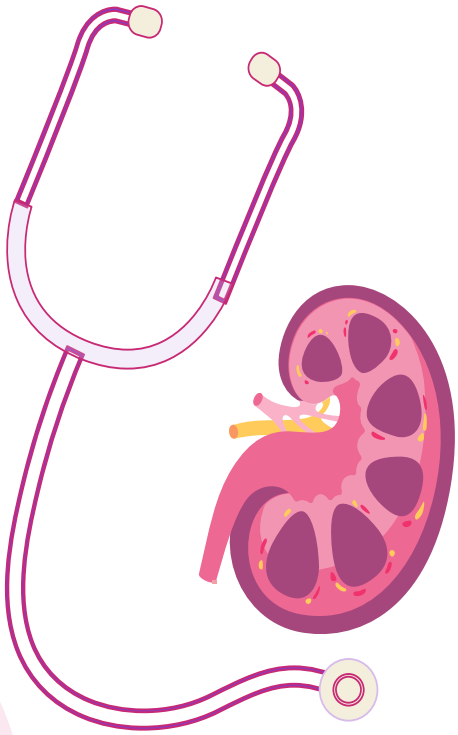


UGS Physiology Summary

By: Shahd Alahmad

Homeostasis of Electrolytes:

🔁 Normal potassium distribution

Compartment	Amount	Conc.
Intracellular	3920 mEq / 28L	140 mEq/L
Extracellular	59 mEq / 14L	4.2 mEq/L
Daily intake	~100 mEq/day	Must = output

Question

- A 26-year-old woman recently adopted a healthier diet to eat more fruits and vegetables. As a result, her potassium intake increased from 80 to 160 mmol/day. Which of the following conditions would you expect to find 2 weeks after she increased her potassium intake, compared with before the increase?

	Potassium Excretion Rate	Sodium Excretion Rate	Plasma Aldosterone Concentration	Plasma Potassium Concentration
A)	↔	↔	↑	Large increase (>1 mmol/l)
B)	↔	↓	↑	Small increase (<1 mmol/l)
C)	↑ 2×	↔	↑	Small increase (<1 mmol/l)
D)	↑ 2×	↑	↓	Large increase (>1 mmol/l)
E)	↑ 2×	↑	↔	Large increase (>1 mmol/l)

⚖️ Defenses against K^+ imbalance

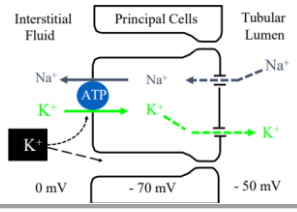
Line	Mechanism
First	Intracellular ↔ extracellular shift
Second	Urinary excretion (hormonal regulation)

☠️ K^+ imbalance effects

Condition	Consequences
Hyperkalemia	- Partial Cell depolarization - Cardiac toxicity (VF, asystole)
Hypokalemia	- Hyperpolarization - Weakness, fatigue - Hypoventilation - Delayed ventricular repolarization

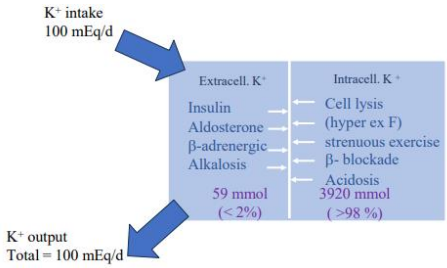
🔁 Cellular K^+ uptake – regulators

Factor	Mechanism
Insulin	Stimulates Na^+/K^+ ATPase → ↑ K^+ uptake post-meal
Aldosterone	↑ Na^+ reabsorption, ↑ K^+ secretion
β-adrenergic	Stimulates Na^+/K^+ ATPase = stimulus for k+ uptake



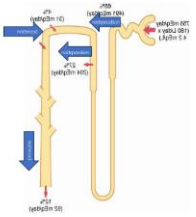
📉 K^+ release from cells

Factor	Effect
Cell lysis	Tissue injury or hypotonic ECF → K^+ shift to ECF
Hypertonic ECF	Water exits cells → ↑ intracellular K^+ → K^+ efflux
Strenuous exercise	↑ K^+ efflux; worsened by dehydration or β-blockers
↓ Na^+/K^+ ATPase	Seen in acidosis → K^+ accumulation in ECF



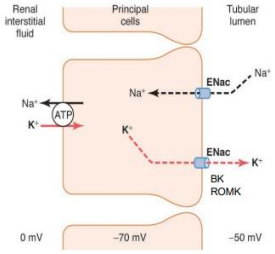
🧪 Renal regulation summary

Step	Notes
Filtration	Depends on GFR × plasma [K^+] (So the filtration aids in the regulation process but it's not enough.)
Reabsorption	- PCT: ~67% - Thick limb: ~25% - Both are Not regulated, we can't control
Secretion	- Late DCT & collecting duct - Aldosterone-dependent - **Main** regulation point



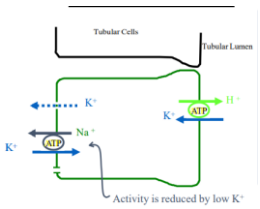
🥯 Principal cells (K^+ secretion)

Channel	Location	Fx.
Na^+/K^+ ATPase	Basolateral	Creates Na^+ gradient, pumps K^+ into cell
ENaC	Apical	Reabsorbs Na^+
ROMK & BK	Apical	Secrete K^+



🪥 Intercalated cells (K^+ reabsorption)

Pump	Condition
K^+/H^+ ATPase	Active in hypokalemia Reabsorbs K^+, secretes H^+



📊 Factors influencing K^+ secretion

Factor	Effect on K^+ secretion
↑ ECF [K^+]	↑ (directly & via aldosterone)
↑ Aldosterone	↑ via ENaC, Na^+/K^+ ATPase, ROMK, BK
↑ Na^+ delivery / flow	↑ via dilution effect
Alkalosis	↑ via ↑ Na^+/K^+ ATPase activity
Acidosis	↓ Na^+/K^+ ATPase → ↓ secretion

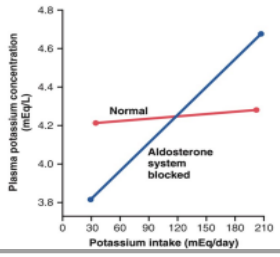
🧪 Aldosterone effects

Trigger	Outcome
↑ Plasma K^+	↑ Aldosterone release
Aldosterone	↑ Na^+/K^+ ATPase, ENaC, K^+ channels ↑ Na^+ reabsorption, ↑ K^+ excretion

⚠️ Aldosterone feedback loop

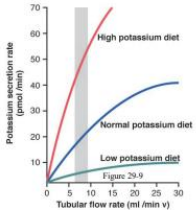
Step	Description

1	↑ Plasma K^+ → ↑ Aldosterone
2	↑ K^+ secretion → ↓ Plasma K^+
3	↓ Aldosterone release (negative feedback)
Block	→ Dangerous K^+ swings (e.g. Addison's disease)



Tubular flow effect

Flow	Impact on K^+
High	- ↓ K^+ in lumen - ↑ Secretion gradient - ↑ K^+ excretion
Clinical	Diuretics → ↑ flow → ↑ K^+ loss

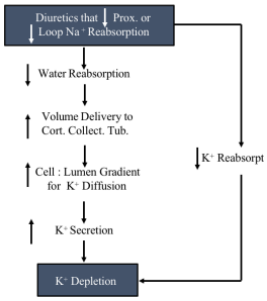


Sodium effect on K^+ *imp*

Na^+ intake	Outcome on K^+ excretion
↑ **Intake**	- ↑ Flow → ↑ K^+ secretion - ↓ RAAS → ↓ aldosterone Net: As a result of these two opposing influences potassium excretion remains relatively **unchanged.** مهم

Clinical note: diuretics

Type	Action
PCT/loop	↓ Na^+ reabsorption → ↑ flow rate
DCT	↓ time for K^+ reabsorption
Result	↑ K^+ loss due to enhanced secretion + limited reabsorption



Acidosis vs alkalosis

State	Effect on K^+
Acidosis	Inhibits Na^+/K^+ ATPase ↓ secretion, ↑ ECF K^+
Alkalosis	Stimulates pump ↑ K^+ secretion & excretion

- Which of the following would cause the most serious hypokalemia?
  A) A decrease in potassium intake from 150 mEq/day to 60 mEq/day
  B) An increase in sodium intake from 100 to 200 mEq/day
  C) Excessive aldosterone secretion plus high sodium intake
  D) Excessive aldosterone secretion plus low sodium intake
  E) A patient with Addison's disease
  F) Treatment with a beta-adrenergic blocker
  G) Treatment with spironolactone

