PIPADOR REVISION

MRCOG Part 1 Revision Notes

Cardiovascular Changes in Pregnancy

PHYSIOLOGY • LECTURE 126

VISUAL EXAMINATION REVISION NOTES

2026 Edition

126

MRCOG PART 1 REVISION NOTES | PHYSIOLOGY

Cardiovascular Changes in Pregnancy

Blood volume, cardiac output, SVR and blood pressure; aortocaval compression; labour and puerperium; clinical applications

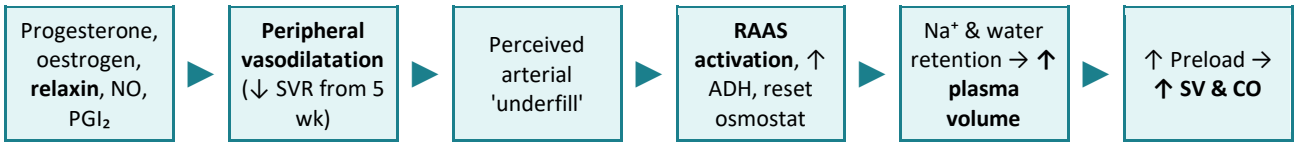
↑ 30–50% Cardiac output	↑ 40–50% Plasma volume	↑ 20–30% Red cell mass
↓ 25–30% Systemic vascular resistance	↑ 10–20 bpm Heart rate	Nadir 16–24 wk Blood pressure (back to baseline by term)

■ KEY FACTS: Learning objectives

- Describe the magnitude and time-course of changes in blood volume, cardiac output, heart rate, stroke volume, SVR and blood pressure.
- Explain the hormonal and vascular mechanisms underlying these adaptations.
- Describe structural cardiac changes and the normal examination findings of pregnancy.
- Explain aortocaval compression and its management.
- Describe haemodynamic changes in labour and the puerperium.
- Apply this physiology to maternal cardiac disease, pre-eclampsia, haemorrhage and anaesthesia.

126.1 Overview — the 'high-flow, low-resistance' state

Pregnancy converts the maternal circulation into a **hyperdynamic, high-volume, low-resistance** system. The primary event is **systemic vasodilatation** (↓ SVR) occurring as early as **5 weeks**, before placentation is complete. This 'underfilling' is sensed as relative arterial underfill, which activates the **RAAS** → sodium and water retention → plasma volume expansion → ↑ preload → ↑ stroke volume and cardiac output.



