

Parathyroid Glands

Normal structure, calcium homeostasis, and clinicopathologic correlations

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Learning objectives

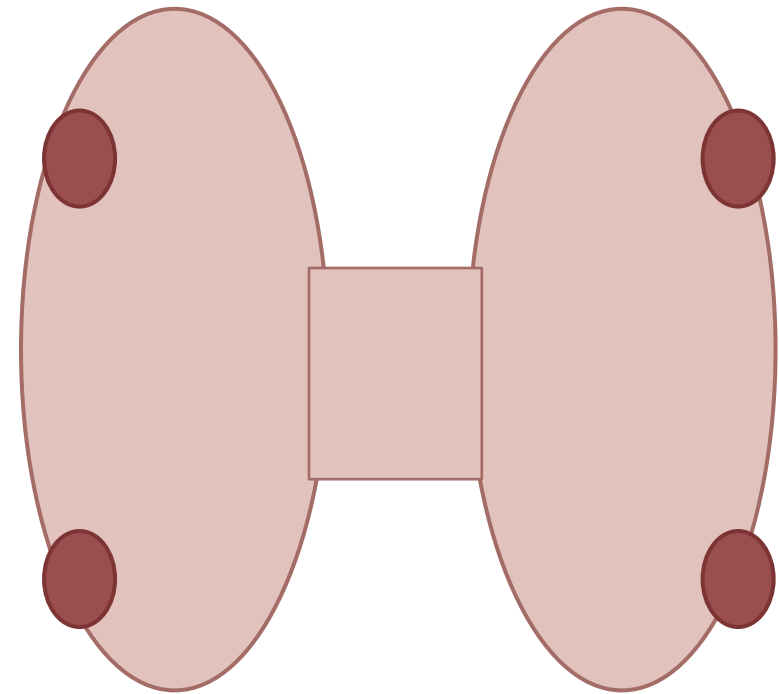
At the end of this lecture, learners should be able to:

- Identify normal parathyroid locations, chief cells, oxyphil cells, and age-related stromal fat.
- Explain how PTH regulates calcium and phosphate through kidney, gut, and bone.
- Distinguish autonomous, compensatory, and deficient parathyroid function.
- Interpret major morphologic and biochemical patterns of parathyroid disease.

Four small glands near the thyroid

Parathyroid anatomy is variable despite a familiar usual arrangement.

- Typically four glands lie near the upper and lower poles of the thyroid lobes.
- Parathyroid tissue is anatomically distinct from thyroid follicular tissue.
- An ectopic gland may be overlooked if only the usual neck locations are considered.

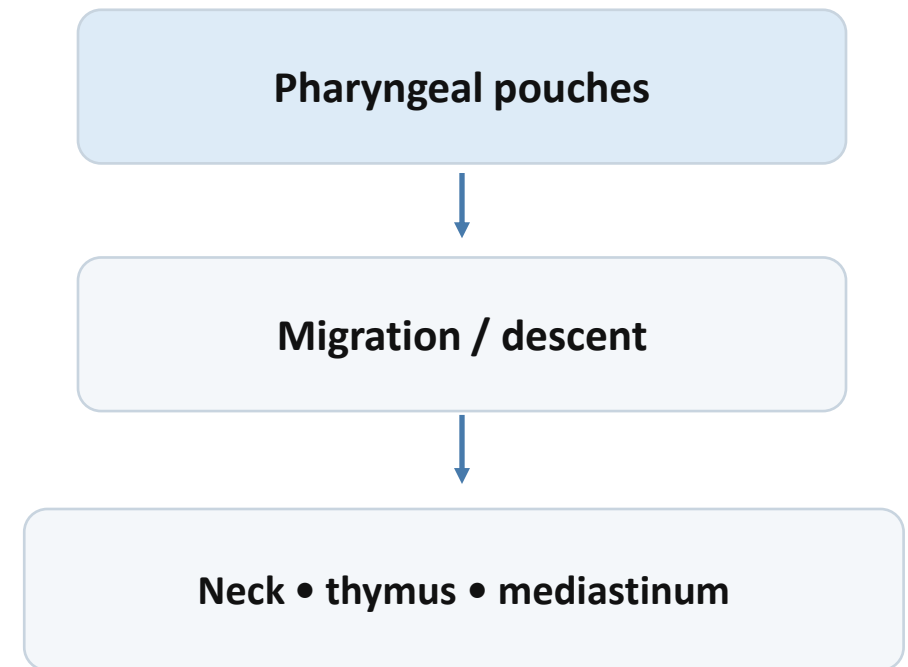


Thyroid lobes
Parathyroids shown in red

Pharyngeal pouch descent explains ectopic sites

Embryologic migration determines the range of normal locations.

