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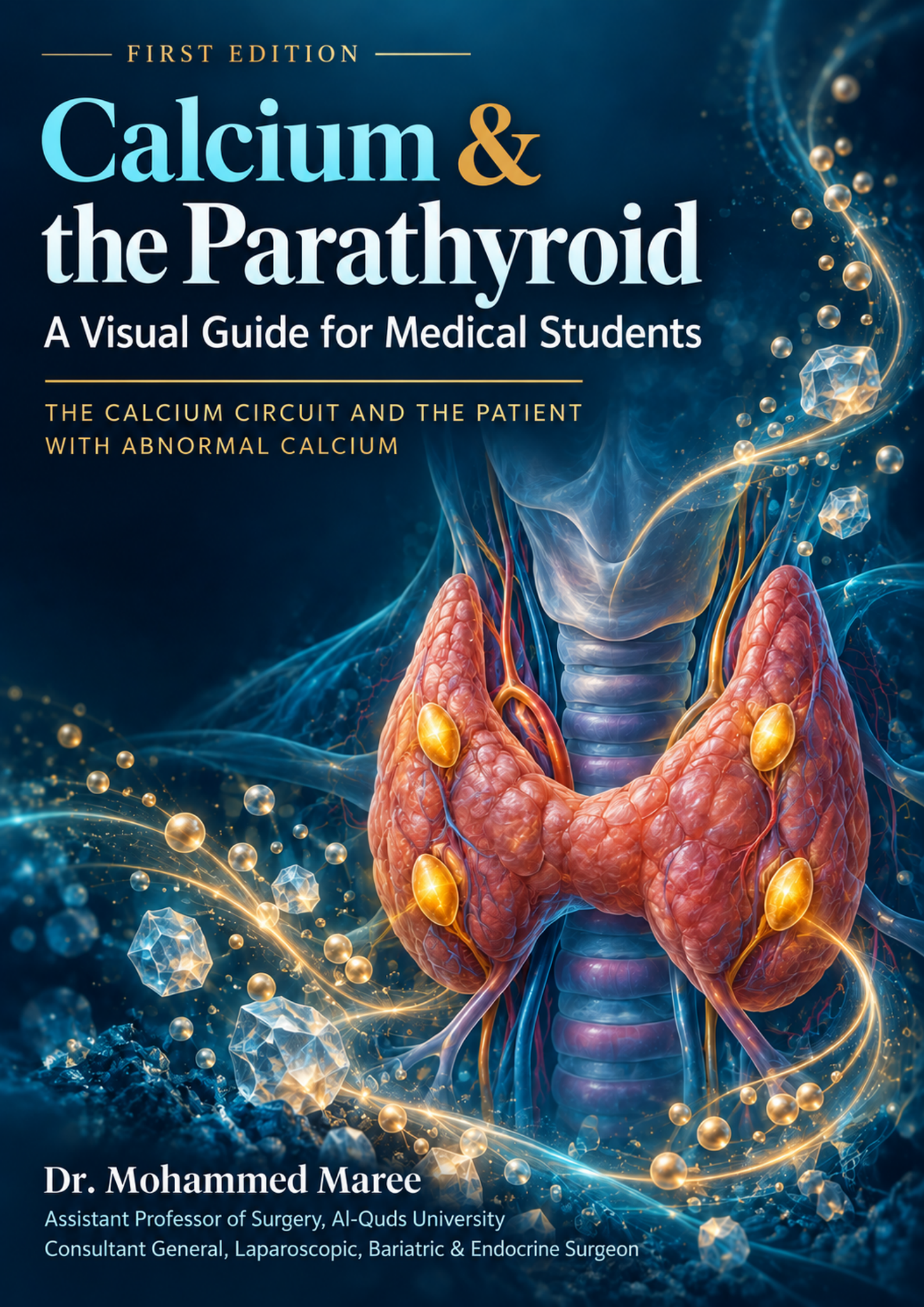
Calcium & the Parathyroid

A Visual Guide for Medical Students

THE CALCIUM CIRCUIT AND THE PATIENT
WITH ABNORMAL CALCIUM

Dr. Mohammed Maree

Assistant Professor of Surgery, Al-Quds University
Consultant General, Laparoscopic, Bariatric & Endocrine Surgeon



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This book is an educational resource for medical students. It does not replace clinical judgement, senior advice or local guidelines. Drug doses and reference intervals must be checked against your own hospital's protocols before use.

Two halves, one idea

Calcium is one of the few things the body defends minute by minute. It is held inside a band roughly one and a half milligrams per decilitre wide, and it is defended by four glands that together weigh less than a paperclip. Almost every calcium problem a surgeon meets — the woman with recurrent stones, the man whose bones ache, the patient whose fingers tingle the night after a thyroidectomy — is that defence system either working too hard or not working at all.

Part 1 — The calcium circuit walks the loop in ten steps. One page, one diagram, one idea per step. Read it once through before you read the clinical half; every clinical decision in Part 2 is simply a step of this circuit failing.

Part 2 — The patient takes the circuit to the bedside: how to read a calcium result, how three numbers give you the diagnosis, how to examine and work up the hypercalcaemic patient, when the disease becomes a surgical one, and how to handle the tingling fingers on the ward round after a neck operation.

Part 3 — Five patients puts both halves to work in five cases: a preoperative diagnosis worked step by step, an intra-operative PTH that does not fall, a family with FHH, a ward round after thyroidectomy, and tertiary hyperparathyroidism after a kidney transplant.

THE FOUR MILESTONES

M1 — Describes the calcium circuit and names the actions of PTH.

M2 — Interprets a calcium, PTH and phosphate panel and reaches a diagnosis.

M3 — Assesses a patient with hypercalcaemia: history, examination, first-line investigations.

M4 — Acts as an intern: recognises hypercalcaemic crisis and post-thyroidectomy hypocalcaemia, starts first-line treatment, and escalates appropriately.

A note on units. All values in this book are given in **mg/dL**, matching the chemistry report you will be handed on the ward. To convert calcium to mmol/L, multiply by 0.25 (10.0 mg/dL = 2.5 mmol/L). Reference intervals are printed on page 17; check them against your own laboratory's sheet before you quote them in an exam or a clinic letter.

Boxes like this one carry what the patient says. Ruled boxes carry what you say to the examiner. Red-ruled boxes carry the thing that, if you miss it, harms someone.

Calcium & the Parathyroid

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WHERE THIS SITS IN THE ROTATION

Read Part 1 before the endocrine tutorial. Read Part 2 alongside the *Thyroid Swelling Trainer* and the thyroid and neck-lump OSCE station — the calcium story is what happens to your OSCE patient the day after her operation.

PART ONE

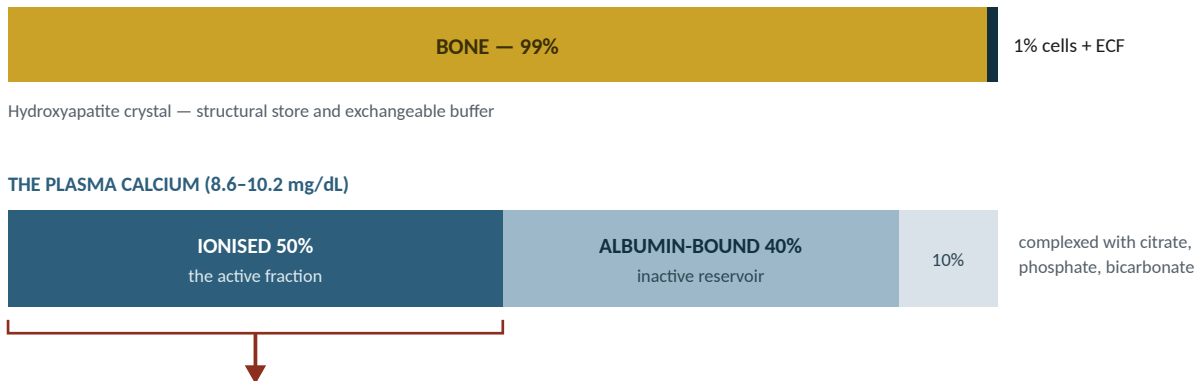
The calcium circuit

Ten steps around a loop that runs every minute of every day, from the sensor on the chief cell to the calcium crossing the gut wall and back again.

Where the calcium is

A kilogram in the skeleton, a gram in the blood, and only half of that gram doing any work.

TOTAL BODY CALCIUM $\approx$ 1 kg



Only this fraction is sensed, and only this fraction causes symptoms.

The laboratory measures the whole bar. If albumin is low, the bar shortens — but the dark blue block, the part that matters, may not have moved at all.

Figure 1. The skeleton holds the stock; the plasma holds the working balance. Of that working balance, only the ionised half is physiologically active — which is why a total calcium can never be read without its albumin.

WHAT IONISED CALCIUM IS FOR

- ◆ **Neuromuscular excitability** — extracellular calcium stabilises the sodium channel. Less calcium, more excitable nerve: tingling, cramp, tetany. More calcium, less excitable: fatigue, constipation, confusion.
- ◆ **Muscle contraction**, including cardiac — hence the QT interval moves with calcium.
- ◆ **Coagulation** — Factor IV. This is why blood for transfusion is citrated, and why massive transfusion drops the ionised calcium.
- ◆ **Secretion and signalling** — the intracellular second messenger for almost everything.

SURGICAL RELEVANCE

In massive transfusion, citrate chelates ionised calcium faster than the liver can clear it. Total calcium may look normal while the ionised falls — the patient becomes hypotensive and coagulopathic. Measure ionised calcium in any patient receiving large volumes of blood.

SAY THIS TO THE EXAMINER

"Ninety-nine per cent of body calcium is in bone. Of the plasma calcium, about half is ionised and physiologically active, about forty per cent is bound to albumin and the remainder is complexed to anions. Only the ionised fraction is regulated, so a total calcium must always be interpreted alongside the albumin."

The thermostat

A receptor on the chief cell reads the ionised calcium every few seconds and sets the hormone accordingly.