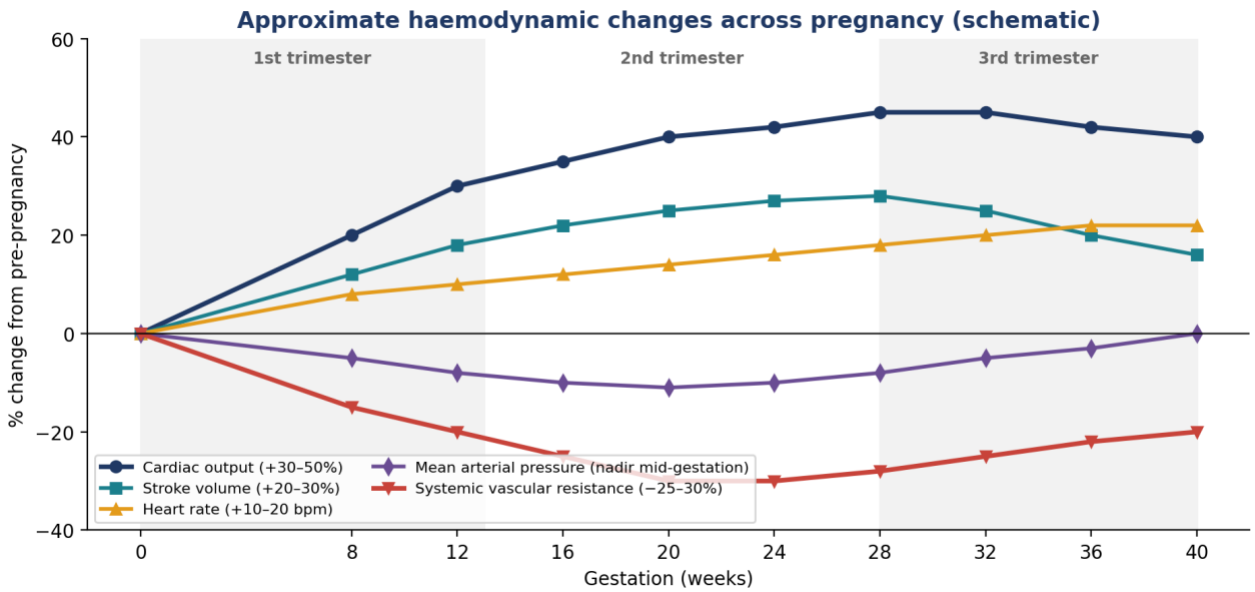


Figure 126.1 — Approximate percentage changes in haemodynamic variables across gestation (schematic; individual studies vary)

126.2 Summary table of haemodynamic changes

Parameter	Change	Magnitude	Timing / notes
Cardiac output	↑	30–50% (≈ 4.5 → 6–7 L/min)	Rises from 5 weeks; ≈ 75% of the rise occurs by the end of the first trimester; peaks ≈ 25–32 weeks then plateaus (may fall slightly at term when supine)
Stroke volume	↑	20–30%	Main driver of the early CO rise; peaks ≈ 20 weeks then plateaus/declines
Heart rate	↑	10–20 bpm (≈ 15–20%)	Progressive; maximal in the third trimester — becomes the main contributor to CO late in pregnancy
SVR	↓	25–30%	Begins ≈ 5 weeks; nadir mid-gestation (≈ 20 weeks); rises towards term but remains below non-pregnant values
Pulmonary vascular resistance	↓	≈ 30%	PA pressure therefore unchanged despite ↑ pulmonary flow
Blood pressure	↓ then ↑	DBP ↓ ≈ 10 mmHg; SBP ↓ less	Fall from first trimester; nadir 16–24 weeks; returns to pre-pregnancy levels by term. Pulse pressure widens
CVP, PCWP	↔	Unchanged	Ventricles dilate to accommodate the extra volume (↑ compliance)
Colloid oncotic pressure	↓	≈ 10–15% (↓ albumin)	COP − PCWP gradient narrows → pulmonary oedema risk, especially with pre-eclampsia, tocolytics, oxytocin and fluid overload
LVEF / contractility	↔ / slight ↑	—	EF essentially unchanged; LV end-diastolic volume ↑
Plasma volume	↑	40–50% (≈ 1.2–1.5 L)	Rises from 6–8 weeks; peaks ≈ 32–34 weeks
Red cell mass	↑	20–30% (≈ 250–450 mL)	Driven by ↑ erythropoietin; greater with iron supplementation
Total blood volume	↑	30–45% (≈ 1.5 L)	Larger with multiple pregnancy; smaller in pre-eclampsia and FGR
Haemoglobin / haematocrit	↓	Physiological 'dilutional anaemia'	Anaemia defined (BSH) as Hb < 110 g/L (1st trimester), < 105 g/L (2nd/3rd trimester), < 100 g/L postpartum
Uterine blood flow	↑↑	≈ 50 → 500–750 mL/min	≈ 10–12% of CO at term; non-autoregulated (pressure-passive)

Parameter	Change	Magnitude	Timing / notes
Renal plasma flow / GFR	↑	RPF ↑ 50–80%; GFR ↑ ≈ 50%	Creatinine and urea fall (creatinine typically < 75–80 µmol/L)

CVP ↔	PCWP ↔	COP ↓	Uterus ×10
Unchanged	Unchanged	Pulmonary oedema risk	≈ 50 → 500–750 mL/min