Sodium & potassium homeostasis under varied intake مهم

Condition	Na^+ effect	K^+ excretion impact
High Na^+ intake	↑ GFR, ↓ proximal Na^+ reabsorption → ↑ distal flow	- ↑ K^+ secretion due to flow - ↓ aldosterone → ↓ K^+ secretion - Net: no significant K^+ excretion change
Low Na^+ intake	↓ GFR, ↑ proximal Na^+ reabsorption → ↓ distal flow	- ↑ aldosterone → ↑ K^+ secretion - ↓ flow → ↓ K^+ secretion - Net: no significant K^+ excretion change

🧪 Acid-base effects on K^+ secretion

Condition	Impact
Acute acidosis	↓ Na^+/K^+ ATPase activity + ↓ membrane K^+ permeability → ↓ K^+ secretion
Chronic acidosis	↓ Na^+ reabsorption → ↑ tubular flow → ↑ K^+ secretion
Alkalosis	↑ Na^+/K^+ ATPase → ↑ intracellular K^+ → ↑ K^+ secretion/excretion → hypokalemia risk

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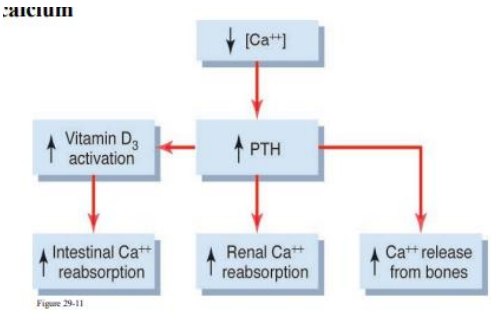
🧠 Clinical causes of potassium disturbances مهم جدا

Hyperkalemia	Hypokalemia
- Renal failure	- Very low K^+ intake
- ↓ distal nephron flow (e.g. heart failure, NSAIDs)	- GI loss (diarrhea)
- ↓ aldosterone/effect (Addison's, K^+-sparing diuretics)	- Alkalosis مهم
- Acidosis (mild hyperkalemia)	- Excess insulin
- Diabetes	- ↑ distal flow (diuretics, nephropathies)
	- ↑ aldosterone/mineralocorticoids

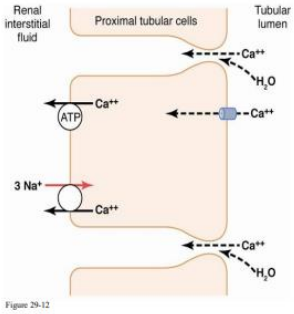
🦴 Calcium regulation by PTH

Organ	Action
Kidney	↑ Ca^{2+} reabsorption (DCT)
Bone	↑ osteoclast activity via osteoblasts → ↑ Ca^{2+} release
Gut (via Vit D)	↑ Ca^{2+} absorption (↑ Ca^{2+}-binding protein synthesis)



🫓 Calcium reabsorption in nephron

Segment	Mechanism
PCT	~80% via paracellular route with water
PCT (transcellular)	20% via Ca^{2+} channels → Ca^{2+}-ATPase + Na^+/Ca^{2+} exchanger
Thick ascending limb	Paracellular *also there's reabsorption in DCT*

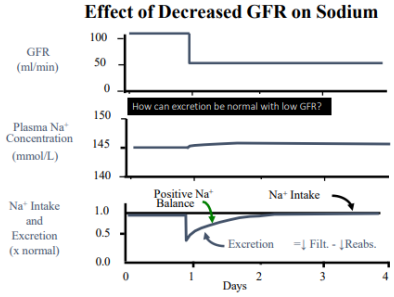


🧠 Renal integration: fluid & electrolyte regulation

Formula	Notes
Excretion = Filtration − Reabsorption + Secretion	Applies to Na^+, K^+, Ca^{2+}
Steady-state	Intake = Output

🌀 Effects of ↓ GFR

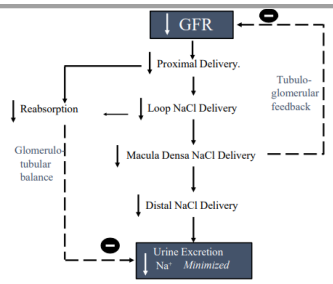
Solute	Result
Na^+	↓ excretion initially .. Tubular reabsorption ↓ → restores balance



Creatinine	Plasma ↑ ↑ filtered load → excretion returns to match production

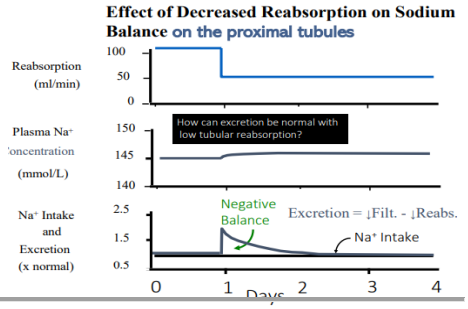
🔁 Compensatory mechanisms

Response type	Includes
Local renal	GFR, tubular reabsorption/secretion changes
Systemic	Hormones, BP, sympathetic tone, blood composition



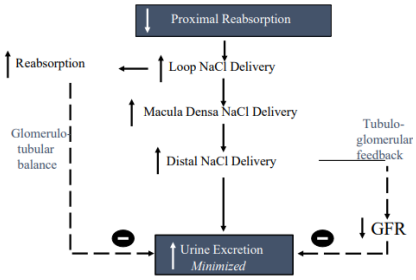
💉 Na^+ balance with altered reabsorption

Case	Response
↓ reabsorption	Initially ↑ Na^+ excretion → autoreg ↓ GFR → Na^+ balance restored



📈 High Na^+ intake: integrated renal response

Effect	Mechanism
Slight ↑ GFR	↑ filtration
↓ Na^+ reabsorption	- ↓ RAAS - ↑ peritubular pressure - ↑ natriuretic peptides
Net result	↑ Na^+ excretion → maintains homeostasis



Acid-Base Regulation in the Kidney:

⚖️ Acid-base balance

System	Function
Respiratory + Renal	Work in harmony to maintain acid-base balance
Buffer systems	Act immediately to resist changes in pH

- Body pH must be between 7.2–7.4 for enzyme function
- H+ is regulated at 3–5 x 10^{-8} mol/L
- Metabolism produces acids:
    - Volatile: removed via CO2
    - Non-volatile: organic, titrated then excreted

🛡️ Systems regulating H^+ in body fluids

Line	System	Mechanism

1st	Chemical buffers	Bicarbonate, ammonia, proteins, phosphate (fast but temporary)
2nd	Lungs	Eliminate volatile acids via CO2 (rapid, incomplete)
3rd	Kidneys	Eliminate non-volatile acids by: - H^+ secretion - HCO_3^- reabsorption - New HCO_3^- generation

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🧪 Buffer systems in body (60-70% of buffering is in the cells)

Buffer	Location	Rx.
Bicarbonate	ECF	$H_2O + CO_2 \leftrightarrows H_2CO_3 \leftrightarrows H^+ + HCO_3^-$
Phosphate	Renal tubules	$HPO_4^{2-} + H^+ \leftrightarrows H_2PO_4^-$
Ammonia	Renal tubules	$NH_3 + H^+ \leftrightarrows NH_4^+$
Proteins	Intracellular (e.g Hb)	$H^+ + Hb \leftrightarrows HHb$

Buffer effectiveness depends on:

- Buffer concentration
- Proximity of pKa to pH

🧱 Henderson-Hasselbalch equation

Term	Definition
pH	Depends on ratio of HCO_3^- to CO_2
pKa	Bicarbonate buffer ≈ 6.1
When $[HCO_3^-] = [CO_2]$	pH = pKa

- Normal point for bicarbonate buffer = pH 7.4 (not optimal, but highly regulated)

📉 Non-volatile acid burden

Metric	Value
Normal $[H^+]$	0.00004 mmol/L
Acid produced	60–80 mmol/day
Need for buffering	Buffer 47,500x normal H^+ concentration
Viable pH limits	6.8–8

🌬️ Respiratory compensation

Condition	Response
Acidosis	↑ Ventilation → ↓ CO_2 → ↓ H^+
Alkalosis	↓ Ventilation → ↑ CO_2 → ↑ H^+
Feedback gain	1.0–3.0 → 50–75% correction only

🧪 Renal compensation

Mechanism	Notes
H^+ secretion	Mainly by intercalated cells
HCO_3^- reabsorption	1:1 with H^+ secretion
New HCO_3^- generation	When H^+ exceeds titration capacity

- **Kidneys eliminate non-volatile acids (H_2SO_4, H_3PO_4) (~ 80 mmol/day)**
- Filtration of HCO_3^- (~ 4320 mmol/day)
- Secretion of H^+ (~ 4400 mmol/day)
- Reabsorption of HCO_3^- (~ 4319 mmol/day)
- Production of new HCO_3^- (~ 80 mmol/day)
- Excretion of HCO_3^- (1 mmol/day)

