PIPADOR REVISION

MRCOG Part 1 Revision Notes

Fetal Renal Physiology

PHYSIOLOGY • LECTURE 153

VISUAL EXAMINATION REVISION NOTES

2026 Edition

153

MRCOG PART 1 REVISION NOTES | PHYSIOLOGY

Fetal Renal Physiology

Development of the urinary tract, fetal renal function, amniotic fluid, congenital anomalies and neonatal transition

9–10 wk	≈ 16 wk	34–36 wk	< 24 h
Fetal urine production begins	Urine becomes main source of amniotic fluid	Nephrogenesis complete	First void in most newborns

■ KEY FACTS: Learning objectives

- Describe the embryological development of the kidneys, ureters and bladder.
- Describe fetal renal function and the fetal contribution to amniotic fluid.
- Describe neonatal renal maturation and its clinical consequences.
- Relate fetal renal physiology to congenital anomalies, oligohydramnios and pulmonary hypoplasia, and to fetotoxic drugs.

153.1 Development of the urinary tract

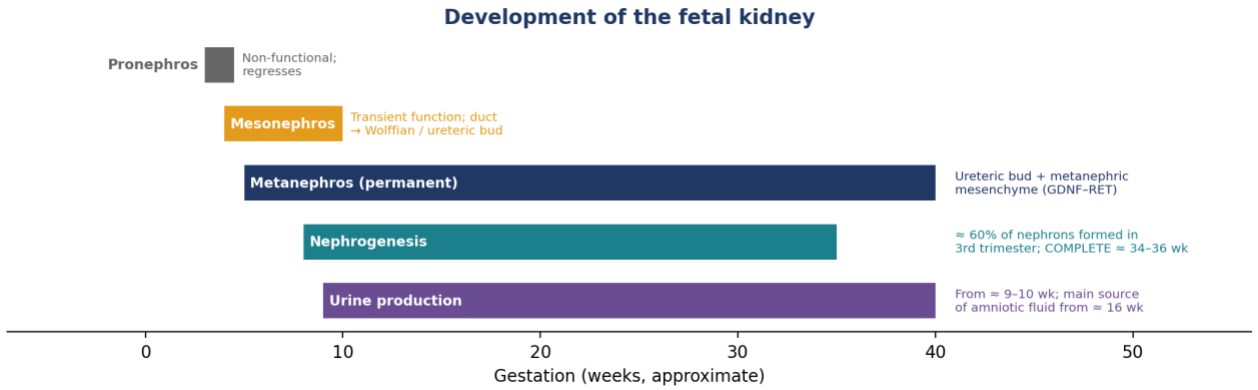


Figure 153.1 — Stages of kidney development

Structure	Origin	Key points / anomalies
Pronephros	Cervical intermediate mesoderm (≈ wk 4)	Rudimentary, regresses
Mesonephros	Thoracolumbar (≈ wk 4–8)	Transiently functional; mesonephric (Wolffian) duct persists → ureteric bud; male genital ducts
Metanephros	Sacral region (from ≈ wk 5)	Ureteric bud → ureter, pelvis, calyces, collecting ducts; metanephric mesenchyme (blastema) → glomeruli, tubules (to DCT). Reciprocal induction (GDNF–RET, WT1, PAX2). Failure → renal agenesis; abnormal → multicystic dysplastic kidney
Ascent and rotation	Pelvis → lumbar (wk 6–9)	Pelvic kidney; horseshoe kidney (lower poles fuse, trapped under the inferior mesenteric artery; ↑ in Turner)
Bladder & urethra	Urogenital sinus (cloaca divided by urorectal septum)	Allantois → urachus (median umbilical ligament) — patent urachus/cyst; posterior urethral valves (males)
Ureteric bud branching	Early bifurcation	Duplex kidney ± ectopic ureter/ureterocele (Weigert–Meyer rule: upper-pole ureter obstructs, lower-pole refluxes)

153.2 Fetal renal function

- The **placenta** performs excretion and fluid–electrolyte homeostasis — a fetus with absent kidneys has normal biochemistry at birth.
- Fetal kidneys receive only **≈ 3–5% of cardiac output** (high renal vascular resistance); **GFR is low** but rises with nephron number and renal blood flow.
- Fetal urine is **hypotonic and dilute** (immature concentrating ability: short loops of Henle, low urea gradient, reduced ADH responsiveness) and contains relatively high sodium.
- Urine production rises from ≈ 5 mL/h at 20 weeks to **≈ 30–50 mL/h at term (≈ 700–1200 mL/day)** — the major source of amniotic fluid from ≈ 16 weeks (Lecture 142).
- The fetal **RAAS is active** and important for renal development and GFR — the basis of **ACE inhibitor/ARB fetopathy**.

