PIPADOR REVISION

MRCOG Part 1 Revision Notes

Testicular Hormones

ENDOCRINOLOGY • LECTURE 181

VISUAL EXAMINATION REVISION NOTES

2026 Edition

181

MRCOG PART 1 REVISION NOTES | ENDOCRINOLOGY

Testicular Hormones

Testosterone, DHT and oestradiol in males, AMH, inhibin B and INSL3, and disorders of androgen production and action

≈ 95%	≈ 100×	≈ 80%	8–12 wk
Male testosterone from Leydig cells	Intratesticular vs serum testosterone	Male oestradiol from peripheral aromatase	hCG-driven fetal testosterone peak

■ KEY FACTS: Learning objectives

- Describe testicular hormone synthesis (testosterone, DHT, oestradiol, AMH, inhibin B, INSL3) and regulation.
- Describe the actions of testosterone, DHT and oestradiol in males across the lifespan.
- Describe male hypogonadism and disorders of androgen action relevant to O&G.

181.1 Testicular hormones and their control

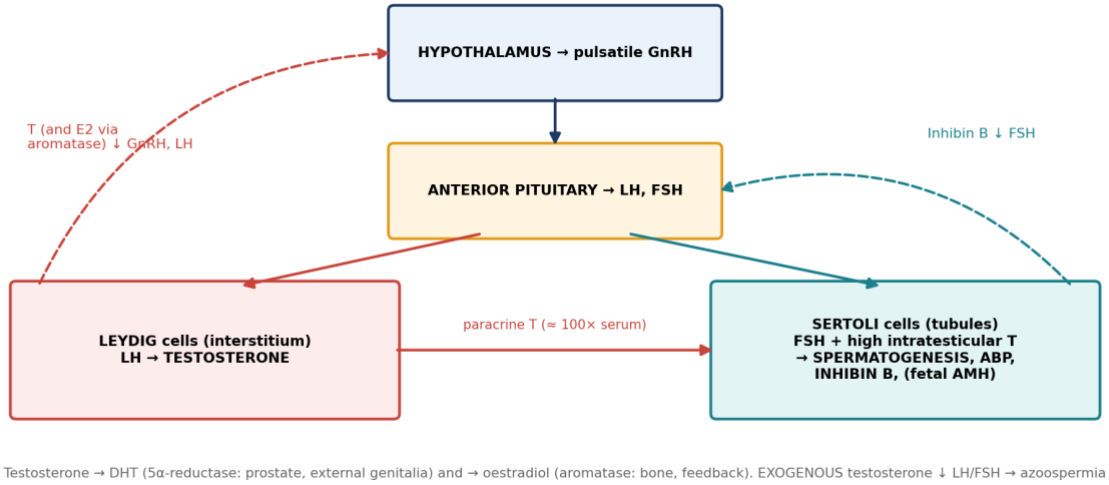


Figure 181.1 — The hypothalamic–pituitary–testicular axis

Hormone	Source	Actions
Testosterone	Leydig cells (LH → StAR, CYP11A1, CYP17, 17β-HSD3)	Spermatogenesis (high local levels with FSH), Wolffian ducts, muscle, bone, libido, erythropoiesis; feedback on GnRH/LH
DHT	5α-reductase type 2 (prostate, genital skin)	External genitalia and prostate (fetus); prostate growth, male-pattern hair and baldness (adult)
Oestradiol	Aromatase (adipose, bone, brain; ≈ 20% testicular)	Epiphyseal closure, bone density, libido, negative feedback on LH; excess → gynaecomastia
AMH	Sertoli cells (fetal)	Müllerian duct regression (≈ 8 wk) — absence → persistent Müllerian duct syndrome
Inhibin B	Sertoli cells	↓ FSH; marker of spermatogenesis
INSL3	Leydig cells	Transabdominal testicular descent (gubernaculum)

