GM26 · GOLD-STANDARD SERIES

BASIC SCIENCE MODEL ANSWERS

Chapter 1 — Cardiovascular Physiology

Cornell-format exam model answers · Pareto 20/80 highlighting
16 questions · MD / DNB Medicine · PYQ 2009–2024

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How to use this chapter

Each question is answered in **Cornell layout**: the **gold cue column** (left) holds recall triggers, mnemonics and headings; the wide column holds the **model answer** you would write to score maximum marks. **Yellow highlight** marks the Pareto 20% — the phrases that earn most of the marks. Colour-coded boxes: **red = definition/emergency**, **teal = clinical pearl**, **gold = mnemonic/cram**. Answers follow the standard theory skeleton (define → mechanism → clinical features → investigations → management) adapted per question.

Q	Topic	Marks	PYQ
1	Cardiac cycle & exercise	10M	STD
2	Wigger's diagram	10M	2x
3	Conduction · re-entry · RF ablation	10M	STD
4	Bradycardia · pacemaker indications	10M	STD
5	SA node physiology · SND	10M	STD
6	Frank–Starling law	5M	STD
7	Pulsus paradoxus	10M	2x
8	Coronary circulation & regulation	10M	2x
9	PSVT pathogenesis	5M	STD
10	Cerebral autoregulation	5M	2x
11	Thermoregulation · hypothermia	10M	STD
12	Effective circulatory volume	10M	STD
13	IVC anatomy & obstruction	10M	STD
14	SVC tributaries · SVC syndrome	10M	STD
15	Pulsus alternans · acute LVF	10M	STD
16	Re-entry · atrial fibrillation	10M	2x

Q1. Cardiac cycle: physiological basis & changes in exercise

10M
DNB Oct-2024
STD

Define · phases

DEFINITION of CARDIAC CYCLE

The **sequence of electrical and mechanical events in one heartbeat**, from the start of one atrial contraction to the next. At HR 75/min it lasts **0.8 s** (**systole 0.3 s, diastole 0.5 s**).

Seven phases (one cycle)

- **Atrial systole** — 'atrial kick', ~20% of filling; produces **S4** if stiff ventricle
- **Isovolumetric contraction** — all valves shut, sharp LV pressure rise; onset of **S1**
- **Rapid ejection** — aortic valve opens (LV>aorta ~80 mmHg), ~70% SV ejected
- **Reduced ejection** — flow falls, LV pressure peaks ~120 mmHg then declines
- **Isovolumetric relaxation** — AV closes (**S2**, dicrotic notch), all valves shut
- **Rapid filling** — MV opens, ventricle fills passively; **S3** if volume overload
- **Reduced filling / diastasis** — slow filling before next atrial systole

Numbers to quote

Parameter	Value
EDV / ESV	~130 / ~50 mL
Stroke volume	70–80 mL
Ejection fraction	55–65%
Aortic / LV peak	~120 mmHg
Cardiac output (rest)	~5 L/min

Exercise: how CO rises

Cardiovascular response to exercise

CO can rise from **~5 to 20–25 L/min** (up to 35 in elite athletes). **CO = HR × SV** — both rise, but their contributions differ with intensity:

- **Heart rate** — the **dominant** factor; early rise by **vagal withdrawal**, later by **sympathetic** drive (HRmax ≈ 220 − age)
- **Stroke volume** — rises via ↑preload (muscle & respiratory pump → venous return, Frank–Starling) and ↑contractility (sympathetic, ↓ESV)
- **a-v O2 difference** widens (↑extraction); **flow redistributes** to muscle & myocardium, away from splanchnic/renal beds
- **SVR falls** (metabolic vasodilation in muscle); MAP rises modestly, **pulse pressure** widens

Sub-max vs maximal

	Sub-maximal	Maximal
HR	Rises linearly with work	Reaches HRmax
SV	Rises, plateaus ~40–50% VO2max	May fall slightly (short filling time)
CO	Linear rise	Plateaus at VO2max
Main driver	SV + HR	Almost entirely HR

APPLIED

In **heart failure / chronotropic incompetence**, the SV reserve and HR reserve are blunted → reduced exercise capacity; this is the physiological basis of the **6-minute walk test** and **CPET (peak VO2)** used for prognosis and transplant listing.

Take-home

CRAM

CO↑ in exercise = **HR (main) + SV (preload↑, contractility↑, ESV↓) + extraction↑**. At maximal effort, **HR carries the rise; SV plateaus**.

Q2. Cardiac cycle — Wigger's diagram (labelled)

10M
MDS 2024 · DNB J-2018
2x REPEAT

Draw & label

The **Wigger diagram** plots, on one time axis, the **aortic, left-ventricular and left-atrial pressures**, **LV volume**, **ECG** and **heart sounds** through a single cycle. Reproduce it accurately — a correctly labelled figure carries most of the marks.