126.3 Blood volume and red cell changes

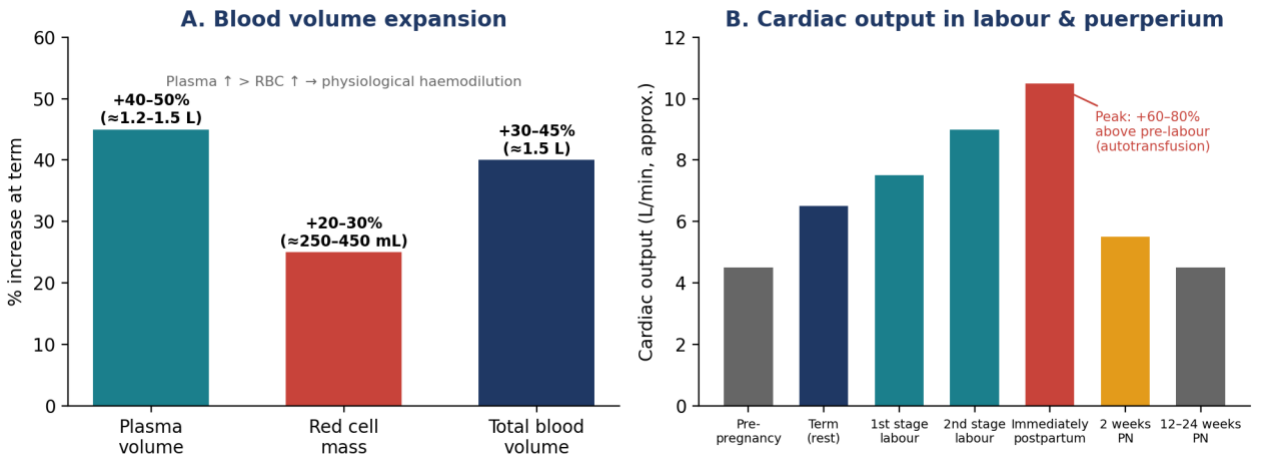


Figure 126.2 — (A) Blood volume expansion at term; (B) approximate cardiac output in labour and the puerperium

Plasma volume expansion — mechanisms

- **Vasodilatation-driven RAAS activation**: renin, angiotensinogen (oestrogen-induced hepatic synthesis), angiotensin II and **aldosterone all increase** — but pregnant women are relatively **resistant to the pressor effect of angiotensin II** (↑ PGI_2/NO; loss of this refractoriness precedes pre-eclampsia).
- **Osmoregulatory reset**: thirst and ADH thresholds fall by ≈ 10 mOsm/kg → plasma osmolality falls to ≈ 280 mOsm/kg; serum Na^+ falls by ≈ 4–5 mmol/L.
- **Progesterone** antagonises aldosterone at the tubule (natriuretic) — offset by ↑ aldosterone; net retention ≈ 900 mmol Na^+ and **6–8 L total body water** by term.
- Placental hormones (oestrogen, hPL), relaxin and ↑ ANP also contribute.

Why it matters

- **Reserve for delivery blood loss** — average vaginal delivery ≈ 500 mL; caesarean ≈ 1000 mL.
- **Placental perfusion** — inadequate volume expansion is associated with **FGR and pre-eclampsia**.
- Reduced blood viscosity → ↓ resistance, improved placental flow; but ↓ viscosity and ↑ velocity produce **flow murmurs**.
- Iron requirement ≈ 1000 mg in total (≈ 500 mg red cell expansion, ≈ 300 mg fetus/placenta, ≈ 200 mg basal losses); iron absorption increases in the 2nd/3rd trimesters.

126.4 Cardiac output, heart rate and stroke volume

- **Early** pregnancy: ↑ CO driven mainly by ↑ **SV** (↑ preload, ↓ afterload, ↑ LV end-diastolic volume).
- **Late** pregnancy: ↑ **HR** sustains CO while SV plateaus or declines (partly IVC compression).
- Measurement position matters: CO in late pregnancy is markedly lower **supine** than in the **left lateral** position (see 126.6).
- **Multiple pregnancy**: CO ≈ 15–20% higher than singleton; larger blood volume and greater fall in Hb.