Reabsorption & H^+ secretion (nephron segments)

Segment	Action
PCT	Reabsorbs 70–80% of HCO_3^-
Thin Henle	No HCO_3^- change
Thick Henle	Reabsorbs 10%
Distal/Collecting tubules	Fine-tune HCO_3^- reabsorption

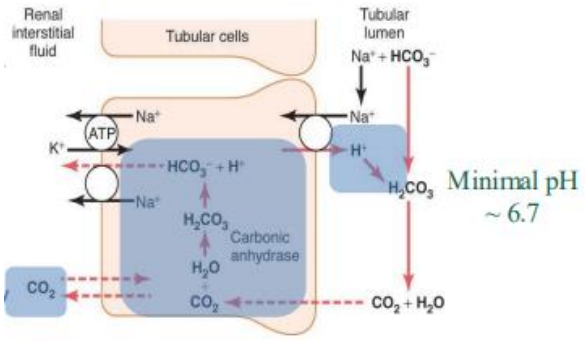
Key:

- Total filtered HCO_3^-: ~4320 mmol/day
- ~1 mmol/day HCO_3^- excreted; 4319 mmol H^+ secreted

Proximal tubule & thick loop transport

Side	Transporters
Basolateral	Na^+/K^+ ATPase, HCO_3^-/Na^+ cotransport
Apical	Na^+/H^+ exchanger

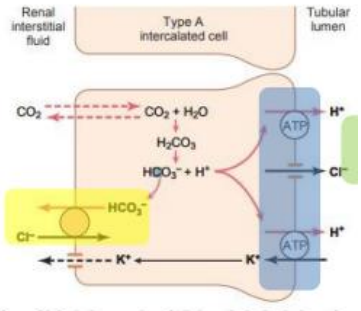
- Carbonic anhydrase drives cycle:
  - $H^+ + HCO_3^- \rightarrow H_2CO_3 \rightarrow CO_2 + H_2O \rightarrow$ diffuses into cell $\rightarrow$ reforms $HCO_3^- + H^+$
  - Repeat cycle
- Result: H^+ secreted, HCO_3^- reabsorbed



Intercalated cells (Distal/Collecting tubules)

Type	Transporters
Type A	Apical: H^+ ATPase, H^+/K^+ antiport Basolateral: HCO_3^-/Cl^- exchanger

- H^+ secreted, HCO_3^- reabsorbed
- Minimal urine pH = 4.5



Regulation of renal H^+ secretion

Stimulus	Effect
↑ Plasma CO_2	↑ H^+ secretion (respiratory acidosis)
↑ Extracellular H^+	↑ H^+ secretion (acidosis)
↑ Tubular buffer	↑ H^+ secretion (adaptive response)

Generating new bicarbonate

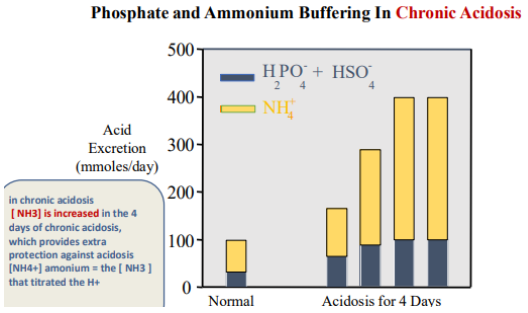
Context	Mechanism
All HCO_3^- reabsorbed	Excess H^+ remains
Buffering w/ phosphate or ammonia	Generates new HCO_3^-
End result	Increases systemic HCO_3^- even without reabsorbing it directly

Phosphate buffer role

Process	Notes
In lumen	$NaHPO_4^{2-} + H^+ \leftrightarrows NaH_2PO_4$
Result	Each titrated $H^+ \rightarrow$ new HCO_3^- formed
Buffering capacity	~30 mmol/day
Chronic acidosis	Not regulated → less useful * not the major tubular buffer in chronic acidosis*

Ammonia buffer role

Source	Action
Proximal tubule	Glutamine $\rightarrow NH_4^+ + HCO_3^-$
Collecting duct	NH_3 binds $H^+ \rightarrow NH_4^+$ (excreted)
Result	NH_4^+ excretion → new HCO_3^-
Chronic acidosis	Ammonia increases; phosphate does not



Quantification summary

Value	Description
Total H^+ secretion	4380 mmol/day (4320 HCO_3^- + 60 non-volatile)
Titratable acid	30 mmol/day (phosphate)
NH_4^+ excretion	30 mmol/day
HCO_3^- excreted	1 mmol/day
Net H^+ excretion	59 mmol/day

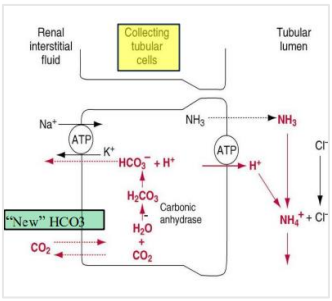
- 

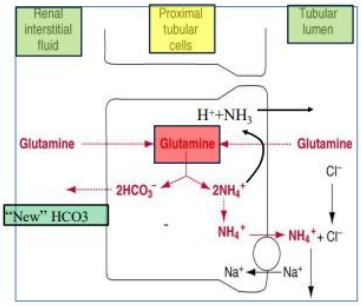
H^+ buffering and new bicarbonate generation

Process	Description
$H^+ + NH_3$ / $NaHPO_4^-$	**- Forms NH_4^+ or $H_2PO_4^-$** **- Excretes H^+ safely** **- Adds new HCO_3^- to blood**

🧬 NH_4^+ & HCO_3^- production sites

Segment	Action
Proximal tubule	- Glutamine → $NH_4^+ + HCO_3^-$ - NH_4^+ secreted via Na^+ exchange - HCO_3^- reabsorbed with Na^+ - NH_4^+ can → $NH_3 + H^+$
Thick limb & distal tubule	Continue HCO_3^- generation via NH_4^+
Collecting tubule	- NH_3 from capillaries/cells binds H^+ - Forms NH_4^+ → excreted - Prevents HCO_3^- loss → net new HCO_3^-





📊 Renal acid-base regulation values

Parameter	Normal Value
Total H^+ secretion / HCO_3^- reabsorption	~4320 mmol/day
Nonvolatile acid excretion	~60 mmol/day
Titratable acid + NH_4^+ excretion	~60 mmol/day
Net H^+ excretion = new HCO_3^-	~59 mmol/day

القيمة (تقريبية)	شو معناته؟	Parameter
mmol/day 4320 ≈	كمية H⁺ اللي الكلى تتعامل معه يوميًا (إما تطلع H⁺ أو تعيد امتصاص HCO₃⁻)	Total H⁺ secretion / HCO₃⁻ reabsorption
mmol/day 60 ≈	كمية الأحماض غير الطيّارة اللي الجسم لازم يتخلص منها عن طريق الكلى (مش عبر الرئتين)	Nonvolatile acid excretion
mmol/day 60 ≈	مجموع الطرق اللي الكلية تطرد فيها H⁺ فعليًا (titratable acids + ammonium ـك)	TA + NH₄⁺ excretion
mmol/day 59 ≈	الفرق الصافي = كمية H⁺ اللي خرجت فعلًا وخلّت الجسم يكسب بيكربونات جديدة	Net H⁺ excretion = new HCO₃⁻

📈 Compensation changes

Condition	Titratable acid	NH_4^+	HCO_3^- excretion	Total
Acidosis	35	165	0	200
Alkalosis	0	0	80	80

🧪 Net acid & HCO_3^- addition formula مهم

Equation	Meaning
Net H^+ excretion = TA + NH_4^+ − HCO_3^- loss	H^+ buffered = new HCO_3^-
Addition of HCO_3^- = TA + NH_4^+ − HCO_3^- excretion	Same as above

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⚖️ Compensation directions

Disorder	Compensation
Respiratory acidosis	↑ Renal HCO_3^- reabsorption, H^+ excretion
Respiratory alkalosis	↓ H^+ secretion, ↑ HCO_3^- loss

Metabolic acidosis	**Hyperventilation → ↓ PCO_2 + renal buffer excretion**
Metabolic alkalosis	**↓ HCO_3^- reabsorption + ↑ excretion**

Buffer roles summary

Buffer	Effect
HCO_3^-	**Reabsorbed and used in initial buffering**
NH_3/NH_4^+	**Buffers H^+ without using HCO_3^- → new HCO_3^- added**
$NaHPO_4^-$	**Titrates H^+ → forms titratable acid**