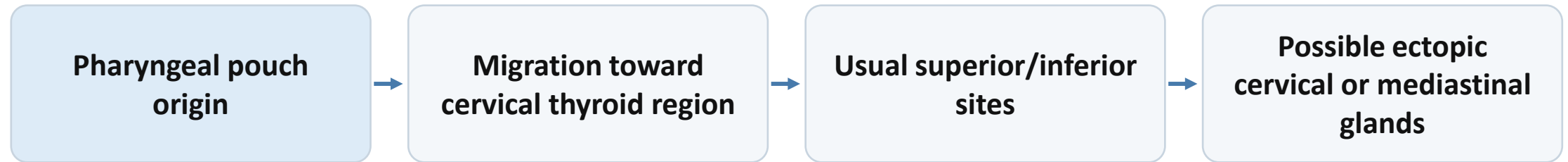
- The glands arise from pharyngeal pouches related embryologically to the thymus.
- Parathyroid tissue may occur along the descent pathway.
- Documented ectopic locations include carotid sheath, thymus, and anterior mediastinum.



Schematic — not a histologic specimen

From embryology to operative anatomy

The route of descent creates practical implications for gland localization.

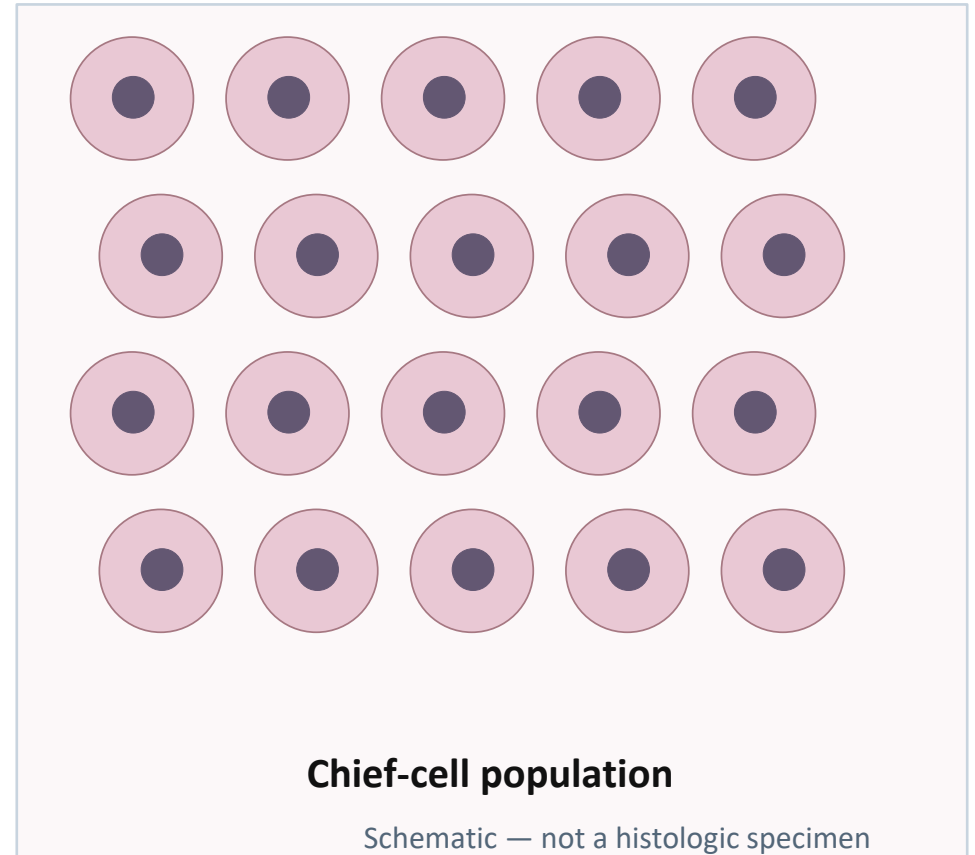


An anatomically “missing” gland may be ectopic rather than absent.