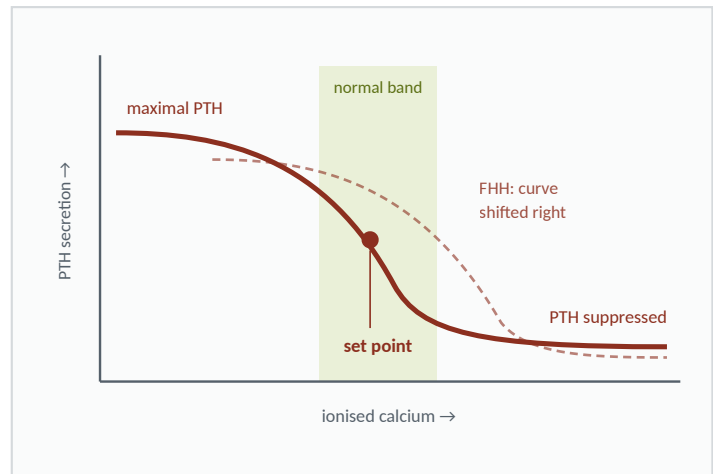
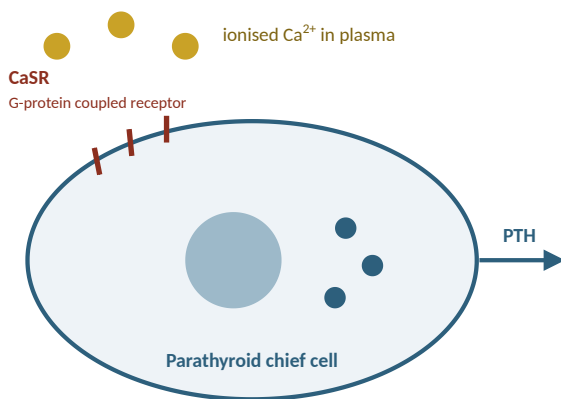


Figure 2. The relationship between ionised calcium and PTH is a steep inverse sigmoid. A fall of a few tenths of a milligram produces a large rise in hormone — which is why the system corrects so fast, and why PTH is so informative when calcium is abnormal.

THREE PROPERTIES WORTH REMEMBERING

- ◆ **It is fast.** The receptor acts within seconds on hormone already stored in granules.
- ◆ **It is steep.** Small deviations in calcium produce large swings in PTH.
- ◆ **It has a set point** that can be moved — by mutation, by drugs, and by chronic disease.

MOVING THE SET POINT

- ◆ **Familial hypocalciuric hypercalcaemia (FHH)** — an inactivating CASR mutation. The gland thinks calcium is low when it is high: mild lifelong hypercalcaemia, PTH normal or mildly raised, and — the giveaway — **low urine calcium**.
- ◆ **Cinacalcet** — a calcimimetic. It sensitises the receptor, so the gland thinks calcium is higher than it is, and PTH falls.

DO NOT OPERATE ON FHH

A patient with FHH who has a parathyroidectomy remains hypercalcaemic and has been harmed for nothing. Before any patient is listed for parathyroid surgery, ask for a 24-hour urine calcium or a calcium-to-creatinine clearance ratio. A ratio below 0.01 points to FHH, not to an adenoma.

Four glands, two origins

Together they weigh about 150 mg. Finding them is the whole operation.

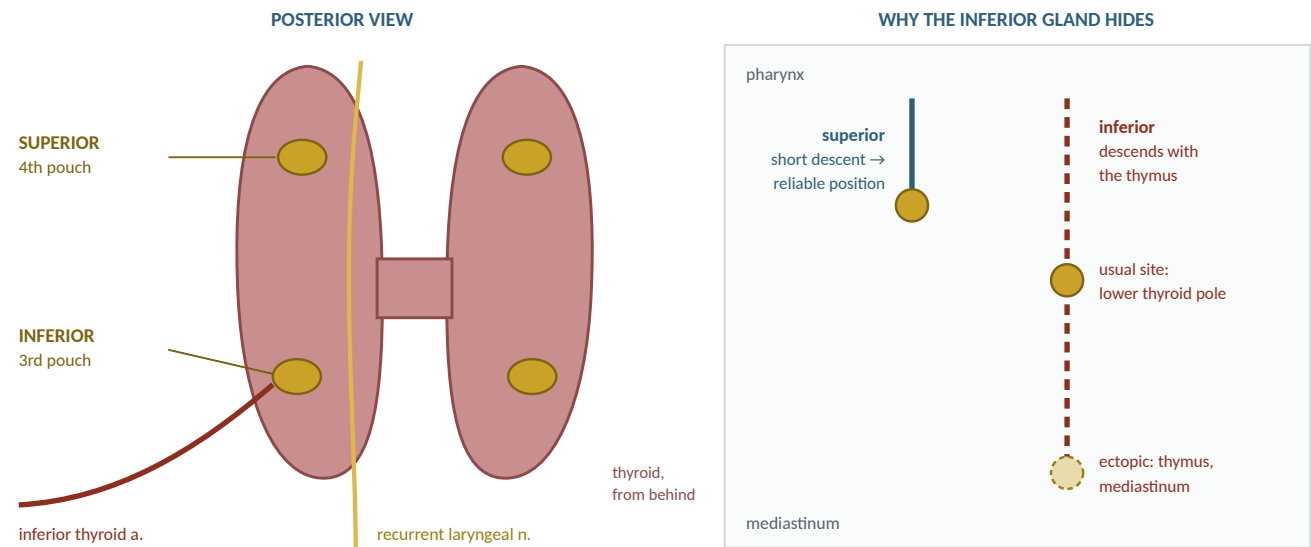


Figure 3. The superior glands come from the fourth pouch and travel almost nowhere, so they sit predictably at the cricothyroid junction. The inferior glands descend with the thymus from the third pouch, and can end up anywhere along that path.

	Superior pair	Inferior pair
Origin	4th pharyngeal pouch	3rd pharyngeal pouch (with thymus)
Usual site	Posterior, at cricothyroid junction, ~1 cm above where the inferior thyroid artery crosses the nerve	Near the lower thyroid pole, or within the thyrothymic ligament
Relation to RLN	Posterior (deep) to the nerve	Anterior (superficial) to the nerve
Position	Constant — easier to find	Variable — the usual culprit at re-exploration

SURGICAL RELEVANCE

Blood supply is mainly from the **inferior thyroid artery**, for all four glands. During thyroidectomy, ligate the branches on the thyroid capsule rather than the trunk of the artery, and the parathyroids keep their supply. A gland that turns dusky should be minced and autotransplanted into the sternocleidomastoid. Up to 13% of people have a fifth gland.

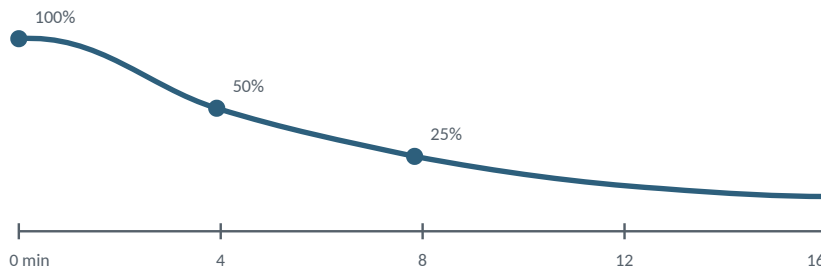
SAY THIS TO THE EXAMINER

"Four glands, about 40 mg each, tan-coloured. The superior pair arise from the fourth pouch and lie posterior to the recurrent laryngeal nerve; the inferior pair arise from the third pouch with the thymus and lie anterior to it. All are supplied mainly by the inferior thyroid artery, which is why capsular dissection matters in thyroid surgery."

Parathyroid hormone

Eighty-four amino acids with a half-life of about four minutes — a hormone built for minute-to-minute control.

A HORMONE THAT DISAPPEARS IN MINUTES



This is what makes intra-operative PTH monitoring possible.

Remove the adenoma and the hormone is gone from the blood before the wound is closed.

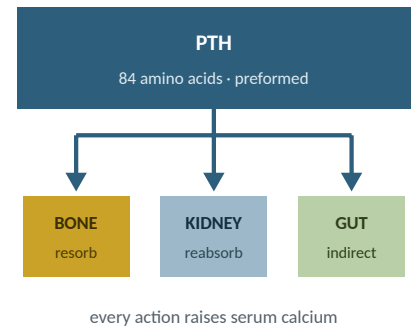


Figure 4. PTH is stored ready-made and cleared fast. Three target organs, one direction of travel: calcium up.

FOUR THINGS PTH DOES, IN ONE SENTENCE EACH

- ◆ **Bone** — drives osteoclastic resorption, releasing calcium *and* phosphate.
- ◆ **Kidney, distal tubule** — reabsorbs calcium.
- ◆ **Kidney, proximal tubule** — dumps phosphate into the urine.
- ◆ **Kidney, 1 α -hydroxylase** — activates vitamin D, which then absorbs calcium from the gut.

Why phosphate goes the opposite way. Bone resorption releases calcium and phosphate together. If phosphate were allowed to stay, it would complex the calcium straight back out of solution. So PTH simultaneously throws phosphate into the urine. This single fact explains the classic primary hyperparathyroid panel: **calcium high, phosphate low**.

SURGICAL RELEVANCE — THE MIAMI CRITERION

Sample PTH before incision and again 10 minutes after the suspect gland is removed. A fall of more than **50% from the highest pre-excision value**, into the normal range, predicts cure; the neck can be closed. If it does not fall, there is more disease — keep exploring.

PTH on bone

PTH never touches the osteoclast. It speaks to the osteoblast, and the osteoblast gives the order.

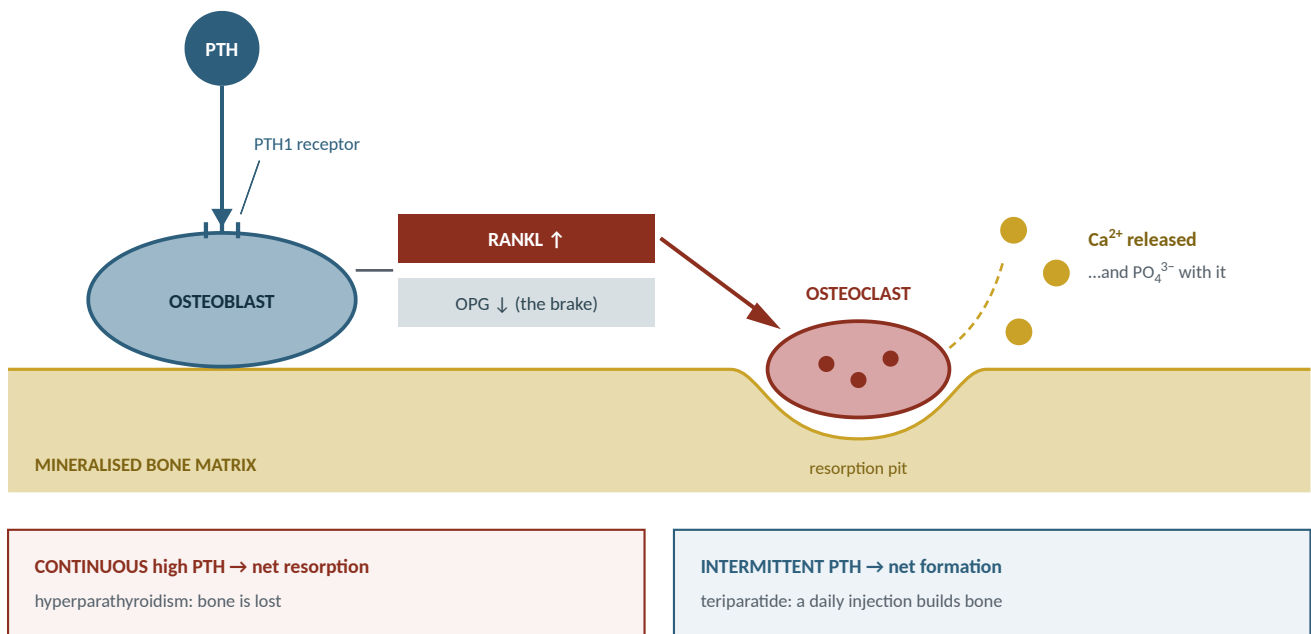


Figure 5. The osteoblast carries the receptor; RANKL carries the message; the osteoclast does the digging. Whether bone is gained or lost depends not on how much PTH there is, but on whether it arrives continuously or in pulses.

