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The Cardiac Cycle

How the Heart Fills, Contracts and Pumps Blood

The cardiac cycle in animals is the repeating sequence of one heartbeat: the ventricles contract (systole) to eject blood, then relax (diastole) to refill.

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IN SHORT

The **cardiac cycle in animals** is the repeating sequence of one heartbeat: the ventricles **contract (systole)** to eject blood, then **relax (diastole)** to refill. It runs in four phases — **isovolumetric contraction → ejection → isovolumetric relaxation → filling** — and the whole thing is driven by **pressure gradients** that open and close the valves. Those valve closures are what you hear on auscultation: **S1 ("lub")** as the AV valves shut at the start of systole and **S2 ("dub")** as the aortic and pulmonic valves shut at its end. Master the pressures, volumes and sounds of one cycle and cardiac output, murmurs and heart failure all fall into place.

KEY POINTS

1. The **cardiac cycle in animals** is one heartbeat split into **systole** (the ventricles contract and eject) and **diastole** (the ventricles relax and fill) — and unqualified, "systole" and "diastole" always refer to the ventricles.
2. It runs as **four phases**: **isovolumetric contraction** (all valves shut, pressure rises, volume fixed) → **ejection** → **isovolumetric relaxation** (all valves shut, pressure falls) → **filling**.
3. Every valve movement is driven by a **pressure gradient**: the **AV valves** (mitral, tricuspid) shut when ventricular pressure rises above atrial; the **semilunar valves** (aortic, pulmonic) open when ventricular pressure rises above arterial.
4. The two heart sounds are valve closures: **S1 ("lub")** is AV-valve closure at the *start* of systole, and **S2 ("dub")** is semilunar-valve closure at its *end* — so ventricular systole is the interval between S1 and S2.
5. In a resting dog each ventricle holds ~**60 mL at end-diastole (EDV)**, ejects ~**30 mL (stroke volume)** and keeps ~**30 mL (ESV)** — an **ejection fraction of ~50–65%**; the left ventricle peaks at ~120 mmHg, the right at only ~20.
6. **Cardiac output = heart rate × stroke volume** (a resting dog: ~80 bpm × ~30 mL ≈ 2.4 L/min), and it is tuned three ways — **preload** (Frank–Starling), **contractility** and **afterload**.
7. Most ventricular filling (**~80%**) happens passively *before* the atria contract, so the atrial "kick" adds only the final ~20% — which is why an animal in **atrial fibrillation** can still pump a near-normal stroke volume at rest.
8. **Species point:** the third and fourth heart sounds (**S3, S4**) can be entirely normal in horses and cattle, but in a dog or cat the same sounds are abnormal — a **gallop rhythm** that flags heart disease.

01 Why the cardiac cycle matters in veterinary medicine

Every time you rest a stethoscope on an animal's chest you are listening to the **cardiac cycle in animals** — the repeating, beautifully choreographed sequence of one heartbeat. The heart is a two-stage pump, and one turn of that pump is all the cardiac cycle is: the ventricles **contract** to push blood out to the lungs and body (this is **systole**), then **relax** and refill ready for the next beat (this is **diastole**). Understand what happens to the pressures, the volumes and the valves during a single cycle and an enormous amount of clinical cardiology — the heart sounds, the murmurs, cardiac output, the changes of heart failure — suddenly makes sense.

The elegant part is that the whole performance is driven by one simple rule: blood flows down pressure gradients, and each valve opens or shuts the instant the pressure across it reverses. There is no separate nervous command telling the valves what to do. Get that idea firmly in mind and the cycle almost predicts itself. This guide walks through it from the ground up — systole and diastole, the four phases, the valve events, the pressure and volume curves, the heart sounds, murmurs, and how it all rolls up into cardiac output — with the numbers for a resting dog and the ways the cycle differs across species. It is the natural sequel to how the heart is triggered (the cardiac action potential) and how that electrical trigger is read (the ECG): this is the mechanical beat those electrical events set in motion.

02 Systole and diastole: the two halves of every heartbeat

Split one heartbeat in two and you have the whole framework. **Systole** is ventricular contraction: the ventricular muscle shortens, pressure inside the chamber shoots up, and blood is forced out through the semilunar valves into the aorta and pulmonary artery. **Diastole** is ventricular relaxation: pressure falls, the AV valves open, and blood pours back in from the atria to refill the chambers before the next beat. A crucial convention: when a clinician says "systole" or "diastole" without qualification, they mean the *ventricles*. The atria have their own, gentler cycle — the atria contract right at the end of ventricular diastole, giving the nearly full ventricle a final top-up.

The two halves are not equal in length, and the split matters clinically. In a resting dog one cardiac cycle lasts about **0.75 second**; in a resting cow, whose heart beats around 60 times a minute, the cycle lasts about **1 second**, of which systole takes roughly **0.35 s** and diastole the remaining **0.65 s**. Diastole is normally the longer phase — and that is a good thing, because filling and the heart's own blood supply both happen mainly in diastole. When heart rate climbs, diastole is shortened far more than systole, which is exactly why a very fast heart can paradoxically fill and pump less well.