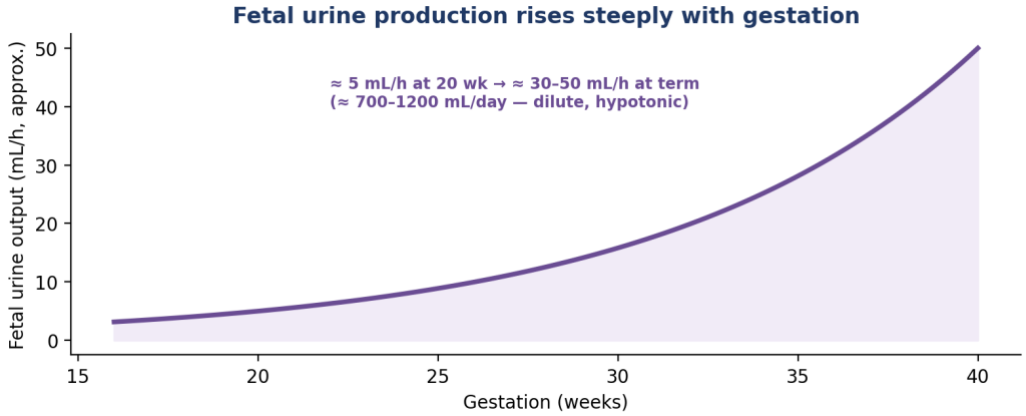


Figure 153.2 — Fetal urine output across gestation

Drug	Fetal renal effect
ACE inhibitors / ARBs (2nd/3rd trimester)	Renal tubular dysgenesis, anuria → oligohydramnios, pulmonary hypoplasia, skull ossification defects (hypocalvaria), limb contractures, neonatal hypotension/renal failure. 1st-trimester exposure: switch, low teratogenic risk
NSAIDs / indomethacin	↓ Fetal renal blood flow and urine output → oligohydramnios (+ ductus arteriosus constriction)
Aminoglycosides	Nephrotoxicity/ototoxicity
Lithium	Fetal nephrogenic DI → polyhydramnios

153.3 Oligohydramnios and pulmonary hypoplasia

Bilateral renal agenesis, bilateral MCDK, ARPKD or **LUTO** (posterior urethral valves) ▶ No/low fetal urine from ≈ 16 wk ▶ **Anhydramnios / severe oligohydramnios** ▶ Fetal compression + absent lung distension ▶ **Potter sequence**: pulmonary hypoplasia (lethal), flattened facies, limb deformities (talipes), IUGR

Condition	Prenatal ultrasound features	Notes
Bilateral renal agenesis	Anhydramnios from 16 wk; absent kidneys/bladder; absent renal arteries on colour Doppler; 'lying-down' adrenals	Lethal (pulmonary hypoplasia); unilateral agenesis compatible with normal life
Multicystic dysplastic kidney	Multiple non-communicating cysts, no normal parenchyma	Unilateral usually; check contralateral kidney (VUR, PUJO)
ARPKD (PKHD1)	Bilaterally enlarged, echogenic kidneys, oligohydramnios	Autosomal recessive; liver fibrosis; 25% recurrence

Condition	Prenatal ultrasound features	Notes
ADPKD	Enlarged echogenic kidneys (often normal liquor)	Autosomal dominant — scan parents
Pelviureteric junction obstruction	Renal pelvic dilatation without ureteric dilatation	Commonest cause of significant fetal hydronephrosis
Posterior urethral valves (LUTO)	Male; 'keyhole' sign (dilated bladder + posterior urethra), thick bladder wall, bilateral hydronephrosis, oligohydramnios	Fetal urine biochemistry for prognosis; vesico-amniotic shunting in selected cases
Renal pelvic dilatation (RPD)	Anteroposterior diameter ≥ ≈ 7 mm in 3rd trimester (thresholds vary)	Soft marker; postnatal ultrasound; consider VUR

Favourable fetal urine biochemistry in LUTO (serial vesicocentesis): Na^+ < 100 mmol/L, Cl^- < 90 mmol/L, osmolality < 200 mOsm/kg, β_2-microglobulin < 6 mg/L — hypotonic urine reflects preserved tubular function.

