PIPADOR REVISION

MRCOG Part 1 Revision Notes

Fetal Circulation and Physiology

PHYSIOLOGY • LECTURE 128

VISUAL EXAMINATION REVISION NOTES

2026 Edition

128

MRCOG PART 1 REVISION NOTES | PHYSIOLOGY

Fetal Circulation and Physiology

Fetal shunts, oxygen transport, cardiac physiology, response to hypoxia and the transition at birth

DV	FO	DA	P50 2.5 kPa
Ductus venosus: bypasses the LIVER	Foramen ovale: RA → LA	Ductus arteriosus: bypasses the LUNGS	HbF left-shifted (adult 3.6 kPa)

■ **KEY FACTS: Learning objectives**

- Trace the course of blood through the fetal circulation and explain the role of the three shunts.
- Describe fetal oxygen transport: fetal haemoglobin, the double Bohr and Haldane effects.
- Describe fetal cardiac physiology and control of fetal heart rate.
- Explain the fetal cardiovascular response to hypoxia and its Doppler correlates.
- Describe the circulatory transition at birth and the adult remnants of fetal structures.
- Interpret umbilical cord blood gases and relate fetal physiology to clinical scenarios (PDA, PPHN, duct-dependent lesions, NSAIDs).

128.1 Principles of the fetal circulation

In the fetus the **placenta**, not the lungs, is the organ of gas exchange. The lungs are fluid-filled with a **high pulmonary vascular resistance (PVR)**, whereas the placenta provides a **low-resistance** systemic bed. The fetal circulation is therefore arranged **in parallel** (both ventricles pump into the systemic circulation) rather than in series, with three shunts directing the best-oxygenated blood to the heart and brain.

