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— STUDY NOTES

How Blood Pressure Is Controlled in Animals

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.

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How Blood Pressure Is Controlled in Animals

STUDY NOTES · BASIC SCIENCES · PHYSIOLOGY · UPDATED 2026-07-06

CONTENTS

LEARNING OBJECTIVES

After working through these notes you will be able to:

- ✓ Define blood pressure regulation in animals as the coordinated defence of mean arterial pressure (MAP) so every organ is perfused.
- ✓ Write the equation MAP = CO x TPR and give resting systolic values for a dog, a cat and a horse.
- ✓ List the three time-scales of BP control and state which mechanisms operate on each.
- ✓ Describe the baroreceptor reflex arc: receptors, afferents, medullary centre and efferent branches, with a resting-BP example.
- ✓ Walk the RAAS pathway from renin release to angiotensin II's four effects, and state where ACE is located.
- ✓ Explain ADH's dual action at V1 (vascular) and V2 (renal aquaporin-2) receptors and its main triggers.
- ✓ State the Guyton principle of pressure natriuresis and why it sets the long-term BP mean.
- ✓ Give the ACVIM 2018 thresholds for hypertension in cats and list the top two secondary causes in cats and in dogs.
- ✓ Match antihypertensive drug classes (ACE inhibitor, ARB, CCB, beta-blocker) to their physiological targets.

TL;DR

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.

AT A GLANCE

WHAT BP REGULATION IS	Keeping **MAP** stable so every organ is perfused — irrespective of posture, blood loss, exercise or sleep
THE CORE EQUATION	**MAP = CO × TPR** — change cardiac output or peripheral resistance, and MAP moves with them
SPECIES NORMALS	Dog systolic ~100–160 mmHg · cat ~120–160 · horse ~90–140 (all resting, awake)
SECONDS — BAROREFLEX	Carotid sinus + aortic arch stretch receptors → CN IX + CN X → medulla NTS → heart + vessels
MINUTES-HOURS — RAAS	JG cells → renin → Ang I → **ACE** (lung) → **Ang II** → vasoconstriction + aldosterone + thirst + ADH
MINUTES-HOURS — ADH	Hypothalamic osmoreceptors + atrial volume receptors → posterior pituitary → V1 (vessels) + V2 (kidney)
DAYS — KIDNEY	**Pressure natriuresis** (Guyton) sets the long-term BP set-point through Na+/water excretion
HYPERTENSION IN DOGS AND CATS	Cats: CKD, hyperthyroidism (systolic > 160 borderline, > 180 hypertensive). Dogs: CKD, Cushing's, phaeochromocytoma
DRUG LOGIC	**ACE inh** (enalapril/benazepril) · **ARB** (telmisartan) · **CCB** (amlodipine — cats) · **β-blocker** (hyperthyroid cats)

01 Why BP control matters

- **Blood pressure regulation in animals** = the whole body's job of holding **MAP** steady enough to perfuse every organ — brain, kidney, heart, gut — second by second, hour by hour, day by day.
- **MAP** ~= **CO × TPR**. The body's controllers act on one lever or the other. See cardiac cycle for CO.
- **Failure = perfusion loss** (shock, syncope, ischaemic organs) or **chronic overload** (hypertension: retinal detachment, encephalopathy, LVH, CKD).

02 MAP = CO × TPR (species normals)

- **MAP** (mean arterial pressure) ≈ DBP + 1/3·(SBP − DBP).
- **Dog:** systolic ~100–160 mmHg · **Cat:** ~120–160 · **Horse:** ~90–140 · **Cow:** ~100–180. Awake, unstressed.
- **CO** = HR × SV (heart lever). **TPR** = arteriolar tone (vessel lever), set by sympathetic + Ang II + ADH V1.