WHAT CHRONIC PTH EXCESS DOES TO THE SKELETON

- ◆ **Cortical bone suffers first.** Measure the distal radius on DEXA as well as spine and hip — it is the site most affected in hyperparathyroidism and the one most often omitted.
- ◆ **Subperiosteal resorption** on the radial side of the middle phalanges — the classic radiographic sign.
- ◆ **Salt-and-pepper skull**, loss of the lamina dura around the teeth.
- ◆ **Osteitis fibrosa cystica** and **brown tumours** — now rare, seen in long-neglected or severe disease.

SAY THIS TO THE EXAMINER

"PTH acts on the PTH1 receptor on osteoblasts and osteocytes, not on osteoclasts directly. It increases RANKL and reduces osteoprotegerin, so osteoclast differentiation and activity increase and mineral is released. Sustained elevation causes net bone loss, preferentially cortical."

The kidney: calcium in, phosphate out

Two tubular segments, two opposite instructions — and the explanation for the classic biochemical picture.

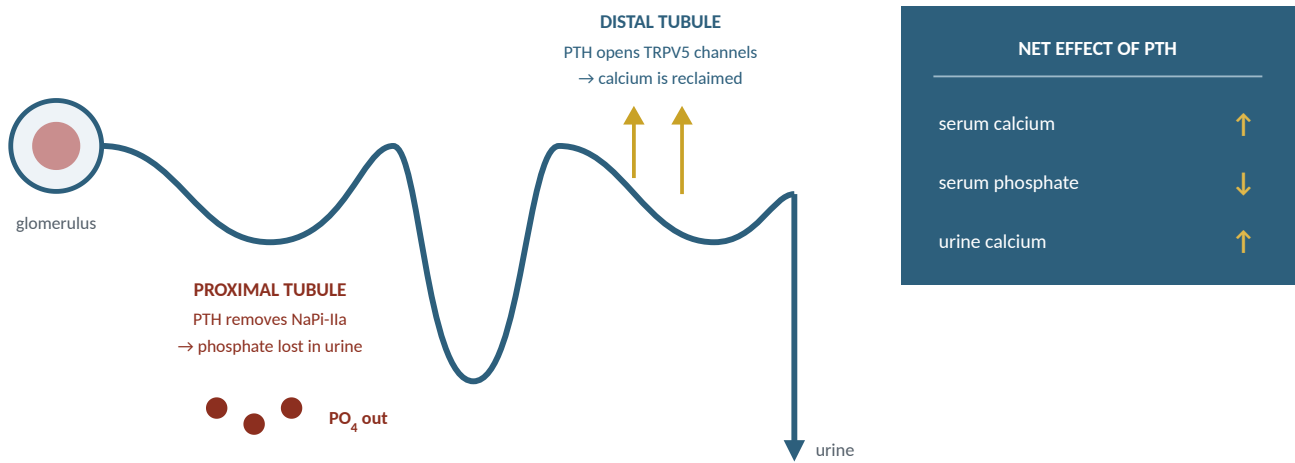


Figure 6. PTH reclaims calcium distally and wastes phosphate proximally. Note the third line of the box: urine calcium rises despite increased reabsorption, because so much more calcium is being filtered. That is where the stones come from.

The apparent contradiction. "PTH makes the kidney keep calcium — so why do these patients form stones?" Because the filtered load rises far more than the fractional reabsorption does. Reabsorbing 99% of a much larger number still leaves more calcium in the urine than before.

TWO DRUGS THAT MOVE CALCIUM

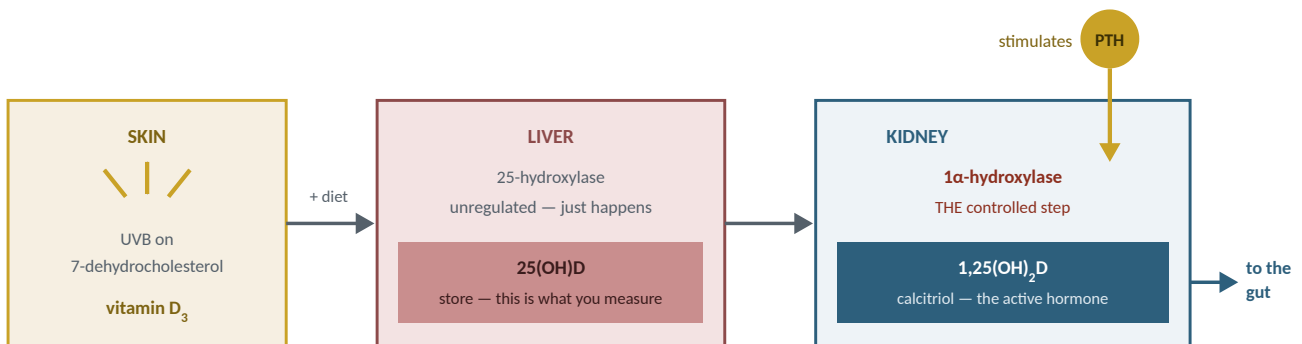
Thiazides reduce urinary calcium and can unmask or mimic hypercalcaemia — stop them and repeat before working anyone up.
Loop diuretics do the opposite and increase calcium loss.

SAY THIS TO THE EXAMINER

"PTH increases calcium reabsorption in the distal convoluted tubule and inhibits phosphate reabsorption in the proximal tubule. The result is a raised serum calcium with a low serum phosphate — and, because the filtered calcium load rises, hypercalciuria and a risk of nephrolithiasis."

Making the active vitamin D

Three organs, two hydroxylations — and only the last one is under hormonal control.



WHEN THE KIDNEY FAILS, THIS STEP FAILS

No 1α -hydroxylation → no calcitriol → gut absorbs less calcium → serum calcium falls → the four glands are driven hard, permanently. That is secondary hyperparathyroidism (page 25).

Figure 7. Sunlight starts it, the liver stores it, the kidney switches it on. Measure 25(OH)D to assess status — not calcitriol, whose level is often normal or even high in deficiency because PTH is driving the last step hard.

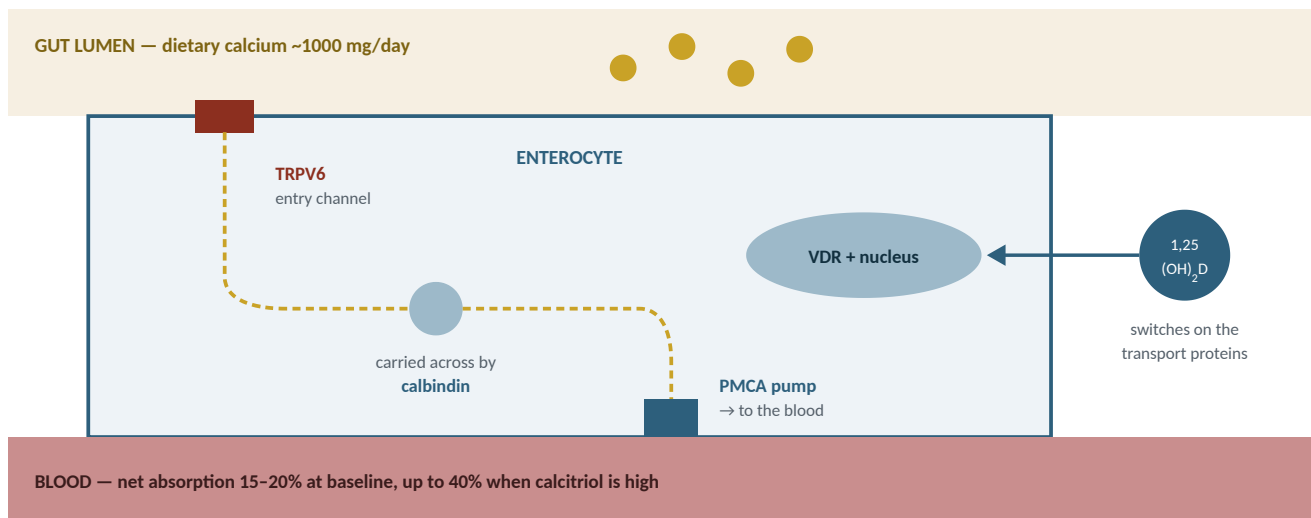
Which vitamin D do I request? 25(OH)D — the storage form with a half-life of weeks — is the test of vitamin D status. $1,25(\text{OH})_2\text{D}$ has a half-life of hours and is requested only in specific situations: suspected granulomatous disease or lymphoma, where macrophages make calcitriol outside the kidney's control and cause a PTH-independent hypercalcaemia.

SURGICAL RELEVANCE

Vitamin D deficiency is common and must be corrected *before* a calcium panel is called normal or abnormal — deficiency drives PTH up and can produce a picture indistinguishable from early primary hyperparathyroidism. Equally, a patient with primary hyperparathyroidism *and* deficiency has a hungrier skeleton, and a higher risk of severe hypocalcaemia after operation.

Calcitriol on the gut

The only place new calcium can enter the body — and the first segment of bowel a bariatric operation bypasses.



Most active absorption happens in the DUODENUM and PROXIMAL JEJUNUM; passive paracellular uptake continues along the ileum.

Figure 8. Calcitriol does not push calcium across the wall — it instructs the enterocyte to build the machinery that does. That is why its effect takes hours to days, not minutes.

SURGICAL RELEVANCE — THE BYPASSED DUODENUM

Roux-en-Y gastric bypass, and more so a duodenal switch, excludes exactly the segment where active, calcitriol-dependent absorption occurs, and reduces the gastric acid that frees calcium from its salts.

Two practical consequences:

- ◆ Prescribe **calcium citrate**, not carbonate — citrate does not need acid to dissolve.
- ◆ Expect a rise in PTH with a normal calcium — a mild secondary hyperparathyroidism that quietly removes bone. Check calcium, albumin, 25(OH)D and PTH at least annually after bypass.

Balance, for scale. Of roughly 1000 mg eaten each day, about 200 mg net is absorbed; the same 200 mg leaves in the urine. The skeleton exchanges around 500 mg a day in each direction. The circuit is not about gaining calcium — it is about holding a number steady.

Closing the loop

Calcium rises, the receptor notices, the hormone stops. Plus two minor players worth naming.

