PIPADOR REVISION

MRCOG Part 1 Revision Notes

Cardiovascular Physiology

PHYSIOLOGY • LECTURE 125

VISUAL EXAMINATION REVISION NOTES

2026 Edition

MRCOG PART 1 REVISION NOTES | PHYSIOLOGY

125 Cardiovascular Physiology

Conduction, action potentials, the cardiac cycle, cardiac output, haemodynamics and blood pressure control

5 L/min	55–70%	MAP = CO × SVR	$R \propto 1/r^4$
Resting cardiac output (HR 70 × SV 70 mL)	Ejection fraction (SV ÷ EDV)	≈ DBP + ⅓ pulse pressure	Halve radius → 16 × resistance

125.1 Circulation and conduction

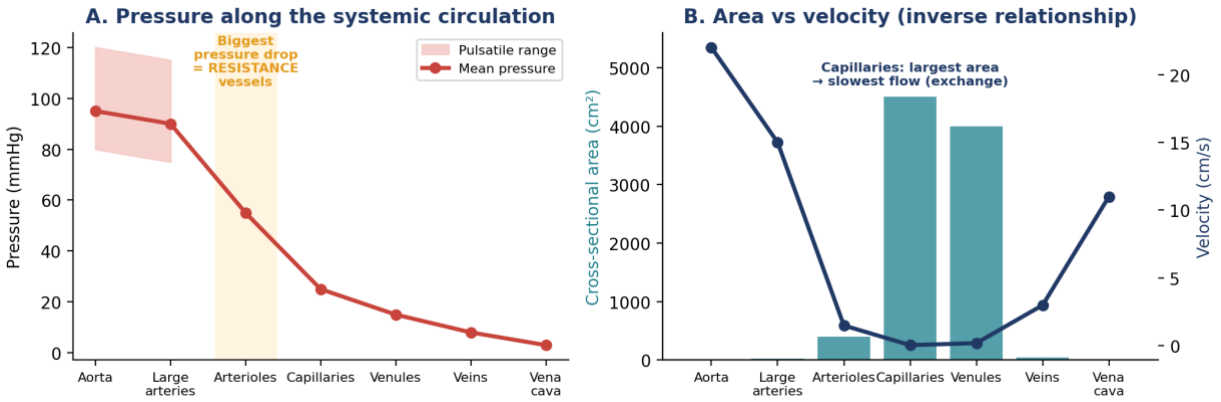


Figure 125.1 — Pressure falls most across the arterioles; capillaries have the largest area and slowest flow

Vessel	Role	Key fact
Elastic arteries	'Windkessel' recoil → continuous flow	Maintain diastolic pressure
Arterioles	Resistance vessels (α_1 sympathetic)	Main site of SVR and BP control
Capillaries	Exchange vessels	Largest cross-sectional area
Veins	Capacitance vessels	Hold ≈ 60–70% of blood volume
Pulmonary	Low resistance; hypoxic vasoconstriction	PA ≈ 25/10 mmHg

SA node 60–100/min ▶ Atria ▶ **AV node** delay ≈ 0.1 s (PR) ▶ Bundle of His → bundle branches ▶ **Purkinje** fastest conduction

Latent pacemakers: AV junction 40–60/min, ventricle 20–40/min (escape rhythm of complete heart block). The AV node has the **slowest conduction**, protecting the ventricles from fast atrial rates.