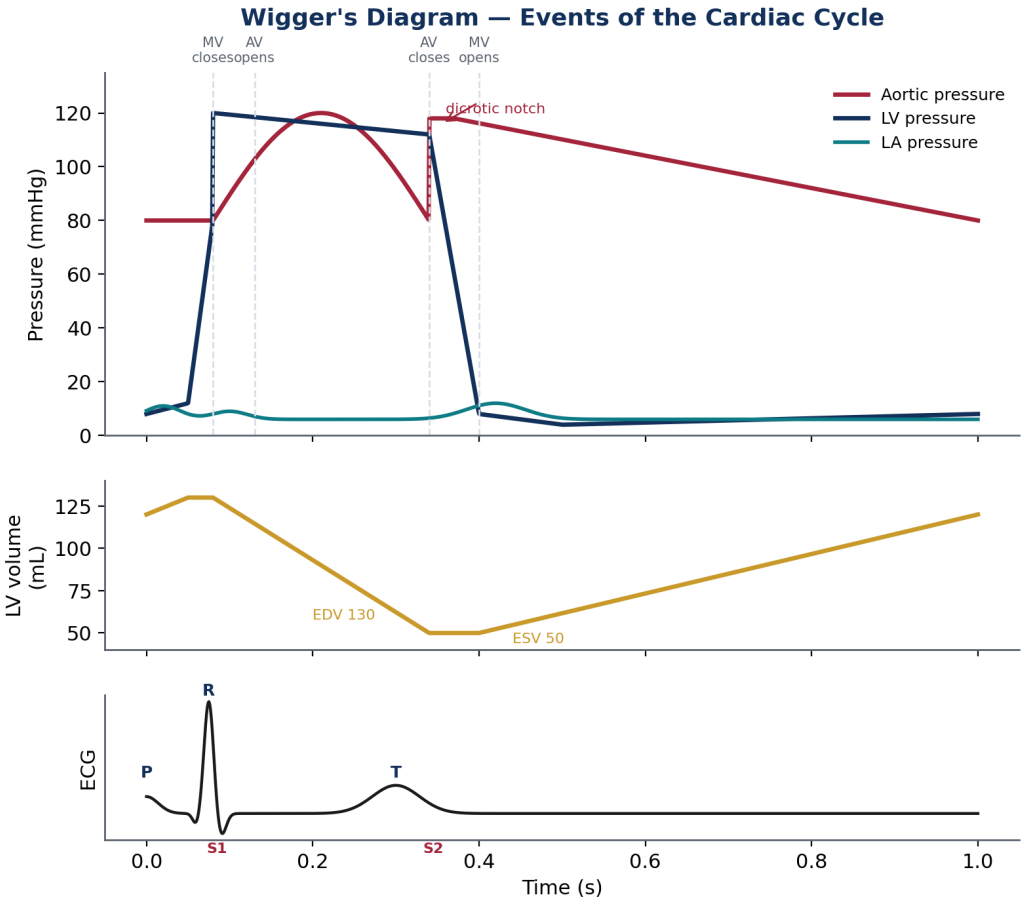

Fig. Wigger's diagram — pressures, volume, ECG and heart sounds.

Read the curves

- **P wave** → atrial systole → small **a-wave** bump in LA/LV pressure
- **QRS** → isovolumetric contraction → **MV closes = S1**
- LV pressure > aortic → **AV opens** → ejection (no sound; valve opening is silent)
- **T wave** → repolarisation → ejection ends → **AV closes = S2** + **dicrotic notch**
- LV pressure < LA → **MV opens** → rapid filling → **S3** (if present)

Link to JVP

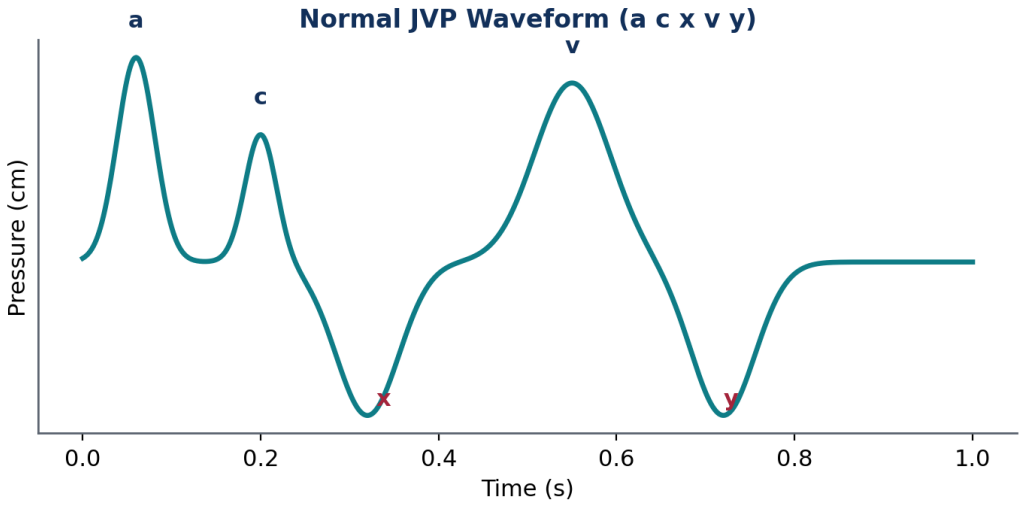

Fig. JVP waveform — a (atrial systole), c (TV bulge), x descent, v (atrial filling), y descent.

VIVA LINK

a wave = atrial systole; **cannon a waves** in complete heart block/VT (AV dissociation); **absent a wave** in AF; **large v wave** in tricuspid regurgitation.

Common mistake

COMMON MISTAKE

Do not label valve opening events as heart sounds — **only valve CLOSURE makes S1/S2**. Also keep LA pressure below LV during systole and crossing it during filling.