The following data were taken from a patient:
urine volume = 1.0 liter/day
urine HCO_3^- concentration = 2 mmol/liter
urine NH_4^+ concentration = 15 mmol/liter
urine titratable acid = 10 mmol/liter

net acid excretion = Titr. Acid + NH_4^+ excret - HCO_3^-
= (10 x 1) + (15 x 1) - (1 x 2)
= 23 mmol/day

net rate of HCO_3^- addition to body = 23 mmol/day

Renal responses by disorder

Condition	Renal reaction
Respiratory acidosis	**- ↑ PCO_2 → ↑ tubular H^+ - ↑ HCO_3^- reabsorption - Net new HCO_3^- made**
Metabolic acidosis	**- ↓ HCO_3^- → ↓ filtered load - Full HCO_3^- reabsorbed - H^+ buffered by NH_4^+, $NaHPO_4^-$ - Net HCO_3^- added**
Respiratory alkalosis	**- ↓ PCO_2 → ↓ H^+ secretion. - Excess filtered HCO_3^- not reabsorbed - ↑ HCO_3^- excretion → ↓ pH**
Metabolic alkalosis	**- ↑ HCO_3^- filtered → ↓ reabsorption - ↑ Excretion of HCO_3^- - ↓ H^+ secretion due to less titration**

Acid-base disorder classification

Condition	pH	HCO_3^-	PCO_2	Primary Issue	Compensation
Metabolic acidosis	↓	↓	↓ (comp)	↓ HCO_3^-	↑ ventilation ↑ renal HCO_3 production
Respiratory acidosis	↓	↑ (comp)	↑	↑ PCO_2	↑ renal HCO_3 production
Metabolic alkalosis	↑	↑	↑ (comp)	↑ HCO_3^-	↓ ventilation ↑ renal HCO_3 excretion
Respiratory alkalosis	↑	↓ (comp)	↓	↓ PCO_2	↑ renal HCO_3 excretion

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Mixed acid-base disturbances

pH	HCO_3^-	PCO_2	Diagnosis
7.09	**15**	**50**	**Mixed acidosis**
7.60	**29**	**30**	**Mixed alkalosis**
7.34	**15**	**29**	**Metabolic acidosis w/ resp. compensation**

Test	Normal	Decrease Value	Increase Value
pH	7.35-7.45	Acidosis	Alkalosis
PaCO2	35-45	Alkalosis	Acidosis
HCO3	22-26	Acidosis	Alkalosis
PaO2	80-100	Hypoxemia	O2 therapy

7.34	31	60	Respiratory acidosis

📊 Anion gap tool ($Na^+ - Cl^- - HCO_3^-$) مهم

الفكرة:
إذا الـ AG عالي = في أحماض جديدة بالجسم (مش تعويضية)
إذا AG طبيعي بس Cl^- عالي = الجسم خسر بيكربونات وعوّضها بكلورايد

Type	AG	Cl^-	Causes
High AG (normochloremic)	>16	Normal	DKA, lactic acidosis, salicylates, methanol, starvation
Normal AG (hyperchloremic)	8–16	↑	Diarrhea, RTA, Addison's, CA inhibitors

Example: $Na^+ = 142$, $Cl^- = 102$, $HCO_3^- = 12$ → AG = 28 → High AG → metabolic acidosis

---

🧠 Clinical case diagnostics مهم

pH	HCO_3^-	PCO_2	Anion gap	Interpretation
7.12	18	50	—	Mixed acidosis
7.60	29	30	—	Mixed alkalosis
7.15	8	24	—	Metabolic acidosis (partial resp. comp.)
7.25	12	28	AG = 28	Metabolic acidosis w/ resp. comp. (e.g diabetic ketoacidosis)
7.34	15	29	AG = 9	Normal AG → hyperchloremic metabolic acidosis (e.g diarrhea)

📜 Common disorder triggers

Disorder	Examples
Metabolic acidosis	DKA, diarrhea, RTA, salicylates, CA inhibitors
Respiratory acidosis	Brain injury, pneumonia, emphysema, lung disorders
Metabolic alkalosis	Vomiting, diuretics, aldosterone excess, $NaHCO_3$ overdose
Respiratory alkalosis	High altitude, fear, hyperventilation

🧬 Aldosterone & metabolic alkalosis

Mechanism
↑ Aldosterone → ↑ tubular K^+ secretion → K^+ depletion → ↑ H^+ secretion → ↑ HCO_3^- reabsorption & new HCO_3^- generation

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💊 Diuretic overuse & alkalosis

Mechanism
Diuretics → ↓ ECV → ↑ RAAS → ↑ aldosterone → ↑ K^+ & H^+ loss → ↑ HCO_3^- retention and production

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📌 Normal reference values

Metric	Range
pH	**7.35–7.45**
$PaCO_2$	**35–45 mmHg**
HCO_3^-	**22–26 mEq/L**
AG	**8–16 mEq/L**

pH	HCO_3^-	PCO_2	Acid-Base Disorder ?
7.34	15	29	Metabolic acidosis
7.49	35	48	Metabolic alkalosis
7.34	31	60	Respiratory acidosis
7.62	20	20	Respiratory alkalosis
7.09	15	50	Acidosis: respiratory + metabolic

Female Reproductive System:

🧬 Female reproductive functions

Fx.	Details
1. Prepares for conception	Through hormonal cycles and structural development
2. Maintains pregnancy	Supports fertilized ovum to develop fetus

---

🫓 Female reproductive organs

Organs	Notes
Ovaries, Fallopian tubes, Uterus, Vagina, Mammary glands	Coordinate with hormones for reproduction

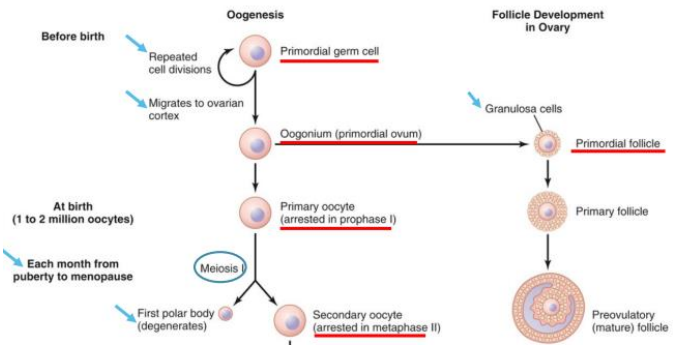
- Ovum expelled mid-cycle → abdominal cavity → fallopian tube → uterus
- If fertilized, develops into fetus/placenta/membranes

🧪 Oogenesis

Stage	Details
Primordial germ cells	Migrate to ovary surface, divide, become oogonia
Oogonia	Surrounded by granulosa cells → form primordial follicles
Primary oocyte	Arrested in prophase I
First meiotic division	Completed post-puberty: forms secondary oocyte + polar body
Second meiotic division	Completed if fertilization occurs

📊 Oocyte timeline

Time	Oocyte status
5th fetal month	Mitosis ends, meiosis starts
Birth	~1–2 million primary oocytes
Puberty	~300,000 remain
Reproductive years	Only 400–500 mature and ovulate
Menopause	Few remain; all degenerate

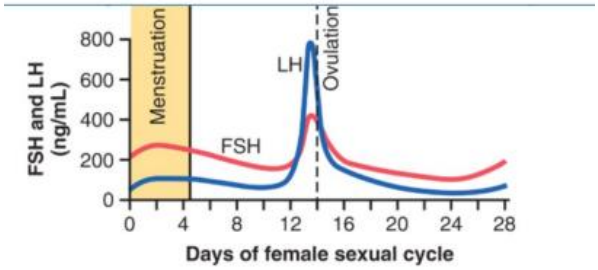


🧬 Gonadotropic hormones

Hormone	Effects
FSH & LH	Stimulate ovarian function post-puberty
No FSH/LH	Ovaries inactive (e.g in childhood)
Puberty onset	Pituitary secretes more FSH/LH (age 9–12)
Menarche	First menstrual cycle

📈 Hormonal cycle dynamics

Phase	Hormonal changes
Early cycle	FSH > LH, both rise gradually
Mid-cycle	Estrogen peaks → LH surge
Ovulation	Triggered by LH surge
Post-ovulation	Estrogen drops, progesterone increases



🌱 Follicular development

Follicle type	*Characteristics*
Primordial	Single granulosa layer; oocyte in prophase secrete an oocyte maturation-inhibiting factor that keeps the ovum suspended in its primordial state
Primary	Granulosa cells proliferate; zona pellucida forms. (after puberty)
Secondary	Antrum forms; granulosa become corona radiata
Vesicular	Rapid growth due to FSH + estrogen + LH

Secondary follicle

The theca differentiates into two layers:

1) Theca interna, epithelioid characteristics, secrete additional steroid sex hormones.
2) The theca externa, develops into a highly vascular connective tissue capsule.