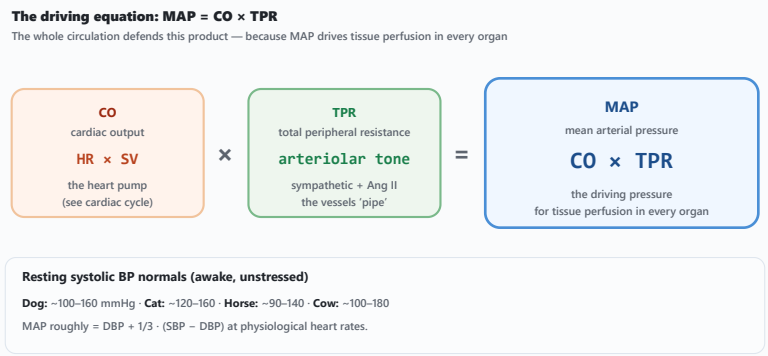

*Fig 1 — MAP = CO × TPR. The two levers the body pulls to keep **blood pressure regulation in animals** on target.*

03 Three time-scales of BP control

- **Seconds:** baroreflex + chemoreflex + CNS ischaemic response. Neural.
- **Minutes – hours:** RAAS + ADH. Hormonal.
- **Hours – days:** renal **pressure natriuresis** (Guyton). Volume-based. Sets the long-term mean.
- **They layer, don't compete** — a single fall in BP triggers all three; each dominates over a different window.

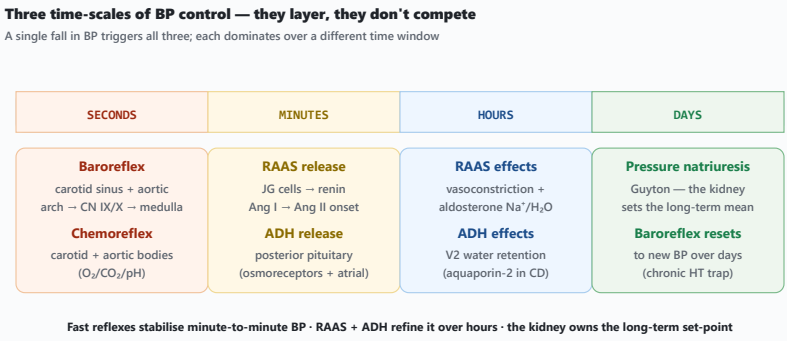

Fig 2 — The three time-scales of BP control — seconds → minutes → hours → days.

04 The baroreceptor reflex

- **Receptors**: stretch receptors in the **carotid sinus** (dilation of internal carotid) + **aortic arch** wall.
- **Afferents**: **CN IX** (glossopharyngeal) from carotid sinus + **CN X** (vagus) from aortic arch → medulla **NTS**.

- **Efferents**: NTS → RVLM (sympathetic outflow T1–L2) + nucleus ambiguus (parasympathetic via CN X).
- **If BP falls**: baroreceptor firing drops → sympathetic UP → HR↑, contractility↑, TPR↑, venoconstriction → BP returns.
- **If BP rises**: baroreceptors fire more → parasympathetic UP (vagus to SA node) → HR↓, sympathetic tone↓ → BP returns.
- **Trap:** the baroreflex **resets** over days at chronic hypertension — the alarm goes quiet. A hypertensive cat has lost its guardrail.

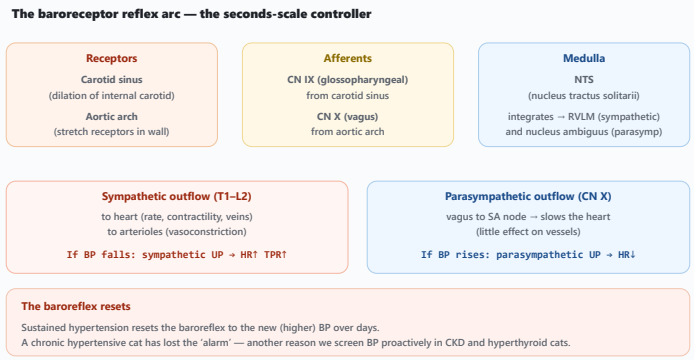

Fig 3 — The baroreceptor reflex arc: carotid sinus + aortic arch → CN IX + X → NTS → sympathetic & parasympathetic outflow.

05 Chemoreflex + CNS ischaemic response

- **Peripheral chemoreceptors** (**carotid** + **aortic bodies**) sense ↓O_2, ↑CO_2, ↓pH → sympathetic surge → TPR ↑.
- **CNS ischaemic response** = the emergency backup. Kicks in when MAP falls below ~**50 mmHg** and the medulla itself becomes ischaemic — massive sympathetic surge (Cushing's triad if intracranial pressure is the cause).