Q3. Conduction system; re-entrant tachycardia; radiofrequency ablation

10M
DNB J-2020

STD

Conduction pathway

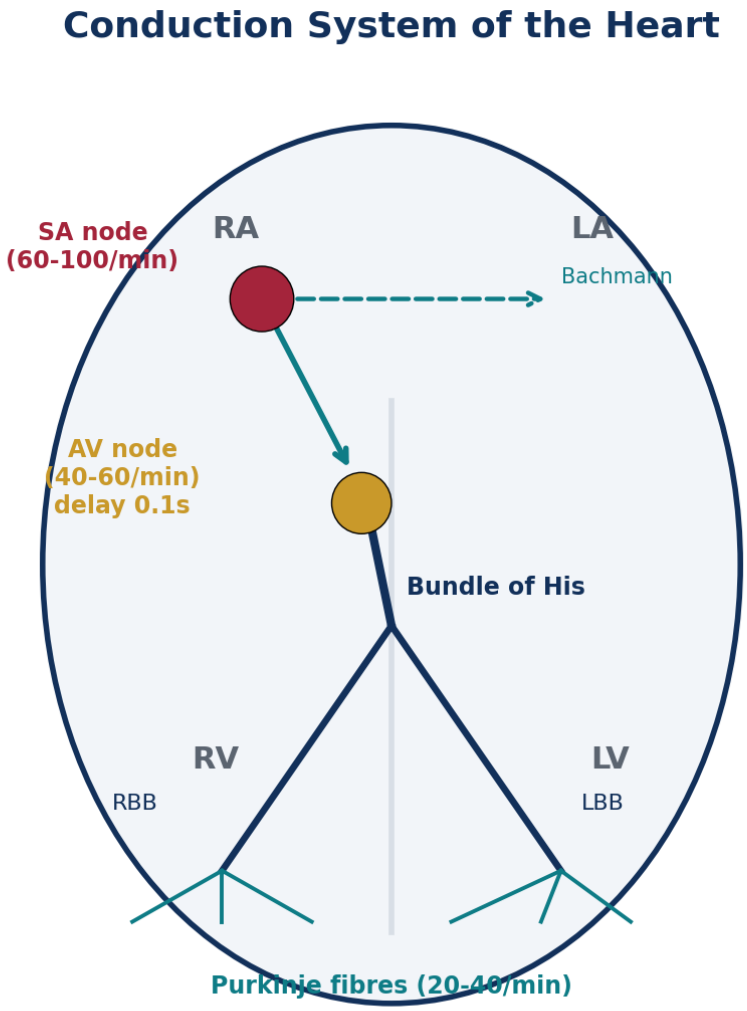

Fig. SA node → AV node → His → bundle branches → Purkinje.

Structure	Intrinsic rate	Blood supply
SA node	60–100/min	RCA 60% / LCx 40%
AV node	40–60/min	RCA 90% (AV nodal a.)
His–Purkinje	20–40/min	LAD septals + RCA

Re-entry: 3 needs

Mechanism of re-entrant tachycardia

Re-entry needs **three conditions**: (1) **two anatomically or functionally distinct pathways** forming a circuit, (2) **unidirectional block** in one limb, and (3) **slow conduction** in the other so the blocked limb recovers excitability — the impulse then re-enters and circulates (**circus movement**).

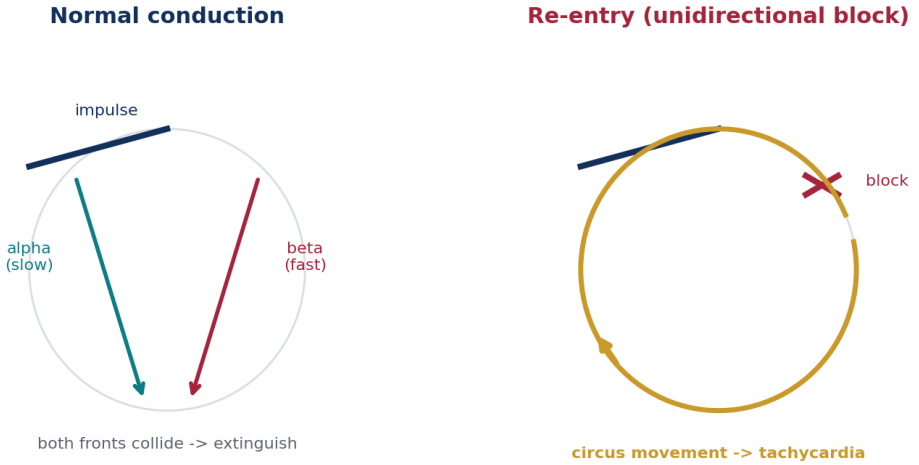

Fig. Normal collision vs re-entrant circuit with unidirectional block.

RF ablation

Radiofrequency ablation — essentials

• **Principle**: RF energy heats tissue to **~50–60°C** → focal **coagulation necrosis** that interrupts the circuit or destroys the focus

• **Indications**: AVNRT (slow-pathway modification), AVRT/WPW (accessory pathway), typical atrial flutter (**cavotricuspid isthmus**), AF (**pulmonary vein isolation**), VT

• **Guidance**: electroanatomical (3-D) mapping; entrainment for macro-re-entry

• **Complications**: tamponade, AV block (needing PPM), PV stenosis, stroke, vascular injury

HIGH-YIELD

AVNRT is the commonest cause of paroxysmal SVT → slow-pathway ablation is **>95% curative**.

Q4. Conduction anatomy; causes of bradycardia; pacemaker indications

10M
DNB D-2018
STD

Bradycardia = HR<60

DEFINITION

Bradycardia = resting heart rate < 60/min. Two mechanisms: **failure of impulse generation** (SA node dysfunction) or **failure of conduction** (AV block).

Causes of bradycardia

Group	Examples
Physiological	Athletes, sleep, vagal tone
Intrinsic	SND, idiopathic fibrosis (Lev–Lenègre), ischaemia (inferior MI), infiltrative
Drugs	Beta-blockers, non-DHP CCB, digoxin, amiodarone, ivabradine
Metabolic	Hypothyroidism, hyperkalaemia, hypothermia
Other	Raised ICP (Cushing), obstructive sleep apnoea, cholestasis

PPM indications

Permanent pacemaker — class I indications

• **Symptomatic SA node dysfunction** (incl. drug-induced when drug is essential)
• **Third-degree** and advanced **second-degree** AV block (symptomatic, or with asystole >3 s / escape <40)
• **Mobitz type II** AV block, even if asymptomatic
• Alternating bundle branch block; persistent post-MI high-grade block
• Chronotropic incompetence with symptoms

COMMON MISTAKE

Mobitz I (Wenckebach) is usually benign — pace only if symptomatic; **Mobitz II** and complete block need pacing regardless of symptoms.

