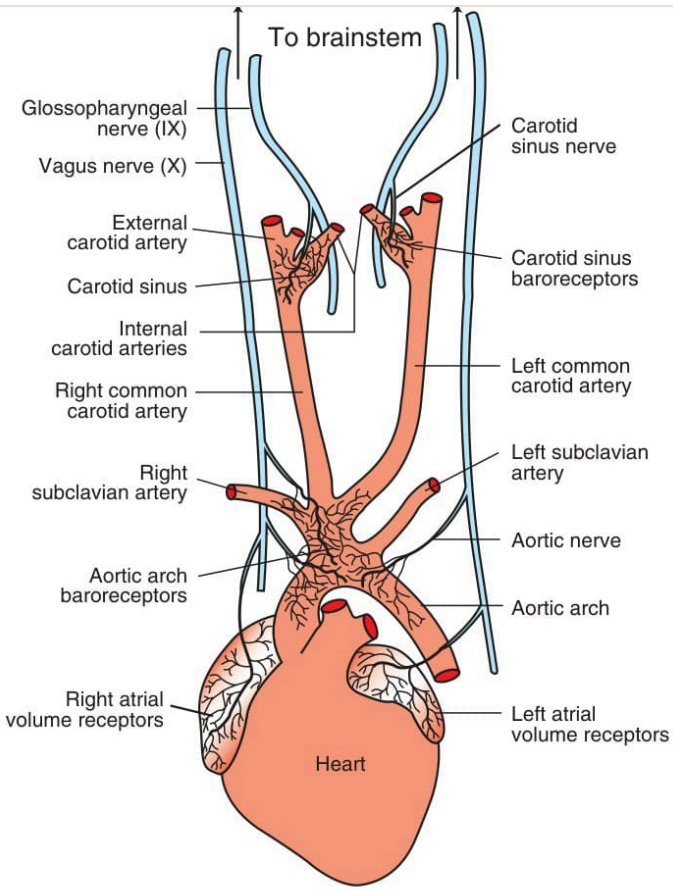


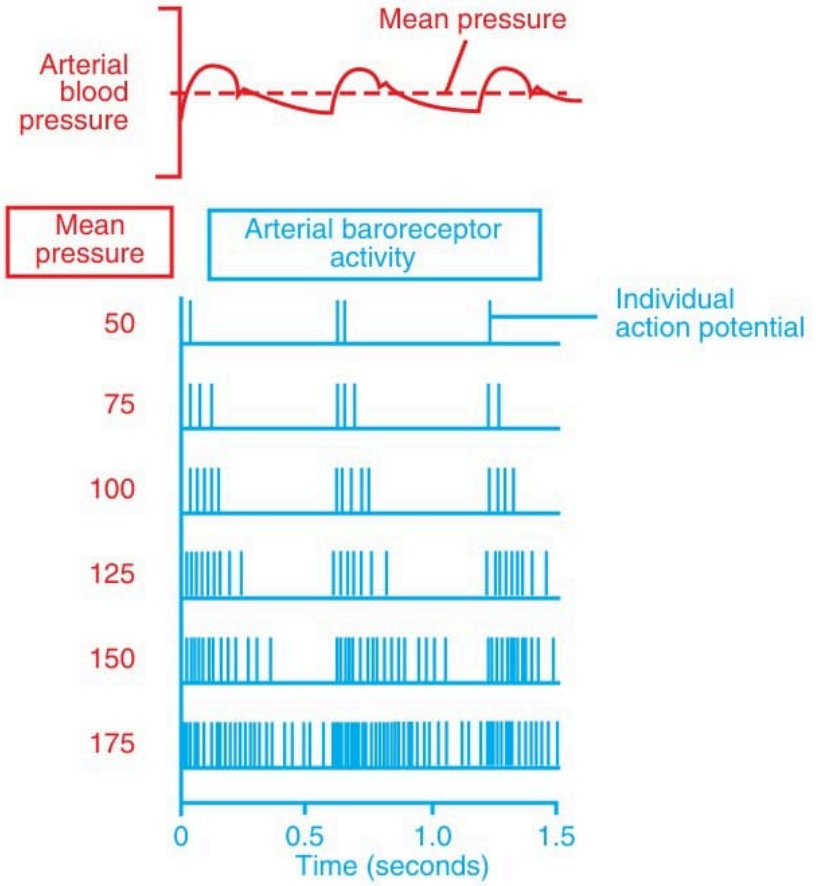
Fig 3 — The baroreceptor reflex arc. Receptors in the carotid sinus + aortic arch → afferents in CN IX + CN X → NTS in the medulla → sympathetic (T1–L2) and parasympathetic (CN X) outflow to heart and vessels. Resets over days at chronic higher pressure. (Cunningham 6e Ch25 p275.)



• **Fig. 25.1** Arterial baroreceptors are located in the walls of the carotid sinuses and in the walls of the aortic arch and its major branches. The atrial volume receptors are located in the walls of the right and left atria. See text for a description of the neural pathways followed by the baroreceptor and volume receptor afferents.

Fig 3a — The anatomical picture. Baroreceptors sit in the walls of the **carotid sinuses** (dilated origins of the internal carotid arteries) and around the **aortic arch** and its major branches; the atrial walls carry the low-pressure **volume receptors**. The afferents feed the medulla via CN IX (from carotid sinus) and CN X (from aortic arch and atria).

Source: Cunningham 6e Ch25 Fig 25.1 p265.



• **Fig. 25.2** Each arterial pressure pulse (*top trace, in red*) causes action potentials to be generated in baroreceptor afferent neurons (*lower traces, in blue*). The number of action potentials generated per heartbeat increases dramatically with increases in mean arterial pressure.

Fig 3b — The quantitative reader. Recordings from a baroreceptor afferent neuron at rising mean pressures show that firing frequency (blue traces) scales steeply with MAP (red top trace). This is the 'dial' that the medulla reads to know how firmly to push sympathetic vs parasympathetic outflow.

Source: Cunningham 6e Ch25 Fig 25.2 p265.

05 The chemoreceptor and CNS ischaemic responses

Two more brainstem-level mechanisms contribute to blood pressure regulation, both of them best thought of as *emergency reflexes* for extreme situations. In routine physiology they contribute little; when the animal is in trouble they are decisive.

The peripheral chemoreceptor reflex. Small clusters of glomus cells in the **carotid bodies** (near the carotid sinus, though anatomically distinct) and **aortic bodies** sense arterial blood chemistry. They fire faster when arterial oxygen tension drops, when carbon dioxide rises, or when pH falls. Their afferents travel with the baroreceptor afferents (CN IX and CN X) but project to a distinct set of medullary output neurones. The primary output is a strong sympathetic surge to peripheral vessels — sending blood centrally to defend brain and heart perfusion — along with respiratory drive changes (increased ventilation). In practice the peripheral chemoreflex contributes little to BP regulation in a healthy animal at rest, but is dramatic in *hypoxaemia* (upper-airway obstruction, severe pneumonia, severe anaemia at altitude) and in severe metabolic acidosis (DKA, severe uraemia).