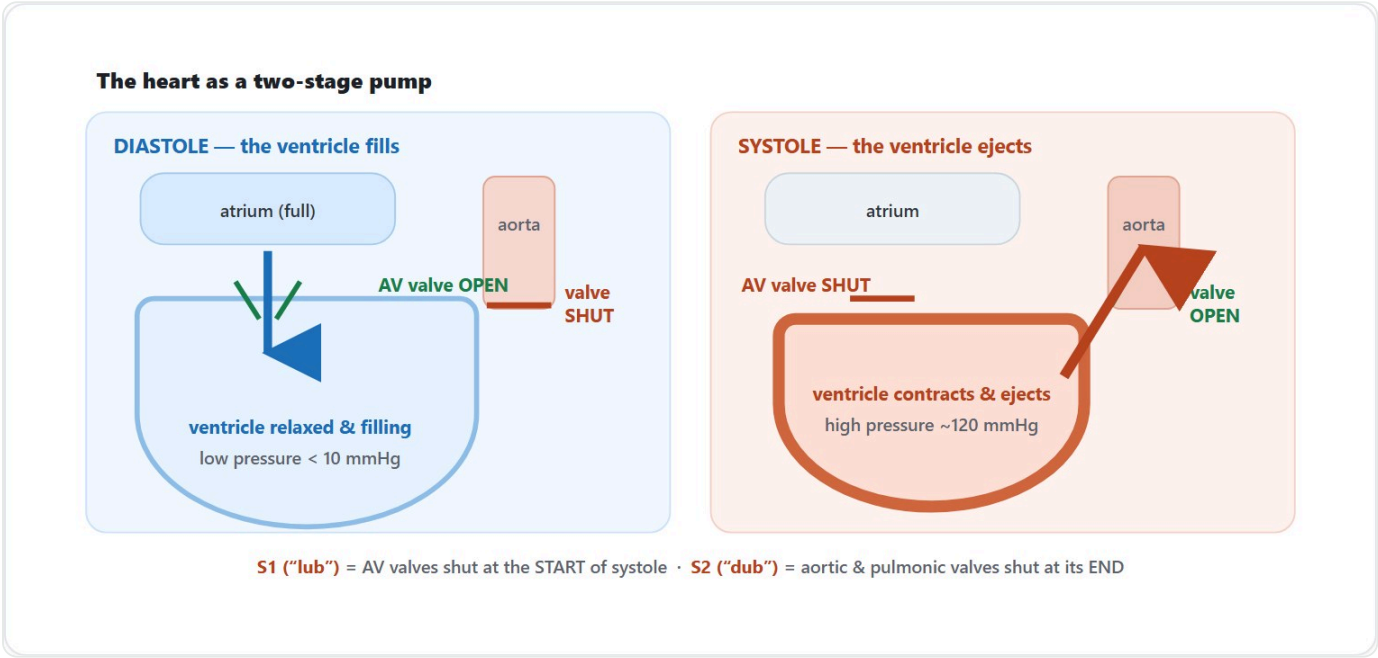


Fig 1 — The heart as a two-stage pump. In diastole the ventricle is relaxed and full, the AV valve open and the aortic valve shut; in systole the ventricle contracts, the AV valve shuts (S1) and the aortic valve opens to eject blood, closing again at end-systole (S2).

03 The four phases of the cardiac cycle

Zoom in and each heartbeat resolves into **four phases**, two in systole and two in diastole. They always run in the same order, and the trick to remembering them is that twice during every cycle *all four valves are momentarily shut*, trapping the blood so the ventricle changes pressure without changing volume — these are the "isovolumetric" phases.

1. **Isovolumetric contraction.** Systole begins. The ventricle starts to contract and its pressure rises almost instantly above atrial pressure, snapping the AV valves shut. But pressure is not yet high enough to open the aortic valve, so for a brief moment *every valve is closed* and the trapped blood volume cannot change — the ventricle simply builds pressure, like tensing against a locked door.
2. **Ejection.** The instant ventricular pressure exceeds the pressure in the aorta (and pulmonary artery), the semilunar valves are pushed open and blood is ejected — a burst of **rapid ejection** as pressure peaks, then **reduced ejection** as the ventricle passes its peak. During reduced ejection the ventricular pressure actually dips slightly *below* aortic pressure, yet blood keeps flowing outward under its own **momentum** — which is why the aortic valve does not slam shut the instant the two pressures cross.

3. **Isovolumetric relaxation.** At the end of ejection the falling ventricular pressure lets aortic pressure win, and a brief backflow snaps the aortic valve shut — the event that marks the end of systole (and makes the second heart sound). The ventricle then relaxes with its pressure still above atrial pressure, so the AV valves stay shut too: once again *all four valves are closed* and pressure plummets with no change in volume. (Some textbooks count this relaxation as the tail end of systole; here we follow the common convention that it opens diastole.)
4. **Ventricular filling.** When ventricular pressure finally falls below atrial pressure, the AV valves open and diastole's main event begins — classically in three roughly equal thirds: a **rapid-filling** first third as the blood that piled up in the atria rushes in, a quiet middle third (**diastasis**) where blood trickles straight through the atria from the veins, and a final third — the **atrial kick** — as the atria contract and top the ventricle up.

One fact from this sequence is worth committing to memory because it explains a great deal of clinical cardiology: **most ventricular filling — roughly 80% — happens passively, before the atria contract**. The atrial kick adds only the final fifth or so (textbooks cite anywhere from about 10% to 30%, depending on species and heart rate). That is why an animal in atrial fibrillation — where coordinated atrial contraction is lost — can still pump a nearly normal stroke volume at rest, and only runs into trouble during exercise: the fast heart rate leaves so little diastolic filling time that the missing atrial kick finally counts, and the animal shows exercise intolerance.

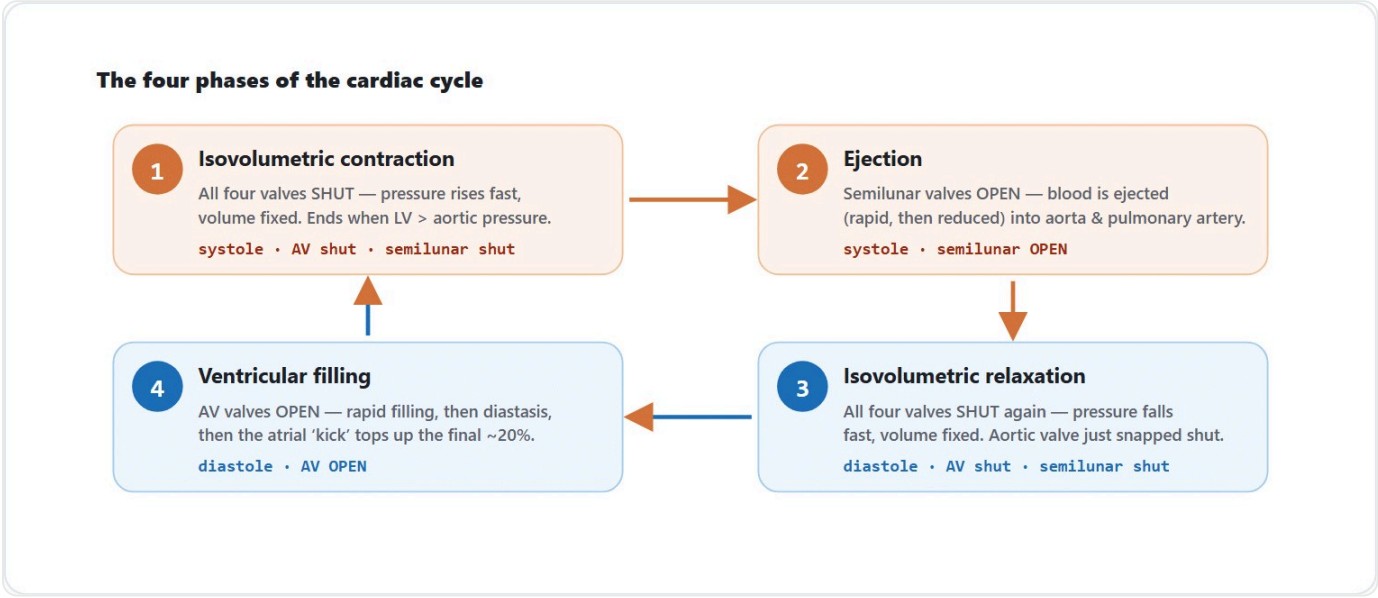


Fig 2 — The four phases as a loop: isovolumetric contraction (all valves shut, pressure rising) → ejection (semilunar valves open) → isovolumetric relaxation (all valves shut, pressure falling) → ventricular filling (AV valves open), then back to the start.