06 RAAS — the minutes-to-hours axis

- **Triggers** (all three release renin from JG cells): ↓ renal perfusion pressure · ↓ NaCl at macula densa · sympathetic β_1 drive.
- **Renin** (JG cells) → cleaves liver **angiotensinogen** → **Angiotensin I**.
- **ACE** (pulmonary endothelium) → **Angiotensin II**. Blocked by ACE inhibitors (enalapril, benazepril).
- **Angiotensin II — 4 effects, all → BP ↑:** (A) **vasoconstriction** (systemic arterioles — via vascular AT_1; ~**40× more potent** than noradrenaline); (B) **aldosterone** release (adrenal zona glomerulosa) → Na^+/H_2O retention (DCT/CD); (C) stimulates **thirst**; (D) releases **ADH**.

- **Drug logic:** ACE inhibitors block synthesis; **ARBs** (telmisartan) block AT_1 receptor. Both stop A + B + D.

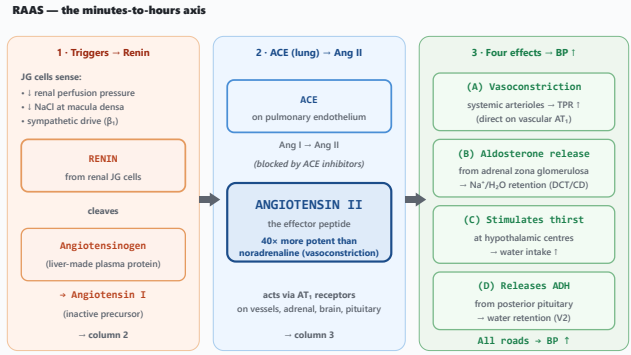

Fig 4 — RAAS: triggers → renin → angiotensin II → four effects → BP ↑.

07 ADH (vasopressin) — the water lever

- **Triggers**: hypothalamic **osmoreceptors** (↑ plasma osmolality above ~280 mOsm/kg) + atrial low-pressure receptors (↓ venous return / volume).
- **Site**: made by supraoptic + paraventricular hypothalamic nuclei; released from **posterior pituitary**.
- **V1 receptor** (vascular smooth muscle) → vasoconstriction (mainly at high dose — severe haemorrhage/shock) → TPR↑.
- **V2 receptor** (collecting duct principal cell) → inserts **aquaporin-2** channels in apical membrane → water reabsorbed → blood volume↑ → BP↑.
- **Trap:** at "physiological" plasma levels ADH is primarily a water hormone. It is a vasoconstrictor only when levels are high (severe volume loss).

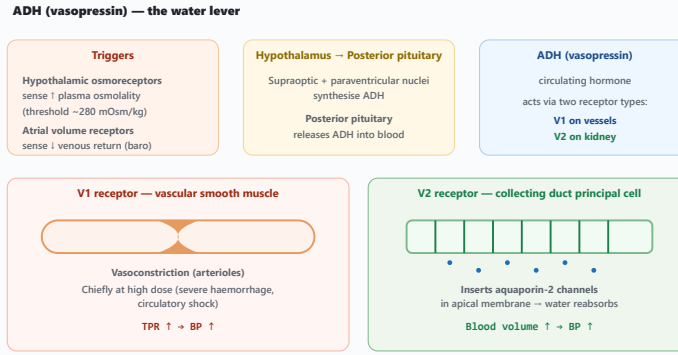

Fig 5 — ADH acts on two receptors: V1 (vessels) & V2 (collecting duct aquaporin-2).

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

- **Rule:** as arterial pressure rises, the **kidney excretes more Na^+ and water**. Blood volume falls. BP falls.
- Conversely: low BP → less Na^+/H_2O excreted → volume rises → BP rises.
- **Days-to-weeks scale** — the kidney owns the long-term mean; every other mechanism just trims around it.
- **Clinical:** when the kidney fails (CKD), pressure natriuresis stops working — the animal loses the anchor → hypertension.

09 Putting it together — haemorrhage in a dog

- **Second 0–30:** BP drops (loss of ~20% volume) → baroreceptors fire less → sympathetic surge → HR↑, TPR↑, venoconstriction. Pale mm, cold extremities.
- **Minute 1–60:** RAAS releases — renin, Ang II vasoconstricts, aldosterone begins retaining Na^+. ADH released (V1 vasoconstriction if severe; V2 water retention).
- **Hour 1–24:** RAAS + ADH fully engaged. Interstitial fluid shifts into vessels. Thirst drives water intake.
- **Day 1–7:** renal pressure natriuresis + erythropoiesis + healing. Volume restored. Baroreflex re-sensitised.