Chief cells dominate normal parathyroid

Chief cells are the principal hormone-secreting cell population.

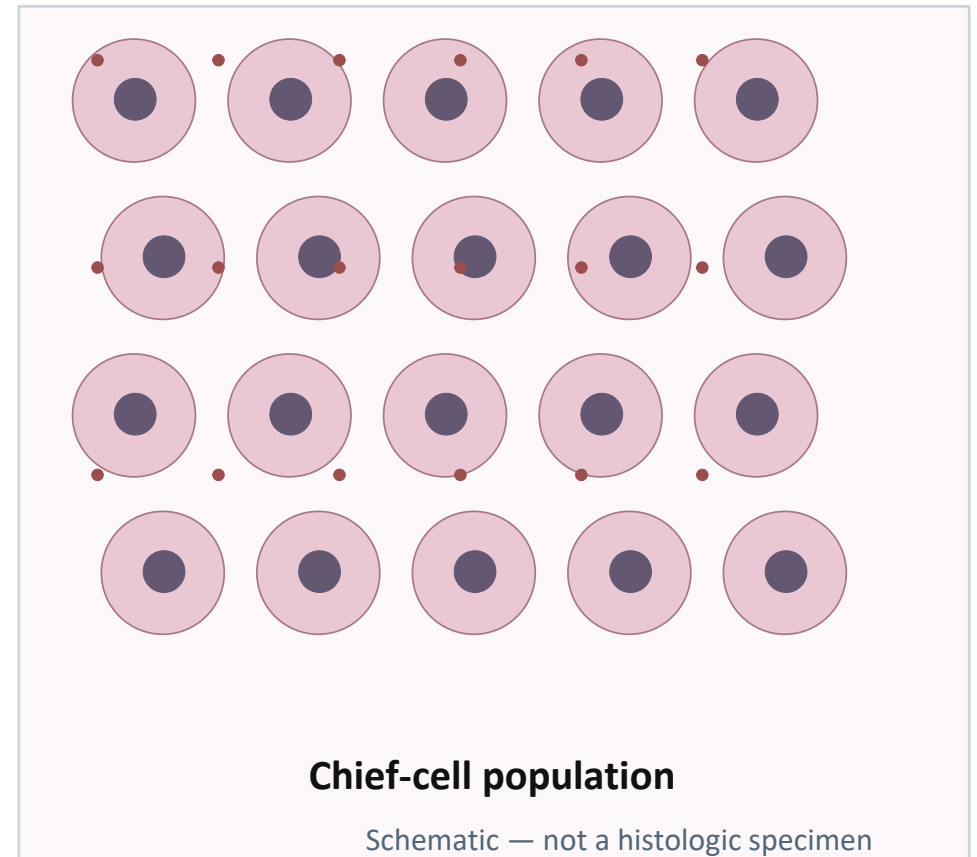
- Polygonal cells approximately 12–20 μm in diameter.
- Central, round, relatively uniform nuclei.
- Light-to-dark pink cytoplasm; Cytoplasmic secretory granules contain PTH.



Chief-cell granules contain PTH

Function is linked directly to normal cell architecture.