04 The heart valves: when they open, when they close, and why

Four one-way valves keep blood moving forwards, and understanding them is simply understanding the pressure gradients that work them. The two **atrioventricular (AV) valves** guard the entrances to the ventricles: the **mitral (bicuspid) valve** on the left and the **tricuspid valve** on the right. The two **semilunar valves** guard the exits: the **aortic valve** on the left and the **pulmonic valve** on the right. No valve has any muscle of its own; each is a passive flap that is pushed open or slammed shut purely by the blood pressure on its two sides.

So the rule is mechanical and absolute. The **AV valves shut** the moment the contracting ventricle's pressure rises above the atrium's (the start of systole), and they **open** again when the relaxing ventricle's pressure falls below the atrium's (the start of filling). The **semilunar valves open** when the ventricle's pressure rises above the artery's (the start of ejection), and they **shut** when arterial pressure exceeds the falling ventricular pressure at the end of systole. That last closure produces a signature you can see on a pressure trace: as the aortic valve snaps shut, the column of blood recoiling against it is abruptly arrested, and its momentum bounces back as a small blip in aortic pressure — the **dicrotic notch** (incisura). Right-sided events mirror the left exactly, with one difference: the pressures are far lower, because the right ventricle pumps into the low-resistance lungs.

05 Pressures through the cycle: the Wiggers diagram

Lay the pressure curves of one cycle on a single time axis and you get the classic **Wiggers diagram** — the single most useful picture in cardiac physiology, because every valve event and every heart sound can be read straight off the pressure crossings. Track three curves: **aortic pressure**, **ventricular pressure** and **atrial pressure**. During filling, the relaxed ventricle sits at a low pressure of under **10 mmHg** — about **5 mmHg** in the left ventricle and **3** in the right — essentially equal to atrial pressure because the AV valve is wide open. (That end-diastolic

pressure *is* the ventricle's **preload**.) Then systole fires: ventricular pressure rockets up, crosses atrial pressure (AV valve shuts), and shortly after crosses aortic pressure to fling the aortic valve open.

In a dog's **left ventricle**, pressure peaks at about **120 mmHg** during ejection; on the **right**, the ventricle peaks at only about **20 mmHg**, which is why the same cycle is drawn on two very different pressure scales. Aortic pressure, meanwhile, rises with the ventricle during ejection to its systolic peak, then falls back — but never below about **80 mmHg**, because the closed aortic valve holds the arterial column back and the elastic aorta keeps squeezing blood onward through diastole. That maintained diastolic pressure is what perfuses the coronary arteries and the rest of the body between beats. The four labelled crossings — AV valve closes (A), aortic valve opens (B), aortic valve closes (C), AV valve opens (D) — are the skeleton of the entire cycle.

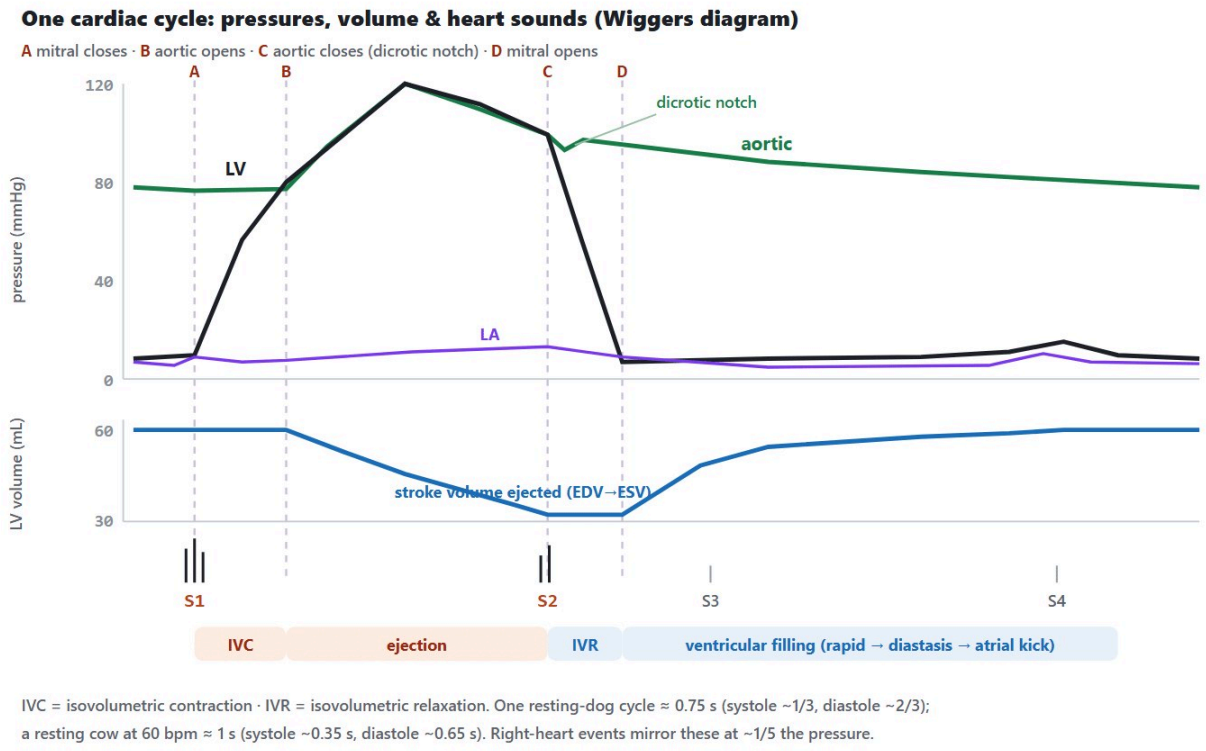


Fig 3 — A Wiggers diagram of one cardiac cycle: aortic, left-ventricular and left-atrial pressures on top, ventricular volume below, and the heart sounds at the bottom, all aligned in time. A/B/C/D mark the four valve events; the dicrotic notch marks aortic-valve closure.