10 Hypertension in dogs & cats

- **Idiopathic HT is rare** in animals (unlike humans). Almost always **secondary** — find and treat the cause.
- **Cats:** 1) CKD (commonest), 2) hyperthyroidism, 3) Cushing's, 4) diabetes.

- **Dogs:** 1) CKD, 2) hyperadrenocorticism, 3) diabetes, 4) phaeochromocytoma / adrenal tumours.
- **ACVIM 2018 thresholds (systolic):** < 140 normotensive · 140–159 pre-HT · 160–179 borderline · ≥ 180 severe.
- **End-organ damage:** eye (retinal detachment, blindness) · brain (encephalopathy, seizures) · heart (LVH) · kidney (proteinuria, CKD progression).
- **Cats: confirm on 2–3 visits** before diagnosing — single readings are often falsely high (white-coat effect).

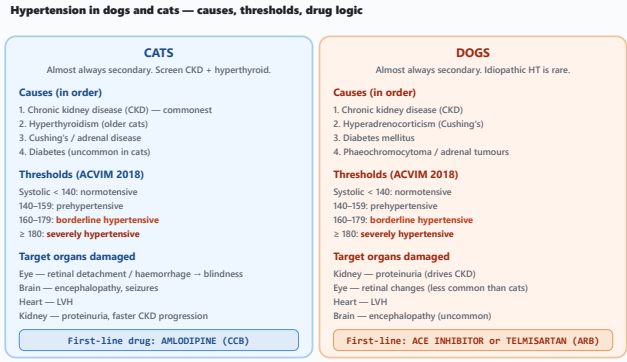

Fig 6 — Hypertension in dogs & cats: causes, ACVIM thresholds, target organs, first-line drug.

11 Drug logic

- **Cats (first-line): amlodipine** (calcium-channel blocker) — arteriolar vasodilator. Add telmisartan for proteinuric CKD cats.
- **Dogs (first-line): ACE inhibitor** (enalapril, benazepril) or **ARB** (telmisartan). Add amlodipine if BP still > 160.
- **Hyperthyroid cats:** add **atenolol** (β-blocker) for tachycardia + HT.
- **Emergency HT (retinal detachment, encephalopathy):** IV amlodipine or hydralazine; monitor closely.
- **Treat cause too:** methimazole for hyperthyroid cats; trilostane for Cushing's; renal diet for CKD.
- **Target BP:** systolic < 160 mmHg (ideally < 140 in most patients).

> **RED FLAG**
> Acute blindness with dilated pupils and a systolic > 180 mmHg — hypertensive retinal detachment. Emergency BP lowering + ophthalmology referral; some retinas re-attach if treated within 24–48 h.

> *Every animal defends MAP with the same layered system — baroreflex in seconds, RAAS and ADH in hours, the kidney over days. Break one, the others compensate; break them all, tissue perfusion fails.*
>
> — after Cunningham 6e Ch25 & PDA 3e Ch11.

KEY TERMS — QUICK GLOSSARY

MAP (mean arterial pressure)	The average pressure driving tissue perfusion over one cardiac cycle — MAP = CO × TPR. Roughly DBP + 1/3 (SBP−DBP) at physiological rates.
CO (cardiac output)	Volume of blood pumped per minute; CO = HR × SV. See the cardiac cycle article for the pump side.
TPR (total peripheral resistance)	The combined resistance of all systemic arterioles — set by arteriolar tone (mostly sympathetic + angiotensin II).
Baroreceptor	Stretch-sensitive nerve ending in the wall of the carotid sinus and aortic arch that fires more as vessel wall stretches with rising pressure.
Baroreflex	The reflex loop from baroreceptors → medulla → heart + vessels that stabilises BP on a second-to-second time-scale.
Carotid sinus	Slight dilation of the internal carotid artery at its origin; densest concentration of arterial baroreceptors.
Aortic arch	Second major baroreceptor site; afferents travel in the aortic depressor nerve (branch of CN X).
NTS (nucleus tractus solitarii)	Medullary integration centre for baroreceptor and chemoreceptor afferents; sends signals to the rostral ventrolateral medulla (RVLM) and nucleus ambiguus.
CN IX (glossopharyngeal)	Afferent nerve of the carotid sinus baroreceptors and carotid body chemoreceptors.
CN X (vagus)	Afferent nerve of aortic-arch baroreceptors and aortic-body chemoreceptors; also the efferent parasympathetic nerve to the heart.
Chemoreceptor	Carotid body + aortic body sensor of arterial O_2, CO_2 and pH; fires with low O_2, high CO_2, low pH.
CNS ischaemic response	Last-ditch reflex — medullary hypoperfusion at MAP < ~50 mmHg triggers massive sympathetic surge.
RAAS	Renin-angiotensin-aldosterone system: JG cells → renin → Ang I → ACE → Ang II → vasoconstriction + aldosterone + thirst + ADH.
Renin	Enzyme released from renal juxtaglomerular (JG) cells; cleaves angiotensinogen to angiotensin I.