The CNS ischaemic response. When the medulla itself becomes hypoperfused — typically at a MAP below about **50 mmHg** — a last-ditch, extremely powerful sympathetic surge is triggered directly by the ischaemic brainstem. This is the physiological response you see in a dog in profound haemorrhagic (hemorrhagic) or septic shock as MAP crashes: a huge sympathetic release drives HR to its ceiling, vasoconstricts every non-critical bed (skin, gut, muscle) and tries to shunt what blood is left to the brain and heart. It is a survival reflex, not a fine-tuning mechanism. Anaesthetists respect it: any drug that abolishes it (deep general anaesthetic, spinal blocks) removes the animal's last defence

against catastrophic hypoperfusion. Cunningham (Ch25 p276) covers it briefly but explicitly; PDA (Ch11) reinforces that it is the most powerful sympathetic response the animal can mount.

06 The renin-angiotensin-aldosterone system (RAAS) — the minutes-to-hours axis

Where the baroreflex is fast and local, the **renin–angiotensin–aldosterone system (RAAS)** is the second layer — a circulating hormonal cascade that operates over minutes to hours and raises MAP by two levers at once: peripheral vasoconstriction (TPR) and sodium/water retention (blood volume, and hence CO). The system is drug-targeted more than any other endocrine axis in veterinary medicine because five of the commonest antihypertensives in the pharmacopoeia work on one of its steps.

The trigger — renin release. RAAS begins in the kidney. Modified smooth-muscle cells in the wall of the afferent arteriole, called **juxtaglomerular (JG) cells**, sense three things simultaneously. First, *renal perfusion pressure*: JG cells are themselves stretch receptors, and when perfusion falls, they release more renin. Second, *NaCl load at the macula densa*: the macula densa is a small cluster of specialised distal tubular cells that sit against the JG apparatus and sample the tubular fluid; when NaCl is low (as it is when GFR has fallen), the macula densa signals the JG cells (via adenosine and prostaglandin messengers) to release renin. Third, *sympathetic drive*: β_1 receptors on the JG cells respond directly to sympathetic activation, so any fall in BP that triggers the baroreflex sympathetic surge also releases renin. All three inputs converge on the same output: **renin**, an enzyme released into the circulation.

The cascade — renin to angiotensin II. In the plasma renin encounters **angiotensinogen**, a large peptide made continuously by the liver. Renin cleaves ten amino acids off the front of angiotensinogen to make **angiotensin I**, an inactive precursor. As angiotensin I flows through any capillary bed containing **angiotensin-converting enzyme (ACE)** — and ACE is present on the surface of vascular endothelium everywhere, but most densely on **pulmonary endothelium** — ACE cleaves two more amino acids off angiotensin I to make **angiotensin II**, the active vasoconstrictor peptide.

The four effects of angiotensin II. Angiotensin II acts at **AT_1 receptors** distributed across many tissues, and it does four important things — every one of them raising MAP:

1. **Direct vasoconstriction of arterioles → TPR up.** Angiotensin II is a spectacularly potent vasoconstrictor; Cunningham (Ch25 p278) gives the memorable comparison that on a per-mole basis it is about **40× more potent than noradrenaline**. It acts on smooth muscle throughout the systemic arteriolar bed and is especially strong at the efferent arteriole of the renal glomerulus (which is why ACE inhibitors and ARBs reduce glomerular capillary pressure and slow proteinuric CKD).
2. **Aldosterone release → Na^+/H_2O retention → blood volume up.** Angiotensin II acts on the **zona glomerulosa** of the adrenal cortex to release **aldosterone**, a mineralocorticoid. Aldosterone travels to the distal tubule and cortical collecting duct, where it activates principal cells to insert more Na^+/K^+-ATPase and ENaC channels — increasing sodium and water reabsorption from the urine, and secreting potassium in exchange. Over hours this raises extracellular fluid volume, plasma volume and thus CO.
3. **Stimulation of thirst.** Angiotensin II acts on subfornical organ neurones in the hypothalamus to trigger thirst behaviour — more water drunk → more volume replaced.
4. **Release of ADH from the posterior pituitary.** Angiotensin II is a mild ADH secretagogue itself, layering onto the osmoreceptor and volume-receptor inputs that also drive ADH release. This provides yet another route to water retention.

The clinical relevance of every step of this cascade is enormous. **ACE inhibitors** (enalapril, benazepril) block the conversion of angiotensin I to angiotensin II — taking all four effects off the table. **Angiotensin receptor blockers (ARBs)** such as **telmisartan** block the AT_1 receptor itself — especially useful in feline proteinuric CKD because they cut angiotensin II's effect at the efferent arteriole. **Aldosterone antagonists** (spironolactone) block the mineralocorticoid receptor and are used in advanced heart failure. And any disease that overactivates the RAAS — renovascular disease, primary hyperaldosteronism (Conn's syndrome; rare in cats), a phaeochromocytoma driving sympathetic tone — drives hypertension.