Distribution of the increased cardiac output

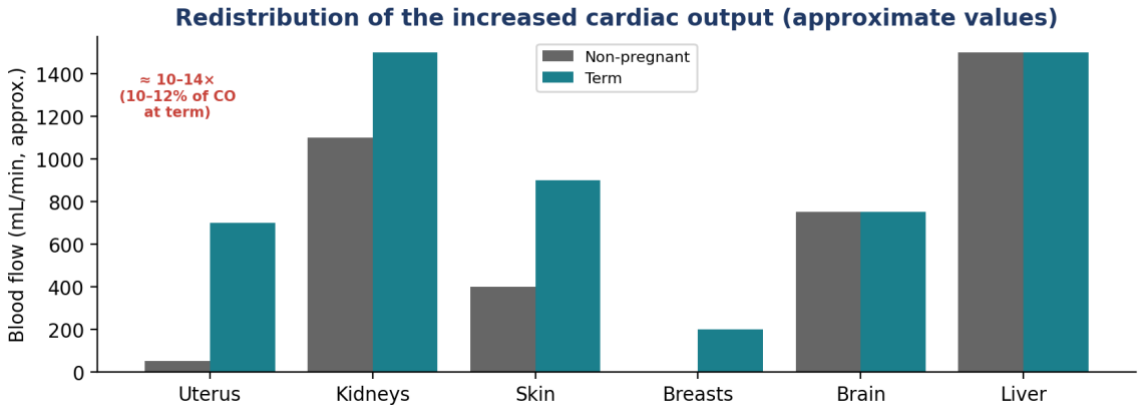


Figure 126.3 — Organ blood flow in non-pregnant women and at term (approximate)

Organ	Change	Comment
Uterus/placenta	≈ 50 → 500–750 mL/min	Low-resistance bed from spiral artery remodelling (endovascular and interstitial trophoblast invasion, loss of smooth muscle) — failure underlies pre-eclampsia and FGR
Kidneys	↑ 50–80% RPF	Glycosuria, ↓ urea/creatinine, ↓ uric acid early
Skin	↑ markedly	Heat dissipation of fetal metabolism; warm extremities, palmar erythema, spider naevi, nasal congestion/epistaxis
Breasts	↑	Mammary hypertrophy and lactation preparation
Brain, liver	Essentially unchanged	Liver receives a smaller proportion of the increased CO

126.5 Blood pressure and vascular resistance

Vasodilator mediator	Action in pregnancy
Progesterone	Smooth-muscle relaxation; ↓ vascular tone; also venous dilatation (varicosities, haemorrhoids)
Nitric oxide	↑ eNOS activity (oestrogen, shear stress) → ↓ SVR; mediates refractoriness to angiotensin II
Prostacyclin (PGI_2)	↑ Production; PGI_2:TXA_2 ratio favours vasodilatation (reversed in pre-eclampsia — basis of low-dose aspirin prophylaxis)
Relaxin (corpus luteum)	Early systemic vasodilatation, ↑ arterial compliance, ↑ renal flow via endothelin-B/NO pathway
Oestrogen	↑ NO, ↑ uterine blood flow, ↑ angiotensinogen
Low-resistance uteroplacental circulation	Acts as an arteriovenous shunt in parallel with the systemic circulation (later contribution)

■ CLINICAL CORRELATION

- Measure BP **seated or semi-recumbent**, using an appropriately sized cuff; use **Korotkoff V** (disappearance) for diastolic (NICE).
- Pre-eclampsia is characterised by **↑ SVR, vasoconstriction, ↓ plasma volume, endothelial dysfunction and ↑ capillary permeability** — the reverse of normal adaptation.
- A BP that fails to fall in the second trimester may be an early marker of later hypertensive disease.