125.2 Action potentials and excitation–contraction coupling

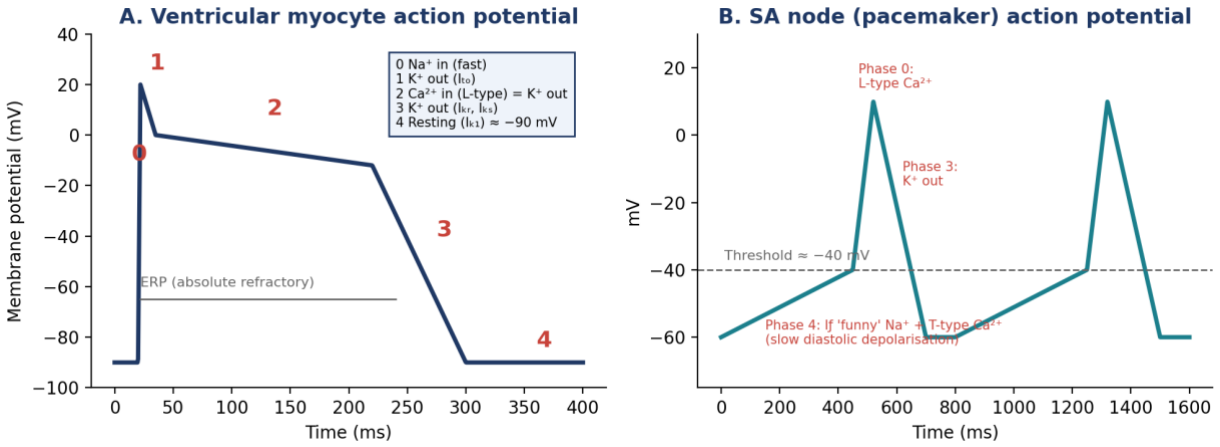


Figure 125.2 — Ventricular myocyte vs SA nodal action potentials

■ KEY FACTS

- **Phase 2 plateau** = L-type Ca^{2+} influx → long refractory period → **heart cannot be tetanised**.
- **Pacemaker phase 4** = funny current (If, Na^+) + T-type Ca^{2+}; upstroke = L-type Ca^{2+} (no fast Na^+).
- **Sympathetic (β_1)** steepens phase 4 → ↑ HR; **vagal (M_2)** hyperpolarises and flattens phase 4 → ↓ HR.
- **Hyperkalaemia** depolarises the resting membrane → wide QRS, asystole/VF.

T-tubule depolarisation ▶ L-type Ca^{2+} entry (**trigger**) ▶ Ca^{2+}-induced Ca^{2+} release via **RyR2** ▶ Ca^{2+} binds **troponin C** → cross-bridges ▶ Relaxation: **SERCA** (phospholamban) + Na^+/Ca^{2+} exchanger