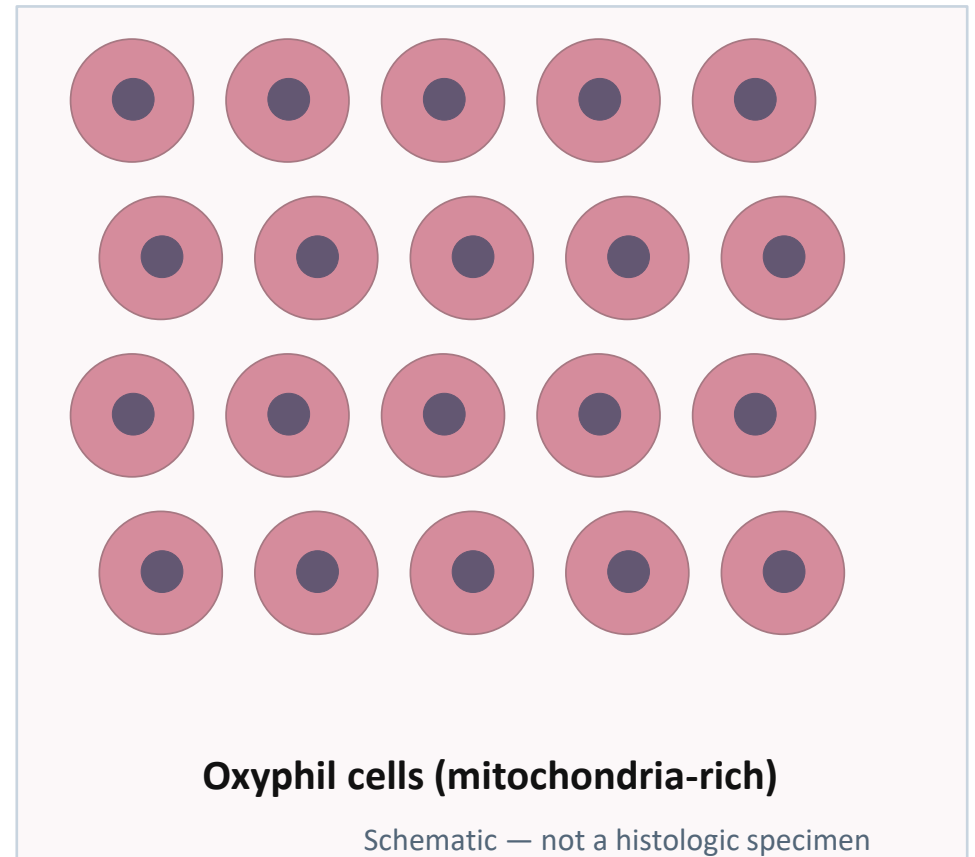
- The hormone is stored in secretory granules in chief cells.
- Circulating ionized calcium regulates synthesis and release of PTH.
- Normal endocrine morphology must be interpreted together with calcium–PTH physiology.



Oxyphil cells have distinctive cytoplasm

The oxyphil population differs from chief cells in size and organelles.

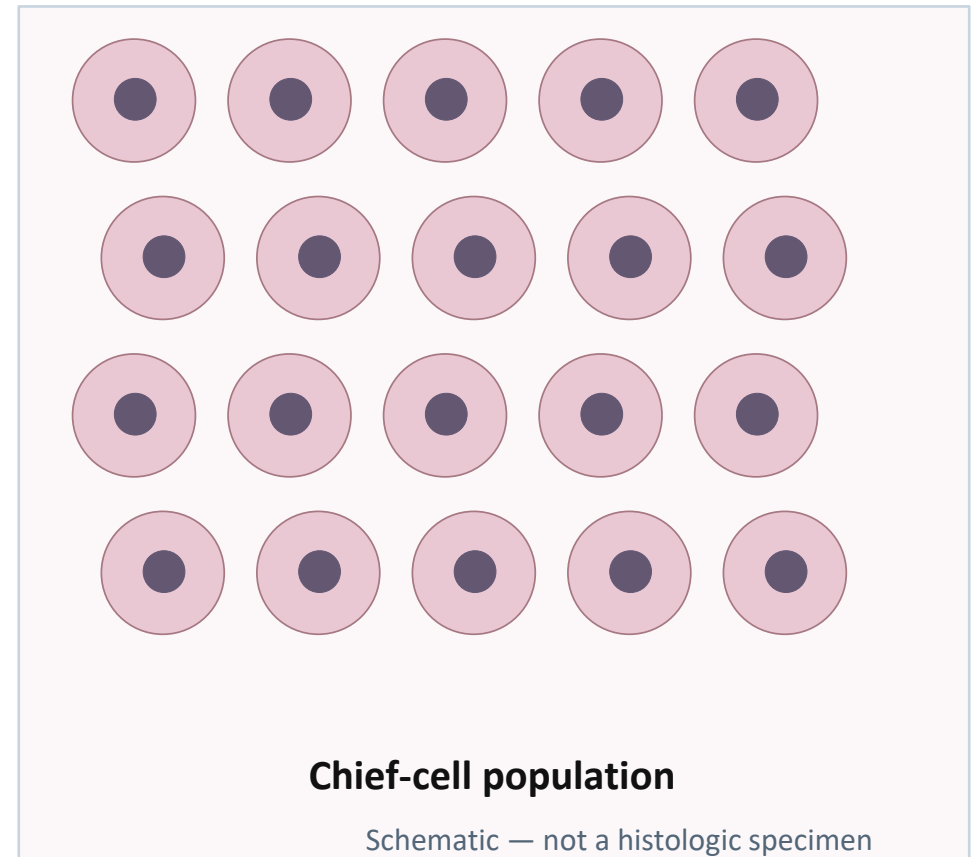
- Oxyphil and transitional oxyphil cells occur singly or in small clusters.
- They are slightly larger than chief cells, with eosinophilic cytoplasm.
- The eosinophilia corresponds to numerous mitochondria; Secretory granules are sparse or absent.