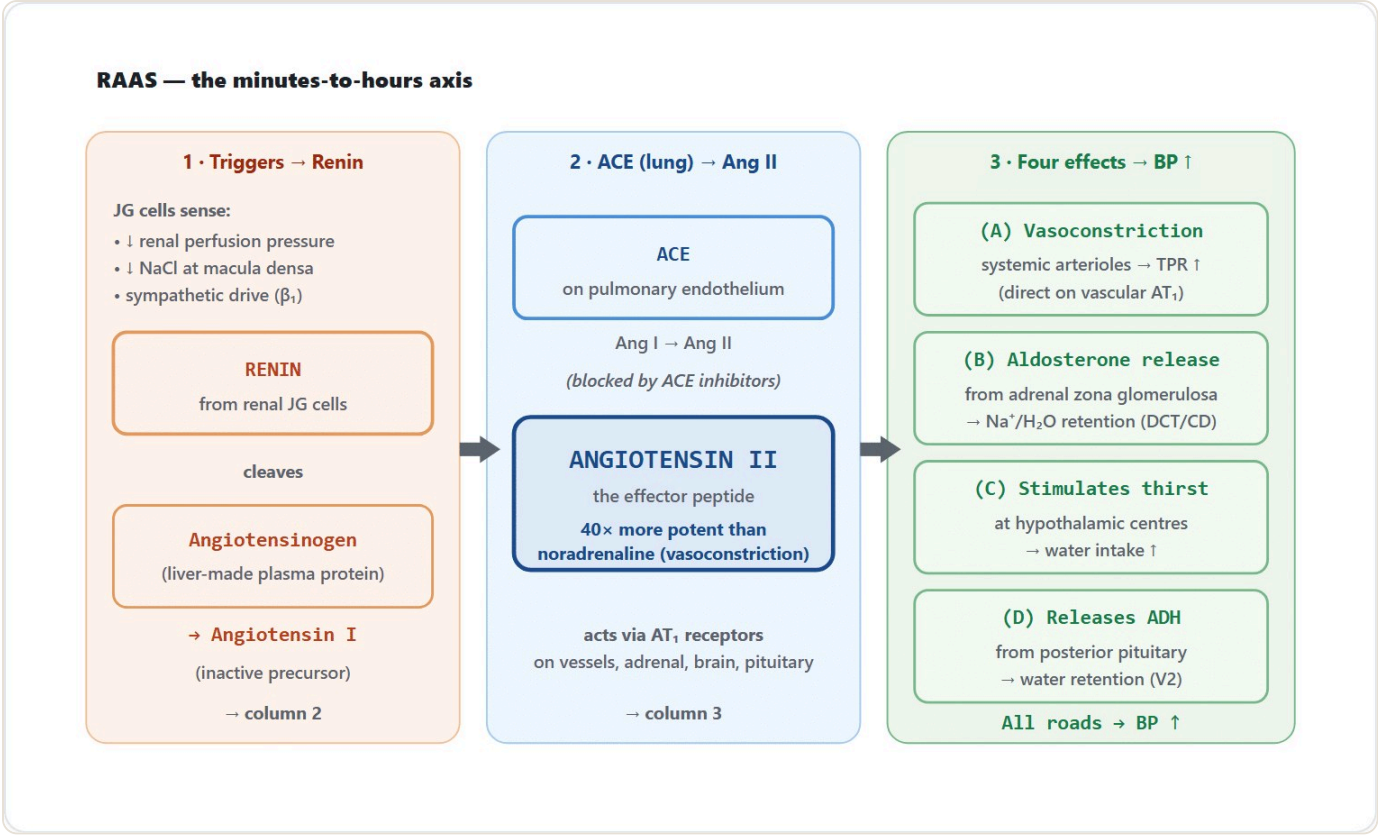


Fig 4 — The RAAS cascade. JG cells release renin → angiotensinogen → angiotensin I → pulmonary ACE → angiotensin II. Angiotensin II then does four things: vasoconstriction (40× noradrenaline), aldosterone release, thirst and ADH release — all raising BP. (Cunningham 6e Ch25 p278.)

07 ADH (vasopressin) — the water lever

The second circulating axis in the minutes-to-hours band is **antidiuretic hormone (ADH)**, also called **vasopressin**. If RAAS is the salt-and-vessel-tone lever, ADH is the pure-water lever. It is one of the smallest of the endocrine peptides (nine amino acids) but its effect on urine concentration, plasma osmolality and blood volume — and thus on BP — is out of all proportion to its size.

Synthesis and release. ADH is synthesised in the cell bodies of magnocellular neurones in the **supraoptic and paraventricular nuclei** of the hypothalamus. The peptide is transported down these neurones' axons and stored in the **posterior pituitary (neurohypophysis)** as vesicles ready for release into the systemic circulation. Two main stimuli release it. First, **hypothalamic osmoreceptors** sense a rise in plasma osmolality of only a few mOsm/kg above the set-point (around 280 mOsm/kg in a resting dog) and trigger ADH release — this is why a dehydrated animal makes concentrated urine even if its blood pressure is normal. Second, **atrial low-pressure (volume) receptors** in the walls of the atria and great veins signal changes in venous return; a fall in venous return (haemorrhage, volume depletion) drops their firing rate and triggers ADH release. Angiotensin II adds a third, smaller input as noted above.

Actions — V1 and V2 receptors. ADH acts on two G-protein coupled receptors with different jobs.

- **V1 receptors** sit on vascular smooth muscle. When ADH engages them (chiefly at the high circulating concentrations reached in severe haemorrhage or shock) they trigger vasoconstriction, raising TPR and MAP. In routine physiology the V1 effect is minor; in shock physiology it is important, and this is why the human ICU sometimes uses vasopressin as an adjunctive vasopressor in septic shock — the physiology is preserved in dogs and cats too.
- **V2 receptors** sit on the basolateral membrane of the principal cells of the renal collecting duct. ADH binding activates a Gs-cAMP cascade that inserts pre-formed **aquaporin-2** water channels into the apical membrane. Water then flows from the tubular lumen (dilute) through the aquaporins, across the cell, out through the constitutive aquaporin-3/4 on the basolateral side, and into the hypertonic medullary interstitium — leaving concentrated urine behind. This is the mechanism by which a dehydrated animal makes urine 5–10× more concentrated than plasma, and by which ADH raises blood volume.

The clinical consequences follow naturally. **Central diabetes insipidus** is a failure of ADH release (hypothalamic or posterior pituitary damage) — the animal cannot make concentrated urine, produces massive volumes of dilute urine, and drinks compulsively to keep up. **Nephrogenic diabetes insipidus** is failure of the collecting-duct response (V2 or aquaporin-2 defect) — the picture is the same, but ADH

itself is normal or high. And any process that removes the ADH response — furosemide (loop diuretic acting upstream), certain drugs, hypercalcaemia (hypercalcemia) — produces a similar pattern of polyuria and polydipsia.