- Only one follicle fully matures each cycle; others undergo atresia
- The early growth of the primary follicle up to the antral stage is stimulated mainly by FSH alone.مهم

Ovulation مهم

Trigger	Effects
LH surge	- Final follicle maturation - Granulosa/theca → progesterone secretion - Estrogen falls - Ovum released

Ovulation mechanism

Step	Action
1	LH surge
2	Theca externa enzymes weaken wall
3	Neovascularization, prostaglandin release
4	Follicle swells
5	Stigma ruptures, ovum discharged

Positive feedback: estrogen & LH surge

Trigger	Effect
Sustained high estrogen	Switches from negative to positive feedback → LH surge
Granulosa secretes progesterone	May enhance LH release

- LH increases 6–8x; FSH increases ~2x

Female reproductive cycle :

Phase	events
Follicular	- FSH stimulates follicle growth - Estrogen secreted - Endometrial proliferation
Ovulation	- LH surge - Ovum released
Luteal	- Corpus luteum forms - Secretes progesterone, estrogen, inhibin - Negative feedback ↓ FSH/LH
Menstruation	- Hormone drop → endometrial necrosis - Vasospasm, prostaglandins, leukocytes - 50–150 mL flow with fibrinolysin

Corpus luteum & luteal phase

Topic	Details

Corpus luteum	- From granulosa & theca cells - Fills with lipids → yellow (lutein) - Secretes estrogen, progesterone, inhibin
Involution	- Begins ~12 days after ovulation - Becomes corpus albicans - Replaced by CT and absorbed over months
Pregnancy	- hCG (placenta) prolongs corpus luteum life (2–4 months) *imp* بالتالي لو تم ازالة الجسم الأصفر بعد هالمدة عادي ما رح يصير اجهاض ويستمر الحمل

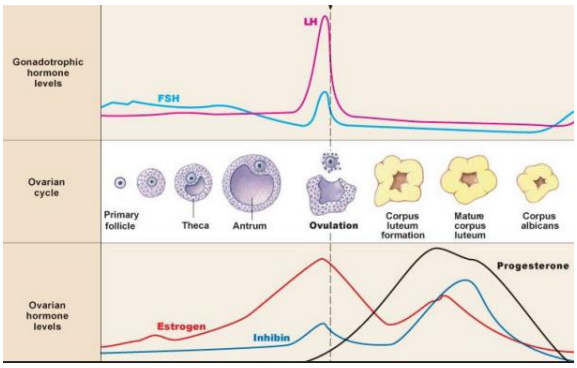
🔁 Gonadotropins & puberty

Hormone	Role
FSH & LH	- Required for ovarian cycle - Absent during childhood
GnRH	- Pulsatile, every 90 min - Stimulates FSH/LH release
Puberty	- Starts age 9–12 - Menarche = first menstruation • During growth of the follicles, estrogen is mainly secreted.

Another hormone with almost exactly the same properties as LH, chorionic gonadotropin, which is secreted by the placenta, can act on the corpus luteum to prolong its life—usually maintaining it for at least the first 2 to 4 months of pregnancy. مهم

🧪 Female hormones in cycle مهم

Day	Hormonal changes
Days 1–5	FSH ↑ → follicle growth, estrogen starts rising
~Day 12–14	Estrogen peak → LH surge → ovulation
Post-ovulation	↑ Progesterone ↓ Estrogen Inhibin released
Late luteal	↓ All hormones *especially progesterone*, → menstruation



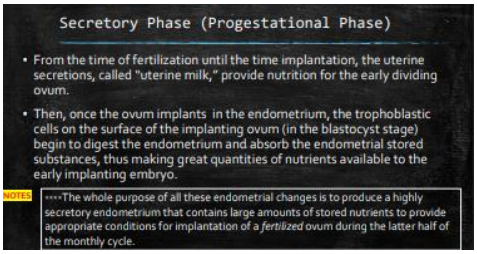
🫓 Endometrial cycle

Phase	Description
Proliferative	- Estrogen-driven - Epithelial/stromal proliferation - Thickness ↑ to 3–5 mm by ovulation
Secretory	- Progesterone-driven - Glands: tortuous, secrete glycogen - lipid and glycogen deposits increase greatly in the stromal cells - Vessels proliferate, edema ↑ - Thickness peaks at 5–6 mm *check the black pic below*
Menstruation	- Hormone withdrawal - Vasospasm, necrosis, bleeding - Flow: 50–150 mL with leukocytes

---

Functions of estrogens مهم

System	Effects
Genitalia	- Maturation of ovaries, tubes, uterus, vagina - Stratified epithelium (vagina)
Endometrium	- Gland/stroma proliferation - Prepares for implantation
Breast	- Ductal growth - Fat deposition - Stromal tissue ↑
Bone	- ↓ Osteoclast activity - Epiphyseal fusion (growth stops earlier) - Post-menopause → osteoporosis
Metabolism	- ↑ Protein deposition - ↑ Basal metabolic rate - ↑ Fat in breast, buttocks, thighs



Functions of progesterone

Target	Effect
Uterus	- Secretory transformation of endometrium - ↓ Myometrial contractions (maintain pregnancy)
Breasts	- Lobulo-alveolar development - ↑ Size, fluid retention - Prepares for lactation (but prolactin needed for milk)

Fertilization and Implantation:

ملاحظة: غيّرت تسلسل المحاضرات ابتداءً من هون بحيث حطيت المحاضرات الي بتحكي عن الـFemale ورا بعض عشان التسلسل يكون أفضل

Maturation and fertilization of the ovum

Stage	Details
Ovum entry	- Secondary oocyte enters fallopian tube via fimbriae - Cilia activated by estrogen - 98% success - Slow fluid current guides ovum
Fertilization site	- Occurs in ampullae of fallopian tube
Sperm transport	- Aided by: 1. Prostaglandins in semen stimulate uterus/tube contractions 2. Oxytocin released during female orgasm

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Capacitation of spermatozoa

Change	Description

Pre-capacitation	- Sperm inhibited by genital tract factors (cholesterol-rich) - Tough acrosomal membrane
In female tract *imp to know that this happens in Female genital tract not in epididymis*	- Inhibitory factors washed away - Cholesterol loss weakens head of the sperm - Ca^{2+} influx ↑ flagellar motility + Ca^{2+} enables acrosome enzyme release - Capacitation: 1–10 hours

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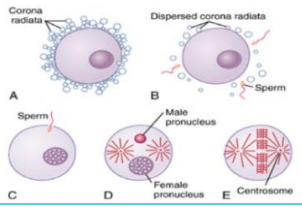
Acrosome reaction

Process	Details
Enzymes	- Hyaluronidase: breaks down granulosa cell matrix - Proteolytics: digest tissue proteins
Action	- Binds to zona pellucida receptors - Acrosome dissolves, releases enzymes - Pathway opens for sperm entry - Sperm and oocyte membranes fuse within 30 minutes - Genomes combine to form zygote (23 pairs)



Polyspermy prevention

Trigger	Calcium influx following sperm-oocyte fusion
Response	- Cortical granule exocytosis - Enzymatic alteration of zona pellucida - Blocks polyspermy (prevents entry of additional sperm)

Fertilized ovum transport

Feature	Notes
Duration	3–5 days to reach uterus
Mechanism	- Fluid current + cilia beat toward uterus - Isthmus remains contracted ~3 days (Progesterone relaxes it)
Nutrition	Tube secretions nourish blastocyst
Division	Blastocyst forms (≈100 cells) before reaching uterus

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Implantation in uterus

Feature	Notes
Timeline	5–7 days post-ovulation - 1–3 days in uterus pre-implantationبكون داخل الرحم لكن ما انزرع

Nutrition	Uterine milk (endometrial secretion) feeds blastocyst
Mechanism	- Trophoblast enzymes digest endometrium - Trophoblast absorbs nutrients

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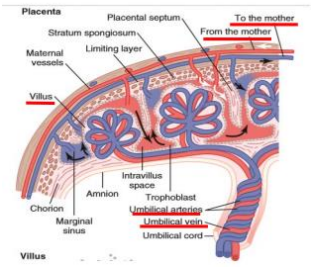
Placental development

Event	Timeline/Feature
Trophoblast + adjacent cells proliferate	Placenta and membranes form
Capillaries grow in cords	Fetal heart pumps blood by day 21
Maternal sinuses form	Surround trophoblastic cords
Placental villi grow	Interface for maternal-fetal exchange