JG cells (juxtaglomerular cells)	Modified smooth-muscle cells in the wall of the afferent arteriole; sense renal perfusion + sympathetic drive.
Macula densa	Distal tubular cells that sense NaCl load; signal JG cells to release renin when NaCl is low.
Angiotensinogen	Liver-made plasma precursor cleaved by renin to angiotensin I.
ACE (angiotensin-converting enzyme)	Enzyme on the surface of pulmonary vascular endothelium; converts Ang I to Ang II. Blocked by ACE inhibitors (enalapril, benazepril).
Angiotensin II	Potent vasoconstrictor (40× noradrenaline); releases aldosterone; stimulates thirst; releases ADH. Acts via AT_1 receptors.
Aldosterone	Mineralocorticoid released from the adrenal zona glomerulosa in response to Ang II; drives Na^+/water retention at the distal tubule + collecting duct.
ADH (vasopressin)	Posterior-pituitary hormone released in response to plasma osmolality + atrial low-pressure receptors. V2 = renal water reabsorption; V1 = vasoconstriction.
V1 receptor	Vascular receptor for ADH; high-dose vasoconstriction.
V2 receptor	Renal collecting-duct receptor for ADH; inserts aquaporin-2 water channels → water reabsorption.
Aquaporin-2	Water channel inserted into the apical membrane of collecting-duct principal cells under ADH/V2 signalling.
Pressure natriuresis	Guyton's principle: as arterial pressure rises at the kidney, Na+/water excretion rises — the long-term BP set-point mechanism.
Hypertension in cats	Systolic > 160 mmHg borderline, > 180 mmHg hypertensive (ACVIM 2018); confirm on 2–3 visits before diagnosing.
Hypertension in dogs	Systolic > 160 mmHg (with end-organ damage) or > 180 mmHg; almost always secondary in dogs.
End-organ damage	Retinal detachment, encephalopathy, LVH, proteinuria — the reason hypertension is treated even when the animal feels well.

QUICK REVISION — REMEMBER THESE

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. 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.

MEMORY AIDS

BR-AC-CS — the layers of BP control (fast to slow) — **B**aroreflex (seconds) · **R**AAS (minutes-hours) · **A**DH (minutes-hours) · **C**hemoreceptor + **C**NS ischaemic (emergencies) · **S**odium/pressure natriuresis (days — the set-point).

A²VAT — the four actions of angiotensin II — **V**asoconstrict · **A**ldosterone release · **T**hirst · (plus ADH release) — all raise BP.

COT — feline hypertension causes — **C**KD · **O**verthyroid (hyperthyroidism) · **T**umour (adrenal/phaeochromocytoma) — almost always secondary in cats.

MAP = CO x TPR — only two dials — Every BP change goes through **pump** (CO = HR x SV) or **pipe** (TPR = arteriolar tone). Any drug or reflex touches one, the other, or both.

TEST YOURSELF — ACTIVE RECALL

Cover the answers and try to retrieve each one from memory first — self-testing beats re-reading.

1. **Write the equation for mean arterial pressure and explain each term.**
2. **Name the receptors, afferent nerves, medullary centre and effector branches of the baroreceptor reflex.**
3. **Walk the RAAS pathway from renin release to angiotensin II's four effects.**
4. **Where does ADH act — vasculature vs kidney — and via which receptors?**
5. **What is pressure natriuresis and why is it the long-term BP controller?**
6. **In a cat with a repeatable systolic of 195 mmHg — what workup, what drug, what target BP?**
7. **Why is angiotensin II approximately 40× more potent than noradrenaline as a vasoconstrictor?**
8. **What are the ACVIM 2018 thresholds for hypertension in cats, and what end-organ damage should you look for?**