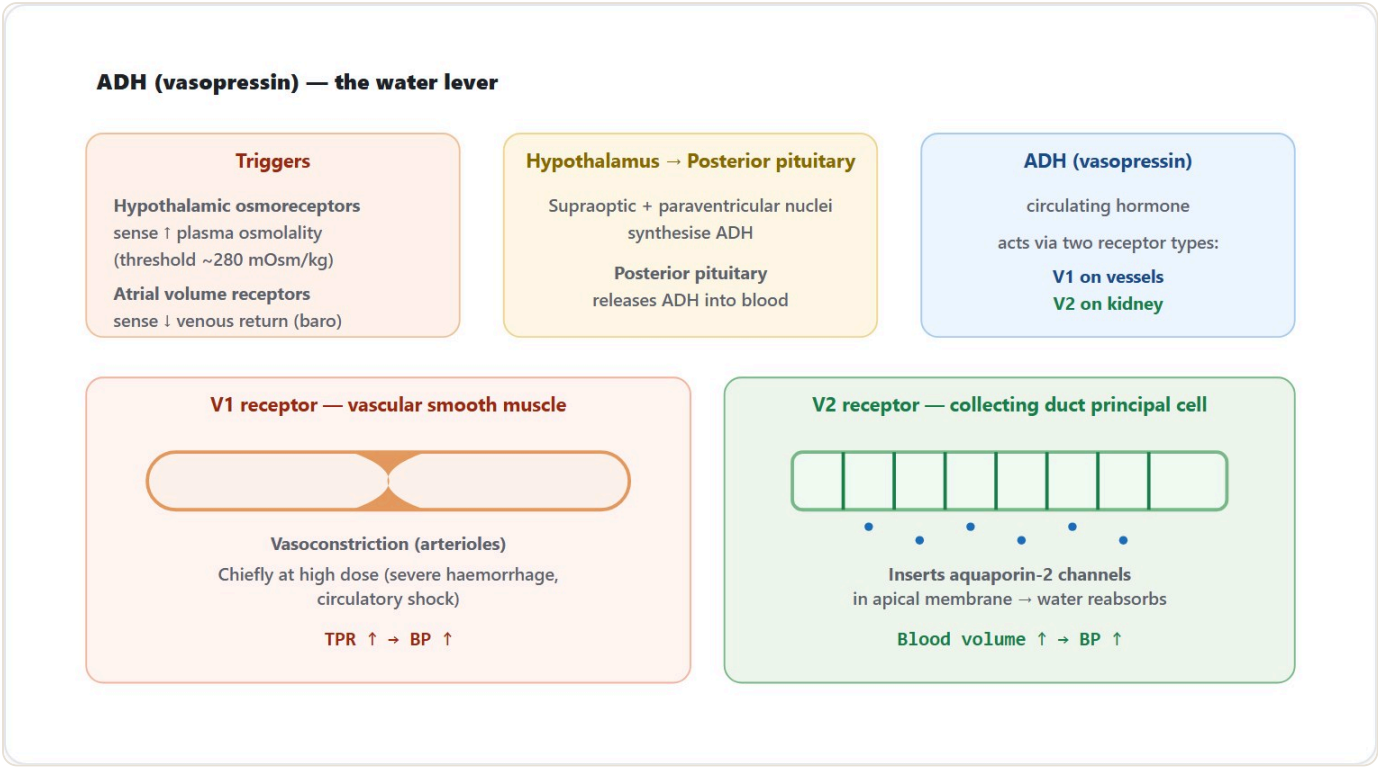


Fig 5 — ADH (vasopressin) — the water lever. Hypothalamic osmoreceptors + atrial volume receptors trigger ADH release from the posterior pituitary. In the periphery: V1 receptors on vessels drive vasoconstriction (mainly high dose); V2 receptors on collecting-duct principal cells insert aquaporin-2 water channels for water reabsorption. (Cunningham 6e Ch25 p280.)

08 Pressure natriuresis — the long-term set-point (Guyton)

Underneath the baroreflex, RAAS and ADH sits a slower, more fundamental control that keeps the arterial pressure set-point stable over days, weeks and years. It is the phenomenon known as **pressure natriuresis**, worked out in detail by Arthur Guyton in the mid-twentieth century (and covered in Cunningham Ch25 pp281–283). The principle is simple, but its implications are profound.

The principle. Raise the arterial pressure at the renal artery and the kidney, purely on the basis of the higher hydrostatic pressure at the glomerular capillary, will excrete more sodium and water — even before any reflex or hormonal signal has changed. Higher BP → higher GFR → higher tubular flow → less time to reabsorb sodium → more Na^+/H_2O in the urine → falling blood volume → falling CO → falling BP toward the set-point. The reverse is also true: at low arterial pressure the kidney retains sodium and water, expanding volume until pressure recovers.

Why it is the long-term controller. The baroreflex resets over days, so it cannot enforce a long-term set-point. RAAS and ADH habituate and are modulated by many other inputs. But pressure natriuresis is a physical mechanism — the higher the pressure at the kidney, the more Na^+ excreted — and it does not habituate. Whatever the reflexes and hormones do in the short term, the kidney gets the final vote over hours to days: it will keep excreting or retaining Na^+/H_2O until arterial pressure returns to whatever set-point the renal machinery favours. This is why any disease that damages the kidney's ability to excrete Na^+ (chronic kidney disease, hyperaldosteronism, primary renal disease) shifts the renal set-point upward and drives chronic hypertension — the whole rest of the circulation resets around a higher MAP because the kidney will not let it settle lower.

Clinically this is the single most important idea in the whole article. When you see a hypertensive cat with CKD, do not think of the CKD and the hypertension as two coincident diseases. They are one disease: the kidney has lost its ability to enforce the low-pressure set-point, and the whole circulation has resettled higher. Treatment is not just antihypertensives, it is also renal support and, where possible, correction of the underlying renal disease. In dogs, the same logic explains why Cushing's disease (which raises circulating cortisol, which has mineralocorticoid effects and shifts renal Na^+ handling) so often causes systemic hypertension.

09 Putting it together — a haemorrhage case in a dog

The best way to see all these mechanisms in action is to walk through one clinical scenario in real time. Consider a 25 kg Labrador hit by a car. Its abdomen is bruised, its gums are tacky, and it has lost approximately 20% of its blood volume into a splenic haemorrhage. Let us follow blood pressure regulation over seconds, minutes, hours and days.

Seconds — the baroreflex. Loss of 400–500 mL of blood drops venous return, then stroke volume, then MAP by 15–20 mmHg within a couple of beats. Firing at the carotid sinus and aortic arch baroreceptors falls; the medullary NTS senses the drop; the RVLM fires the sympathetic outflow harder and the vagal parasympathetic drive falls away. Within 3–5 seconds the dog's heart rate has risen from ~90 to ~140 bpm, contractility has risen, venoconstriction has raised venous return again, and arteriolar vasoconstriction in the skin, gut and muscle has raised TPR by about 30%. MAP is partially defended — often within 5–10 mmHg of baseline — on the strength of the baroreflex alone.