06 Volumes: EDV, ESV, stroke volume and ejection fraction

Now follow the **volume** of blood in the ventricle instead of the pressure, and four numbers tell the whole story. At the end of filling, just before it contracts, each ventricle of a resting dog holds about **60 mL** — the **end-diastolic volume (EDV)**. During ejection it pushes out about half of that, and roughly **30 mL** is left behind — the **end-systolic volume (ESV)**. The blood actually ejected on that beat is the difference:

> **Stroke volume (SV) = EDV − ESV**
>
> — about 60 − 30 = **30 mL** in a resting dog. The **ejection fraction (EF) = SV ÷ EDV**, here about **50%** — these are rounded teaching figures; a healthy resting dog runs **50–65%**. A falling ejection fraction is a classic marker of failing ventricular contractility.

These volumes are not fixed — they are exactly the levers the body pulls to change how much blood the heart pumps. Fill the ventricle more (raise EDV) and it ejects more; make it empty more completely (lower ESV) and it also ejects more. During vigorous exercise it is mostly the second lever that does the work: a dog's **EDV stays about the same or even dips slightly (~60→55 mL)** while **ESV falls sharply (~30→15 mL)**, so stroke volume climbs to ~40 mL and the ejection fraction rises from ~50% to over 70%. Combined with a racing heart rate, that lifts cardiac output roughly four-fold — from ~2.4 to ~9.6 L/min in a large dog. The extra output comes mainly from emptying more completely, not from filling more.

How readily a ventricle fills is its **compliance** (the volume it gains per unit of filling pressure). A healthy ventricle is very compliant up to its normal EDV, then stiffens sharply: its walls, held in check by the surrounding fibrous **pericardium**, reach an elastic limit around **90 mL**, beyond which extra filling pressure buys almost no extra volume — a built-in guard against overstretch. This becomes critical in disease: a **stiff, non-compliant ventricle** needs an abnormally high filling pressure to reach even a normal EDV, and that back-pressure floods the circulation upstream. A stiff *left* ventricle raises left-atrial and pulmonary pressures and drives **pulmonary oedema**; a stiff *right* ventricle backs up into the body as **systemic (peripheral) oedema**. It is the mechanism behind much of what we call diastolic heart failure.

The numbers for a resting dog, and why the right side is drawn on a different scale from the left.

QUANTITY	LEFT VENTRICLE	RIGHT VENTRICLE	NOTE
Peak pressure in ejection	~120 mmHg	~20 mmHg	The right pumps into the low-resistance lungs, so the same cycle needs two pressure scales
Filling pressure	~5 mmHg	~3 mmHg	Essentially equal to atrial pressure, because the AV valve is wide open
End-diastolic volume (EDV)	~60 mL	~60 mL	What the ventricle holds just before it contracts
End-systolic volume (ESV)	~30 mL	~30 mL	What is left behind after ejection
Stroke volume (SV)	~30 mL	~30 mL	EDV − ESV. Both sides must eject the same volume or blood banks up
Cardiac output	~2.4 L/min	~2.4 L/min	Heart rate × stroke volume — about 80 bpm × 30 mL

07 The heart sounds: S1, S2, S3 and S4

Here is where the physiology reaches your ears. The heart sounds are not the muscle contracting — they are the **valves closing**, and the brief vibrations that closure sets up in the valve leaflets, the blood and the chamber walls. The **first heart sound, S1 ("lub")**, is the closure of the **AV valves** (mitral and tricuspid) at the very start of systole; it is relatively *low-pitched and long*. The **second heart sound, S2 ("dub")**, is the closure of the **semilunar valves** (aortic and pulmonic) at the end of systole; it is *briefer, sharper and higher-pitched*. Because S1 opens systole and S2 closes it, the interval between "lub" and "dub" *is* ventricular systole — a fact you can use at the bedside to time any extra sound or murmur. Sometimes the aortic and pulmonic valves close a fraction apart and you hear a **split S2**.

Two quieter sounds can appear in diastole, and here species matters enormously. The **third heart sound (S3)** comes from the turbulent rush of *rapid ventricular filling* in early diastole; the **fourth heart sound (S4)** comes from *atrial contraction* driving blood into the ventricle in late diastole. In large animals — **horses and cattle** — S3 and S4 are often perfectly **normal** findings. In **dogs and cats**, however, an audible S3 or S4 is **abnormal**: it is called a **gallop rhythm** and signals a dilated, stiff or failing ventricle. Never call a gallop without first asking what species you are listening to.