Cram

CRAM

Pace the **block below the AV node** (Mobitz II, 3°, alternating BBB) and the **symptomatic slow** — spare the benign Wenckebach.

Q5. Physiology of the SA node; SA node dysfunction; diagnostic approach

10M
DNB J-2017

STD

Pacemaker current

Why the SA node sets the rhythm

• No stable resting potential — **slow phase-4 diastolic depolarisation** to threshold
• **Funny current I_f** (HCN channels, Na influx) initiates depolarisation
• **I_CaT then I_CaL** complete the upstroke; K efflux repolarises
• Fastest intrinsic rate → SA node is the **dominant pacemaker** (overdrive suppression)
• **Autonomic control**: vagus (ACh, M2) slows; sympathetic (NA, β1) accelerates

SND spectrum

SA node dysfunction (sick sinus syndrome)

Type	Cause
Intrinsic	Age-related fibrosis, ischaemia, infiltration, cardiomyopathy, congenital
Extrinsic	Drugs, ↑vagal tone, hypothyroid, hyperkalaemia, hypoxia, hypothermia

Manifestations: **sinus bradycardia, sinus pause/arrest, SA exit block**, **tachy-brady syndrome**, and **chronotropic incompetence**.

Diagnose

Approach to diagnosis

• Confirm symptom–rhythm correlation: **12-lead ECG**, **Holter**, **event/loop recorder** for infrequent symptoms
• **Exercise test** — for chronotropic incompetence
• Exclude **reversible** causes (drugs, thyroid, electrolytes) — always first
• **EP study** (SNRT, SACT) if diagnosis unclear

MANAGEMENT

Withdraw offending drugs and correct reversible causes; **permanent pacing** is the definitive treatment for symptomatic, irreversible SND.

Q6. Frank–Starling law and its clinical significance

5M
DNB 2022 (P1)

STD

Statement

DEFINITION

Within physiological limits, the **force of ventricular contraction (stroke volume) is proportional to the initial length of the muscle fibre (end-diastolic volume / preload)**. 'The heart pumps what it receives.'

Mechanism

- Optimal sarcomere length **~2.0–2.2 µm** → maximal actin–myosin overlap
- **Length-dependent activation** — stretch ↑ troponin-C Ca^{2+} sensitivity
- Beyond the optimum → **descending limb** (over-distension, seen in failure)

Curve

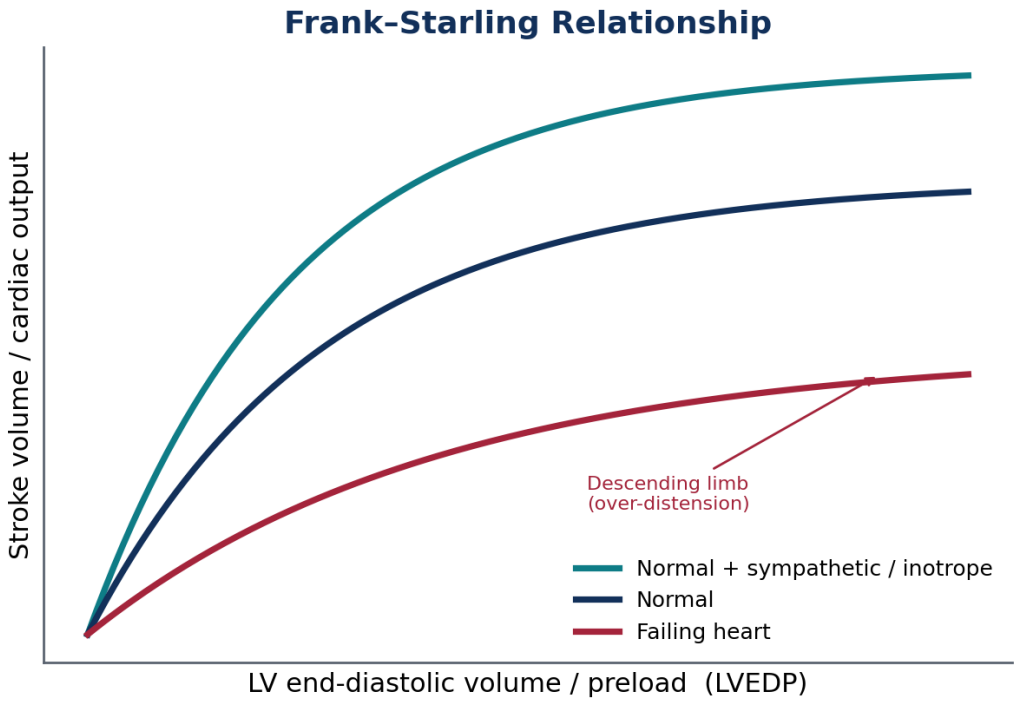

Fig. Frank–Starling curves — sympathetic/inotrope shift up, failing heart shifts down/right.

Clinical use

Clinical significance

- **Balances output** of the two ventricles beat-to-beat
- Underlies the response to **↑venous return** (exercise, posture, pregnancy)
- Rationale for **preload reduction** (diuretics, venodilators) in congestion
- Explains benefit of **optimising filling** in the ICU (fluid responsiveness)

CRAM

Stretch → stronger beat, up to a point. Failing heart works on a **flat / descending** curve.

Q7. Pulsus paradoxus — diagram, clinical findings, causes

10M
DNB D-2020 · J-2020

2x REPEAT

Definition

DEFINITION

An **exaggerated inspiratory fall in systolic BP of > 10 mmHg** (normal < 10). The pulse is 'paradoxical' — it weakens/disappears on inspiration despite the heartbeat continuing.