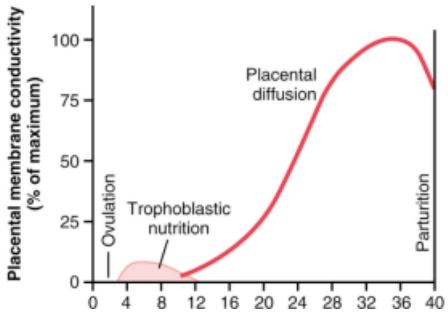


Placental circulation

Fetal side	Maternal side
2 umbilical arteries → capillaries in villi → 1 umbilical vein back to fetus	**Uterine arteries → sinuses → uterine veins**

Early embryo nutrition *imp*

Stage	Source
Week 1	Decidual tissue only مهم
Up to week 8	Continued decidual digestion
After day 16	Placenta begins support (partial)
Later pregnancy	Placenta thins and grows → ↑ diffusion capacity



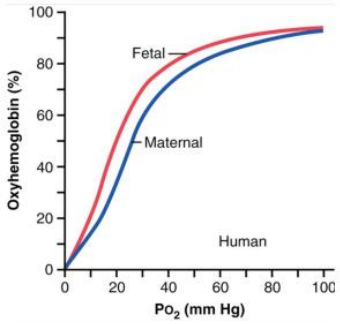
Placental functions

Fx.	Detail
Respiration	Simple diffusion of O_2 (maternal → fetal)
Nutrition	Glucose by facilitated diffusion
Excretion	Waste elimination
Endocrine	HCG, etc
Protection	Barrier function

Oxygen transfer via placenta

الخلاصة انه الجنين قادر يعيش حتى لو الاكسجين مو عالي جدًا لانه عنده وسائل تكيف (زي HbF)

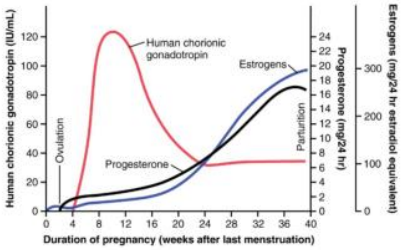
Feature	Detail
PO_2 gradient	20 mmHg (mother ≈50, fetus ≈30)
Fetal hemoglobin	-Binds 20–50% more O_2 than maternal Hb -curve shifts to the left ***imp***
Hb concentration	Fetal blood has 50% more Hb
Bohr effect	CO_2 loss → alkalinity → ↑ O_2 affinity



📈 Hormones in pregnancy

Hormone	Source & function
HCG	- From trophoblast.. Detected day 8–9 post-ovulation - Peaks 10–12 weeksمهم, declines by 16–20 - Maintains corpus luteum - Stimulates feta l testes to produce testosterone
Estrogen	- From placenta (via fetal DHEA مهم) - Enlarges uterus, breast, genitalia - Relaxes pelvic joints
Progesterone	- From corpus luteum early(involutes slowly after the 13th to 17th week), placenta later - Supports endometrium - ↓ Uterine contractility - Stimulates tube & uterus secretion - Prepares breast for lactation

The placenta converts the androgen hormone produced by the fetal adrenal cortex, dehydroepiandrosterone (DHEA), into estrogen.
** The placenta cannot produce estrogen until the fetus has developed to the point that its adrenal cortex is secreting DHEA into the blood. The placenta extracts DHEA from the fetal blood and converts it into estrogen, which it then secretes into the maternal blood.



Pregnancy, Labor and Lactation:

🧬 Response of the mother's body to pregnancy

Category	Notes
Weight gain	- Average: 10–15 kg - Mostly in last 2 trimesters - Fetus ~3.5 kg, amniotic fluid 1.5 kg - Enlarged uterus, breasts, blood volume, fat storage
Appetite	- Increased desire for food - Caused by fetal nutrient draw + hormones - Risk of excess weight gain if uncontrolled (up to 75 lb)

🔥 Metabolism during pregnancy

Factor	Details
Hormones	- Thyroxine, adrenocortical, sex hormones ↑

BMR	- Increases ~15% (latter half of pregnancy) - Extra energy for muscle activity - Sensation of overheating

Endocrine glands

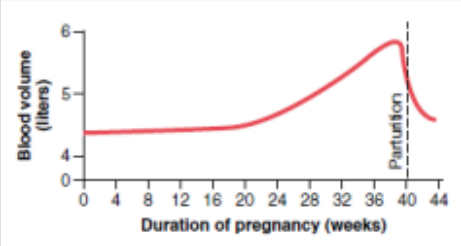
Gland	Response
Anterior pituitary	- Enlarges ~50% *imp* - ↑ ACTH, TSH, prolactin - ↓ FSH, LH (inhibited by estrogen & progesterone)
Adrenal cortex	- ↑ Glucocorticoids, aldosterone
Thyroid	- Enlarges, ↑ T_4 secretion
Parathyroid	- Enlarges - ↑ Ca^{2+} absorption from bone - Needed for fetal bone development - ↑ secretion during lactation

Nutrition during pregnancy

Nutrient	Importance
Protein, calcium, phosphate, iron	- Required in large amounts - Stored in placenta & maternal reserves
Iron	- Deficiency → hypochromic anemia
Vitamin D	- Helps absorb calcium (normally poorly absorbed)
Vitamin K	- Prevents neonatal hemorrhage (esp. brain)
Fetal growth	- Greatest in 3rd trimester (weight almost doubles in last 2 months)

Cardiovascular changes

Factor	Change
Cardiac output	- ↑ by 30–40% (peaks by week 27) - Drops slightly last 8 weeks
Placental blood flow	- ~625 ml/min
Blood volume	- ↑ by ~30% Caused by aldosterone, estrogens - Extra RBCs from active marrow - Buffer for delivery bleeding (~1–2 L extra, 350 ml lost)



Therefore, at the time of the birth of the baby, the mother has about 1 to 2 liters of extra blood in her circulatory system. Only about 350 ml is normally lost through bleeding during delivery of the baby, thereby allowing a considerable safety factor for the mother.

Respiratory changes

Factor	Effect
O_2 use	↑ 20% due to ↑ BMR + size
Ventilation	↑ minute ventilation
Diaphragm	Pressed up → ↑ RR
Progesterone	↑ sensitivity to CO_2 at brainstem

🧪 Renal changes

Function	Notes
Urination	↑ due to fluid load and excretion
Tubular function	↑ Na^+, Cl^-, water reabsorption
GFR	↑ via vasodilation (relaxin)

🤰 Labor & parturition

Trigger	Mechanism
Estrogen/progesterone	- Estrogen > progesterone → ↑ uterine contractility - Estrogen ↑ gap junctions
Oxytocin	- ↑ Receptors near term - ↑ Secretion during labor - Cervical stretch → positive feedback
Fetal hormones	- Fetal oxytocin, cortisol, prostaglandins all ↑ contractions
Mechanical	- Uterine stretch ↑ excitability - Cervical stretch = reflex uterine contraction - Twins born ~19 days earlier

**Note especially that twins are born, on average, 19 days earlier than a single child, which emphasizes the importance of mechanical stretch in eliciting uterine contractions stretching or irritation of nerves in the cervix initiates reflexes to the body of the uterus, OR result simply from myogenic transmission of signals from the cervix to the body of the uterus.

🔁 Positive feedback in labor

Step	Process
1	Fetal head stretches cervix
2	Cervical stretch → ↑ uterine contractions
3	Contractions push fetus → more stretch
4	Feedback loop continues until delivery
Failure	Weak contractions → feedback halts

💪 Abdominal muscles during labor

- Pain from strong uterine contractions → neurogenic reflexes → trigger abdominal contraction → aids expulsion

---

🕑 Stages of labor

Stage	Duration
1st	True labor onset → full cervical dilation
2nd	Full dilation → birth of baby
3rd	Baby delivery → placenta delivery
4th	Placenta delivery → maternal stabilization (~6 hrs)

وَوَصَّيْنَا ٱلْإِنسَٰنَ بِوَٰلِدَيْهِ إِحْسَٰنًا ۖ حَمَلَتْهُ أُمُّهُۥ كُرْهًا وَوَضَعَتْهُ كُرْهًا ۖ وَحَمْلُهُۥ وَفِصَٰلُهُۥ ثَلَٰثُونَ شَهْرًا ۚ حَتَّىٰٓ إِذَا بَلَغَ أَشُدَّهُۥ وَبَلَغَ أَرْبَعِينَ سَنَةً قَالَ رَبِّ أَوْزِعْنِىٓ أَنْ أَشْكُرَ نِعْمَتَكَ ٱلَّتِىٓ أَنْعَمْتَ عَلَىَّ وَعَلَىٰ وَٰلِدَىَّ وَأَنْ أَعْمَلَ صَٰلِحًا تَرْضَىٰهُ وَأَصْلِحْ لِى فِى ذُرِّيَّتِىٓ ۖ إِنِّى تُبْتُ إِلَيْكَ وَإِنِّى مِنَ ٱلْمُسْلِمِينَ ﴿١٥﴾

[الأحقاف]

كُرهًا = مشقة وتعب

🩸 Placenta delivery

Event	Notes
Separation	Opens sinuses → bleeding (~350 ml)
Uterus contraction	Constricts vessels - Prostaglandins help - Shearing detaches placenta (10–45 min)

😣 Labor pain

Phase	Cause
Early	Uterine hypoxia from blood vessel compression
Late	Cervical & perineal stretch, vaginal tearing

**It is fortunate that the contractions of labor occur intermittently, because strong contractions impede or sometimes even stop blood flow through the placenta and would cause death of the fetus if the contractions were continuous. Indeed, overuse of various uterine stimulants, such as oxytocin, can cause uterine spasm rather than rhythmic contractions and can lead to death of the fetus.