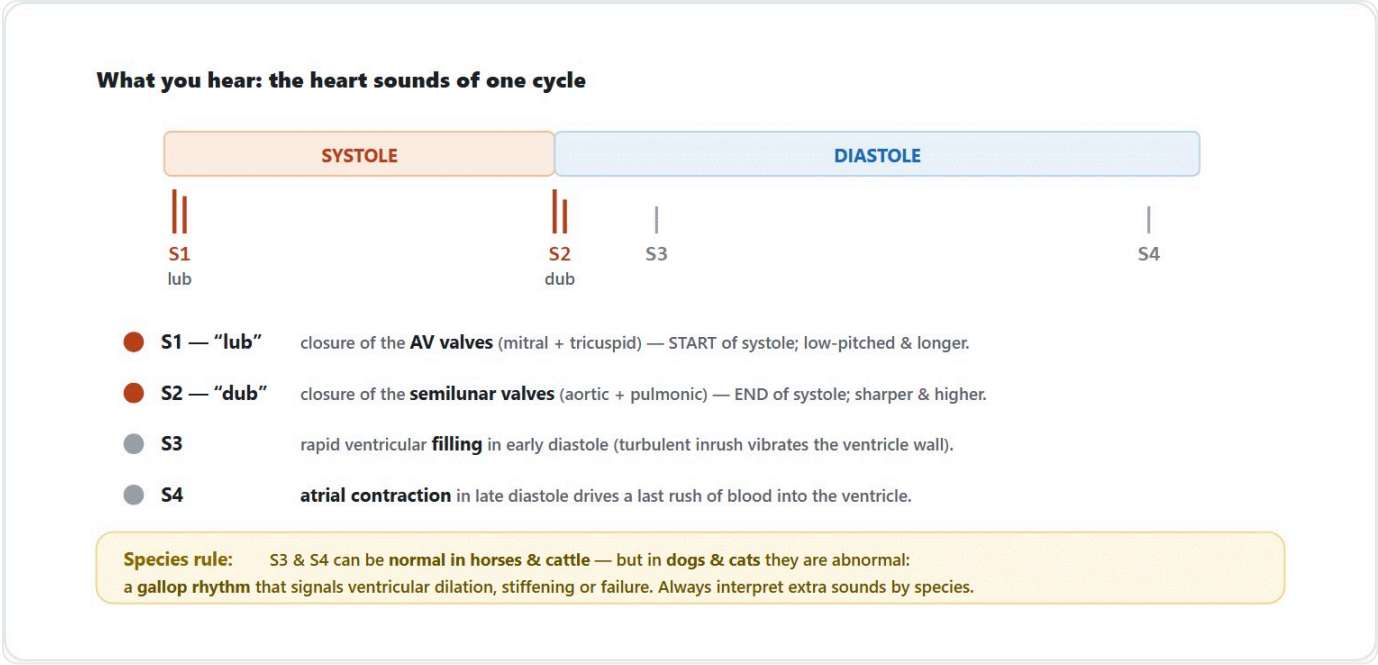


Fig 4 — The heart sounds of one cycle. S1 ("lub") is AV-valve closure at the start of systole; S2 ("dub") is semilunar-valve closure at its end. S3 (rapid filling) and S4 (atrial contraction) are normal in horses and cattle but a gallop rhythm in dogs and cats.

08 Murmurs: what turbulent flow tells you

Normal blood flow is smooth (laminar) and therefore silent. A **murmur** is the sound of **turbulent** flow, and turbulence appears whenever blood is forced at high velocity through an abnormal opening — a valve that will not open properly (**stenosis**), a valve that will not close properly and leaks backward (**insufficiency** or **regurgitation**), or a hole between chambers (a septal defect or a patent ductus arteriosus). Turbulence can even appear with entirely normal valves — in **severe anaemia (anemia)**, the thin, low-viscosity blood and the raised cardiac output together tip flow into turbulence (a functional or "flow" murmur). Because you now know the pressure timeline, you can place any murmur in the cycle and reason back to its cause.

The logic is clean. A valve that should be *open* during ejection but is narrowed makes a noise *during systole*: so **aortic or pulmonic stenosis** gives a **systolic murmur**. A valve that should be *shut* during systole but leaks backward also makes a systolic noise: so **mitral or tricuspid regurgitation** is **systolic** too. Flip it for diastole: a leaking aortic or pulmonic valve gives a **diastolic murmur**, and a stenotic mitral or tricuspid valve gives a diastolic murmur as the ventricle fills through the narrowed opening. A **patent ductus arteriosus** shunts continuously and gives the classic **continuous "machinery" murmur**, heard best over the left heart base and common in young female dogs. These patterns have real prevalence in practice: mitral regurgitation is present in roughly **8% of dogs over five years old** — the mitral valve is especially prone to age-related and degenerative disease, and in the **Cavalier King Charles Spaniel** it is almost a breed signature — while aortic regurgitation is common in horses but rare in dogs.

09 Stroke volume and cardiac output

Everything so far serves one purpose: delivering blood to the tissues. The measure of that delivery is **cardiac output (CO)** — the volume each ventricle pumps per minute — and it is the product of just two things:

> **Cardiac output = heart rate × stroke volume.**
>
> A resting dog: ~80 beats/min × ~30 mL ≈ **2.4 L/min**. A 500 kg horse runs around **30 L/min** at rest and can drive that **6–8× higher — up to ~250 L/min** — during hard exercise.

Heart rate is set by the autonomic nervous system acting on the pacemaker. Stroke volume is tuned three ways, and they are worth knowing by name. **Preload** is the degree of end-diastolic stretch (essentially the EDV); by the **Frank–Starling law of the heart**, stretching the ventricular fibres (fibers) more makes them contract more forcefully, so "more in" automatically gives "more out." This is what keeps the left and right ventricles matched — over successive beats, if one side pumps a little more, the other fills a little more and catches up. Preload itself depends on **venous return**, the rate at which blood flows back to the heart, which is raised by the **skeletal-muscle pump** (contracting limb muscles squeezing blood along the veins, one-way valves keeping it moving heart-ward), the **respiratory pump** (each inspiration

drawing blood toward the chest), **sympathetic venoconstriction** (the veins hold most of the blood volume and can be tightened to return more of it), and simply a larger blood volume — which together explain why exercise, with its working muscles and deep breathing, fills the heart so well.

Contractility is the intrinsic vigour of contraction, independent of stretch; sympathetic nerves and adrenaline raise it (via more calcium in each beat), emptying the ventricle more completely and lowering ESV — while many **anaesthetic (anesthetic) agents**, β-blockers and calcium-channel blockers *depress* it, a real consideration when anaesthetising a patient with a weak heart. **Afterload** is the arterial pressure the ventricle must overcome to eject; a sudden rise transiently reduces stroke volume (Frank–Starling soon compensates), and a sustained high afterload drives the ventricle to thicken. A useful rule here: **pressure work is harder for the heart than volume work** — pumping against a high pressure wastes far more energy as heat and provokes much greater hypertrophy than simply pumping a larger volume. One last caution: forcing the heart faster does *not* by itself keep raising output. If the rate is driven up *artificially* — by a pacemaker or a tachyarrhythmia — without the matching sympathetic changes, the diastolic filling time eventually becomes so short that EDV, stroke volume and even cardiac output fall. In genuine **exercise** this does not happen, because the same sympathetic surge that speeds the heart also shortens systole and boosts contractility, so a galloping dog can reach 240 beats/min with its output still climbing.