153.4 Nephron endowment and fetal programming

Each kidney has **≈ 1 million nephrons (range ≈ 200,000–2 million)**; ≈ 60% form in the 3rd trimester and **no new nephrons form after ≈ 34–36 weeks** (in preterm infants nephrogenesis continues briefly after birth but is often abnormal). **FGR, prematurity and maternal diabetes** reduce nephron number → glomerular hyperfiltration → **hypertension and CKD** in adult life (**Brenner hypothesis**; Lecture 146).

153.5 Neonatal renal transition

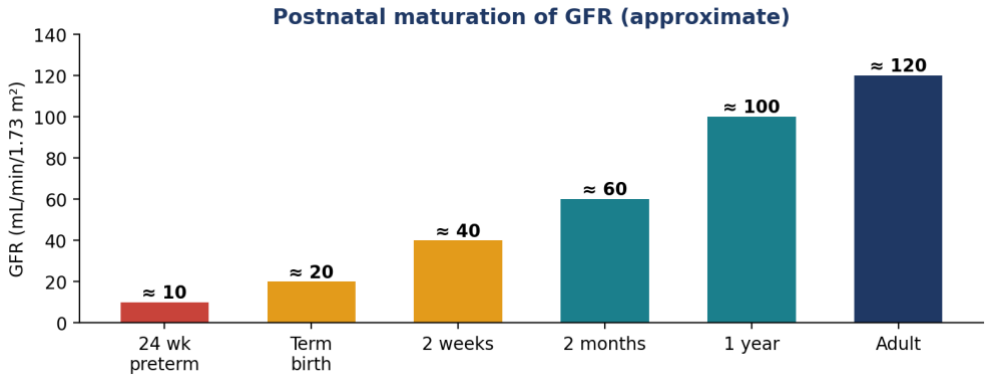


Figure 153.3 — GFR maturation after birth

Feature	Neonate	Clinical consequence
GFR	≈ 20 mL/min/1.73 m² at term; doubles by 2 weeks; adult values by 1–2 years	Prolonged half-life of renally excreted drugs (gentamicin, vancomycin — dose interval adjusted)
Concentrating ability	Max ≈ 600–700 mOsm/kg	Dehydration risk; hypernatraemia with poor feeding
Sodium	Preterm: tubular Na^+ wasting	Hyponatraemia of prematurity — supplementation
Acid–base	Low bicarbonate threshold, limited NH_4^+ excretion	Mild metabolic acidosis in preterm
Creatinine at birth	Reflects maternal creatinine (crosses placenta); falls over first week	Rising creatinine after 48 h suggests AKI
First void	≈ 90–95% within 24 h; ≈ 99% by 48 h	Delay → PUV, agenesis, dehydration

153.6 Must-know summary

■ EXAM TIP

- Pronephros → mesonephros → **metanephros**: **ureteric bud** = collecting system; **metanephric mesenchyme** = nephrons.
- Fetal urine from **9–10 wk**; main amniotic fluid source from **≈ 16 wk**; ≈ 700–1200 mL/day at term.
- Placenta, not the fetal kidney, maintains fetal homeostasis; fetal kidneys get ≈ 3–5% of CO.
- **Nephrogenesis complete by 34–36 wk**; low nephron number (FGR, preterm) → adult hypertension.
- ACEi/ARBs and NSAIDs → **oligohydramnios**; bilateral agenesis/LUTO → **Potter sequence**.

■ MNEMONIC / MEMORY AID

Potter sequence — 'POTTER': Pulmonary hypoplasia, Oligohydramnios, Twisted face (flattened), Twisted skin (wrinkled), Extremity defects, Renal agenesis. **Horseshoe kidney 'hooked' by the IMA**.