126.6 Aortocaval compression and supine hypotensive syndrome

Supine position > ≈ **20 weeks** ▶ Gravid uterus compresses **IVC** (± aorta) ▶ ↓ Venous return → ↓ preload ▶ ↓ SV and **CO (by up to ≈ 25–30%)** ▶ Maternal hypotension, pallor, sweating, nausea, syncope ▶ ↓ Uteroplacental perfusion → **fetal heart rate decelerations**

- Compression begins from ≈ 20 weeks (earlier with multiple pregnancy/polyhydramnios) and occurs in **most women at term**; only ≈ 10% become symptomatic (supine hypotensive syndrome) because of collateral venous return (azygos and paravertebral veins) and sympathetic compensation.
- **Aortic compression** reduces flow to the uterus and lower limbs even when brachial BP is maintained ('concealed' hypotension).
- Compensation is impaired by **regional (spinal/epidural) or general anaesthesia** (sympathetic block) — hence routine **left lateral tilt (≈ 15°)** or left uterine displacement for caesarean section.

> 20 wk
Onset of significant IVC compression

↓ CO 25–30%
When supine at term

15° tilt
Left lateral / manual uterine displacement

4–5 min
Perimortem CS: start by 4, deliver by 5 min

■ RED FLAGS

- In **maternal cardiac arrest** (> ≈ 20 weeks or a palpable fundus at/above the umbilicus) use **manual left uterine displacement** during CPR — a lateral tilt compromises chest compression efficacy.
- Perform **perimortem caesarean section (resuscitative hysterotomy)**, aiming for delivery within **5 minutes** of arrest (start by 4 minutes) if there is no return of spontaneous circulation — primarily to improve **maternal** resuscitation by relieving aortocaval compression.

126.7 Structural cardiac changes and normal clinical findings

Structural change	Detail
Position	Heart displaced upward, to the left and rotated anteriorly by the elevated diaphragm; apex beat displaced laterally (≈ 4th intercostal space); ↑ cardiothoracic ratio on CXR
Chamber size	All four chambers dilate; LV end-diastolic dimension ↑
LV mass	↑ ≈ 30–50% (physiological eccentric hypertrophy); regresses postpartum
Valves	Annular dilatation → mild physiological tricuspid, pulmonary and mitral regurgitation on echo
Pericardium	Small, asymptomatic pericardial effusion is common in the third trimester
Diastolic function	Altered filling (E/A ratio changes in late pregnancy)

Normal in pregnancy	Requires investigation (echo, cardiology review)
Ejection systolic (flow) murmur at the left sternal edge/pulmonary area (> 90% of women)	Any diastolic murmur
Third heart sound (S3)	Fourth heart sound (S4)
Loud S1 with exaggerated splitting	Loud (grade ≥ 3/6), harsh or pansystolic murmur; murmur radiating to the carotids
Mammary souffle (continuous; disappears with pressure of the stethoscope)	Fixed splitting of S2 (ASD)
Venous hum; bounding, collapsing pulse	Syncope on exertion; chest pain

Normal in pregnancy	Requires investigation (echo, cardiology review)
Peripheral oedema; mildly raised JVP with prominent waves	Orthopnoea, PND, persistent tachycardia > 110–120 bpm, ↓ SpO_2
Atrial/ventricular ectopics, sinus tachycardia	Sustained arrhythmias; breathlessness disproportionate to gestation
Palpitations, dizziness, ↓ exercise tolerance	Signs of heart failure (basal crepitations, raised JVP with hepatomegaly)

■ EXAM TIP

- Physiological breathlessness of pregnancy is due to **progesterone-driven hyperventilation** (↑ tidal volume) and is **not** associated with tachypnoea, hypoxia or exercise-limiting symptoms.