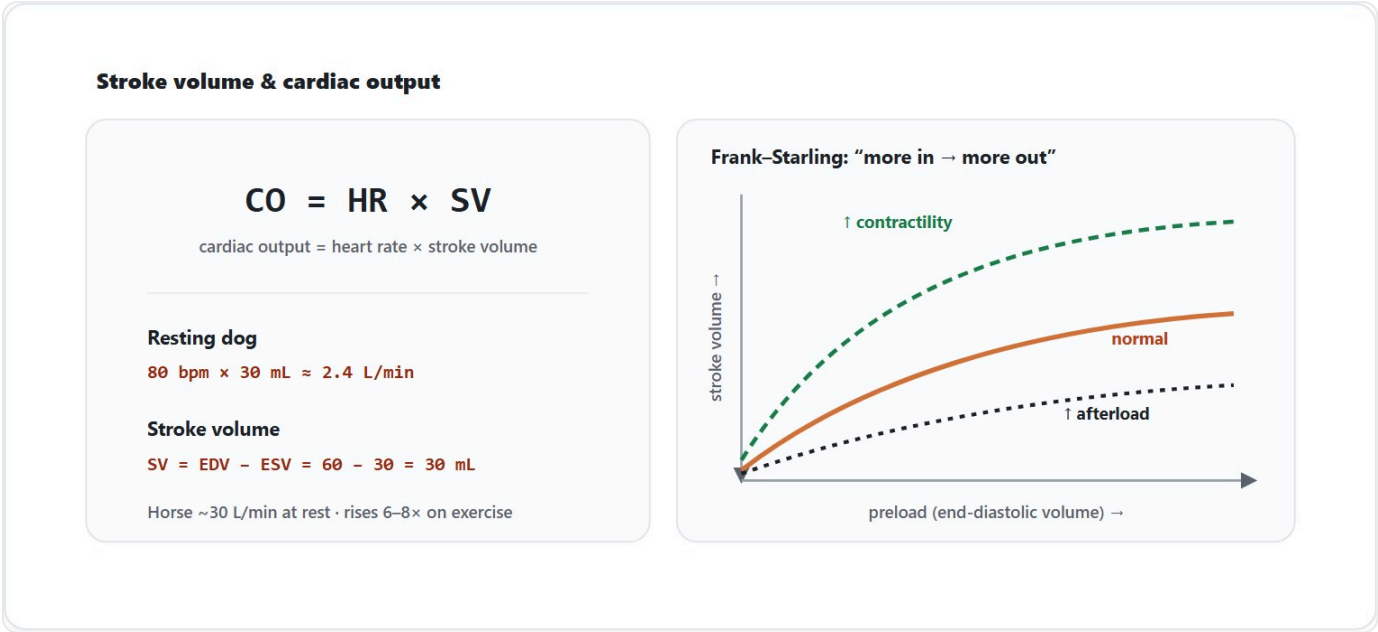


Fig 5 — Cardiac output = heart rate × stroke volume (a resting dog: 80 bpm × 30 mL ≈ 2.4 L/min). The Frank–Starling curves show stroke volume rising with preload; more contractility lifts the curve, more afterload lowers it.

10 Putting it together: the pressure–volume loop

The most complete single picture of the cardiac cycle plots ventricular **pressure against volume** for one beat, tracing a closed **pressure–volume loop** that you travel anticlockwise. Start at the bottom-right corner, at end-diastolic volume and low pressure, the moment the **mitral valve closes (A)**. Isovolumetric contraction runs straight *up* the right side — volume fixed, pressure soaring — until the **aortic valve opens (B)**. Ejection sweeps *leftward* across the top as blood leaves and volume falls, until the **aortic valve closes (C)** at end-systolic volume. Isovolumetric relaxation drops straight *down* the left side, until the **mitral valve opens (D)**, and filling carries you *rightward* along the bottom back to the start.

The loop is not just elegant — it is quantitative. Its **width** is the stroke volume (EDV minus ESV). Its enclosed **area** is the mechanical (stroke) work the ventricle does that beat. And you can see disease in its shape: a leaking mitral valve widens and distorts the loop, a stiff non-compliant ventricle shifts its filling limb upward, and falling contractility shrinks it. Every corner is a valve event you have already met, which is why the loop is the perfect capstone for the whole cycle.