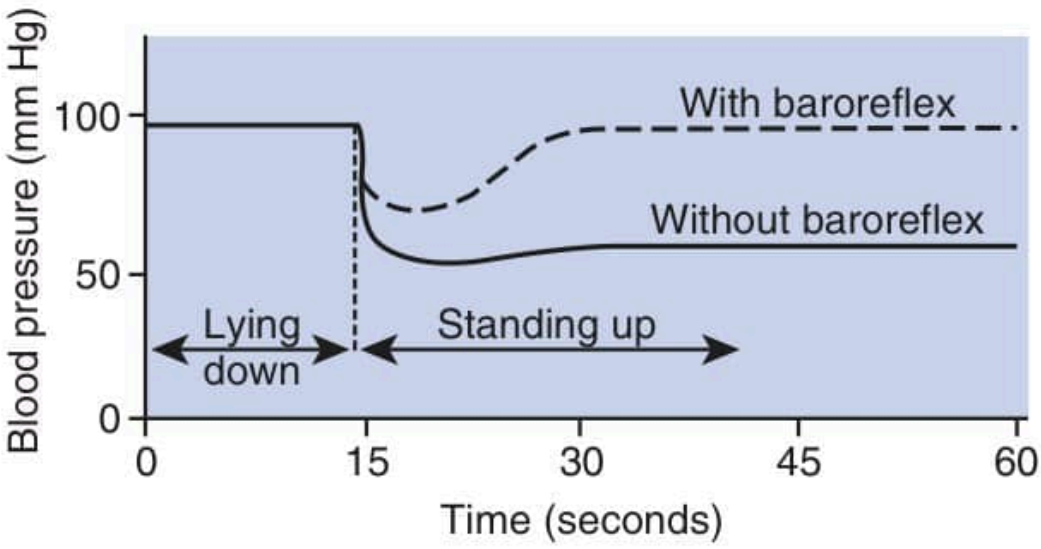


Fig. 25.4 The baroreflex is essential for normal, moment-to-moment stability of blood pressure. Dogs in which the baroreflex has been eliminated exhibit much larger swings in blood pressure in response to postural changes than do dogs with an intact baroreflex.

Fig 4a — A live example. In an intact dog, standing up drops BP briefly (dashed line), then the baroreflex corrects within 15 seconds. In a dog with the baroreflex eliminated, the same postural change drops BP by ~40 mmHg and it stays there — syncope territory. The same physiology defends the haemorrhaging Labrador.

Source: Cunningham 6e Ch25 Fig 25.4 p266.

Minutes — RAAS + ADH begin. Reduced renal perfusion and sympathetic drive have already triggered renin release from the JG cells. Within a few minutes plasma angiotensin II is rising, adding potent vasoconstriction to the sympathetic vasoconstriction already in place. Adrenal aldosterone release starts. The hypothalamic osmoreceptors sense a slight relative haemoconcentration (as tissue water shifts into vessels to replace lost volume) and, together with atrial volume receptors sensing the drop in venous return, drive ADH release. Circulating ADH begins to add V2 water retention at the kidney and, at the high circulating levels reached here, a little V1 vasoconstriction.

Hours — volume replacement. Aldosterone-driven sodium and water reabsorption at the distal tubule and collecting duct steadily rebuilds blood volume over the next few hours. ADH-driven water retention concentrates the urine to the maximum the kidney can achieve (~1200–1500 mOsm/kg in a dog). Angiotensin II-driven thirst has made the animal drink (if it is well enough), replacing water intake. The dog's plasma volume is rising back toward baseline, slowly, over hours.

Days — long-term recovery. Over the following days, if bleeding has stopped, erythropoietin from the kidney drives red-cell replacement, blood volume returns to normal, RAAS and ADH activation subside, and the baroreflex — which had briefly been operating at the higher-than-normal sympathetic output required for compensation — returns to its normal set-point. The kidney's pressure-natriuresis machinery, unchanged throughout, ensures the animal settles at exactly its previous MAP.

Callout — a graduate-level rule. This is why aggressive fluid loading in a haemorrhaging dog before surgical source control can be counter-productive. Raising BP too fast can dislodge a stabilising clot and worsen bleeding. The physiological compensation described above is *working*; permissive hypotension (a moderate, controlled BP target while you get to surgery) is often preferred in the acutely haemorrhaging patient. Fluids replace what the baroreflex, RAAS and ADH cannot; they do not replace them.

10 Measuring it: Doppler, oscillometric, and why the technique changes the number

Every threshold below assumes a **systolic blood pressure** obtained properly. In a nervous cat the method and the handling move the reading further than most diseases do, so the technique is not a detail — it is the measurement. What you are sampling is the product of cardiac output and **total peripheral resistance**, also called **systemic vascular resistance**, at one moment in one limb.

Doppler uses an ultrasonic crystal taped over a peripheral artery, an inflatable cuff and an aneroid manometer: you inflate until flow stops, deflate slowly, and the pressure at which the whoosh returns is the systolic. It needs an operator but tolerates movement and small patients well, which is why it remains the preferred method in cats. **Oscillometric** devices inflate and deflate automatically and read the amplitude of arterial wall oscillation, returning systolic, diastolic and mean pressures without an operator — convenient, but less reliable in small, hypotensive, arrhythmic or shivering patients, exactly the ones you most want a number from.

The protocol matters as much as the machine. Use a cuff whose width is about **30–40% of the circumference of the limb** — too narrow reads falsely high, too wide falsely low. Let the animal settle in a quiet room for five to ten minutes, ideally with the owner present; keep the limb at the level of the heart; take five to seven readings, **discard the first**, and average the rest once they are consistent. Record the cuff site, cuff size, position and the patient's demeanour, so the next person can repeat it the same way.

The white-coat effect is not a myth It is a genuine sympathetic response — the baroreflex working exactly as described above — and it can add tens of mmHg. Never diagnose hypertension on a single high reading in an anxious patient. Repeat it on a separate visit, unless there is already **target organ damage**, in which case the eyes have made the diagnosis for you.

11 Hypertension in dogs and cats

If blood pressure regulation in animals is the coordinated defence of MAP, **hypertension in dogs and cats** is the failure of that regulation in the direction of too much pressure — sustained, and damaging. The physiology of hypertension is the mirror of the physiology of BP defence: it is what happens when one or more of the mechanisms above becomes locked at 'on'. In veterinary medicine, unlike in humans, idiopathic (essential) hypertension is uncommon; almost every hypertensive dog or cat has a secondary cause. Diagnosis is a two-step process: (1) confirm the pressure is really elevated, and (2) find the underlying disease.

Confirming the pressure. The ACVIM 2018 consensus (Acierno et al., *J Vet Intern Med* 2018;32:1803–1822) is the working reference. In cats, the categories are: **normotensive < 140 mmHg** systolic; **prehypertensive 140–159 mmHg**; **borderline hypertensive 160–179 mmHg**; **severely hypertensive ≥ 180 mmHg**. Because cats are famously stress-reactive ('white-coat hypertension' is not just a human phenomenon), the consensus emphasises confirmation on **2–3 visits** before diagnosing and treating chronic hypertension — unless target-organ damage (e.g. sudden blindness from retinal detachment) is already present, in which case treatment starts immediately. In dogs the thresholds are broadly similar, but the picture is often more urgent because the underlying disease (Cushing's, phaeochromocytoma) is itself demanding of attention.