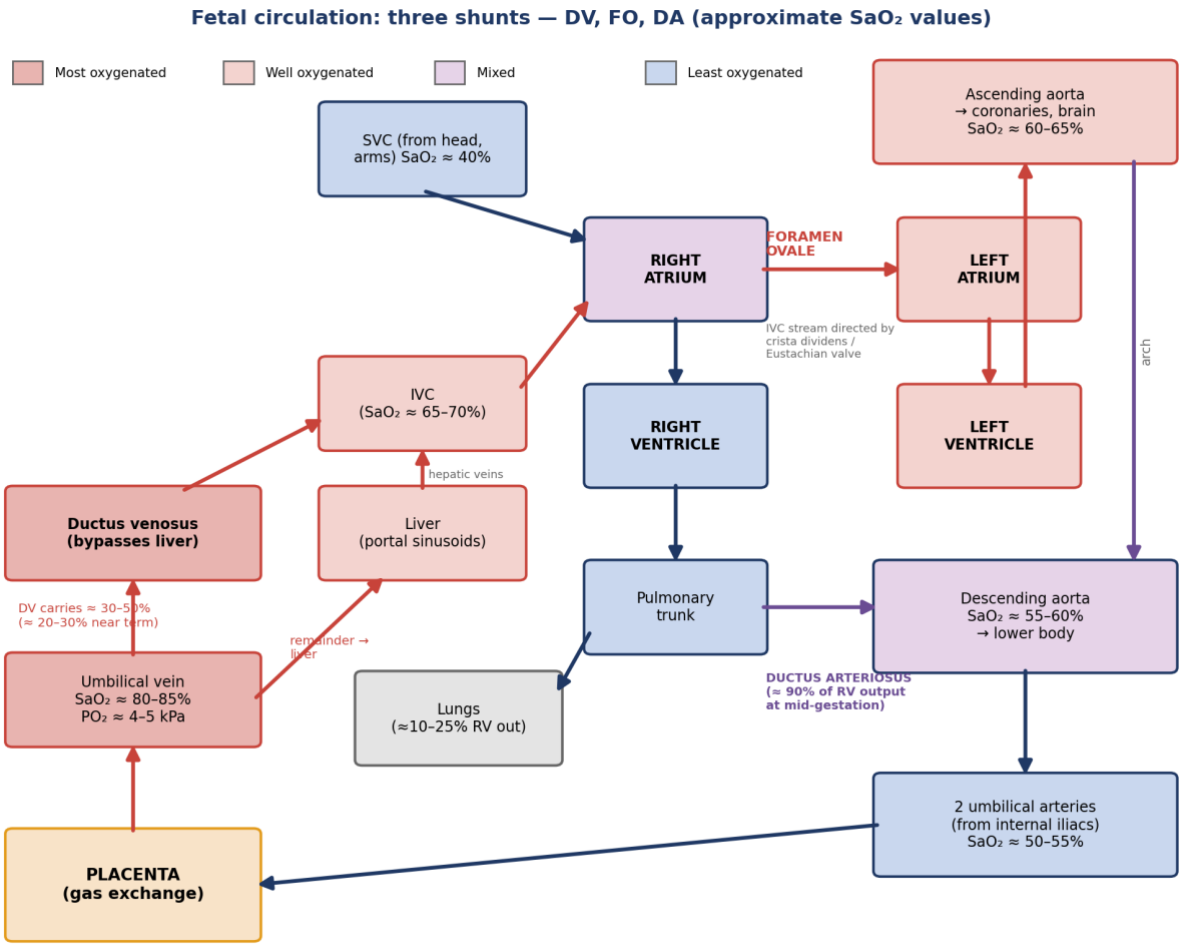


Figure 128.1 — Schematic of the fetal circulation with approximate oxygen saturations

Shunt	Location / connects	Function	Closure after birth	Adult remnant
Ductus venosus (DV)	Umbilical vein (left portal vein) → IVC	Diverts well-oxygenated umbilical venous blood past the liver (≈ 30–50% mid-gestation; ≈ 20–30% near term); high-velocity jet streams preferentially towards the foramen ovale	Functional closure within hours–days (loss of umbilical flow); fibrosed by ≈ 1–3 weeks	Ligamentum venosum
Foramen ovale (FO)	Right atrium → left atrium (flap = septum primum)	Crista dividens and the Eustachian valve direct the oxygen-rich IVC/DV stream across to the LA → LV → ascending aorta (coronaries and brain)	Functional closure at birth (LA pressure > RA pressure); anatomical fusion over months	Fossa ovalis (patent foramen ovale in ≈ 25% of adults)
Ductus arteriosus (DA)	Pulmonary trunk → descending aorta (distal to left subclavian)	Diverts ≈ 90% of RV output away from the high-resistance lungs (mid-gestation)	Functional closure ≈ 12–72 h (↑ PO_2, ↓ PGE_2); anatomical closure ≈ 2–3 weeks	Ligamentum arteriosum (related to the left recurrent laryngeal nerve)

Other fetal vessel	Remnant
Umbilical vein (single, left)	Ligamentum teres (round ligament of the liver), in the free edge of the falciform ligament
Umbilical arteries (two, from internal iliac arteries)	Distal: medial umbilical ligaments; proximal: patent as superior vesical arteries
Allantois / urachus	Median umbilical ligament

■ MNEMONIC / MEMORY AID

Oxygenation order (best → worst): umbilical vein > DV/IVC stream > LA/LV/ascending aorta (brain, heart) > descending aorta > umbilical arteries > SVC.

'**Veins carry oxygen to the fetus, Arteries take it Away**' — the umbilical vein carries the most oxygenated blood; the umbilical arteries return deoxygenated blood to the placenta.

Remnants: '**V**enosum ← **V**enosus; **A**rteriosum ← **A**rteriosus; **teres** = umbilical vein; **ovale → fossa**'.

128.2 The path of blood — step by step

1. Oxygenated blood leaves the placenta in the **umbilical vein** (SaO_2 ≈ 80–85%, PO_2 ≈ 4–5 kPa) and enters the fetus at the umbilicus.
2. At the liver, part of the flow passes through the **portal/hepatic sinusoids** (left lobe preferentially), but a high-velocity stream crosses the **ductus venosus** directly to the IVC.
3. In the IVC, DV blood forms a **preferential stream** that remains relatively unmixed with deoxygenated blood from the lower body and liver.
4. The **crista dividens** (lower edge of the septum secundum) splits IVC flow: the oxygen-rich stream crosses the **foramen ovale** to the LA; the rest enters the RA.
5. LA → LV → **ascending aorta** (SaO_2 ≈ 60–65%) → coronary arteries and brachiocephalic vessels — **the heart and brain receive the best-oxygenated blood**.
6. Deoxygenated **SVC** blood (SaO_2 ≈ 40%) passes preferentially through the tricuspid valve → RV → pulmonary trunk.
7. Because PVR is high, most RV output passes through the **ductus arteriosus** into the **descending aorta** (SaO_2 ≈ 55–60%), supplying the lower body.
8. Blood returns to the placenta via the two **umbilical arteries** (branches of the internal iliac arteries).
9. Pulmonary venous return (from the small pulmonary flow) → LA.