125.3 The cardiac cycle

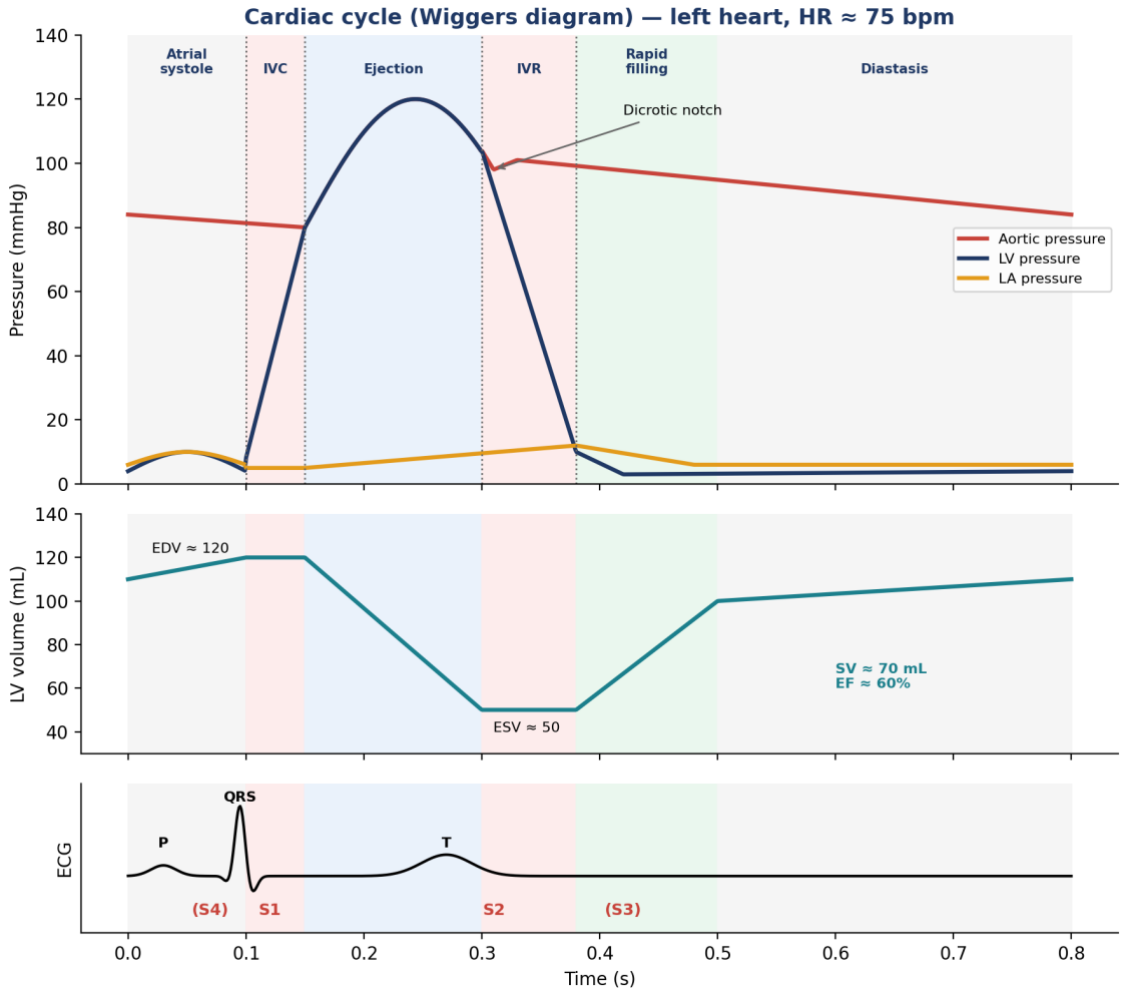


Figure 125.3 — Wiggers diagram: pressures, LV volume, ECG and heart sounds

Phase	Valves	Key events
Atrial systole	AV open	Tops up filling (≈ 15–20%); a wave; S4 if stiff ventricle; lost in AF
Isovolumetric contraction	All closed	Starts with S1 (mitral/tricuspid closure) at QRS; c wave
Ejection	Semilunar open	Opens when LV > aortic diastolic pressure; ≈ 70 mL ejected; x descent
Isovolumetric relaxation	All closed	Starts with S2 (dicrotic notch); v wave
Rapid filling → diastasis	AV open	Most filling (70–80%); y descent; S3 (normal in pregnancy). Diastole shortens most as HR rises

S1	S2	S3	S4
AV valves close (start of systole)	Aortic then pulmonary close split widens on inspiration	Rapid filling — normal in children, athletes, PREGNANCY	Atria → stiff ventricle ALWAYS pathological

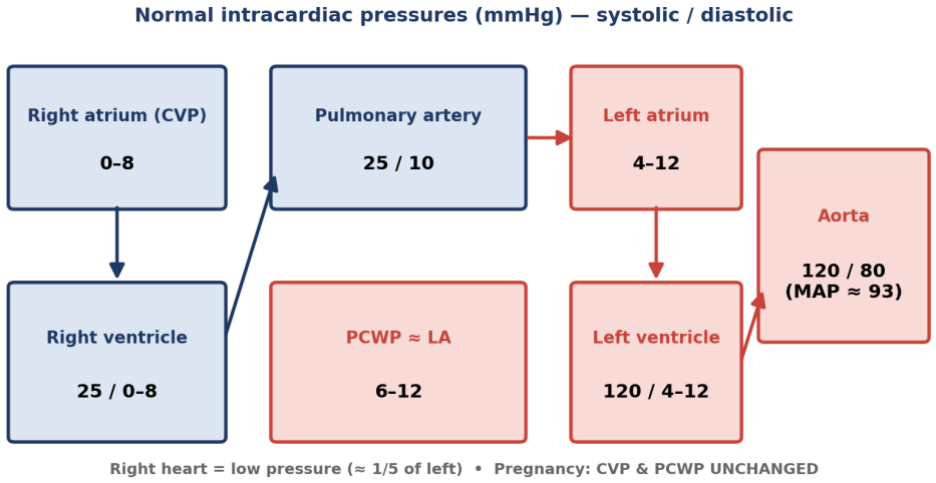


Figure 125.4 — Normal intracardiac pressures

125.4 Cardiac output and its determinants

■ KEY EQUATIONS

CO = HR × SV; **SV = EDV − ESV** (120 − 50 = 70 mL); **cardiac index** = CO ÷ BSA ≈ 2.5–4 L/min/m²

SVR = 80 × (MAP − CVP) ÷ CO ≈ 900–1300 dyn·s·cm^{-5}; **Fick**: CO = VO_2 ÷ (CaO_2 − CvO_2)

Laplace: wall stress = P × r ÷ 2h (dilated ventricle → ↑ O_2 demand); **Reynolds**: turbulence ↑ with ↑ velocity, ↓ viscosity (**anaemia, pregnancy** → flow murmurs)