Causes in cats. The commonest cause of hypertension in cats is **chronic kidney disease**. CKD damages both the excretory and the endocrine machinery of the kidney, disturbs pressure natriuresis, and drives systemic hypertension in roughly 20–65% of affected cats depending on stage. The next commonest is **hyperthyroidism**: elevated circulating thyroid hormone increases heart rate, contractility and CO, and adds a mild peripheral vasodilation with net rise in systolic BP. Other causes include Cushing's-like disease (rare in cats), primary hyperaldosteronism (Conn's; rare but increasingly recognised), and, occasionally, phaeochromocytoma. When you see a hypertensive cat, your workup is a renal panel plus urine protein:creatinine ratio, a total T4, and a fundoscopic examination for end-organ damage.

Causes in dogs. In dogs the top causes are **chronic kidney disease** (as in cats), **hyperadrenocorticism (Cushing's syndrome)**, **diabetes mellitus**, and **phaeochromocytoma or other adrenal tumours**. Cushing's is a particularly consistent cause because cortisol has mineralocorticoid effects (Na^+ retention), promotes vascular reactivity, and can drive systemic hypertension in more than half of affected dogs. Any dog with new-onset hypertension should have a renal panel, an ACTH stimulation test or low-dose dexamethasone suppression test, and (if signs are present) imaging of the adrenals.

End-organ damage. The reason hypertension is treated even when the animal feels well is the damage that sustained high pressure does to four organs. The **eye** is the most dramatic in cats: hypertensive retinopathy can cause retinal haemorrhage, retinal detachment and sudden bilateral blindness — a cat presenting with sudden-onset blindness always warrants a BP measurement before any other workup. The **brain** is affected by hypertensive encephalopathy (seizures, altered mentation), especially at systolic pressures above ~200 mmHg. The **heart** develops left ventricular hypertrophy over time as it labours against high afterload. The **kidney** is both cause and victim: glomerular hypertension drives proteinuria and accelerates CKD progression, closing the loop of self-perpetuation.

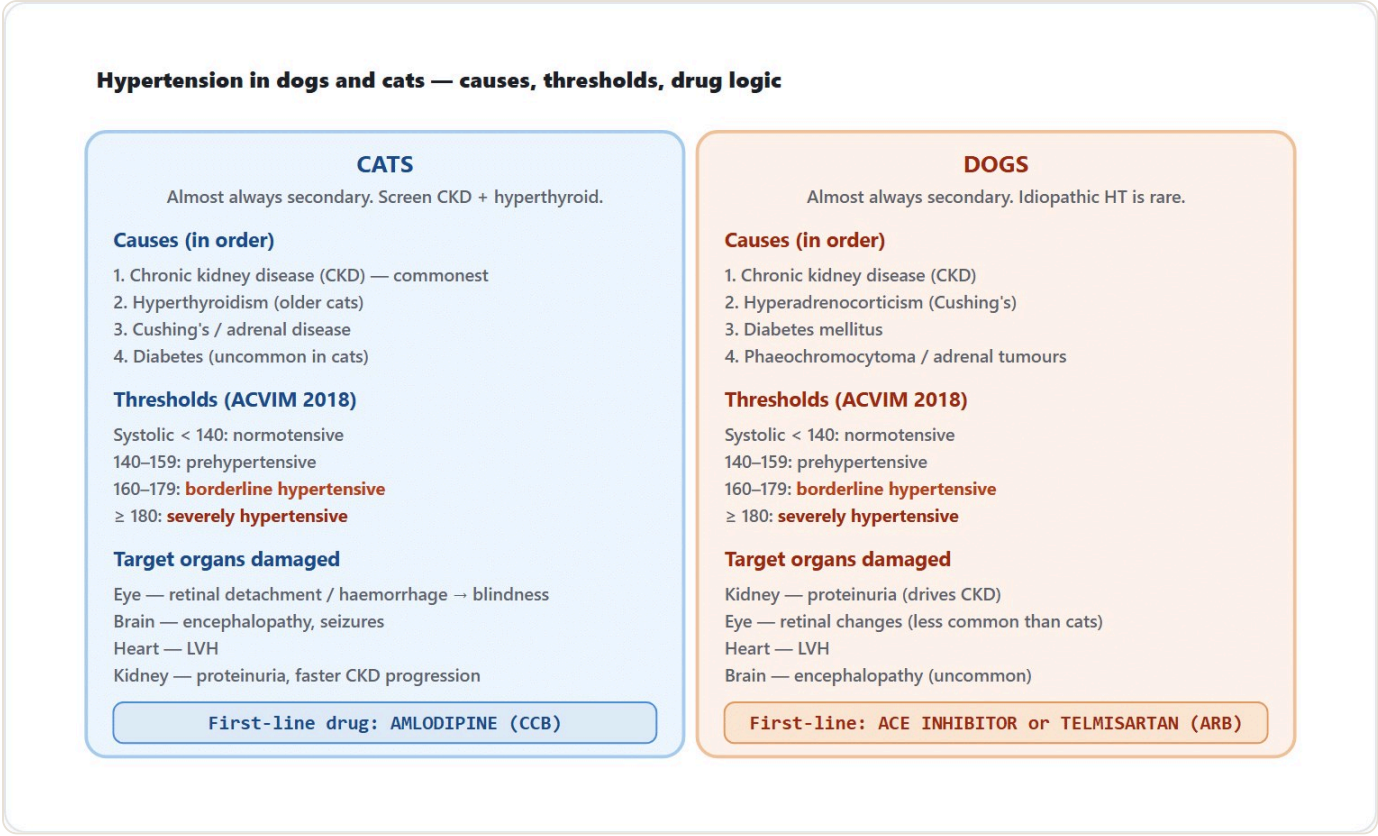


Fig 6 — Hypertension in dogs and cats. Cats: CKD & hyperthyroidism are the top causes, amlodipine (CCB) is first-line. Dogs: CKD, Cushing's, diabetes, phaeochromocytoma; ACE inhibitor or telmisartan (ARB) first-line. ACVIM 2018 thresholds: >160 borderline, >180 hypertensive; treat at ≥160 with end-organ damage.

12 Clinical reasoning + treatment logic

Because **hypertension in dogs and cats** is almost always secondary, the first line of treatment is always *treat the cause*. Correct the hyperthyroidism (methimazole or radio-iodine), correct the Cushing's (trilostane or surgery), stabilise the CKD (renal diet, phosphate binders), remove the phaeochromocytoma. In many cases the BP falls back into the normal range as the primary disease is controlled, and no antihypertensive is needed. Only when the pressure remains elevated after correcting the cause, or when the animal has end-organ damage, or when the underlying disease cannot be corrected in the short term, do you reach for the antihypertensive drugs. And here the drug logic falls straight out of the physiology.