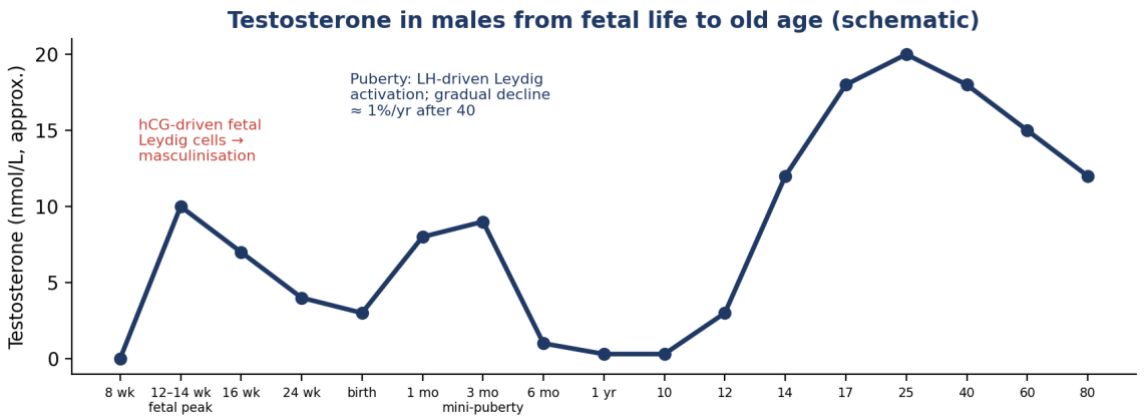


Figure 181.2 — Testosterone across the male lifespan

181.2 Actions across life

Period	Key events
Fetal	Placental hCG stimulates Leydig cells (8–14 wk) → masculinisation; fetal LH maintains later; testicular descent (INSL3 then androgens; complete by ≈ 35 wk)
Mini-puberty (1–6 months)	Transient HPG activation — penile growth, Sertoli proliferation; diagnostic window for hypogonadism
Puberty	Testicular enlargement (≥ 4 mL), genital growth, voice, muscle, growth spurt, spermatogenesis, libido, acne
Adult	Spermatogenesis, sexual function, bone and muscle, erythropoiesis
Ageing	Testosterone falls ≈ 1%/year after 40; SHBG ↑ → free T ↓; late-onset hypogonadism (obesity, diabetes)

181.3 Disorders

Category	Examples	Hormones
Primary (hypergonadotrophic) hypogonadism	Klinefelter (47,XXY), mumps orchitis, chemotherapy/radiotherapy, torsion, cryptorchidism	↓ T, ↑ LH/FSH, ↓ inhibin B
Secondary (hypogonadotrophic)	Kallmann, pituitary tumours, hyperprolactinaemia, obesity, opioids, anabolic steroids	↓ T, ↓ LH/FSH
Androgen resistance	Complete/partial AIS (46,XY; female/ambiguous phenotype, testes, no uterus)	↑ T, ↑ LH (resistant feedback)
5α-reductase deficiency	46,XY undervirilised at birth, virilises at puberty	Normal/↑ T, ↑ T:DHT ratio
Oestrogen excess	Obesity, liver disease, drugs (spironolactone), hCG-secreting tumours	Gynaecomastia

■ CLINICAL CORRELATION

- **Exogenous testosterone/anabolic steroids** suppress LH/FSH → **azoospermia** (may take months–years to recover); hCG/FSH can restore spermatogenesis.
- **AIS** (Lecture 150): gonadectomy after puberty (malignancy risk), vaginal dilators, HRT; **testicular feminisation** term obsolete.
- Fetal exposure to **anti-androgens** (finasteride, spironolactone, cyproterone) → undervirilisation of a male fetus — contraception required.

181.4 Must-know summary

■ EXAM TIP

- Leydig (LH) → testosterone; Sertoli (FSH) → spermatogenesis, **inhibin B, AMH**; Leydig **INSL3** → descent.
- **DHT** → external genitalia/prostate; **oestradiol** → epiphyseal closure and feedback (from aromatase).
- Fetal testosterone is **hCG-driven** (8–14 wk); mini-puberty at 1–6 months.
- Klinefelter: ↓ T, ↑ FSH/LH; Kallmann: ↓ T, ↓ FSH/LH; AIS: ↑ T, ↑ LH.

■ MNEMONIC / MEMORY AID

Testicular hormones — 'TEA-I-I': Testosterone, oEstradiol (via aromatase), AMH, Inhibin B, INSL3.