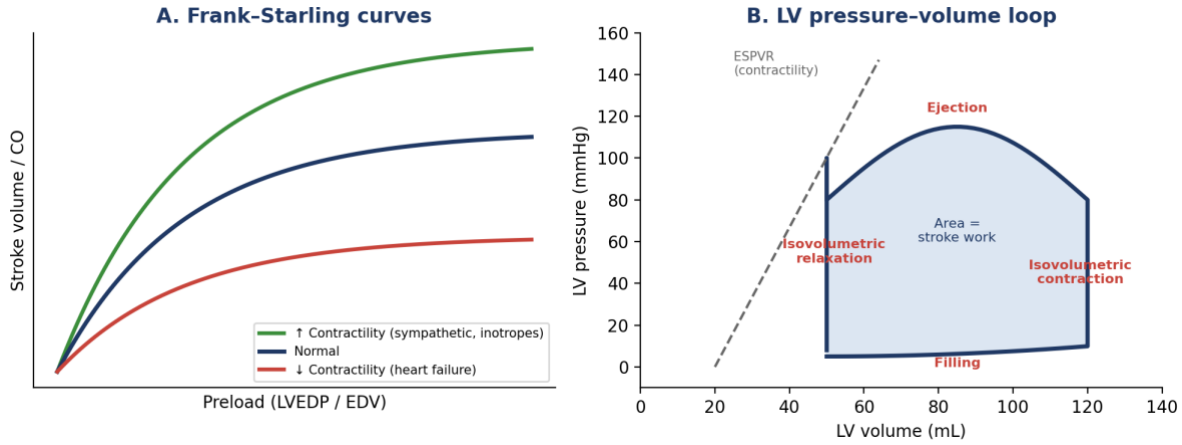


Figure 125.5 — Frank–Starling curves and the LV pressure–volume loop

Determinant	Meaning	↑ by	↓ by
Preload	End-diastolic stretch (EDV; CVP/PCWP)	↑ Venous return, volume, pregnancy	Haemorrhage, spinal block, aortocaval compression, Valsalva
Afterload	Wall stress during ejection (≈ SVR)	Hypertension, aortic stenosis	Vasodilators, pregnancy, sepsis
Contractility	Force independent of load	β_1, inotropes, digoxin	Hypoxia, acidosis, β-blockers, ↑ Mg^{2+}
Heart rate	Chronotropy	Sympathetic, pregnancy	Vagal tone; very high HR ↓ filling

Frank–Starling law: within limits, ↑ fibre length (EDV) → ↑ force of contraction (optimal sarcomere ≈ 2.2 µm; ↑ troponin Ca^{2+} sensitivity). It matches right and left outputs beat-by-beat.

125.5 Microcirculation and local control

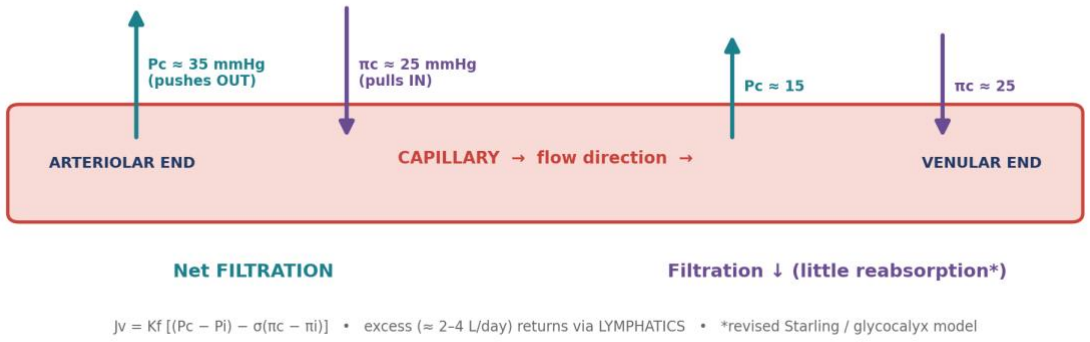


Figure 125.6 — Starling forces across the capillary

Oedema mechanism	Obstetric examples
↑ Hydrostatic pressure	Fluid overload, uterine compression of pelvic veins
↓ Oncotic pressure	Pregnancy (albumin ↓ ≈ 10 g/L), nephrotic syndrome
↑ Permeability	Pre-eclampsia, sepsis, TRALI
Lymphatic obstruction	Lymphadenectomy, radiotherapy