- **Calcium-channel blockers — amlodipine.** The commonest first-line in *cats*. Amlodipine is an L-type calcium-channel blocker that acts predominantly on *arteriolar* smooth muscle, dropping TPR without much direct cardiac effect. In hypertensive cats systolic BP typically falls by 30–50 mmHg on standard doses (0.625–1.25 mg/cat SID). Adverse effects are uncommon; a mild reflex tachycardia is occasionally seen. Amlodipine is the drug of choice for feline hypertension because it is potent, well tolerated, and works regardless of the underlying cause.
- **ACE inhibitors — enalapril, benazepril.** First-line in many *dogs*, especially those with concurrent proteinuric CKD or heart failure. ACE inhibitors block angiotensin II production, taking off vasoconstriction, aldosterone drive, thirst and ADH release in one go. The BP effect in dogs is real but modest (~10–20 mmHg systolic); the anti-proteinuric effect at the efferent arteriole may be even more valuable. Adverse effects: azotaemia (azotemia) can rise slightly (a physiological response to the loss of efferent arteriolar constriction — not a reason to stop the drug unless marked).
- **Angiotensin receptor blockers (ARBs) — telmisartan.** Telmisartan blocks the AT_1 receptor directly, giving all the benefits of ACE inhibition without the small proportion of angiotensin II that escapes ACE (there are non-ACE conversion routes). It is especially useful in *feline proteinuric CKD*, where large randomised studies have shown reduced proteinuria and BP with once-daily dosing (1 mg/kg PO SID). Increasingly it is used first-line in cats where the hypertension is CKD-driven.
- **β-blockers — atenolol.** Reserved for *hyperthyroid cats* where the tachycardia is the driver, or the occasional dog with tachyarrhythmia-driven hypertension. β-blockers drop HR and contractility (i.e. CO) but do little to arteriolar tone, so they are second-line as pure antihypertensives.
- **Aldosterone antagonists — spironolactone.** Adjunctive in advanced heart failure or refractory hypertension; not usually a first-line antihypertensive.

Traps to avoid. Do not start α-blockers or diuretics as first-line antihypertensives in a dog or cat without a specific reason: α-blockers can cause profound orthostatic hypotension and are best reserved for the specific case of a phaeochromocytoma (where phenoxybenzamine has a clear role). Diuretics as antihypertensives are unhelpful in most cats because they can dehydrate a CKD patient and accelerate azotaemia. And in a hyperthyroid cat with hypertension, do not just add amlodipine and consider the case closed — treat the thyroid disease, and the BP will often normalise on its own. The physiology is doing the work; your job is to help it, not to override it.

13 Quiz: test yourself

Fifteen questions across MAP = CO × TPR, the three time-scales, the baroreflex, RAAS, ADH, pressure natriuresis and hypertension. Answers are beneath each question — cover them and work down.

1. What equation defines mean arterial pressure?**Answer: MAP = CO × TPR** — cardiac output times total peripheral resistance. Every mechanism in the article works on one of those two terms.2. What are normal systolic pressures in dog, cat, horse and cow?**Answer:** Dog about **120 mmHg** systolic and 80 diastolic (healthy range roughly 100–160); cats slightly higher at **120–160**; horses slightly lower at **90–140**; cows **100–180**.3. What are the three time-scales on which the circulation defends MAP?**Answer: Seconds** — the baroreflex. **Minutes to hours** — RAAS and ADH. **Days to years** — pressure natriuresis. They layer rather than compete.4. Where are the arterial baroreceptors? **Answer:** Stretch-sensitive nerve endings in two places: the **carotid sinus** — a slight dilation of the internal carotid just past its origin — and the **aortic arch**.5. A dog's blood pressure falls. What does the baroreflex do?**Answer:** Baroreceptors fire **more slowly**, so the medulla raises **sympathetic** and lowers **parasympathetic** outflow: heart rate up, contractility up, venoconstriction, TPR up.6. How do the chemoreceptor and CNS ischaemic responses differ from the baroreflex?**Answer:** Both are **emergency reflexes for extremes**, not everyday control. Their afferents travel with the baroreceptor afferents in **CN IX and CN X** but project to a distinct set of medullary output neurones.7. What are the three stimuli to renin release?**Answer: Renal perfusion pressure** — the JG cells are themselves stretch receptors; **macula densa** NaCl delivery; and **sympathetic drive** on β_1 receptors, so any baroreflex surge also releases renin.8. What triggers ADH release, and how sensitive is the sensor?**Answer: Hypothalamic osmoreceptors**, which detect a rise of only a **few mOsm/kg** above a set-point near **280 mOsm/kg** in a resting dog. It is why a dehydrated animal makes concentrated urine even when its blood pressure is normal.9. What is pressure natriuresis, and who worked it out?**Answer:** The kidney excreting more sodium and water as arterial pressure rises — the slow control that sets the long-term pressure set-point over days, weeks and years. Worked out by **Arthur Guyton**.10. How should you size a blood-pressure cuff, and what happens if you get it wrong?**Answer:** Width about **30–40% of the limb circumference**. **Too narrow reads falsely high; too wide reads falsely low.**11. Why is essential hypertension a poor first guess in a hypertensive cat?**Answer:** Because in veterinary medicine, unlike human, **idiopathic hypertension is uncommon** — almost every hypertensive dog or cat has a **secondary cause**.12. What is the two-step diagnostic process in suspected hypertension?**Answer: (1) Confirm the pressure is really elevated** — the ACVIM 2018 consensus is the working reference — and **(2) find the underlying disease**.13. What is the first line of treatment for hypertension in a dog or cat?**Answer: Treat the cause.** Correct the hyperthyroidism, the hyperadrenocorticism, stabilise the CKD, remove the phaeochromocytoma. In many cases the pressure falls back to normal and no antihypertensive is needed.14. A 25 kg Labrador loses 20% of its blood volume. What happens in the first seconds?**Answer:** Losing **400–500 mL** drops venous return, then stroke volume, then MAP by **15–20 mmHg** within a couple of beats. Baroreceptor firing falls, the RVLM drives sympathetic outflow harder and vagal tone falls away — so within **3–5 seconds** heart rate has gone from about **90 to 140 bpm**, with raised contractility and venoconstriction.15. When do you reach for an antihypertensive, and what is first-line in the cat?**Answer:** Only when pressure stays high **after** correcting the cause, when there is **end-organ damage**, or when the primary disease cannot be corrected quickly. **Amlodipine**, a calcium-channel blocker, is the commonest first-line in cats.

14 Blood pressure regulation in animals — frequently asked questions

How is blood pressure regulated in animals?

Blood pressure in animals is regulated on three time-scales that layer together. In *seconds*, the baroreceptor reflex (from stretch receptors in the carotid sinus and aortic arch, via CN IX and CN X to the medullary NTS) adjusts sympathetic and parasympathetic outflow to heart and vessels. In *minutes to hours*, the renin-angiotensin-aldosterone system (RAAS) and ADH (vasopressin) drive vasoconstriction and sodium/water retention. Over *days*, the kidney enforces the long-term set-point through pressure natriuresis (Guyton). All defend one number: mean arterial pressure, so every organ is perfused.