Mechanism

Pathophysiology

• Inspiration → ↑systemic venous return → **RV filling ↑**
• In a fixed space (tamponade), the **septum shifts left** → **LV filling ↓** (**ventricular interdependence**)
• ↑pulmonary vascular capacitance **pools blood** in lungs → less LV preload
• Net effect: **LV stroke volume and SBP fall in inspiration**

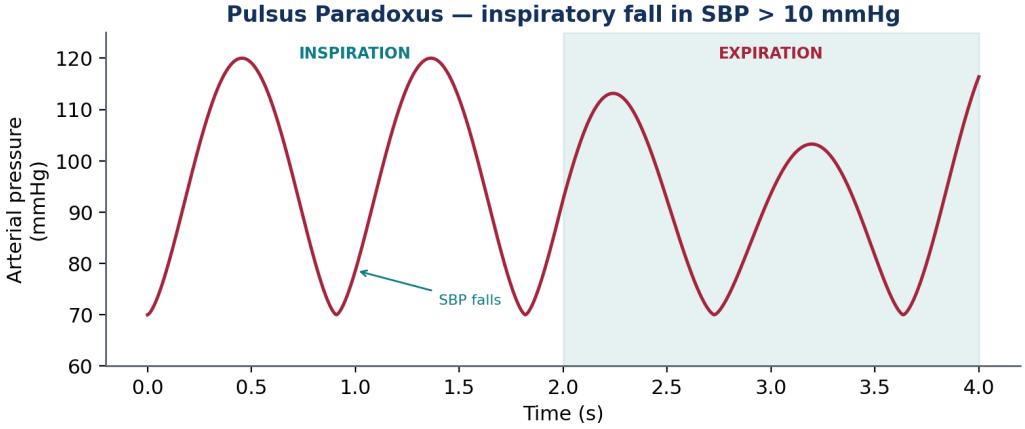

Fig. Inspiratory fall in arterial pressure > 10 mmHg.

Elicit & causes

How to elicit

Inflate cuff above SBP, deflate slowly: Korotkoff sounds first heard only in **expiration**; the gap between that and when they are heard **throughout** the respiratory cycle is the paradox (>10 mmHg = positive).

Causes

Common	Others
Cardiac tamponade	Constrictive pericarditis (variable)
Severe asthma / COPD	Massive pulmonary embolism
Tension pneumothorax	Hypovolaemic shock, obesity

EMERGENCY / RED FLAG

With hypotension + ↑JVP + muffled heart sounds (**Beck triad**) and pulsus paradoxus, suspect **cardiac tamponade** → urgent echo and pericardiocentesis.

Q8. Coronary circulation and its regulation

10M
MD State · DNB D-2018

2x REPEAT

Anatomy

Anatomy

- **LCA** → LAD (anterior wall, septum, apex) + LCx (lateral wall)
- **RCA** → RV, inferior wall; gives **PDA in ~85%** = right dominance
- Flow **~250 mL/min (≈5% of CO)**; venous return via coronary sinus → RA
- **LV perfused mainly in diastole** (systolic compression); RV in both phases

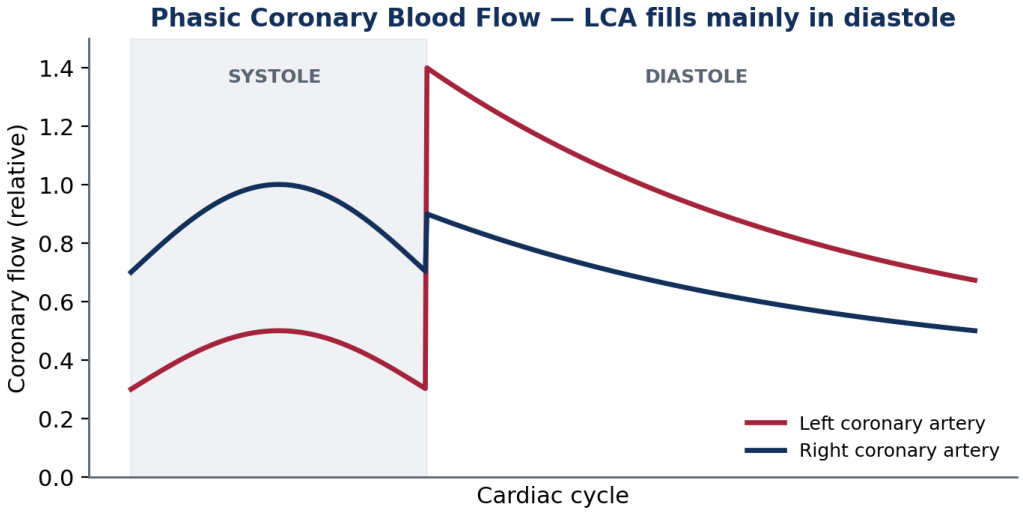

Fig. Phasic coronary flow — LCA fills in diastole.

Regulation

Regulation of coronary flow

Mechanism	Key mediators / effect
Metabolic (dominant)	Adenosine, hypoxia, ↑CO2, ↑K+ → vasodilation matches O2 demand
Autoregulation	Constant flow over CPP ~60–180 mmHg
Endothelial	NO, prostacyclin, EDHF (dilate) vs endothelin (constrict)
Neural	Sympathetic α (constrict) & β2 (dilate); vagal minor
Myogenic	Bayliss response to wall stretch

Reserve & MVO2

Coronary flow reserve = ratio of maximal (hyperaemic) to resting flow; falls in microvascular disease and significant stenosis. Myocardial O2 demand (**MVO2**) is set by **heart rate, contractility and wall tension (afterload)**.

APPLIED

Because the LV fills in diastole, **tachycardia** (short diastole) and **low aortic diastolic pressure** both worsen ischaemia — the rationale for **beta-blockers** in angina.

Q9. Pathogenesis of paroxysmal supraventricular tachycardia

5M
DNB D-2022
STD

Define

DEFINITION

PSVT = sudden-onset, regular, narrow-complex tachycardia (rate 150–250) arising above the bundle of His, usually by **re-entry**.