🍼 Lactation & breast development

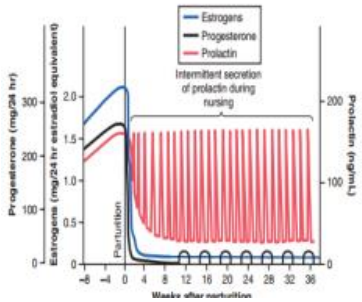
Hormone	Fx.
Estrogen	- Puberty: ductal growth + fat - Pregnancy: extensive ductal branching
Progesterone	- Lobule-alveolar development - Secretory characteristics
Prolactin	- ↑ 10–20x during pregnancy - Milk production post-delivery - Suppressed by estrogen/progesterone during pregnancy - Colostrum (pre-milk) produced first - If nursing does not continue, the breasts lose their ability to produce milk within 1 week or so. مهم

characteristics in the cells of the alveoli.

NOTES

Once the ductal system has developed, progesterone—acting synergistically with estrogen, as well as with the other hormones just mentioned—causes additional growth of the breast lobules, with budding of alveoli and development of secretory characteristics in the cells of the alveoli.

Oxytocin	- Milk ejection (let-down) - Suckling → hypothalamic signal → oxytocin release - Myoepithelial contraction - When the baby suckles, it receives virtually no milk for the first half minute or so.



🕒 Suppression of ovarian cycle

Cause	Effect
Prolactin + breast stimulation	↓ GnRH → ↓ FSH & LH → no ovulation during nursing

Male Reproductive System:

🧬 GnRH and pituitary hormone control

Hormone	
GnRH	- Secreted intermittently every 1–3 hours - Stimulus intensity depends on frequency & quantity
LH	- Mirrors GnRH pulses closely - Stimulates Leydig cells to produce testosterone
FSH	- Slower, steadier changes - Binds Sertoli cells → supports spermatogenesis
Inhibin	- From Sertoli cells - ↓ FSH when spermatogenesis is high

** Furthermore, when tumors develop from the interstitial cells of Leydig, great quantities of testosterone are secreted. When the germinal epithelium of the testes is destroyed by x-ray treatment or excessive heat, the Leydig cells, which are less easily destroyed, often continue to produce testosterone.

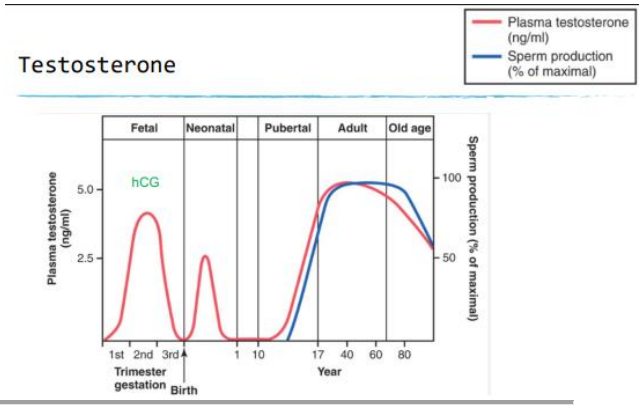
🧪 Spermatogenesis

Phase	Description
Initiation	- Starts at puberty - Needs FSH + testosterone
Duration	- ~74 days total
Location	- Seminiferous tubules
Hormonal control	- LH → Leydig cells → testosterone - FSH → Sertoli cells → spermatogenic substances
Feedback	- ↓ sperm → ↑ FSH - Fast sperm production → ↓ FSH via inhibin

🥯 Testosterone production

Stage	Testosterone status

Fetal	hCG stimulates Leydig cells to make testosterone مهم
Newborn	Leydig cells active for a few months
Childhood	Leydig cells mostly inactive
Puberty onward	Pituitary LH drives production Pituitary LH → active Leydig cells
Peak	Ages 20–50
Decline	Drops to 20–50% by age 80



👶 Testosterone in fetal life

Function	Role
SRY gene	- Triggers differentiation into testes - Leads to testosterone production
Organ development	- Penis, scrotum formation - Testes descent in last 2–3 gestational months

💪 Testosterone adult effects مهم

Trait	Effect
Genitalia	Penis, scrotum, testes enlarge post-puberty
Hair	↑ Pubic, facial hair; ↓ scalp hair in baldness-prone individuals
Voice	Larynx enlargement, deeper voice
Skin	Thicker skin, potential acne
Muscle	↑ Protein formation and muscle mass
Bone	↑ Matrix, Ca^{2+} retention, thickness
Metabolism	↑ Basal rate ~15%
RBCs	↑ by 15–20%
Electrolytes	↑ Na^{+} reabsorption in kidney

🧬 Spermatogenesis overview

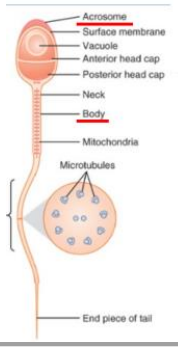
Stage	Description
Location	Seminiferous tubules
Start	After puberty, continues lifelong
Duration	~74 days total

**Spermatogonia that cross the barrier into the Sertoli cell layer become progressively modified and enlarged to form large primary spermatocytes.
**The rate of spermatogenesis is constant and cannot be accelerated by hormones such as gonadotropins or androgens.
** In the female, the mitotic proliferation of germ cells takes place entirely before birth. In the male, spermatogonia proliferate only after puberty and then throughout life
** The entire period of spermatogenesis, from spermatogonia to spermatozoa, takes about 74 days.

Trigger	FSH + testosterone

🔁 Spermatogenesis process (from embryo to adult)

Step	Detail
Embryo	Primordial germ cells migrate → become spermatogonia
At puberty	Spermatogonia undergo mitosis → generate Type A & Type B
Type B → meiosis	→ Primary spermatocytes (2N DNA)
Meiosis	Each produces 4 spermatozoa (2X, 2Y)
From spermatids → spermatozoa	No division; structural change only



⚙️ Spermiogenesis (spermatid → spermatozoon) مهم تمييز الاسم

Part	Feature
Head	Condensed nucleus Acrosome (from Golgi) with enzymes (hyaluronidase, proteases)
Tail	Microtubules, mitochondria (energy supply, membrane)
Fx.	Allows fertilization: enzymes digest ovum's protective layers

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🌡️ Spermatogenesis & temperature

Factor	Effect
Heat ↑	Destroys germ cells besides spermatogonia. ↓ Spermatogenesis
Cold reflex	Scrotum contracts to preserve temp
Function	Scrotum maintains testes ~2°C below core body temp
Storage	24–48h at body temp Weeks refrigerated, Years frozen at −100°C

🩹 Epididymis role

Fx.	Detail
Transit	**Sperm pass slowly (several days)**
Motility	**Achieved after 18–24h, but Epididymal fluid contains proteins that inhibit motility until ejaculation**

🧬 Hormonal factors stimulating spermatogenesis

Hormone	Function
Testosterone	Growth & division of testicular germinal cells
LH	Stimulates Leydig cells → testosterone
FSH	Enables spermiogenesis (spermatids → sperm) *look at Rt.*
Estrogen	Formed by Sertoli cells from testosterone
Growth hormone	Supports metabolic activity & early spermatogonia division

3. Follicle-stimulating hormone → without this stimulation, conversion of the spermatids to sperm (the process of spermiogenesis) will not occur.