ANSWERS

1. **MAP = CO × TPR**. MAP is the average arterial pressure over one cardiac cycle. **Cardiac output (CO)** is the volume of blood the heart pumps per minute (CO = HR × SV). **Total peripheral resistance (TPR)** is the combined resistance of all systemic arterioles, mostly under sympathetic and angiotensin-II control. Move either CO or TPR and MAP moves with it.
2. **Receptors:** stretch-sensitive baroreceptors in the *carotid sinus* (slight dilation of internal carotid) and *aortic arch*. **Afferents:** CN IX (glossopharyngeal, carotid sinus) and CN X (vagus, via aortic depressor nerve for aortic arch). **Medullary centre:** nucleus tractus solitarii (NTS), which signals the rostral ventrolateral medulla (sympathetic output) and nucleus ambiguus (parasympathetic output). **Effectors:** sympathetic to heart (SA node, contractility, veins) and to arterioles (vasoconstriction); parasympathetic via vagus to SA node (bradycardia).
3. **Trigger:** renal JG cells sense ↓ renal perfusion, ↓ NaCl at macula densa, or sympathetic drive. **Release renin**, which cleaves liver-made **angiotensinogen** → **angiotensin I** (inactive). **ACE** on pulmonary endothelium converts Ang I → **angiotensin II**. Angiotensin II does four things: (1) direct vasoconstriction (40× noradrenaline), (2) release of **aldosterone** from adrenal zona glomerulosa → Na^+/water retention at distal tubule + collecting duct, (3) stimulation of thirst, (4) release of ADH from posterior pituitary. All four raise BP.

4. ADH (vasopressin) is released from the posterior pituitary. On **V1 vascular receptors** it causes vasoconstriction (chiefly at high circulating doses, e.g. severe haemorrhage). On **V2 renal receptors** in the collecting-duct principal cells, it inserts aquaporin-2 water channels into the apical membrane, increasing water reabsorption from the tubular fluid down its osmotic gradient. V2 action is why a dehydrated animal makes concentrated urine.
5. **Pressure natriuresis** is the phenomenon (Guyton) that as arterial pressure at the renal artery rises, the kidney excretes more sodium and water — even without any reflex or hormone change. Higher BP → higher Na^+/water excretion → falling blood volume → falling BP. Because the effect operates over hours to days without habituating, it is the ultimate set-point mechanism: no other system can override a persistent renal set-point for long. This is why any process that damages the kidney's ability to excrete Na+ (CKD, hyperaldosteronism, primary renal disease) drives chronic hypertension.
6. **Workup:** confirm on 2–3 visits (cats often stress-spike). Then look for the cause — renal panel + urine protein:creatinine ratio (CKD), total T4 (hyperthyroidism), fundoscopic exam (retinal detachment as end-organ marker), thoracic imaging if cardiomegaly. Idiopathic feline hypertension is uncommon. **Drug:** first-line is **amlodipine** (a calcium-channel blocker; potent arteriolar vasodilator; drug of choice in cats). If the cat is also proteinuric or in CKD, add **telmisartan** (angiotensin receptor blocker). Address the underlying disease (methimazole for hyperthyroidism, renal diet + ACE inh for CKD). **Target:** systolic < 160 mmHg per the ACVIM 2018 consensus.
7. Angiotensin II acts via AT_1 receptors on vascular smooth muscle that are highly potent, long-lived and cover the whole systemic arteriolar bed. Its half-life in plasma (though short) is longer than noradrenaline's, and its integrated effect includes not just direct vasoconstriction but also sympathetic potentiation (Ang II augments noradrenaline release from sympathetic terminals). Cunningham (Ch25 p278) uses the '40×' comparison specifically for the peptide's per-mole potency at the arteriole — a fact worth memorising because it explains why ACE inhibitors and ARBs work so well as antihypertensives.
8. The ACVIM 2018 consensus categorises cats by systolic BP as **< 140 normotensive**, **140–159 prehypertensive**, **160–179 borderline hypertensive**, and **≥ 180 severely hypertensive**. Treatment is generally indicated at persistent systolic ≥ 160 with any end-organ damage, or at ≥ 180 regardless. End-organ damage: *eye* (retinal detachment, retinal haemorrhage → sudden blindness), *brain* (encephalopathy — seizures, altered mentation), *heart* (LVH), *kidney* (proteinuria, accelerated CKD).

WHEN TO REFER OR ESCALATE

- 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.

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