Mechanisms

Type	Frequency	Basis
AVNRT	~60%	Dual AV-nodal pathways (slow–fast re-entry)
AVRT	~30%	Accessory pathway (WPW): orthodromic / antidromic
Atrial tachycardia	~10%	Automatic focus or micro-re-entry

A **premature beat** finds one limb refractory → **unidirectional block** → conduction down the other limb, then retrograde return → sustained **circus movement**.

ACUTE Rx

Vagal manoeuvres → IV adenosine terminate most AVNRT/AVRT by transient AV-nodal block; unstable → **synchronised DC cardioversion**.

Q10. Autoregulation of cerebral blood flow & clinical significance

5M
DNB J-2019 · May-2024

2x REPEAT

Define

DEFINITION

Maintenance of a **relatively constant cerebral blood flow (~50 mL/100 g/min)** across a wide range of **mean arterial / cerebral perfusion pressure (≈60–150 mmHg)**. **CPP = MAP − ICP**.

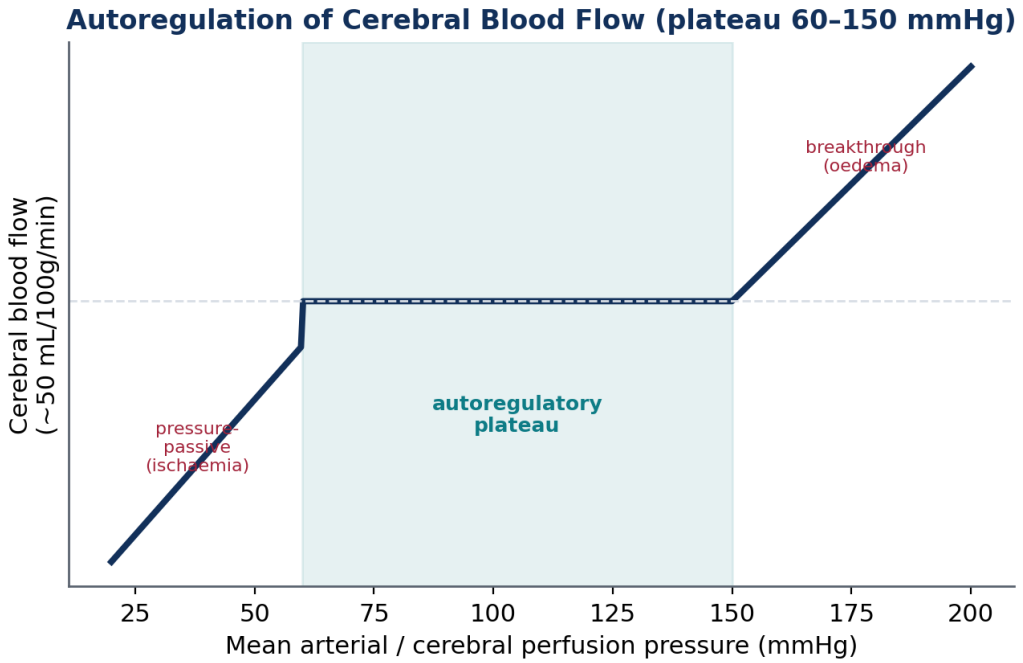

Fig. Autoregulatory plateau 60–150 mmHg; failure at extremes.

Mechanisms & clinic

Mechanisms

- **Myogenic** (Bayliss) — arteriolar constriction to ↑pressure
- **Metabolic** — **PaCO2 is the most potent** dilator (↑CO2 → vasodilation)
- Neurogenic and endothelial (NO) modulation

Clinical significance

- Curve **shifts right** in chronic hypertension → avoid rapid BP lowering
- **Lost** in TBI, stroke penumbra, malignant HTN → flow becomes pressure-passive
- **Hyperventilation** (↓CO2) transiently lowers ICP; **permissive hypertension** in acute ischaemic stroke protects the penumbra

CRAM

Plateau 60–150; **CO2 = strongest lever**; hypertensives sit shifted to the right.

Q11. Physiology of thermoregulation; causes & management of hypothermia

10M
DNB J-2019
STD

Control centre

Thermoregulation

• **Hypothalamus** is the thermostat: **preoptic anterior** drives heat loss, **posterior** drives heat conservation
• **Heat gain**: BMR, shivering, thyroxine, sympathetic non-shivering thermogenesis (brown fat)
• **Heat loss**: **radiation, conduction, convection, evaporation**
• Effectors: skin vasomotion, sweating, behaviour

Grades

DEFINITION

Hypothermia = core temperature < 35°C. Mild **32–35**, moderate **28–32**, severe **< 28°C**.

Causes

Group	Examples
Environmental	Cold exposure, immersion
↓Thermogenesis	Hypothyroid, hypopituitary, hypoglycaemia, malnutrition, sepsis
Impaired control	Autonomic failure, hypothalamic lesion, stroke
Drugs	Alcohol, sedatives, phenothiazines

Manage

Management

• ABC, **handle gently** (irritable myocardium → VF), continuous core-temp & cardiac monitoring
• ECG: **Osborn (J) wave**, bradycardia, prolonged intervals, AF, VF
• **Rewarming** — passive external (mild) → active external (warm blankets, forced air) → active internal (warmed humidified O2, IV fluids, lavage; **ECMO/bypass** for severe/arrest)
• Treat the cause; correct glucose, thyroid, sepsis

EMERGENCY / RED FLAG

In hypothermic arrest, continue resuscitation and rewarm — **"not dead until warm and dead"** (**≥ 32–35°C** before declaring death).

Q12. Effective circulatory volume: definition, altered states, management

10M
DNB Apr-2016
STD

Define

DEFINITION

Effective circulatory volume (ECV) = the part of the ECF within the arterial system that is **effectively perfusing tissues** and is **sensed by baroreceptors** (carotid/aortic, atrial, and renal afferent arteriole/JGA). It is **not directly measurable** and can dissociate from total ECF volume.