Myogenic	Metabolic	NO / PGI_2	ET-1 / TXA_2
Stretch → constriction (brain, kidney)	↑CO_2, H^+, K^+, adenosine → dilatation	Dilators (↓ in pre-eclampsia)	Constrictors (↑ in pre-eclampsia)

125.6 Blood pressure regulation

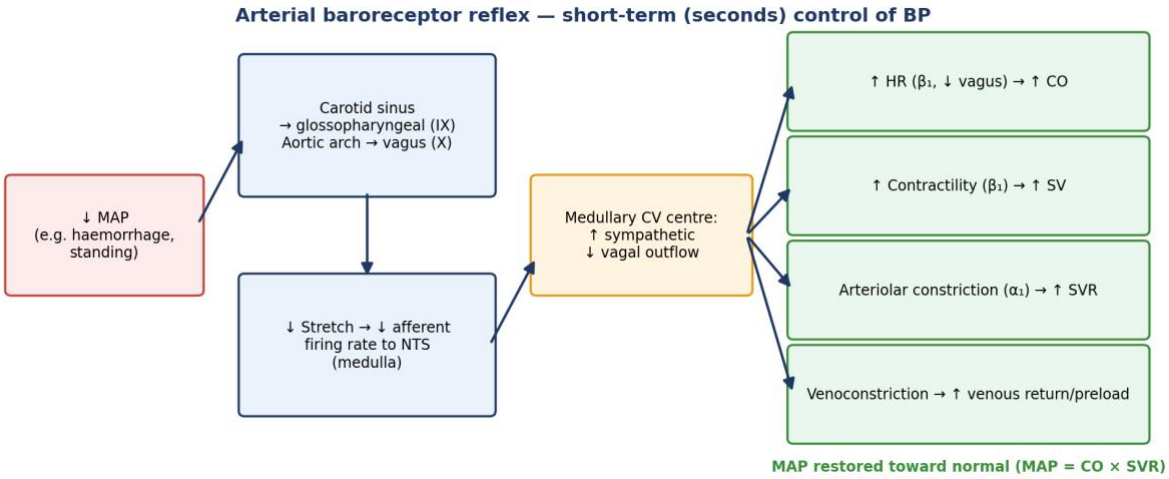


Figure 125.7 — The arterial baroreceptor reflex (carotid sinus → IX; aortic arch → X; both to NTS)

Time-scale	Mechanism	Key point
Seconds	Baroreflex; cardiopulmonary receptors; chemoreflex	Baroreflex resets in days — not long-term control. Bezold–Jarisch: paradoxical bradycardia (severe hypovolaemia, spinal anaesthesia)
Minutes–hours	RAAS; capillary fluid shift	Renin ↑ with ↓ renal perfusion, ↓ macula densa NaCl, β_1
Hours–days	Renal pressure natriuresis, ADH, ANP/BNP	Kidney sets long-term BP

Receptor	Effect (obstetric relevance)
β_1	↑ HR, contractility, renin
β_2	Vasodilatation, bronchodilatation, uterine relaxation (β-agonist tocolysis → tachycardia, pulmonary oedema)
α_1	Vasoconstriction (phenylephrine for spinal hypotension at caesarean)
α_2	Central ↓ sympathetic outflow (methyldopa)
M_2	↓ HR and AV conduction (atropine blocks)