126.8 Labour, delivery and the puerperium

Period	Cardiac output	Mechanism
First stage of labour	↑ ≈ 15% (latent) to 30% (active) above pre-labour	Pain, anxiety → sympathetic activation; each contraction autotransfuses ≈ 300–500 mL from the uterus
Second stage	↑ ≈ 45–50%	Maximal contractions + expulsive effort; Valsalva (↑ intrathoracic pressure, ↓ venous return then rebound)
Immediately after delivery (first 1–2 h)	↑ 60–80% above pre-labour — the highest-risk period	Uterine involution → autotransfusion ≈ 500 mL; relief of IVC compression; mobilisation of extravascular fluid
24–72 hours postpartum	Remains elevated	Fluid shifts; risk of pulmonary oedema in women with cardiac disease or pre-eclampsia
≈ 2 weeks postpartum	Largely returned towards normal (HR and SV fall)	Diuresis (days 2–5)
12–24 weeks postpartum	Complete return to pre-pregnancy values	LV hypertrophy regresses over months

■ CLINICAL CORRELATION

- **Epidural analgesia** attenuates the pain-related ↑ CO (helpful in many cardiac lesions) but causes vasodilatation — beware fixed-output lesions.
- **Oxytocin** bolus causes vasodilatation, hypotension and tachycardia (give slowly/infusion in cardiac disease); **ergometrine** causes vasoconstriction and hypertension — avoided in hypertensive disease and many cardiac lesions; **carboprost** may cause bronchospasm and pulmonary hypertension.
- Supine position, aortocaval compression and haemorrhage interact: always tilt and resuscitate.

126.9 Clinical applications — maternal cardiac disease

Physiological changes predict which lesions are tolerated: **regurgitant lesions** generally do well (↓ SVR reduces regurgitant fraction), whereas **stenotic, fixed-output lesions and pulmonary hypertension** tolerate the ↑ CO, ↑ HR and ↑ volume poorly.

Condition	Why pregnancy is dangerous	Highest-risk time
Mitral stenosis	↑ HR shortens diastolic filling time; ↑ volume ↑ LA pressure → pulmonary oedema, AF	Late 2nd/3rd trimester; immediately postpartum

Condition	Why pregnancy is dangerous	Highest-risk time
Aortic stenosis (severe)	Fixed output cannot rise to meet demand; ↓ SVR and hypovolaemia (haemorrhage, regional block) → collapse, coronary hypoperfusion	Labour, delivery, anaesthesia
Pulmonary arterial hypertension / Eisenmenger	RV cannot cope with ↑ volume; ↓ SVR ↑ right-to-left shunting; thromboembolism; maternal mortality historically ≈ 25–50%	Late pregnancy and first weeks postpartum — pregnancy advised against (mWHO class IV)
Marfan syndrome / aortopathy	↑ CO and SV, hormonal effects on the aortic media → dissection	3rd trimester and postpartum; high risk if aortic root > 4.5 cm (Marfan)
Peripartum cardiomyopathy	LV systolic dysfunction (LVEF < 45%) in last month of pregnancy or within 5 months postpartum, no other cause	Postpartum most common
Ischaemic heart disease	↑ O_2 demand, hypercoagulability; spontaneous coronary artery dissection (SCAD) is a leading cause of pregnancy MI	Third trimester and early puerperium

■ **KEY FACTS**

- **Cardiac disease is the leading single cause of indirect maternal death** in the UK (MBRRACE-UK) — breathlessness, chest pain, orthopnoea and persistent tachycardia must not be dismissed as 'normal pregnancy'.
- Risk stratification uses the **modified WHO (mWHO) classification** (I–IV) together with CARPREG/ZAHARA scores; manage in a multidisciplinary **pregnancy heart team**.

126.10 Quick-revision checklist

■ **MNEMONIC / MEMORY AID**

'Volume up, Resistance down, Pressure dips, Pressures (CVP/PCWP) same'

Plasma > CO > RBC: '45 – 40 – 25' (%)

BP nadir = 'mid-pregnancy' (16–24 weeks); **plasma volume peak = 32–34 weeks**; **CO peak = 25–32 weeks**; **maximum CO = immediately postpartum**.