Decreased ECV

Decreased ECV

Mechanism	Examples
True hypovolaemia	Haemorrhage, GI/renal losses, burns, diuretics
↓Effective (↑total)	Heart failure, cirrhosis, nephrotic syndrome, sepsis (vasodilation)

Response: baroreceptor unloading → **RAAS, ADH, sympathetic activation** → renal **Na^+ and water retention** → oedema despite an already expanded ECF.

Increased ECV

• Primary **hyperaldosteronism**, Cushing, exogenous mineralocorticoid
• Excess salt/water administration; renal Na retention

Manage

Management principle

Distinguish true from effective volume depletion. Give volume for true hypovolaemia; for **low-ECV oedematous states** (HF, cirrhosis, nephrosis) treat the primary disorder and use **diuresis + Na restriction** — not fluids.

COMMON MISTAKE

Do not fluid-load a heart-failure patient because they look 'dry' — total body Na/water is high; only the **effective** volume is low.

Q13. IVC anatomy and clinical features of obstruction by level

10M
DNB 2022 P2

STD

Anatomy

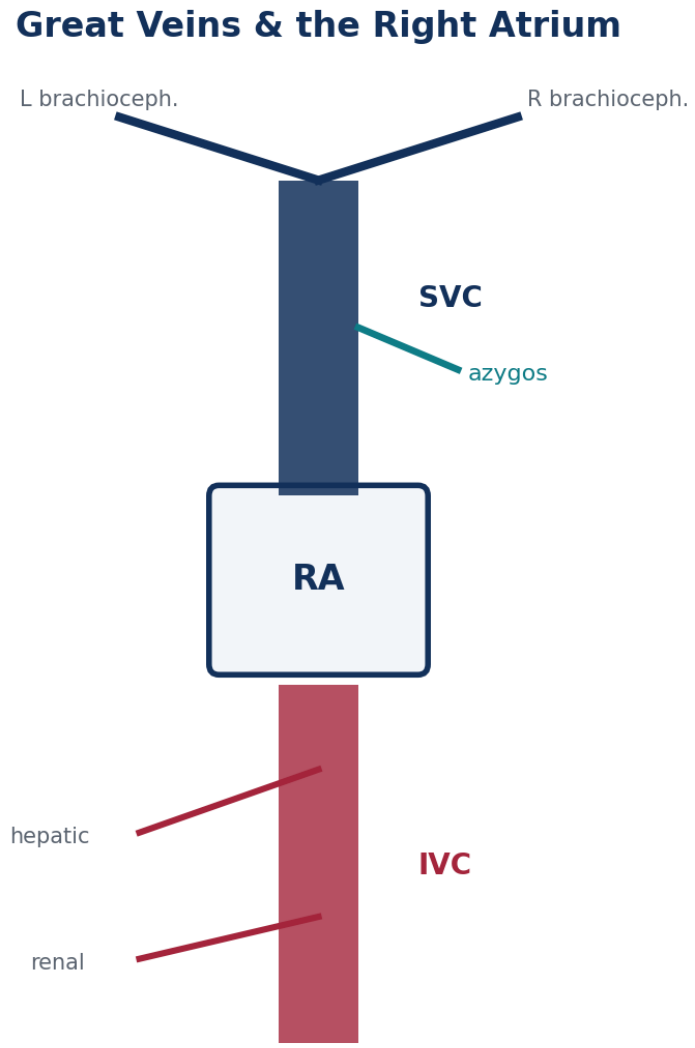

Fig. IVC/SVC and the right atrium (schematic).

Inferior vena cava

- Formed by union of **common iliac veins at L5**; ascends **right of the aorta**
- Pierces the diaphragm at **T8**, enters the RA
- Tributaries: lumbar, **right gonadal, renal, right suprarenal, hepatic, phrenic** veins

Obstruction by level

Level	Clinical features
Infrarenal	Bilateral leg oedema, dilated abdominal-wall veins (flow upward), DVT
Renal	+ Renal vein involvement → proteinuria / nephrotic, RCC extension
Suprarenal / hepatic	Budd–Chiari: tender hepatomegaly, ascites, jaundice
High / RA	Features resembling SVC-type congestion if extensive

Causes: **thrombosis**, tumour (**RCC, HCC**), extrinsic compression, **membranous obstruction (MOVC)**.

CLUE

Abdominal wall venous collaterals with upward flow point to IVC obstruction (vs caput medusae of portal hypertension, which flow away from the umbilicus).

Q14. SVC tributaries (labelled); Superior Vena Cava Syndrome

10M
DNB 2022 P1

STD

Anatomy

Superior vena cava

- Formed by **right + left brachiocephalic veins** behind the **1st right costal cartilage**
- Receives the **azygos vein** just before entering the RA
- Thin-walled, low-pressure → readily compressed by mediastinal disease

SVC syndrome

DEFINITION

SVC syndrome = obstruction of SVC flow → congestion of head, neck and upper limbs.

Features

- **Facial/neck/arm oedema**, plethora, cyanosis
- **Distended, non-pulsatile** neck & chest-wall veins (collaterals)
- Headache, dizziness, **Pemberton sign** (facial congestion on raising arms)
- Dyspnoea, stridor, cough

Causes & Rx

Causes	Management
Malignancy — lung Ca (esp. SCLC), lymphoma	Elevate head, O2; treat cause (RT/chemo)
Catheters / pacemaker leads (thrombosis)	Anticoagulation; remove line if feasible
Fibrosing mediastinitis, goitre	Endovascular stenting (rapid relief)

EMERGENCY / RED FLAG

Airway compromise or cerebral oedema makes SVCS a medical emergency → urgent **stenting** and treatment of the underlying cause.