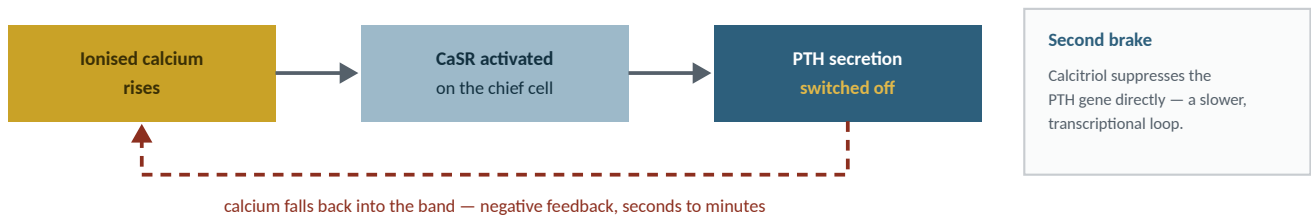


Figure 9. Two brakes of different speed: the receptor acts in seconds on stored hormone; calcitriol acts over hours on hormone production.

FGF23 — THE PHOSPHATE GUARD

- ◆ Secreted by **osteocytes** when phosphate and calcitriol rise.
- ◆ Wastes phosphate in the urine and **inhibits 1 α -hydroxylase**.
- ◆ Rises very early in chronic kidney disease — the first measurable sign of disordered mineral metabolism, before phosphate itself is abnormal.

CALCITONIN — THE ONE THAT BARELY MATTERS

- ◆ From thyroid **C cells**; lowers calcium by inhibiting osteoclasts.
- ◆ In humans it is **physiologically trivial**: total thyroidectomy does not cause hypercalcaemia, and medullary carcinoma with huge calcitonin levels does not cause hypocalcaemia.
- ◆ Useful in two places only: as a **tumour marker** in medullary thyroid carcinoma, and as a short-term drug in hypercalcaemic crisis.

A FAVOURITE EXAMINER'S QUESTION

"If calcitonin lowers calcium, why doesn't a total thyroidectomy make the patient hypercalcaemic?"

"Because calcitonin has no meaningful role in day-to-day calcium homeostasis in adults. Calcium after thyroidectomy falls, if anything — and that is a parathyroid problem, not a calcitonin one."

The circuit on one page

If you can redraw this from memory, you can reason your way through any calcium problem on the ward.

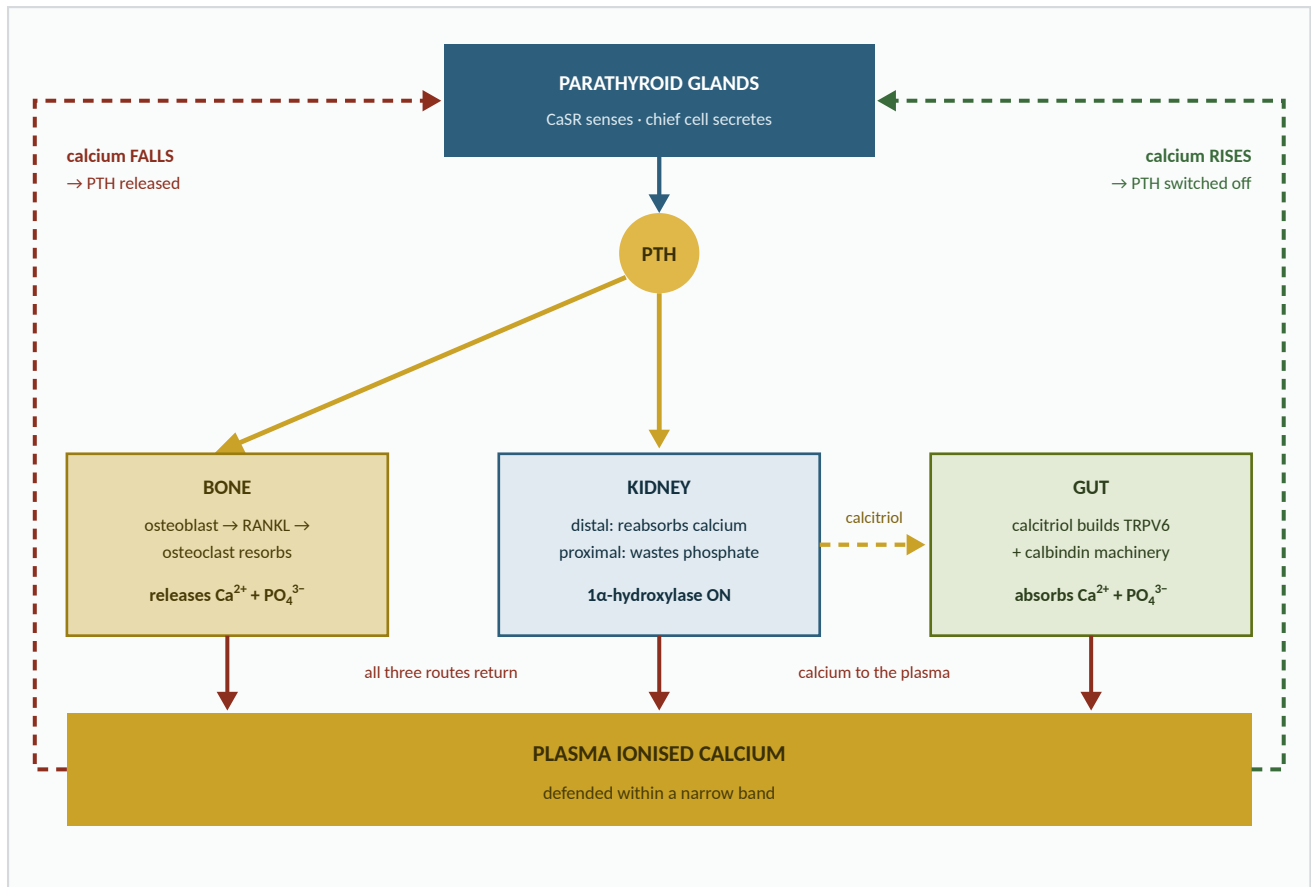


Figure 10. The complete circuit. Four glands watch one number; one hormone acts on three organs; every action returns calcium to the plasma; the rising calcium turns the hormone off.

TEST YOURSELF BEFORE TURNING THE PAGE

Cover the diagram and answer: (1) Which cell carries the PTH receptor in bone? (2) Which tubular segment reabsorbs calcium, and which wastes phosphate? (3) Which hydroxylation step is hormonally controlled, and where does it happen? (4) Why is phosphate *low* in primary hyperparathyroidism but *high* in renal failure?

PART TWO

The patient

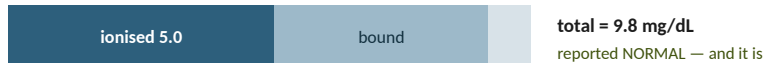
Reading the result, reaching the diagnosis, examining the patient, deciding on the operation — and managing the ward round after it.

Reading a calcium result

Before you diagnose anything, make sure the number in front of you is the number the patient actually has.

THE SAME PATIENT, TWO ALBUMINS

Albumin 4.0 g/dL



Albumin 2.4 g/dL (the sick surgical patient)



CORRECTED CALCIUM (mg/dL)

$$\text{Ca} + 0.8 \times (4.0 - \text{albumin})$$

$$\begin{aligned} \text{Worked: } & 8.5 + 0.8 \times (4.0 - 2.4) \\ & = 8.5 + 1.28 = \mathbf{9.8 \text{ mg/dL — normal}} \end{aligned}$$

The correction is only an estimate — if the answer matters, measure ionised calcium.

Figure 11. Albumin changes the length of the bar without moving the part that matters. Every calcium result must be read next to its albumin — in a surgical ward, where albumin is often low, this is not a formality.

FOUR WAYS A CALCIUM RESULT MISLEADS YOU

- ◆ **Low albumin** — the commonest. Correct it, as above.
- ◆ **Tourniquet and fist-clenching** — venous stasis concentrates protein and falsely raises total calcium. Take the sample without prolonged compression.
- ◆ **Acid–base shifts** — alkalosis increases albumin binding, so ionised calcium falls while total calcium is unchanged. This is why the hyperventilating patient gets perioral tingling and carpal spasm with a "normal" calcium.
- ◆ **The wrong tube** — EDTA contamination chelates calcium and produces a spuriously, sometimes impossibly, low result. Repeat before you treat.

REFERENCE INTERVALS USED IN THIS BOOK

Test	Reference interval	Note
Total calcium	8.6 – 10.2 mg/dL	$\times 0.25 = \text{mmol/L}$
Ionised calcium	4.5 – 5.3 mg/dL	the definitive measure
Albumin	3.8 – 4.4 g/dL	always request with calcium
Phosphate	2.5 – 4.5 mg/dL	the second diagnostic number
Magnesium	1.7 – 2.4 mg/dL	low Mg makes hypocalcaemia refractory
Alkaline phosphatase	35 – 105 U/L	raised with active bone disease
Creatinine	0.6 – 1.2 mg/dL	eGFR drives surgical indications
PTH (intact)	15 – 65 pg/mL	interpret only alongside calcium
25(OH) vitamin D	> 30 ng/mL sufficient	correct deficiency before judging PTH
24-hour urine calcium	100 – 300 mg/24 h	< 100 suggests FHH; > 400 is a surgical indication

Albumin and alkaline phosphatase intervals are those of the hospital General Laboratory; confirm the remainder against your local sheet before quoting them.

Three numbers, one diagnosis

Calcium tells you there is a problem. PTH tells you where it is. Phosphate confirms it.

PLOT THE PATIENT: CALCIUM AGAINST PTH

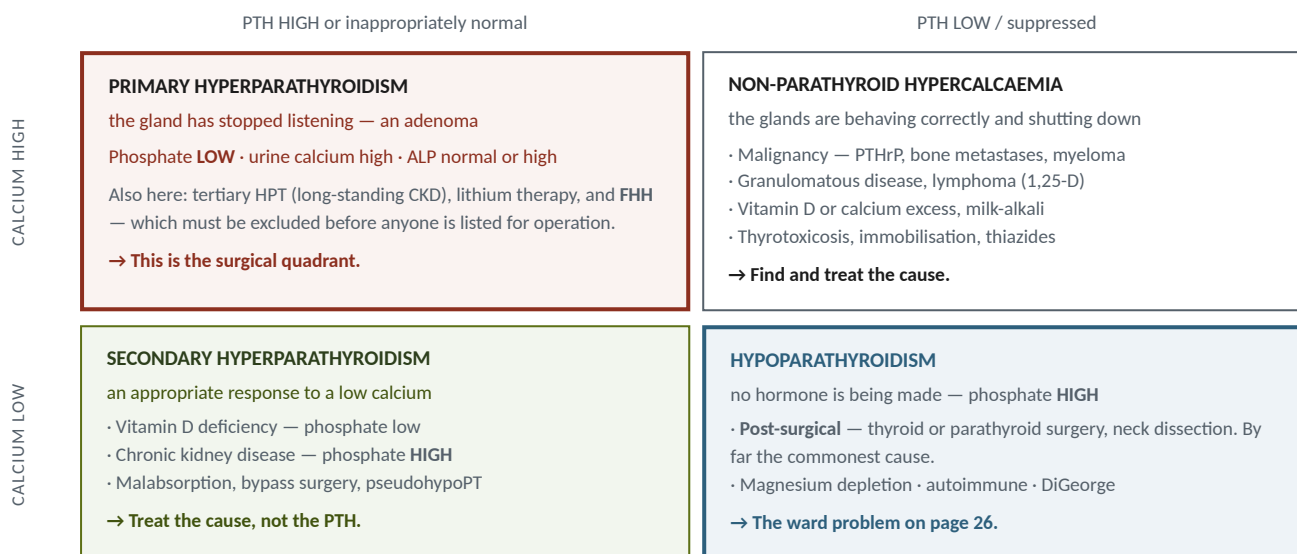


Figure 12. Four quadrants, four diseases. Adding phosphate separates the two causes inside each lower box, and separates PTH-driven hypercalcaemia (phosphate low) from all the rest (phosphate normal or high).