Ventricular output distribution

Parameter	Value (approximate, near term)
Combined ventricular output (CVO)	≈ 400–500 mL/kg/min (several-fold higher per kg than adult)
Right ventricle : left ventricle	RV dominant — RV ≈ 55–60% of CVO; LV ≈ 40–45%
Placental flow	≈ 30–40% of CVO (declines as a proportion near term)
Pulmonary flow	≈ 10% mid-gestation, rising to ≈ 20–25% near term as PVR begins to fall
Ductus arteriosus flow	Most of RV output (≈ 90% mid-gestation)
Brain	≈ 15–20% of CVO (↑ in hypoxia)

128.3 Fetal oxygen transport

The fetus lives at a **low PO_2** ('Everest in utero': fetal arterial PO_2 ≈ 2.5–3.5 kPa) yet maintains adequate oxygen delivery through several adaptations.

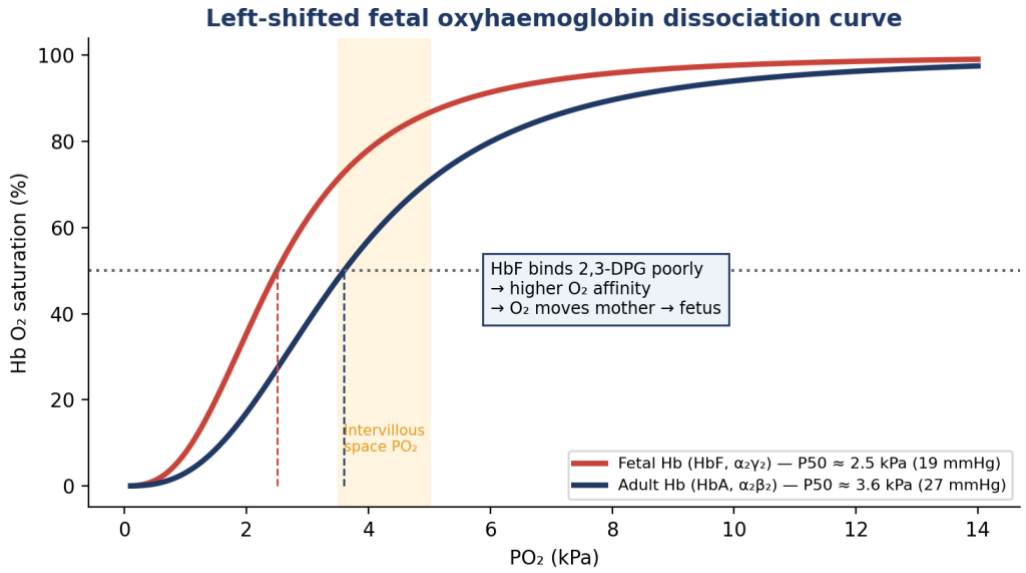


Figure 128.2 — Fetal and adult oxyhaemoglobin dissociation curves

Adaptation	Mechanism / value
Fetal haemoglobin (HbF, $\alpha_2\gamma_2$)	γ-chains bind 2,3-DPG poorly → left-shifted ODC (P50 ≈ 2.5 kPa / 19 mmHg vs adult ≈ 3.6 kPa / 27 mmHg) → higher O_2 affinity
High haemoglobin concentration	Hb ≈ 160–180 g/L at term → ↑ O_2-carrying capacity
High cardiac output and organ blood flow	CVO per kg ≈ 3–4 × adult resting CO
Double Bohr effect	Fetal CO_2 passes to the mother: maternal blood becomes more acidic (ODC shifts right → releases O_2) while fetal blood becomes more alkaline (ODC shifts left → takes up O_2)
Double Haldane effect	Maternal deoxygenation increases her capacity to carry CO_2; fetal oxygenation decreases fetal CO_2 affinity → favours CO_2 transfer fetus → mother
Maternal hyperventilation	Maternal $PaCO_2$ ↓ (≈ 4 kPa) → steep CO_2 gradient for fetal CO_2 elimination
Preferential streaming	DV/FO pathway delivers the best-oxygenated blood to the brain and myocardium