125.7 Regional circulations

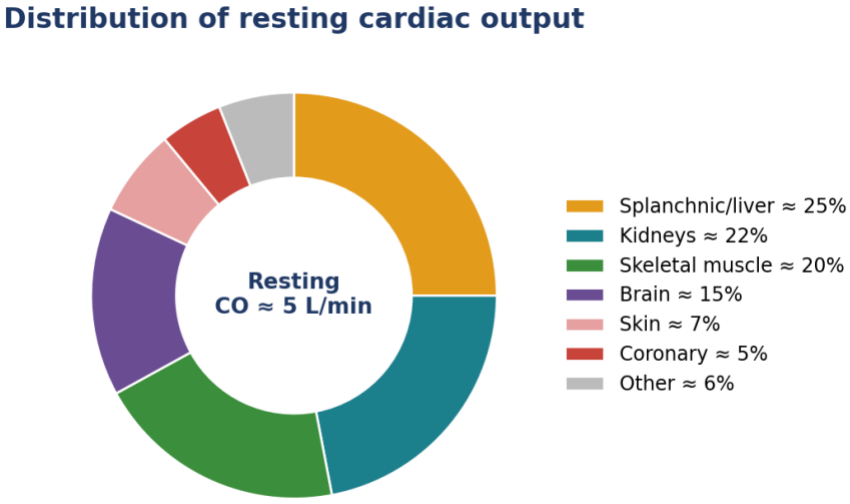


Figure 125.8 — Distribution of resting cardiac output

Circulation	Key features
Coronary	LV perfused in diastole; O_2 extraction ≈ 70–75% at rest → ↑ demand needs ↑ flow (adenosine)
Cerebral	Autoregulation MAP ≈ 60–160 mmHg; most sensitive to $PaCO_2$; loss at high MAP → PRES/eclampsia
Renal	≈ 20–25% of CO; autoregulated; ↑ 50–80% in pregnancy
Uteroplacental	≈ 50 → 500–750 mL/min at term; not autoregulated — maternal hypotension directly ↓ placental flow

125.8 Integrated responses

Haemorrhage → ↓ preload ▶ ↓ SV, CO, MAP ▶ Baroreflex: ↑ HR, contractility, vasoconstriction ▶ RAAS, ADH; plasma refill ▶ Erythropoiesis (days–weeks)

ATLS class	I	II	III	IV
Blood loss	< 15%	15–30%	30–40%	> 40%
Heart rate	Normal	> 100	> 120	> 140
Systolic BP	Normal	Normal	↓	↓↓
Urine output	Normal	Mildly ↓	Oliguria	Negligible

■ CLINICAL CORRELATION

- Pregnant women may lose **1000–1500 mL before hypotension** — tachycardia, narrowing pulse pressure and oliguria come first; **shock index (HR ÷ SBP) ≥ 0.9–1.0** signals significant haemorrhage.

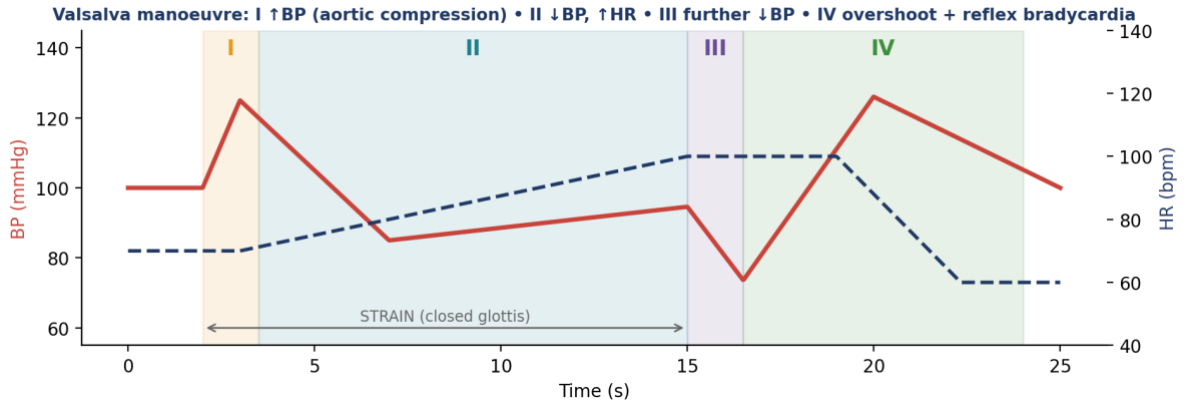


Figure 125.9 — Phases of the Valsalva manoeuvre (relevant to active pushing in the second stage and to vagal termination of SVT)

125.9 Must-know summary

■ EXAM TIP

- Plateau = L-type Ca^{2+}; pacemaker = If + T-type Ca^{2+}. **S3 normal in pregnancy; S4 never normal.**
- Arterioles = resistance; veins = capacitance; capillaries = slowest flow.
- Carotid sinus **IX**, aortic arch **X** → NTS. Cerebral flow most sensitive to **$PaCO_2$**. Coronary flow mainly in **diastole**.
- Plasma oncotic pressure is mainly **albumin** — falls in pregnancy → oedema / pulmonary oedema risk.

■ MNEMONIC / MEMORY AID

JVP 'ACXVY': Atrial contraction, Closure/bulge of tricuspid, relaXation, Venous filling, emptYing. **Myocyte AP 0–4**: 'Na – K – Ca – K – K'.