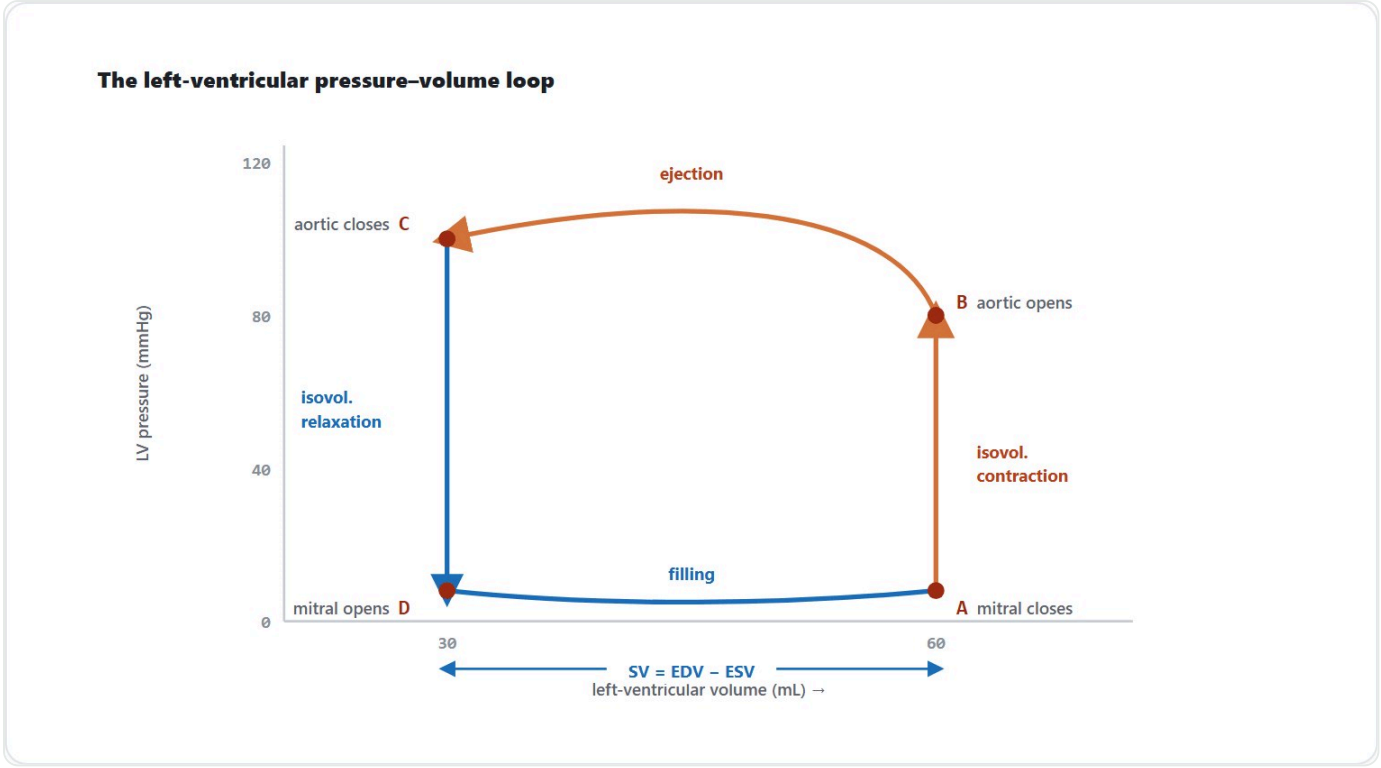


Fig 6 — The left-ventricular pressure–volume loop. A: mitral closes → up (isovolumetric contraction) → B: aortic opens → left (ejection) → C: aortic closes → down (isovolumetric relaxation) → D: mitral opens → right (filling). The loop's width is the stroke volume.

11 The venous side: the jugular pulse you can see

The Wiggers diagram carries an atrial pressure trace that is easy to skip over, and it is the only part of the cardiac cycle visible from across a consulting room. Right atrial pressure rises and falls in three waves, and they are transmitted straight up the jugular vein.

The **a wave** is atrial systole — the atrium contracting against a column of blood. The **c wave** is the tricuspid valve bulging back into the atrium as the ventricle begins isovolumetric contraction. The **v wave** is the atrium filling passively while the tricuspid valve is still shut. Between them the descents (x and y) mark atrial relaxation and then tricuspid opening.

Clinically, three findings are worth having. A **jugular pulse visible above the lower third of the neck** in a standing animal is abnormal, and points to the right side of the heart. **Giant a waves** ("cannon" waves) occur when the atrium contracts against a closed tricuspid valve — complete AV block, or ventricular ectopy — or against a stiff, hypertrophied right ventricle, as in pulmonic stenosis or pulmonary hypertension. **Large v waves with a persistent pulse** suggest tricuspid regurgitation, blood driven back into the atrium during ventricular systole. And **jugular distension without a pulse** is an obstruction rather than a cycle problem — a cranial mediastinal mass or a thrombus.

For vets Check the jugular before you reach for the stethoscope. Right-sided disease that produces ascites and a jugular pulse is easy to miss on auscultation alone, because tricuspid regurgitation is often quiet — a soft right-sided systolic murmur, or none at all.

12 Species differences and the auscultation payoff

The cardiac cycle is the same machine in every mammal, but its tempo and some of its sounds vary in ways that matter at the bedside. **Cycle length tracks heart rate**, and resting heart rates span a huge range: roughly **70–140 beats/min in dogs**, **140–220 in cats**, a slow **28–44 in horses** and **60–80 in cattle** (with tiny mammals faster still — hundreds of beats a minute). The larger and more athletic the animal, the slower and more powerful the resting beat, and the longer each cycle. Remember, too, that as rate rises it is *diastole* that is sacrificed first, so the fast-hearted cat or the exercising dog has proportionally far less filling and coronary-perfusion time than its resting heart rate suggests.

The clinical payoff of knowing the cycle this well is confident **auscultation**. Because S1 opens systole and S2 closes it, you can instantly time any murmur or extra sound against the pulse and reason toward its cause — a systolic murmur at the left apex in an older small-breed dog, for instance, points straight at mitral regurgitation. You will interpret **S3 and S4** correctly by species, calling a gallop in a cat but not in a cow. And you will read the pulse itself as an echo of the pressure curve, feeling the sharp upstroke of ejection and the dicrotic character the aortic valve leaves behind. Master one cardiac cycle and you have the framework for the whole of clinical cardiology.

13 Quiz: test yourself

Nineteen questions across systole and diastole, the four phases, the valve events, the pressures and volumes, the heart sounds and cardiac output. Answers are beneath each question — cover them and work down.

1. What is systole, and what is diastole?**Answer: Systole** — the ventricles contract and push blood to the lungs and body. **Diastole** — they relax and refill ready for the next beat.2. How long is one cardiac cycle in a resting dog, and in a resting cow?**Answer:** About **0.75 s** in a dog. In a cow at about 60 bpm the cycle lasts about **1 s** — roughly **0.35 s systole** and **0.65 s diastole**.3. Which phase is longer, and why is that a good thing?**Answer: Diastole.** Both **filling** and the heart's **own blood supply** happen mainly in diastole.4. Why can a very fast heart pump less well? **Answer:** Because when rate climbs **diastole is shortened far more than systole**, so there is less time to fill and less time to perfuse the myocardium.5. What is an isovolumetric phase, and how many are there?**Answer:** A phase in which **all four valves are shut**, so the ventricle changes pressure without changing volume. There are **two** in every cycle — one in systole, one in diastole.6. What proportion of ventricular filling is passive?**Answer:** Roughly **80%** — it happens **before the atria contract**. That single fact explains a great deal of clinical cardiology.7. What is the dicrotic notch?**Answer:** As the **aortic valve snaps shut**, the recoiling column of blood is abruptly arrested and its momentum bounces back as a small blip in aortic pressure.8. How do right-sided events differ from left?**Answer:** They **mirror them exactly**, with one difference: the **pressures are far lower**, because the right ventricle pumps into the low-resistance lungs.9. Why does aortic pressure never fall below about 80 mmHg?**Answer:** The **closed aortic valve** holds the arterial column back and the **elastic aorta** keeps squeezing blood onward through diastole.10. What are EDV, ESV and stroke volume in a resting dog?**Answer: EDV ~60 mL**, **ESV ~30 mL**, so **SV = EDV − ESV ≈ 30 mL**.11. What is S1, and what makes it?**Answer:** The first heart sound, "lub" — closure of the **AV valves** (mitral and tricuspid) at the very start of systole. Relatively **low-pitched and long**.12. What is S2, and how does it differ from S1?**Answer:** "Dub" — closure of the **semilunar valves** (aortic and pulmonic) at the end of systole. **Briefer, sharper and higher-pitched**.13. What produces a murmur?**Answer: Turbulent flow** — blood moving through a narrowed or leaking orifice, or at abnormal velocity.14. What is the cardiac output equation, and what does it give for a resting dog?**Answer: CO = heart rate × stroke volume**. About **80 bpm × 30 mL ≈ 2.4 L/min**.15. Why is the heart described as a two-stage pump?**Answer:** Because the same cycle drives **two circuits in series** — the right ventricle to the lungs, the left to the body — and both must eject the same stroke volume, at very different pressures.16. What peak pressures does each ventricle reach in a dog?**Answer:** The left peaks at about **120 mmHg** during ejection; the right at only about **20 mmHg**, which is why the two sides are drawn on different pressure scales.17. Trace the four corners of the pressure–volume loop. Which way do you travel?**Answer: Anticlockwise.** From end-diastolic volume at low pressure as the mitral valve closes (A), straight up in **isovolumetric contraction** to aortic valve opening (B), leftward across the top through **ejection** to aortic closure at end-systolic volume (C), then straight down in **isovolumetric relaxation** to mitral opening.18. What are the three waves of the jugular pulse, and what makes each?**Answer:** The **a wave** is atrial systole. The **c wave** is the tricuspid valve bulging back into the atrium as isovolumetric contraction begins. The **v wave** is the atrium filling passively while the tricuspid is still shut. It is the only part of the cardiac cycle visible from across a consulting room.19. How does resting heart rate vary across species, and what follows for filling?**Answer:** Roughly **70–140** bpm in dogs, **140–220** in cats, **28–44** in horses and **60–80** in cattle. The larger and more athletic the animal, the slower and more powerful the beat — and because **diastole is sacrificed first** as rate rises, the fast-hearted cat has proportionally far less filling time.