Haemoglobin and haemopoiesis

Stage	Site of haemopoiesis	Haemoglobin types
Weeks 3–8	Yolk sac (mesoblastic phase)	Embryonic: Gower 1 ($\zeta_2\varepsilon_2$), Gower 2 ($\alpha_2\varepsilon_2$), Portland ($\zeta_2\gamma_2$)
≈ 6 weeks to mid-gestation	Liver (main site, hepatic phase), also spleen	HbF ($\alpha_2\gamma_2$) predominates
From ≈ 18–20 weeks onward	Bone marrow (myeloid phase) becomes dominant in the third trimester	HbF; β-chain synthesis starts rising from ≈ 30–32 weeks
Birth	Bone marrow	HbF ≈ 60–80% of total Hb
6–12 months postnatal	Bone marrow	Adult pattern: HbA ($\alpha_2\beta_2$) ≈ 97%, HbA_2 ($\alpha_2\delta_2$) ≈ 2–3%, HbF < 1%

■ CLINICAL CORRELATION

- **β-thalassaemia major and sickle cell disease** do not manifest at birth because HbF (no β-chains) predominates — symptoms appear at 3–6 months.
- **α-thalassaemia major (Hb Bart's, γ_4)** causes **hydrops fetalis** in utero because α-chains are required for HbF.
- **Kleihauer–Betke test** exploits the resistance of HbF to acid elution to quantify fetomaternal haemorrhage (see 129).

128.4 Fetal cardiac physiology

- **Heart development**: the heart begins to beat at ≈ **day 22 post-conception** (≈ 5 weeks of gestation from LMP); cardiac activity is visible on TVS by ≈ 6 weeks; septation complete by ≈ 8 weeks post-conception.
- **Limited Frank–Starling reserve**: the fetal myocardium has fewer, disorganised contractile elements, immature sarcoplasmic reticulum and T-tubules (relies more on trans-sarcolemmal Ca^{2+}) and is **less compliant** → it operates near the top of its Starling curve; **CO is largely heart-rate dependent**.
- Fetal bradycardia therefore reduces cardiac output substantially.
- Ventricles are **pressure-matched** (RV and LV pressures similar) because the DA links the pulmonary and systemic circulations.
- **Fetal heart rate**: ≈ 170–180 bpm at 9–10 weeks, falling to a baseline of **110–160 bpm** at term as **parasympathetic (vagal) tone matures**; sympathetic innervation develops earlier.
- **Baseline variability** reflects an intact autonomic nervous system interaction (cortex, medulla, vagus) — reduced by hypoxia, sleep cycles (up to ≈ 40–50 minutes), opioids, $MgSO_4$, prematurity.
- **Accelerations** reflect somatic activity and a responsive, non-acidotic fetus (sympathetic).

128.5 Fetal response to hypoxia

Acute hypoxaemia (↓ PaO_2) ▶ Carotid **chemoreceptor** stimulation ▶ Vagal **bradycardia** (deceleration) + sympathetic peripheral **vasoconstriction** ▶ **Redistribution ('brain-sparing')**: ↑ flow to brain, heart, adrenals ▶ ↓ Flow to gut, kidneys, lungs, skin, limbs ▶ Prolonged: anaerobic metabolism → **lactic (metabolic) acidosis**

Consequence of redistribution	Clinical correlate
↓ Renal perfusion → ↓ fetal urine output	Oligohydramnios
↓ Gut perfusion	Meconium passage (± maturity), necrotising enterocolitis after birth
Preferential cerebral flow	Asymmetric growth restriction (head-sparing; ↓ abdominal circumference)
↑ Cerebral flow — vasodilatation	↓ MCA pulsatility index; ↓ cerebroplacental ratio (CPR = MCA PI ÷ UA PI)
↑ Placental resistance (failing placenta)	↑ UA PI → absent → reversed end-diastolic flow
Cardiac decompensation (↑ RA pressure)	Ductus venosus: absent/reversed a-wave; pulsatile umbilical venous flow — late, pre-terminal signs
Myocardial hypoxia / acidosis	Late decelerations, reduced variability, sinusoidal or preterminal CTG