THE PHRASE THAT EARNS MARKS

"A PTH that is *normal* in the face of a *high* calcium is not a normal PTH — it is inappropriately normal, and it means the same thing as a raised one. A healthy gland faced with hypercalcaemia should be completely suppressed."

ORDER OF INVESTIGATION

Calcium and albumin → repeat to confirm → PTH, phosphate, magnesium, renal function, 25(OH)D → then 24-hour urine calcium. Only when this panel says **primary hyperparathyroidism** do you ask for imaging. Imaging tells you where a gland is; it never tells you whether the patient has the disease, and a negative scan does not overturn the biochemistry.

Taking the history

Most are found on a routine panel. The history's job is to decide whether this is a slow gland problem or a fast dangerous one.

PSYCHIC MOANS

fatigue, low mood, poor memory, confusion, in severe cases coma

STONES

renal colic, gravel, nephrocalcinosis, polyuria and thirst

BONES

bone and joint pain, fragility fracture, loss of height

ABDOMINAL GROANS

constipation, anorexia, nausea, peptic ulcer, pancreatitis

...and very often, nothing at all.

Most primary hyperparathyroidism today is found by a routine biochemistry panel.

Figure 13. "Stones, bones, abdominal groans and psychic moans" — useful as a prompt, misleading as an expectation. Ask about each one explicitly; patients rarely volunteer fatigue or constipation as symptoms.

ASK THESE, IN THIS ORDER

- ◆ **How long?** Old normal results in the file mean a chronic, gland-driven problem. A calcium normal six weeks ago and 13 mg/dL now means malignancy until proved otherwise.
- ◆ **Drugs** — thiazides, lithium, calcium and vitamin D supplements, antacids, vitamin A.
- ◆ **Red flags** — weight loss, breast or lung symptoms, bone pain, haematuria, smoking.
- ◆ **Stones and fractures** — the end-organ damage that decides the operation.
- ◆ **Family history** — hypercalcaemia, neck surgery, pituitary or pancreatic tumours, pheochromocytoma, thyroid cancer. MEN1 and MEN2A both start here.
- ◆ **Previous neck surgery or irradiation.**

"I just feel slowed down, doctor. I put it down to my age. I'm always thirsty, I'm up two or three times in the night to pass water, and I haven't opened my bowels properly in years. My mother had her neck operated on for something similar."

PRESENT IT LIKE THIS

"This is a 54-year-old woman with a six-month history of fatigue, polyuria, polydipsia and constipation, with a strong family history of neck surgery for hypercalcaemia. Her calcium is raised at 11.6 mg/dL with an inappropriately normal PTH and a low phosphate. This is primary hyperparathyroidism, and the family history raises the possibility of MEN type 1."

THE ONE DISTINCTION THAT CHANGES TODAY'S MANAGEMENT

Slow and mild (months to years, calcium < 12 mg/dL, few symptoms) — outpatient workup. **Fast and high** (days to weeks, calcium > 13 mg/dL, drowsy, dry) — admit, start intravenous fluids now, and look hard for a cancer.

The examination

You are not examining to find the adenoma. You are examining for the cause you must not miss, and for the damage already done.

Where you look	What you are looking for
General	Hydration — dry mucosae, tachycardia, postural drop. Hypercalcaemia causes a nephrogenic diabetes insipidus, so these patients are almost always volume-depleted. Cachexia or pallor suggests malignancy.
Neck	Usually impalpable — a parathyroid adenoma is rarely felt. A hard, fixed neck mass in a patient with a calcium above 14 mg/dL raises parathyroid carcinoma. Look also for a thyroid nodule and for previous scars.
Hunting a cancer	Breasts, chest, lymph node fields, prostate, abdomen for organomegaly; bone tenderness over spine, ribs and long bones.
Neuromuscular	Proximal muscle weakness, hyporeflexia, drowsiness, confusion. Assess conscious level formally if calcium is high.
Eyes	Band keratopathy at the corneal margin — chronic, rare, and a strong sign when present.
Bedside tests	ECG — short QT interval , and in severe cases bradyarrhythmia. Urine dipstick for blood (stones).

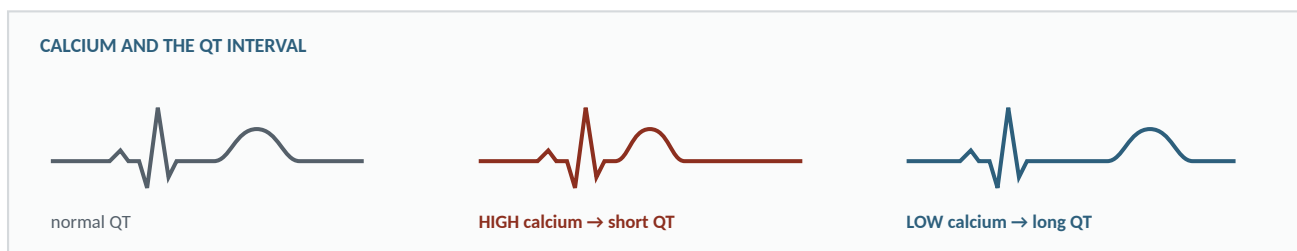


Figure 14. One ECG feature, two directions. Worth knowing in both halves of this book: the post-thyroidectomy patient on page 26 moves to the right of this figure.

CLOSING THE EXAMINATION

"On examination she was clinically dehydrated with a resting tachycardia. The neck was normal with no palpable mass or scar, breast and respiratory examination were normal, and there was no lymphadenopathy or bone tenderness. She had mild proximal weakness. Her ECG showed a shortened QT interval. I would like to start intravenous fluids and complete the biochemical panel."

What the imaging shows

The skeleton, the kidneys and the gland each leave a picture — but imaging stages the disease; it never diagnoses it.

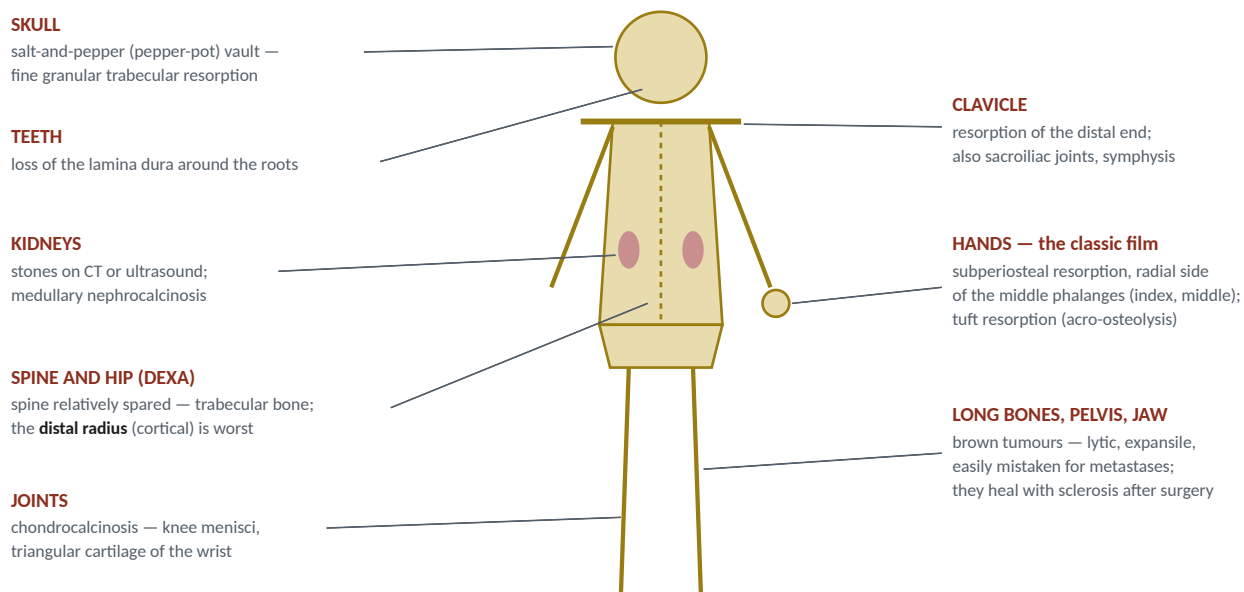


Figure 15. Where hyperparathyroidism shows on imaging. The bone signs of osteitis fibrosa cystica are now rare and mark long-neglected or severe disease; nephrocalcinosis, stones and a low distal-radius score are what you will actually find.

THE GLAND ITSELF — HOW AN ADENOMA LOOKS

Modality	Typical appearance of a parathyroid adenoma
Ultrasound	Oval, well-defined, homogeneously hypoechoic nodule behind or below the thyroid, separated from it by a thin bright fascial line. Doppler shows a polar feeding vessel entering one end, often with a rim of peripheral flow.
Sestamibi SPECT/CT	Tracer taken up by thyroid and adenoma alike, then washed out of the thyroid but retained by the adenoma on the delayed (about 2-hour) images; SPECT/CT gives the focus an anatomical address, including in the mediastinum.
4D-CT	Avid arterial-phase enhancement with washout on the delayed phase — the opposite of a lymph node, which enhances slowly.

TWO TRAPS

A **lytic lesion is not always a metastasis**. In a hypercalcaemic patient, check the PTH before a bone lesion is biopsied or labelled as cancer — a brown tumour looks the same on a plain film. A **normal scan does not exclude the disease**: small adenomas and four-gland hyperplasia are often invisible, and the decision to operate still rests on the biochemistry (page 18).

SAY THIS TO THE EXAMINER

"The pathognomonic radiographic sign is subperiosteal resorption on the radial aspect of the middle phalanges. Other skeletal signs are a salt-and-pepper skull, loss of the lamina dura, distal clavicular resorption and brown tumours. On ultrasound, an adenoma is a hypoechoic oval nodule behind the thyroid with a polar feeding vessel."

Primary hyperparathyroidism

One gland, in most patients, has stopped listening to the receptor — and only an operation cures it.