What is the baroreceptor reflex and what does it do?

The baroreceptor reflex is the fastest of the three BP-regulation mechanisms. Stretch-sensitive baroreceptors in the carotid sinus and aortic arch fire more when arterial pressure rises. Their afferents in CN IX (glossopharyngeal) and CN X (vagus) converge on the nucleus tractus solitarii in the medulla, which reciprocally adjusts sympathetic and parasympathetic outflow. A rise in BP reduces sympathetic drive and increases vagal (parasympathetic) drive, dropping HR, contractility and TPR. A fall in BP does the reverse. The reflex resets to a new set-point over days at chronic higher pressure — which is why chronically hypertensive cats have lost the alarm.

What is the RAAS and why does it matter clinically?

The renin-angiotensin-aldosterone system (RAAS) is the minutes-to-hours arm of BP regulation. Renal juxtaglomerular cells release **renin** when perfusion falls; renin cleaves liver-made angiotensinogen to angiotensin I; angiotensin-converting enzyme (ACE) on pulmonary endothelium converts it to **angiotensin II**; angiotensin II then vasoconstricts (40× more potent than noradrenaline), releases aldosterone (Na^+/water retention), stimulates thirst, and releases ADH. Every step is drug-targeted: ACE inhibitors (enalapril, benazepril) at the ACE step, ARBs (telmisartan) at the AT_1 receptor, aldosterone antagonists (spironolactone) downstream. RAAS is central to hypertension in dogs and cats and to the physiology of heart failure and CKD.

What is normal blood pressure in a dog or cat?

In a resting, awake dog, systolic pressure sits in the range 100–160 mmHg with a diastolic of 60–90 mmHg. In a cat the systolic range is a little higher — 120–160 mmHg. Both species are stress-reactive, so measurements are best made in a quiet room with the animal acclimatised, using an appropriately sized cuff on a tail or limb, and reported as an average of five or more consistent readings. The ACVIM 2018 consensus considers cat systolic > 160 mmHg (repeatedly) as borderline hypertensive and > 180 mmHg as severely hypertensive; dogs use broadly the same thresholds.

What causes hypertension in cats?

Almost always secondary: **chronic kidney disease** is the commonest cause, followed by **hyperthyroidism** (older cats). Cushing's-like disease, primary hyperaldosteronism (Conn's) and phaeochromocytoma are much rarer causes. Idiopathic hypertension is uncommon in cats (unlike humans). Workup for a hypertensive cat is a renal panel plus urine protein:creatinine ratio, a total T4, and a fundoscopic examination for end-organ damage (retinal detachment, retinal haemorrhage). First-line treatment is amlodipine (calcium-channel blocker); telmisartan (ARB) is increasingly first-line in CKD-associated feline hypertension.

How is blood pressure measured in dogs and cats?

Almost always indirectly, giving a **systolic blood pressure**. **Doppler** — a crystal over an artery plus a cuff and manometer — is operator-dependent but copes well with small or moving patients and is preferred in cats. **Oscillometric** machines are automated and also report diastolic and mean pressures, but become unreliable in small, hypotensive or arrhythmic animals. Whichever you use, the cuff should be about 30–40% of limb circumference, the patient should acclimatise for five to ten minutes, the limb should sit at heart level, and you should discard the first reading and average five to seven consistent ones.

Can I download these blood pressure regulation study notes as a free PDF?

Yes — these are complete, illustrated veterinary study notes on blood pressure regulation in animals, free to download as a PDF with no signup. For more veterinary study notes on the cardiovascular system, browse our veterinary physiology hub — including the linked companion articles on the cardiac cycle (where cardiac output is generated) and oedema & the Starling forces (what happens downstream when BP control fails and Pc rises).

When to refer

- A cat with sudden-onset blindness — measure BP first (hypertensive retinal detachment); if systolic > 180 mmHg, treat immediately with amlodipine and work up for CKD or hyperthyroidism.
- A hypertensive cat with confirmed systolic > 160 mmHg on 2–3 visits — refer or workup for **CKD** (biochemistry + UPC) and **hyperthyroidism** (total T4); treat the primary disease and add amlodipine if BP does not normalise.
- A dog with new-onset systemic hypertension — refer or workup for **hyperadrenocorticism (Cushing's)**, CKD, diabetes mellitus and phaeochromocytoma; start with an ACE inhibitor or telmisartan while the underlying disease is characterised.
- A dog in acute haemorrhage (RTA, splenic mass rupture) — support the compensating baroreflex/RAAS/ADH; do *not* over-fluid before surgical source control (risk of dislodging a stabilising clot); target permissive hypotension until the source is controlled.
- A hyperthyroid cat with tachycardia and systolic > 180 mmHg — treat the thyroid disease first (methimazole or radio-iodine); consider β-blocker (atenolol) short-term for symptomatic tachycardia; the BP often normalises on thyroid control alone.

Sources

1. Klein BG (ed). *Cunningham's Textbook of Veterinary Physiology*. 6th ed. St Louis: Elsevier; 2020. **Ch 25 'Neural and Hormonal Control of Blood Pressure and Blood Volume' (pp 273–283)** — primary source for the baroreflex arc, RAAS, ADH, pressure natriuresis and the time-scales of BP control.
2. Sjaastad ØV, Sand O, Hove K. *Physiology of Domestic Animals*. 3rd ed. Oslo: Scandinavian Veterinary Press; 2016. **Ch 11 'The Cardiovascular System'** — secondary source for species differences in resting BP normals, RAAS anatomy and ADH.
3. Acierno MJ, Brown S, Coleman AE, et al. ACVIM consensus statement: guidelines for the identification, evaluation, and management of systemic hypertension in dogs and cats. *J Vet Intern Med* 2018;32(6):1803–1822 — clinical anchor for hypertension thresholds and drug logic.
4. Ettinger SJ, Feldman EC, Côté E. *Textbook of Veterinary Internal Medicine*. 8th/9th ed. Elsevier; 2017–2024. Chapters on systemic hypertension, CKD, hyperthyroidism, hyperadrenocorticism and phaeochromocytoma — clinical anchor for the secondary causes and workup.

Vet eBooks
VET-EBOOKS.COM

MORE FREE STUDY GUIDES · RESOURCES

More free veterinary study guides

This is one of a growing library of free veterinary study guides on vet-ebooks.com — written for practising vets and students, sourced from the standard veterinary texts, and every one free with a downloadable PDF and no sign-up.

3,400+
veterinary books

400+
drug profiles

Study guides
a free PDF with each

All free
no sign-up needed

www.vet-ebooks.com