14 The cardiac cycle — frequently asked questions

What are the phases of the cardiac cycle in order?

Isovolumetric contraction (all valves shut, pressure rising), ejection (semilunar valves open, blood leaves), isovolumetric relaxation (all valves shut, pressure falling), then ventricular filling (AV valves open) — which includes rapid filling, diastasis and the atrial kick. The first two are systole; the last two are diastole.

What is the difference between systole and diastole?

Systole is ventricular contraction, when the ventricles squeeze and eject blood; diastole is ventricular relaxation, when they refill. Unqualified, both terms refer to the ventricles. In a resting dog the whole cycle lasts about 0.75 second, with diastole normally the longer half.

What causes the first and second heart sounds?

S1 ("lub") is closure of the AV valves (mitral and tricuspid) at the start of systole; S2 ("dub") is closure of the semilunar valves (aortic and pulmonic) at the end of systole. The interval between them is ventricular systole. S3 and S4 are quieter diastolic sounds — normal in horses and cattle, but a gallop (abnormal) in dogs and cats.

How do you calculate cardiac output?

Cardiac output = heart rate × stroke volume, where stroke volume = end-diastolic volume − end-systolic volume. A resting dog: about 80 beats/min × ~30 mL ≈ 2.4 L/min. It is adjusted by changing heart rate, preload (Frank–Starling), contractility and afterload.

What causes a visible jugular pulse in a dog?

A jugular pulse visible above the lower third of the neck in a standing animal is abnormal and points to the right heart. Giant "cannon" a waves mean the atrium is contracting against a closed tricuspid valve (complete AV block, ventricular ectopy) or against a stiff right ventricle (pulmonic stenosis, pulmonary hypertension). Large v waves suggest tricuspid regurgitation. Distension *without* a pulse is obstruction — a cranial mediastinal mass or thrombus — rather than a problem with the cycle itself.

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When to refer

- A new **systolic murmur** at the left apex in an older small-breed dog (especially a Cavalier King Charles Spaniel) — degenerative **mitral regurgitation**; work up with echocardiography.
- A **gallop rhythm (S3 or S4)** in a dog or cat — abnormal in small animals; investigate for dilated or hypertrophic cardiomyopathy or failure.
- A **continuous 'machinery' murmur** over the left heart base in a young dog — likely a **patent ductus arteriosus**; refer for closure, ideally young.
- A loud (grade IV–VI) or **thrill**-associated murmur, or any murmur with weakness, exercise intolerance or fainting — characterise fully and refer.
- A resting horse with a **dropped beat** that disappears on exercise is usually normal (physiological second-degree AV block) — but a fast, irregular rhythm needs an ECG for atrial fibrillation.

Sources

1. Stephenson RB. The Heart As a Pump. In: Klein BG, ed. Cunningham's Textbook of Veterinary Physiology. 6th ed. St Louis: Elsevier; 2020. Chapter 21 (pp 216–229): the four phases of the cardiac cycle and the pressure-gradient control of the valves (points A–D); left- and right-ventricular pressures (~120 vs ~20 mmHg) and filling pressures (<10 mmHg); resting-dog EDV 60 mL, ESV 30 mL, stroke volume 30 mL, ejection fraction 50–65%; that 80–90% of filling precedes atrial systole; S1/S2 (and S3/S4) origins and the split S2; the murmur-timing table (stenosis vs regurgitation, systolic vs diastolic) with mitral regurgitation in ~8% of dogs >5 yr and continuous PDA murmurs; cardiac output = heart rate × stroke volume (resting dog HR 80, CO 2.4 L/min), preload/Frank-Starling, contractility and afterload; the pressure-volume loop and the ~160 bpm output ceiling.
2. Sjaastad ØV, Sand O, Hove K. The Pumping Function of the Heart. In: Physiology of Domestic Animals. 3rd ed. Oslo: Scandinavian Veterinary Press; 2016. Chapter 11 (pp 451–470): the cardiac cycle phases and the thirds of ventricular filling (atrial contraction ~20–30%); AV and semilunar valve events and the dicrotic notch as arrested backflow; the heart sounds S1 ('lub') and S2 ('dub'); cardiac output = HR × SV with species values (20 kg dog ~2 L/min, 500 kg horse ~30 L/min, rising to ~250 L/min in exercise); the Frank-Starling mechanism and venous-return determinants; resting cardiac-cycle duration in the cow (~1 s; systole 0.35 s, diastole 0.65 s) and the resting heart-rate table (horse 32–44, cattle 60–70, dog 70–120 bpm); murmurs from stenotic/leaky valves and the mitral-valve susceptibility of the Cavalier King Charles Spaniel.
3. Klein BG (ed.). Cunningham's Textbook of Veterinary Physiology, 6th ed. — Chapters 19–20 for the conduction system and electrocardiogram that time the mechanical cycle described here (see our companion articles on the cardiac action potential and ECG interpretation).

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