WHAT IS ACTUALLY THERE WHEN YOU OPEN THE NECK

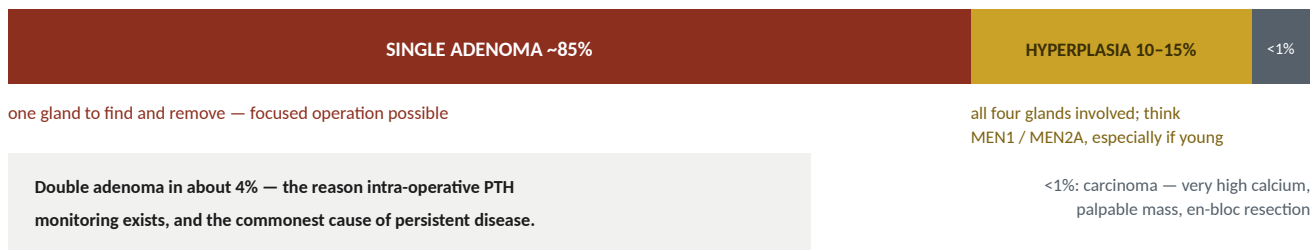


Figure 16. Eighty-five per cent of the disease is one gland. The value of the other fifteen per cent is that it explains every failed operation.

CONFIRM THE DIAGNOSIS

- ◆ Raised corrected calcium on **two** occasions
- ◆ PTH raised or inappropriately normal
- ◆ Phosphate low or low-normal
- ◆ Vitamin D replete before judgement
- ◆ **24-hour urine calcium** to exclude FHH

THEN ASSESS THE END ORGANS

- ◆ **DEXA** — lumbar spine, hip *and* distal radius
- ◆ **Renal imaging** (ultrasound or CT) for stones and nephrocalcinosis
- ◆ **eGFR**
- ◆ Vertebral imaging if height loss or back pain

WHO NEEDS AN OPERATION

Every **symptomatic** patient is offered parathyroidectomy. For the asymptomatic patient, any one of the following is an indication:

Domain	Threshold
Age	Under 50 years
Calcium	More than 1.0 mg/dL above the upper limit of normal
Skeleton	T-score ≤ -2.5 at any site, or a vertebral fracture
Kidney	eGFR < 60 mL/min, stones or nephrocalcinosis, urine calcium > 400 mg/24 h

If the patient is not operated on — annual calcium and creatinine, and DEXA every one to two years. Keep them hydrated, keep vitamin D replete (deficiency makes the hyperparathyroidism worse, not better), avoid thiazides, and do not restrict dietary calcium.

THE QUESTION BEHIND THE QUESTION

"Why operate on someone with no symptoms?" — "Because the end-organ damage is silent: cortical bone loss and renal impairment progress without the patient noticing, and both are arrested by a 40-minute operation with a cure rate above 95%."

Finding it, and taking it out

Imaging is planning, not diagnosis. The decision to operate has already been made in the laboratory.

ULTRASOUND No radiation, cheap, available. Shows the thyroid at the same time — which matters, because a coexisting nodule changes the operation. Blind to mediastinal glands.	SESTAMIBI SPECT/CT Functional: mitochondria-rich adenoma retains the tracer. Finds ectopic and mediastinal glands the ultrasound cannot. Less reliable in hyperplasia.	4D-CT Reserved for discordant or negative first-line imaging, and for re-operative necks. Higher radiation dose — not a first test.
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CONCORDANT ultrasound + sestamibi → focused, minimally invasive parathyroidectomy.

DISCORDANT or negative → four-gland (bilateral) exploration. A negative scan never cancels the operation.

Figure 17. Two concordant tests earn the patient a small incision; anything else means the surgeon looks at all four glands.

THE OPERATION

- ◆ **Focused parathyroidectomy** — small lateral or central incision, single gland excised, intra-operative PTH to confirm cure.
- ◆ **Bilateral exploration** — all four glands identified. For hyperplasia: subtotal (3½ glands) or total parathyroidectomy with forearm autotransplantation.
- ◆ Cure rate **95–98%** in experienced hands; day-case surgery in selected patients.

WHAT CAN GO WRONG

- ◆ **Recurrent laryngeal nerve injury** — hoarseness; check cords if symptomatic.
- ◆ **Hypocalcaemia** — transient, or from **hungry bone syndrome** (see the panel below).
- ◆ **Persistent disease** (<6 months) — missed second adenoma or ectopic gland.
- ◆ **Recurrent disease** (>6 months) — reconsider MEN, hyperplasia, or carcinoma.
- ◆ **Haematoma** — the airway emergency of any neck operation.

HUNGRY BONE SYNDROME

After a long-standing severe hyperparathyroidism is cured, a skeleton that has been dissolving for years suddenly starts rebuilding, and it strips calcium, phosphate and magnesium out of the plasma. Expect it when preoperative calcium and alkaline phosphatase were both high and the adenoma was large. The hypocalcaemia is **profound, prolonged (days to weeks) and requires large doses** of calcium and active vitamin D. Distinguish it from surgical hypoparathyroidism: in hungry bone, phosphate is **low**; in hypoparathyroidism, phosphate is **high**.

Hypercalcaemia of malignancy, and the crisis

Faster, higher, sicker — and it is the intern who starts the treatment.

	Primary hyperparathyroidism	Malignancy
Onset	Months to years	Days to weeks
Calcium	Usually < 12 mg/dL	Often > 13 mg/dL
PTH	High or inappropriately normal	Suppressed
Symptoms	Mild or absent	Marked — drowsy, vomiting, dehydrated
Mechanism	Autonomous gland	PTHrP (squamous, renal, breast) · osteolytic metastases · myeloma · calcitriol from lymphoma
Prognosis	Cured by operation	Marker of advanced disease

MANAGING HYPERCALCAEMIC CRISIS (calcium > 14 mg/dL, or symptomatic at any level)

1 • REHYDRATE — this is the treatment

0.9% sodium chloride, 200–300 mL/h initially, titrated to a urine output of 100–150 mL/h. Reassess hourly for fluid overload in the elderly and in cardiac or renal disease.

2 • STOP what is making it worse

Thiazides, lithium, calcium and vitamin D supplements, antacids. Mobilise the patient. Review digoxin — hypercalcaemia potentiates digoxin toxicity.

3 • TURN OFF THE OSTEOCLAST — once the patient is volume-replete

Zoledronic acid 4 mg IV (dose-adjusted in renal impairment) — acts in 2–4 days, lasts weeks. Add calcitonin for the first 24–48 h if calcium must fall today; denosumab if that fails.

4 • TREAT THE CAUSE — steroids for lymphoma, myeloma and granulomatous disease.

In refractory disease with renal or cardiac failure, consider dialysis.

Figure 18. Fluid first, always. These patients are profoundly volume-depleted from a calcium-induced diabetes insipidus, and restoring the circulating volume alone will drop the calcium substantially.

DO NOT REACH FOR FUROSEMIDE

A loop diuretic given to a dehydrated hypercalcaemic patient makes them more dehydrated and the calcium higher. It has one role only: managing fluid overload in a patient who has already been fully rehydrated. Fluids first, and only then, if the lungs demand it, a diuretic.

Secondary and tertiary

The same four glands, working correctly — until they forget how to stop.

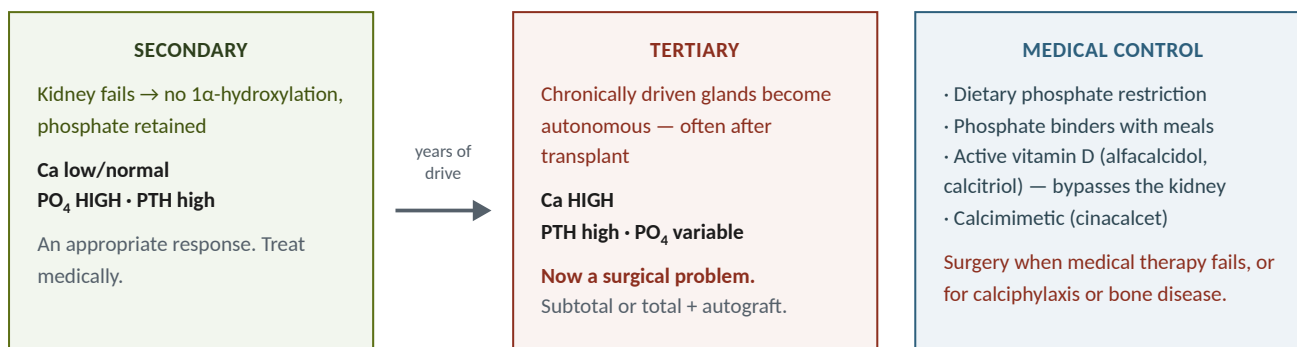


Figure 19. Secondary hyperparathyroidism is the gland doing its job. Tertiary is the gland that has been doing its job so long it can no longer be switched off.

Why alfacalcidol and not ordinary vitamin D? In renal failure the 1α -hydroxylation step is the one that has failed. Giving cholecalciferol simply fills the $25(\text{OH})\text{D}$ store that cannot be activated. Alfacalcidol and calcitriol are already past the missing step — which is also why they are the drugs used after parathyroidectomy, where PTH to drive that step is absent.

THREE LINES THAT SEPARATE THE THREE

Primary — high calcium, high PTH, **low** phosphate. Autonomous adenoma.

Secondary — low or normal calcium, high PTH, **high** phosphate (in renal failure). Appropriate response.

Tertiary — **high** calcium, high PTH, in a patient with long-standing renal disease. Autonomy acquired.

The tingling fingers

Hypocalcaemia after neck surgery — the single most likely calcium problem you will personally manage as an intern.

THE TIMELINE AFTER TOTAL THYROIDECTOMY

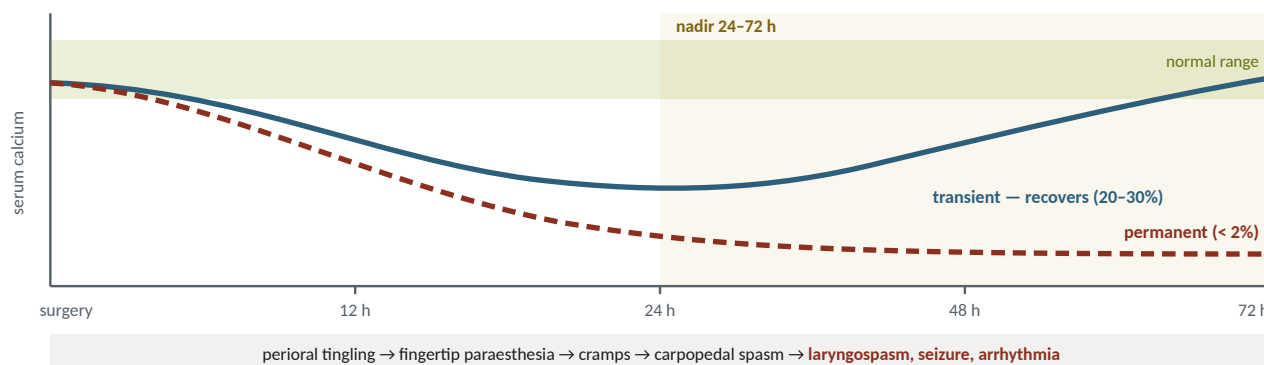


Figure 20. Calcium falls for two to three days before it recovers — so a normal calcium at 6 hours proves nothing. This is why patients are not discharged on the strength of a single early result, and why the symptoms are explained to them before they go home.