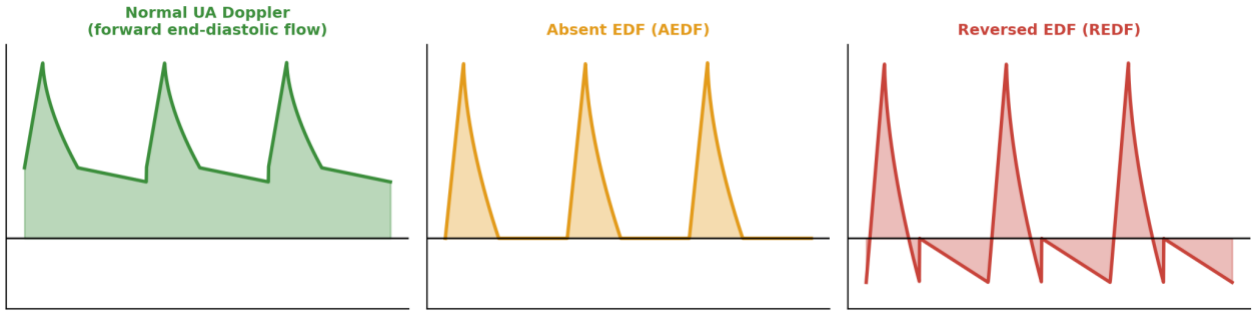


Figure 128.3 — Umbilical artery Doppler waveforms: progressive increase in placental resistance

128.6 Transition to the neonatal circulation at birth

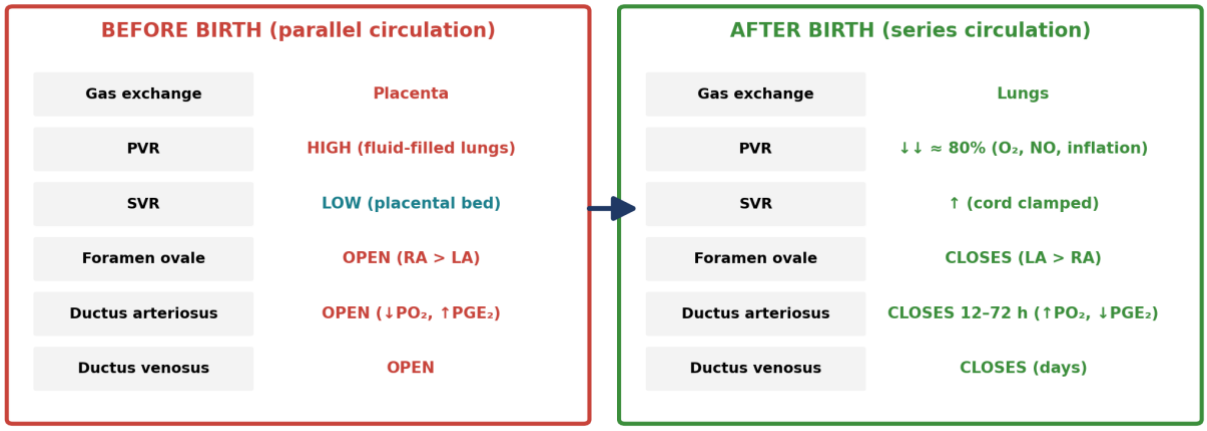


Figure 128.4 — The circulatory transition at birth

First breaths → lung expansion, ↑ PO_2, NO ▶ **PVR ↓↓**, pulmonary flow ↑ 8–10× ▶ Cord clamped → **SVR ↑**, ↓ IVC return ▶ **LA > RA** → foramen ovale closes ▶ ↑ PO_2 + ↓ PGE_2 → **ductus arteriosus** closes

■ EXAM TIP

- **Delayed cord clamping** (≥ 1 minute — NICE/Resuscitation Council UK; ideally after lung aeration) increases neonatal blood volume and iron stores and smooths the cardiovascular transition; it reduces mortality and IVH in preterm infants.
- Ductal patency is maintained in utero by **low PO_2 and circulating PGE_2**; closure is promoted by **↑ PO_2 and ↓ PGE_2**.

