PIPADOR REVISION

MRCOG Part 1 Revision Notes

Diabetes Insipidus

ENDOCRINOLOGY • LECTURE 188

VISUAL EXAMINATION REVISION NOTES

2026 Edition

MRCOG PART 1 REVISION NOTES | ENDOCRINOLOGY

188 Diabetes Insipidus

Classification, ADH physiology in pregnancy, gestational DI, diagnosis and management

> 3 L/day	< 300	Vasopressinase	Desmopressin
Polyuria (dilute urine)	Urine osmolality (mOsm/kg) typical	Placental enzyme degrading ADH	Resistant to vasopressinase

■ KEY FACTS: Learning objectives

- Define and classify diabetes insipidus (cranial, nephrogenic, gestational) and distinguish from primary polydipsia.
- Describe the physiology of ADH and the changes in osmoregulation in pregnancy.
- Describe the investigation and management of DI in pregnancy, labour and the puerperium.

188.1 Physiology and classification

ADH (vasopressin) from the supraoptic/paraventricular nuclei acts on renal **V_2 receptors → aquaporin-2** (Lecture 151). In pregnancy the **osmostat resets ≈ 10 mOsm/kg lower** and the placenta produces **vasopressinase** (cysteine aminopeptidase), increasing ADH clearance ≈ 4-fold — normally compensated by ↑ secretion.

Type	Mechanism	Causes
Cranial (central)	↓ ADH secretion	Idiopathic/autoimmune, pituitary surgery, tumours (craniopharyngioma), trauma, lymphocytic hypophysitis, infiltrative disease; rarely Sheehan's (posterior usually spared)
Nephrogenic	Renal resistance to ADH	Lithium, hypercalcaemia, hypokalaemia, renal disease, X-linked V_2 receptor or AQP2 mutations
Gestational (transient)	↑ Vasopressinase degradation ± ↓ hepatic clearance of the enzyme	3rd trimester; AFLP, HELLP, pre-eclampsia, multiple pregnancy; ≈ 2–4 per 100,000; resolves within days–weeks postpartum
Primary polydipsia	Excess water intake suppresses ADH	Psychiatric illness, drugs causing dry mouth

188.2 Diagnosis

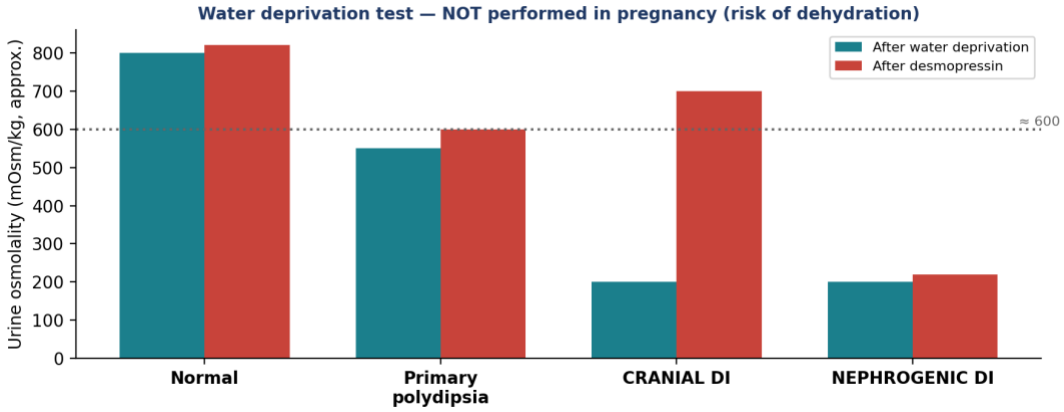


Figure 188.1 — Water deprivation test patterns

- **Polyuria (> 3 L/day), polydipsia, nocturia**, thirst; hypernatraemia if water access is limited (e.g. labour, unconscious).
- Paired **plasma and urine osmolality**: dilute urine (< 300 mOsm/kg) with high-normal/raised plasma osmolality or Na^+ (remember pregnancy's lower normal range).

- **Water deprivation test is avoided in pregnancy** (dehydration, uteroplacental compromise); **copeptin** and a **desmopressin trial** with careful monitoring are used instead. Exclude diabetes mellitus, hypercalcaemia, hypokalaemia; check **LFTs** (AFLP).

188.3 Management in pregnancy

Situation	Management
Pre-existing cranial DI	Often worsens (vasopressinase) — dose of desmopressin may need ↑; monitor Na^+ and fluid balance
Gestational DI	Desmopressin (intranasal/oral; resistant to vasopressinase) — not vasopressin; investigate for AFLP/HELLP; usually resolves after delivery
Nephrogenic DI	Adequate fluids; thiazides; treat cause (stop lithium only with psychiatric input)
Labour / CS	Avoid fluid restriction and also avoid hyponatraemia from desmopressin + IV fluids/oxytocin; strict input–output, Na^+ monitoring; oxytocin action is unaffected
Postpartum / breastfeeding	Desmopressin compatible with breastfeeding; reduce dose as vasopressinase disappears (risk of hyponatraemia)

■ **RED FLAGS**

- Polyuria with **hypernatraemia, abnormal LFTs, coagulopathy or hypoglycaemia** in the 3rd trimester → think **acute fatty liver of pregnancy**.
- Desmopressin overtreatment → **dilutional hyponatraemia and seizures** — especially with oxytocin and IV fluids.

188.4 Must-know summary

■ **EXAM TIP**

- Cranial (↓ ADH), nephrogenic (resistance — lithium, ↑ Ca^{2+}), gestational (**placental vasopressinase**), primary polydipsia.
- Pregnancy: osmostat ↓ 10 mOsm/kg; vasopressinase ↑ ADH clearance.
- No water deprivation test in pregnancy; **desmopressin** is the treatment (vasopressinase-resistant).
- Gestational DI → check for **AFLP/HELLP**; monitor Na^+ in labour and postpartum.

■ **MNEMONIC / MEMORY AID**

DI — 'Dry Inside': lots of dilute urine, high plasma osmolality. **'Desmopressin Defeats vasopressinase'**.