WHY IT HAPPENS

- ◆ **Devascularisation** of the parathyroids — far commoner than removing them.
- ◆ **Inadvertent excision** — found in the specimen at histology.
- ◆ **Hungry bone** after parathyroidectomy for severe disease.
- ◆ **Magnesium depletion** — low magnesium blocks both PTH release and its effect on bone. Hypocalcaemia that will not correct is hypomagnesaemia until proved otherwise.

TWO SIGNS, ONE MORE USEFUL THAN THE OTHER

- ◆ **Chvostek's sign** — tap over the facial nerve in front of the tragus; the ipsilateral lip twitches. Easy, but positive in up to a quarter of normal people.
- ◆ **Trousseau's sign** — inflate a sphygmomanometer cuff above systolic pressure for **three minutes**; carpopedal spasm follows. Uncomfortable, but far more specific.

"My lips have gone funny, like when the dentist numbs you. And my fingers are tingling — both hands."

Managing it

Predict it before it happens; treat by severity, not by number alone.

PREDICT: PTH ON THE EVENING OF SURGERY (or at 4 hours)

PTH < 15 pg/mL predicts hypocalcaemia — start supplements before symptoms. A normal PTH allows early, safe discharge.

MILD / ASYMPTOMATIC — oral

- Calcium carbonate 1–3 g elemental calcium per day, in divided doses, with food
- Add alfacalcidol 0.5–1 µg daily if calcium is slow to respond or PTH is absent
- Check magnesium and replace it
- Recheck calcium daily; warn the patient what symptoms to return with

Wean slowly over weeks, guided by calcium and PTH.

SYMPTOMATIC / SEVERE — intravenous

- **Calcium gluconate 10%, 10 mL** diluted in 50–100 mL of 5% dextrose, over 10 minutes
- Repeat until symptoms settle, then an infusion (e.g. 100 mL of 10% in 1 L over 24 h)
- Cardiac monitoring · correct magnesium
- Start oral calcium and alfacalcidol at the same time — the drip is a bridge, not a cure

Stridor or seizure = emergency. Call for senior help.

Figure 21. Two columns, and the decision between them is clinical. A patient with tetany is treated intravenously whatever the number says; a comfortable patient with a mildly low calcium is treated by mouth.

THREE ERRORS THAT CAUSE HARM

1. Calcium chloride through a peripheral cannula — it is three times as concentrated and causes tissue necrosis if it extravasates. Use gluconate peripherally.
2. Forgetting magnesium — the calcium will not come up until the magnesium does.
3. Rapid intravenous calcium in a patient on digoxin — it can precipitate a fatal arrhythmia. Give it slowly, with monitoring, and tell your senior.

HANDOVER, IN ONE SENTENCE

"Day one after total thyroidectomy, corrected calcium 7.4 mg/dL with perioral paraesthesia and a positive Trousseau's sign. Postoperative PTH was undetectable. I have given 10 mL of 10% calcium gluconate intravenously, started oral calcium with alfacalcidol, sent a magnesium, and put her on cardiac monitoring with four-hourly calcium levels."

Five questions at the bedside

Every abnormal calcium you will ever be handed can be worked through in this order.



Figure 22. The order matters. Questions one to four are outpatient thinking; question five is the one you ask first if the patient in front of you looks unwell.

TEN FACTS TO CARRY INTO THE EXAMINATION

1. Ionised calcium is the regulated fraction; correct for albumin. **2.** CaSR on the chief cell is the sensor; PTH half-life is minutes. **3.** Superior glands lie posterior to the recurrent laryngeal nerve, inferior glands anterior. **4.** PTH raises calcium and lowers phosphate. **5.** The kidney performs the 1α -hydroxylation, and PTH controls it. **6.** Primary hyperparathyroidism is a single adenoma in 85%. **7.** Exclude FHH with a urine calcium before operating. **8.** Imaging localises; it does not diagnose. **9.** Hypercalcaemic crisis is treated with saline first. **10.** Post-thyroidectomy calcium reaches its nadir at 24–72 hours.

PART THREE

Five patients

Five cases that put the circuit and the clinic together — the first abnormal result, the operating theatre, the family that should never have surgery, the ward round, and the transplant clinic. Cover each answer and commit to your own before you read it.

The stone that led to the neck

A raised calcium on a routine panel, worked from the first result to the operating list — in the order you would actually request the tests.

THE PATIENT

Mrs S, 52, a schoolteacher, is referred from urology. A left ureteric stone passed spontaneously last year, and the metabolic stone screen found a calcium of 11.2 mg/dL. Asked directly, she admits to tiredness and thirst but "put it down to work". No regular medication, no family history of calcium problems or endocrine tumours, no previous neck surgery.

1 IS IT REAL?

Albumin 4.1 g/dL, so the corrected calcium is 11.1 mg/dL. A repeat sample, taken without a prolonged tourniquet, is 11.3 mg/dL, and the ionised calcium is 6.0 mg/dL (4.5–5.3). **True hypercalcaemia, confirmed twice.**

2 IS IT PTH-DRIVEN?

PTH 118 pg/mL (15–65) and phosphate 2.2 mg/dL (low). A high calcium with a high PTH and a low phosphate is PTH-dependent hypercalcaemia. Malignancy is effectively excluded at this step — in malignancy the PTH would be suppressed. **Primary hyperparathyroidism is the working diagnosis.**

3 COULD IT BE FHH, OR A MIMIC?

No thiazide, no lithium, no supplements. 25(OH)D is 18 ng/mL — replete it with cholecalciferol and recheck; the calcium stays at 11.2 mg/dL. The 24-hour urine calcium is 360 mg and the calcium-to-creatinine clearance ratio is 0.028. **A ratio above 0.02 argues firmly against FHH.**

4 WHAT HAS IT ALREADY DONE?

Renal ultrasound: a 5 mm non-obstructing left renal stone. eGFR 86. DEXA: spine T –1.3, hip T –1.6, **distal radius T –2.7. Three indications for surgery: a stone, a T-score $\leq$ –2.5, and a calcium more than 1.0 mg/dL above the upper limit.**

5 ONLY NOW — WHERE IS IT?

Ultrasound: a 13 mm oval hypoechoic nodule behind the lower pole of the right thyroid lobe, with a polar feeding vessel. Sestamibi SPECT/CT: a retained focus at the same site on the delayed images. **Concordant imaging — focused right inferior parathyroidectomy with intra-operative PTH.**

PRESENT IT LIKE THIS

"Mrs S has confirmed PTH-dependent hypercalcaemia — calcium 11.3 mg/dL, PTH 118, phosphate low — with a urinary calcium clearance that excludes FHH. She has a renal stone and a distal radius T-score of –2.7, so she meets criteria for parathyroidectomy. Ultrasound and sestamibi agree on a right inferior adenoma, and I would list her for a focused operation with intra-operative PTH monitoring."

Notice that no scan was requested until step 5. The diagnosis was complete before the first image was taken.

The PTH that did not fall

Concordant scans, the expected gland — and a number that says the operation is not finished.

THE PATIENT

Mr K, 61, has primary hyperparathyroidism: calcium 11.8 mg/dL, PTH 210 pg/mL, a previous stone. Ultrasound and sestamibi both point to the **left inferior** gland. He is listed for a focused left inferior parathyroidectomy with intra-operative PTH (IOPTH) monitoring.

INTRA-OPERATIVE PTH (pg/mL)

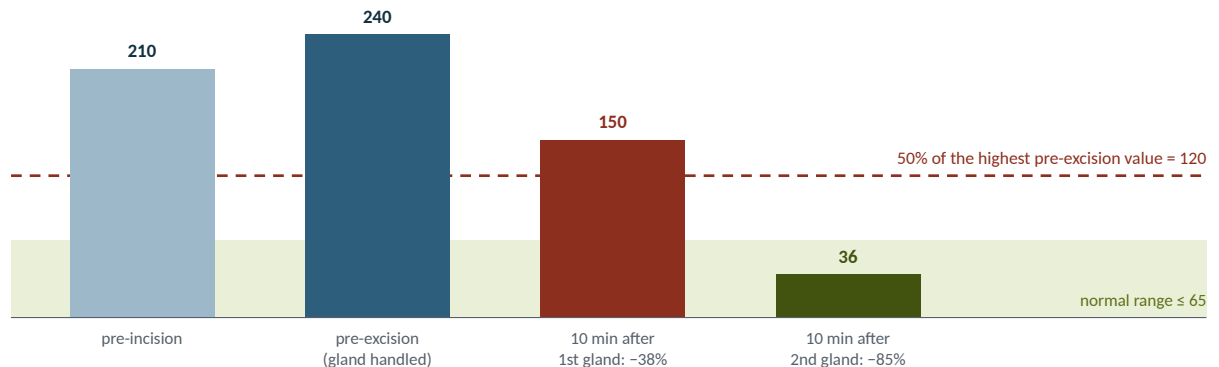


Figure 23. Handling the adenoma releases a surge of PTH, so the fall is measured from the highest pre-excision value. After the first gland the level fell by only 38%; after the second it fell by 85% and into the normal range.

1 THE EXPECTED GLAND

Through a small lateral incision, a 1.1 g left inferior adenoma is found and excised; frozen section confirms hypercellular parathyroid tissue. Blood is drawn 10 minutes later.

2 READ THE NUMBER

PTH 150 pg/mL. The highest pre-excision value was 240, so cure needs a level below 120. **A 38% fall is not enough — there is more disease in the neck.**

3 CONVERT TO BILATERAL EXPLORATION

Identify each recurrent laryngeal nerve first. Left superior and right inferior: normal, about 40 mg, tan — left in place, positions recorded. **Right superior: 0.6 g and dark**, deep to the nerve at the cricothyroid level — excised.

4 CHECK AGAIN

PTH at 10 minutes is 36 pg/mL — 85% below the peak and in the normal range. **Cure: a double adenoma, which no scan had shown.**

IF A GLAND CANNOT BE FOUND

Missing superior: tracheo-oesophageal groove, retro-oesophageal space, carotid sheath, within the thyroid.

Missing inferior: thyrothymic ligament and cervical thymus (transcervical thymectomy), or undescended, high near the carotid bifurcation. Blind thyroid lobectomy is a last resort. Still missing? Close, re-image, and plan again — do not keep exploring.

The operation that should never happen

Familial hypocalciuric hypercalcaemia — a lifelong, harmless high calcium that parathyroidectomy cannot cure.