128.7 Clinical applications

Situation	Physiology	Clinical point
Maternal NSAIDs (e.g. indomethacin tocolysis)	Inhibit COX → ↓ PGE_2 → premature constriction of the DA (sensitivity ↑ after ≈ 30–32 weeks); also ↓ fetal renal output	Oligohydramnios, fetal pulmonary hypertension, tricuspid regurgitation, hydrops — avoid NSAIDs in the third trimester
Patent ductus arteriosus (PDA)	Persistence (prematurity, hypoxia, rubella, high altitude) → left-to-right shunt	Continuous 'machinery' murmur; closure with ibuprofen/indomethacin/paracetamol (COX inhibitors) or surgical/catheter ligation
Duct-dependent congenital heart disease	Pulmonary flow (e.g. pulmonary atresia) or systemic flow (e.g. hypoplastic left heart, critical coarctation) depends on the DA	Collapse when the duct closes (days 2–7): keep it open with prostaglandin E_1 (alprostadil) infusion
Persistent pulmonary hypertension of the newborn (PPHN)	PVR fails to fall (meconium aspiration, sepsis, CDH, asphyxia) → right-to-left shunting at FO and DA	Pre-ductal (right hand) SpO_2 > post-ductal (foot) SpO_2; treat with O_2, ventilation, inhaled NO
Twin–twin transfusion syndrome	Unbalanced placental vascular anastomoses in monochorionic twins	Recipient: polyhydramnios, cardiac strain; donor: oligohydramnios, FGR — laser ablation
Fetal anaemia (red-cell alloimmunisation, parvovirus B19)	↓ Viscosity and ↑ CO → ↑ flow velocity	↑ MCA peak systolic velocity (> 1.5 MoM) predicts moderate/severe anaemia → intrauterine transfusion
Patent foramen ovale in adults	Incomplete fusion (≈ 25%)	Paradoxical embolism (stroke), decompression illness

Umbilical cord blood gases

Variable	Umbilical artery (reflects fetus)	Umbilical vein (reflects placenta)
pH	≈ 7.20–7.30 (mean ≈ 7.26)	≈ 7.30–7.40 (mean ≈ 7.35)
PCO_2	≈ 6.5–7.5 kPa	≈ 5.0–5.5 kPa
PO_2	≈ 2–3 kPa	≈ 3.5–4.5 kPa
Base deficit	< 8–10 mmol/L	< 8 mmol/L

- **Paired samples** are required: the artery has a **lower pH and higher PCO_2** than the vein (arterio-venous pH difference ≈ 0.05–0.1). If values are identical, both samples are probably venous.
- **Pathological fetal acidaemia** (associated with hypoxic–ischaemic encephalopathy): arterial **pH < 7.00 and base deficit ≥ 12 mmol/L** (metabolic acidosis).
- **Respiratory acidosis** (↑ PCO_2, normal base deficit) reflects acute cord compression and is usually benign; **metabolic acidosis** (↑ lactate, ↑ base deficit) reflects prolonged hypoxia.
- Fetal blood sampling during labour: pH ≥ 7.25 normal; 7.21–7.24 borderline; ≤ 7.20 abnormal (lactate ≥ 4.9 mmol/L abnormal).

128.8 Quick-revision checklist

■ KEY FACTS

- Three shunts: **DV** (bypasses liver), **FO** (RA → LA), **DA** (pulmonary trunk → aorta).
- Highest SaO_2: **umbilical vein**; ascending aorta/brain blood is better oxygenated than descending aorta blood.
- HbF: **$\alpha_2\gamma_2$**, binds 2,3-DPG poorly → **left-shifted ODC, P50 ≈ 2.5 kPa**.
- Fetal CO is **rate-dependent**; the RV is dominant.
- At birth: ↓ PVR, ↑ SVR, LA > RA pressure (FO closes), ↑ PO_2 and ↓ PGE_2 (DA closes).
- Remnants: ligamentum venosum (DV), ligamentum arteriosum (DA), fossa ovalis (FO), ligamentum teres (umbilical vein), medial umbilical ligaments (umbilical arteries).