🔬 Semen composition

Source	% / Function
Vas deferens	~10%, contains sperm
Seminal vesicle	~60%, rich in fructose, prostaglandins, fibrinogen
Prostate	~30%, milky fluid: citrate, enzymes, calcium, profibrinolysin. (alkaline fluid)
Bulbourethral glands	Few Mucus secretion
pH	~7.5, alkaline from prostate neutralizes acidic fluids

**Prostaglandins are believed to aid fertilization in two ways: (1) by reacting with the female cervical mucus to make it more receptive to sperm movement and (2) by possibly causing backward, reverse peristaltic contractions in the uterus and fallopian tubes to move the ejaculated sperm toward the ovaries (a few sperm reach the upper ends of the fallopian tubes within 5 minutes).
**A slightly alkaline characteristic of the prostatic fluid may be quite important for successful fertilization of the ovum because the fluid of the vas deferens is relatively acidic owing to the presence of citric acid and metabolic end products of the sperm and, consequently, helps to inhibit sperm fertility. Also, the vaginal secretions of the female are acidic (pH of 3.5 to 4.0). Sperm do not become optimally motile until the pH of the surrounding fluids rises to about 6.0 to 6.5. Consequently, it is probable that the slightly alkaline prostatic fluid helps to neutralize the acidity of the other seminal fluids during ejaculation and thus enhances the motility and fertility of the sperm.
**The average pH of the combined semen is about 7.5, the alkaline prostatic fluid having more than neutralized the mild acidity of the other portions of the semen.

🧠 Fertility support mechanisms

Factor	Action
Prostaglandins	react with cervical mucus, ↑ uterine contractions
Alkalinity	Neutralizes vaginal and vas deferens acidity

⚖️ Spermatogenesis vs oogenesis *imp*

Feature	Males	Females
Germ cell division	After puberty, lifelong	All mitosis before birth
Meiosis products	4 sperms	1 ovum + polar bodies
Post-meiosis	Spermatids mature	No further changes without fertilization

🧠 Male sexual act – nervous control

Phase	Nervous system

Initiation	Spinal reflex (lumbar + sacral) via psychic or tactile stimuli
Erection	Parasympathetic (pelvic nerves) Release ACh, NO, VIP → cGMP ↑ → vasodilation
Emission	Sympathetic (T12–L2) Vas deferens, prostate, seminal vesicles contract
Ejaculation	Rhythmic contraction of genital tract & penile tissue
Resolution	Erection ceases within 1–2 minutes

🧬 Erection physiology

Action	Detail
NO release	Activates guanylyl cyclase → ↑ cGMP
Smooth muscle relaxation	Corpora cavernosa & spongiosum dilate
Blood flow ↑	Sinusoids fill under pressure; venous outflow ↓
Result	Penis becomes hard, elongated (erection)

🧬 Emission & ejaculation: sympathetic roles

Step	Description
Emission	- Vas deferens, ampulla contract → sperm into urethra - Prostate + seminal vesicles release fluids
Ejaculation	- Internal urethra fills - Sensory signals → reflex contraction - Rhythmic contractions expel semen
Orgasm	Emission + ejaculation phase
Resolution	End of erection & sexual excitement within 1–2 min

Abnormalities in the Reproductive System | Last Physio Lec in basic years ♡

🧬 Abnormal spermatogenesis and male fertility

Condition	Details
Sterility	- Due to damage to seminiferous epithelium - Causes: bilateral orchitis (mumps), congenital degeneration, high testicular temperature - Leydig cells may still produce testosterone after damage (not much sensitive to radiation)
Cryptorchidism	- Failure of testes to descend into scrotum - Testes remain abdominal → can't produce sperm - Causes: defective testosterone - Treatment: surgical correction

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🧫 Sperm factors and fertility

Factor	Detail
Sperm count	- Normal: ~120 million/mL m (35 million to 200 million | (total ~400 million in each ejaculate.) - Infertility risk: <20 million/mL الأرقام مهمة ممكن ييجي عليها سؤال -quantity of semen ejaculated bout 3.5 ml.
Morphology	- May be abnormal (2 heads, odd tails, malformed heads) - Even with normal count, infertility possible
Motility	- Structurally normal sperm may be nonmotile or poorly motile - Nonmotility = high infertility risk . - Whenever the majority of the sperm are morphologically abnormal or are nonmotile, the person is likely to be infertile, even though the remainder of the sperm appear to be normal.

🧠 Male sexual function disorders 🚹

Issue	Note
Prostate gland	- Grows at puberty → static until ~50, then involutes - BPH causes urinary obstruction - Cancer stimulated by testosterone . - Estrogens/removal of testes used as treatment
Hypogonadism (fetal)	- Nonfunctional testes → female organ development - Caused by lack of testosterone suppression of female traits
Hypogonadism (pre-pubertal)	- Eunuchism: infantile organs, weak muscle, high-pitched voice, feminized hair distribution The height of an adult eunuch is slightly ***greater*** than that of a normal man.
Hypogonadism (post-pubertal)	- Testes removed → organs regress - Low libido, poor ejaculation, mild voice regression

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🦠 Testicular tumors and hypergonadism

Type	Effect
Leydig cell tumors	- Massive testosterone secretion (100x of testosterone) - In children: rapid growth, early puberty - Skeletal epiphyses fuse early
Germinal epithelium tumors	- More common - Teratomas with mixed tissues (teeth, skin, hair) - Secrete hCG or estrogen

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🧬 Erectile dysfunction (impotence)

Cause	Note
Neurologic	Trauma to parasympathetic nerves (e.g. post-prostate surgery)
Hormonal	Low testosterone
Drug-induced	Nicotine, alcohol, antidepressants
Vascular	Hypertension, diabetes, atherosclerosis

Treatment	PDE-5 inhibitors (e.g. sildenafil) ↑ cGMP → vasodilation → erection

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🧪 Ovarian secretion disorders:

Condition	Detail
Hypogonadism (pre-puberty)	- Female eunuchism: no secondary sex traits - Infantile organs - Prolonged growth of long bones
Hypogonadism (adult)	- Ovary removal → regression of uterus, vagina, breasts, pubic hair

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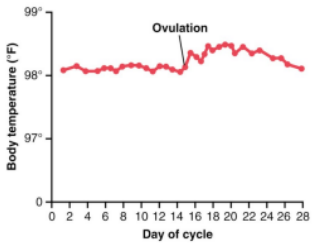
🧬 Female infertility and ovulation issues

Condition	Cause
Female sterility	- Most common: failure to ovulate - Causes: low gonadotropins, ovarian defect
Anovulatory cycles	- LH surge insufficient - No ovulation → no corpus luteum - No progesterone, cycle shortens
Ovulation Detection	- Urine: ↑ pregnanediol (progesterone metabolite) - Body temp: ↑ by 0.5°F during luteal phase (due to progesterone)



💊 Ovulation treatment

Method	Notes
hCG therapy	- Mimics LH - Stimulates ovulation - Overuse: multiple follicle ovulation → multiple births (e.g. 8 babies)

🧠 Polycystic ovarian syndrome (PCOS)

Feature	Detail
Mechanism	- Hormone imbalance in reproductive years - Cysts: fluid sacs with immature follicles - Eggs not released regularly
Possible causes	- Hereditary - Insulin resistance - Excess androgens - Low-grade inflammation
Symptoms	- Irregular periods - Hirsutism - Enlarged polycystic ovaries
Treatment	- Lifestyle change - Hormonal therapy (combination pills, progestins) - Metformin

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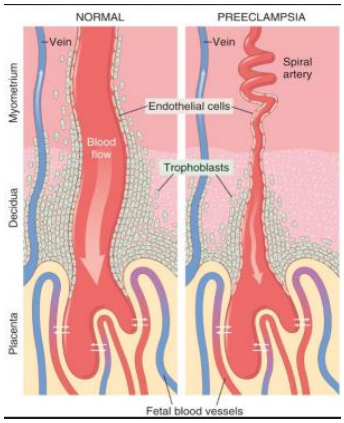
💥 Preeclampsia and eclampsia

Condition	Feature

Preeclampsia	- Pregnancy-induced hypertension (late pregnancy) - Symptoms: proteinuria, edema, wt gain, arterial spasm - Cause: placental ischemia → TNF-α, IL-6 ↑ → endothelial dysfunction
Eclampsia	- Severe preeclampsia: seizures, coma, organ failure - High mortality without treatment
Treatment	- Vasodilators - Emergency delivery → mortality <1% الحل هو توليد المريضة



The End

لا يتَعاظَمهُ شيء؛ هو العظيم الأعظم، ولا يعجزهُ شيء؛ هو القدير القادر، ولا يخفى عليه شيء؛ هو العليم الأعلم. لا يَعظُمُ عليه أمرك، ولا يُعجزه مُرادك، ولا يخفىٰ عليه أنين صدرك، وشدّة احتياجك، وإنّ تلكُ مثقال دمعة من ألمٍ يراها، وإن تلكُ مثقال زفرة من همٍّ يسمعها، هو الأقرب والأرحم والأحكم، أحاط بكل شيءٍ علمًا؛ وسيُعطي حتىٰ يُرضي، وسيجبر، وسيؤتِ كُلّ قلبٍ مؤمنٍ صادقٍ سؤله، فتوكل عليه إنهُ يُحبّ المُتَوكلين.