THE PATIENT

Ms R, 34, is referred for parathyroidectomy with a diagnosis of "primary hyperparathyroidism". She has no symptoms. Her brother had a neck exploration at 29 that "found nothing", and his calcium stayed high afterwards. Her mother has "always had a high calcium".

Calcium **10.9** mg/dL

PTH **58** pg/mL — inappropriately normal

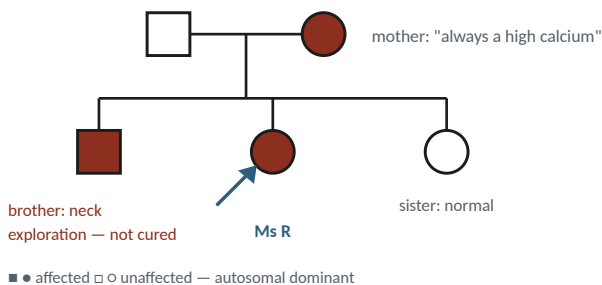
Phosphate **2.9**

Magnesium **2.4** high-normal

25(OH)D **34** ng/mL

Creatinine **0.7**

THE FAMILY TREE



CALCIUM-TO-CREATININE CLEARANCE RATIO

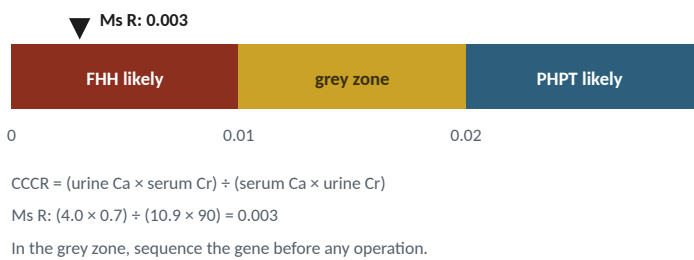


Figure 24. Lifelong hypercalcaemia in first-degree relatives, a failed neck exploration and a very low urinary calcium clearance: the three clues that make FHH the diagnosis.

1

READ THE SERUM AGAIN

A mildly raised calcium with an inappropriately normal PTH fits primary hyperparathyroidism — and fits FHH equally well. **The serum alone cannot separate them.**

2

TAKE THE FAMILY HISTORY SERIOUSLY

Asymptomatic hypercalcaemia in a parent and a sibling, and a brother "not cured" by surgery, is FHH until proved otherwise.

3

MEASURE THE URINE PROPERLY

Only when vitamin D is replete, thiazides and lithium are stopped and renal function is normal. Her 24-hour urine calcium is 62 mg and the ratio is 0.003. **Below 0.01 — FHH.**

4

CONFIRM AND MANAGE

CASR sequencing shows a heterozygous inactivating variant (FHH type 1; types 2 and 3 involve *GNA11* and *AP2S1*). No operation, no routine follow-up. Offer testing to first-degree relatives, record the diagnosis prominently, and tell the obstetric team in pregnancy — an affected neonate can be severely hypercalcaemic.

Day one after a total thyroidectomy

Predict it, treat it by symptoms, correct the magnesium, and send her home with a plan.

THE PATIENT

Mrs A, 45, has a total thyroidectomy for a retrosternal multinodular goitre. The left inferior parathyroid is devascularised and autotransplanted into the sternocleidomastoid. Preoperative calcium 9.4 mg/dL; 25(OH)D 22 ng/mL (insufficient).

CORRECTED CALCIUM (mg/dL) OVER THE ADMISSION

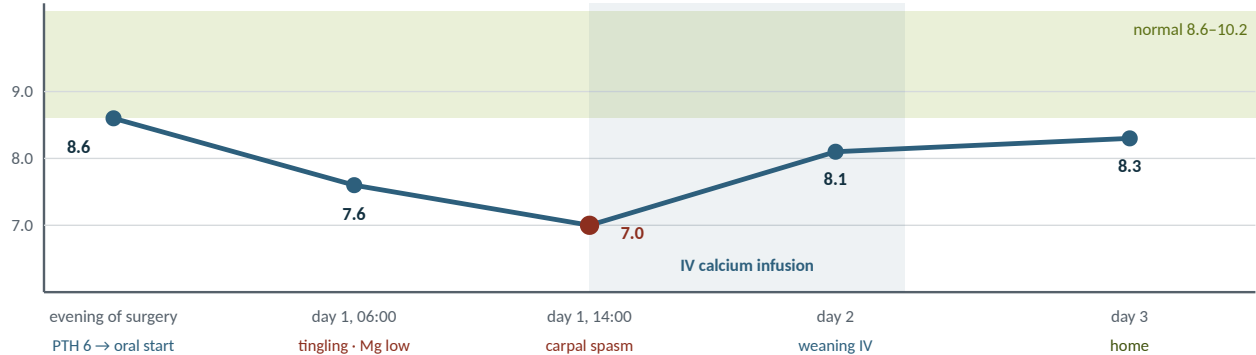


Figure 25. The calcium keeps falling for a day despite oral treatment, as predicted on page 26; intravenous calcium bridges the gap until oral treatment takes over.

1 EVENING OF SURGERY — PREDICT

PTH 6 pg/mL at 4 hours (below 15). Start calcium carbonate, 1 g elemental calcium three times daily, and alfacalcidol 1 µg daily now, before symptoms. Calcium, albumin and magnesium in the morning.

2 DAY 1, 06:00 — TINGLING

Corrected calcium 7.6 mg/dL, perioral tingling, positive Chvostek's sign; magnesium 1.4 mg/dL. **Replace the magnesium first** — intravenous magnesium sulfate, for example 2 g over 1–2 hours — continue the oral regimen, and recheck in 6 hours.

3 DAY 1, 14:00 — CARPAL SPASM

Calcium 7.0 mg/dL, QTc 490 ms. 10 mL of 10% calcium gluconate in 100 mL of 5% dextrose over 10 minutes on a monitor, then 100 mL of 10% gluconate in 1 L over 24 hours. Calcium 4–6-hourly; tell the senior.

4 DAYS 2–3 — WEAN AND DISCHARGE

The infusion is weaned as oral treatment takes over. Twenty-four hours off it she is 8.3 mg/dL and well: home on calcium, alfacalcidol and cholecalciferol, with a written symptom card, a calcium check within a week and clinic at two weeks.

AFTER DISCHARGE

Aim for a **low-normal** calcium (about 8.0–8.5 mg/dL); higher targets cause hypercalciuria. Wean as the glands and autograft recover. Still needing treatment beyond 6–12 months means permanent hypoparathyroidism and lifelong endocrine follow-up.

Eighteen months after the new kidney

The glands were driven for nine years. The kidney has been fixed — and the glands have not noticed.

THE PATIENT

Mr H, 48, received a kidney transplant 18 months ago after nine years of haemodialysis. On dialysis he had severe secondary hyperparathyroidism, treated with phosphate binders, alfacalcidol and cinacalcet; the cinacalcet was stopped at transplant. The graft works well.

Calcium **11.4** mg/dL

PTH **420** pg/mL

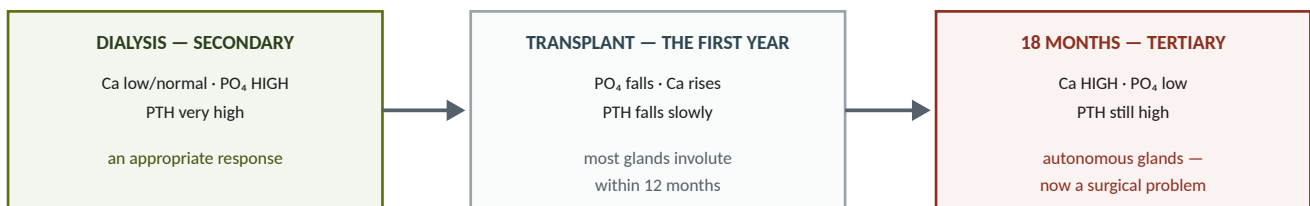
Phosphate **2.1** mg/dL

ALP **190** U/L

Creatinine **1.3** eGFR 60

25(OH)D **32**

THE KIDNEY'S JOURNEY, AND THE GLANDS' RESPONSE



Nine years of drive → hyperplasia, often nodular → the glands no longer switch off when the calcium rises.

Figure 26. The same four glands at three points in one patient's life: working correctly, slowly recovering, and finally out of control.

1 CONFIRM, AND LOOK FOR AN IATROGENIC CAUSE

Repeat calcium 11.5 mg/dL, ionised calcium raised. Check the drug chart: alfacalcidol and calcium supplements are often continued out of habit after transplant — and would raise the calcium. **He takes neither.**

2 RECOGNISE THE PATTERN

High calcium and high PTH after long-standing secondary hyperparathyroidism, now that the kidney has been corrected. The phosphate is low because the new kidney responds to PTH. **Tertiary hyperparathyroidism.** Vitamin D excess would suppress the PTH; a new single adenoma is possible but uncommon.

3 COUNT THE DAMAGE

ALP 190 U/L — high bone turnover. DEXA: hip T −2.6. Graft ultrasound for calcification, and serial creatinine: hypercalcaemia threatens the new kidney.

4 DECIDE ON TIMING, THEN OPERATE

Hypercalcaemia persisting beyond 12 months, severe hypercalcaemia, or damage to graft or bone are all reasons to act; he has all three. Expect multigland hyperplasia: bilateral exploration with **subtotal (3½ gland) parathyroidectomy**, or total with autograft, plus cervical thymectomy. Cinacalcet if he is unfit or declines.

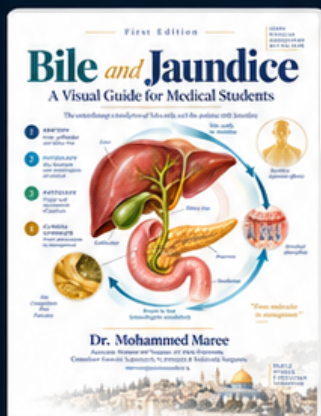
BEFORE AND AFTER THE OPERATION

A high ALP predicts **hungry bone syndrome**: replete vitamin D first and monitor calcium closely afterwards. Tell the transplant team — graft function can dip for a few days after parathyroidectomy.

Four glands weighing less than a paperclip hold one number steady for a lifetime. When they fail, patients may present with kidney stones, fractures, fatigue, neurocognitive symptoms, or tingling after thyroidectomy.

This visual guide follows the calcium circuit in ten illustrated steps, then brings it to the bedside: interpreting an abnormal calcium result, recognising hyperparathyroidism, and responding to hypercalcaemic crisis and postoperative hypocalcaemia.

OTHER VISUAL GUIDES FOR MEDICAL STUDENTS



Bile and Jaundice

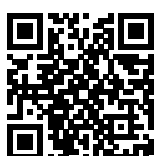
A Visual Guide for Medical Students

DOI: 10.5281/zenodo.23006422

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SCAN TO READ

Bile and Jaundice

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