Q15. Pulsus alternans; acute left ventricular failure

10M
DNB J-2019

STD

Sign

DEFINITION

Pulsus alternans = a **regular rhythm with alternating strong and weak pulse beats**. It signifies **severe LV systolic dysfunction**.

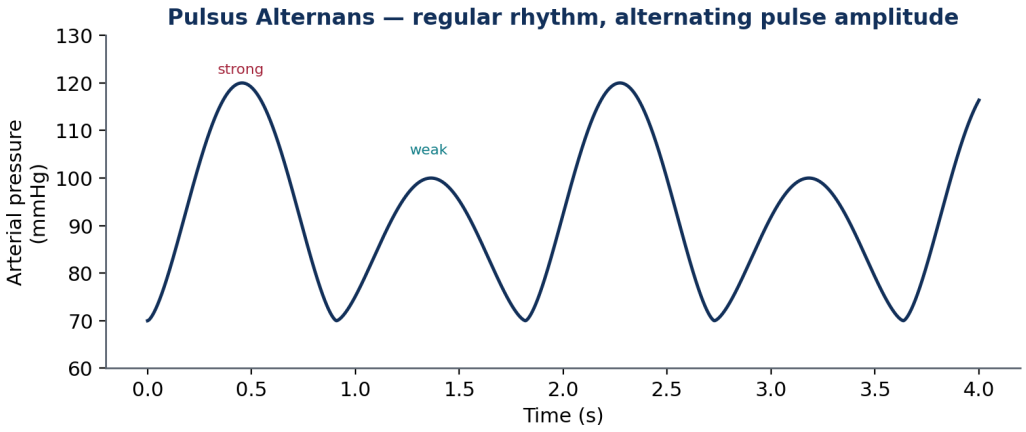

Fig. Regular rhythm, alternating pulse amplitude.

Mechanism: beat-to-beat variation in **intracellular Ca^{2+} cycling** and incomplete mechanical restitution of failing myocytes. Best detected with a **sphygmomanometer** (Korotkoff sounds double as cuff is deflated).

Acute LVF

Acute LV failure / cardiogenic pulmonary oedema

- **Symptoms**: acute dyspnoea, orthopnoea, PND, **pink frothy sputum**, anxiety
- **Signs**: tachypnoea, bibasal fine crepitations, **S3 gallop**, ↑JVP, cold sweaty periphery
- Precipitants: ACS, arrhythmia, uncontrolled HTN, valve failure, non-adherence

Manage

Management (LMNOP)

Step	Action
Position + O2	Sit up; high-flow O2 / NIV (CPAP) if hypoxic
Diuretic	IV furosemide (Loop)
Vasodilator	IV/SL nitrate if SBP adequate → ↓preload & afterload
Morphine	Selective (anxiety, dyspnoea) — caution
Treat cause	ACS, arrhythmia, valve; inotropes/mechanical support if in shock

CRAM

Acute LVF = **LMNOP** — Lasix, Morphine, Nitrates, Oxygen/NIV, Position (sit up).

Q16. Re-entry in arrhythmias; management of atrial fibrillation

10M
DNB 2022 P1 · J-2020

2x REPEAT

Re-entry recap

Re-entry (see Q3) requires **two pathways, unidirectional block and slow conduction** → **circus movement**. It underlies **AVNRT, AVRT, atrial flutter, most VT** and, via **multiple wavelets / rotors**, atrial fibrillation.

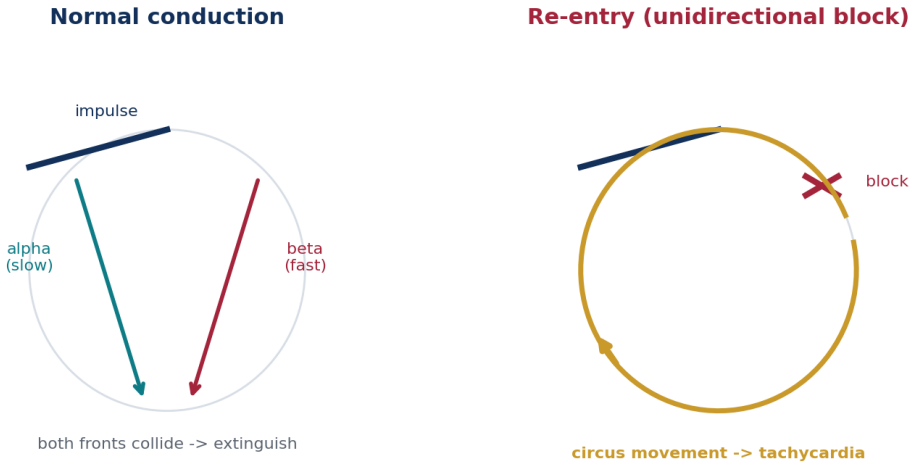

Fig. Re-entrant circuit with unidirectional block.

AF: two questions

Management of AF — the two decisions

Ask: (1) is the patient **haemodynamically unstable**? → immediate **synchronised DC cardioversion**. (2) If stable — **rate vs rhythm control**, plus **stroke prevention** in every case.

Pillars

Pillar	Options
Anticoagulation	Assess CHA2DS2-VASc & HAS-BLED → DOAC (preferred) or warfarin; LAA occlusion if OAC unsuitable
Rate control	Beta-blocker, non-DHP CCB (diltiazem/verapamil), digoxin
Rhythm control	Cardioversion (electrical/pharmacological), amiodarone/flecainide, catheter ablation (PVI)
Treat cause	Thyroid, alcohol, sepsis, valve, OSA, electrolytes

HIGH-YIELD

CHA2DS2-VASc ≥ 2 (men) / ≥ 3 (women) → anticoagulate. Rhythm control is favoured in **younger, symptomatic, or recent-onset** AF and early after diagnosis (EAST-AFNET 4).

Cram

CRAM

AF = **Rate + Rhythm + Anticoagulate + treat the cause**; **unstable → shock**.