Oxyphil biology: Avoid overinterpretation

Oxyphil cells have specialized features, but their principal physiologic role is less defined.

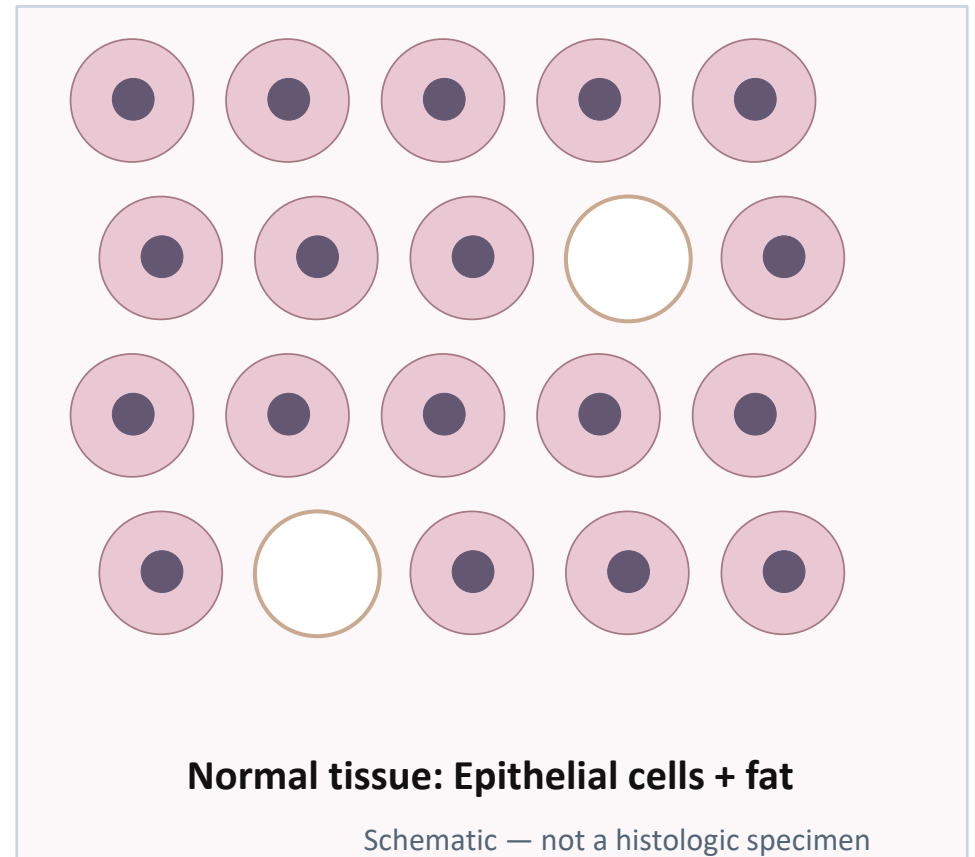
- Oxyphil cells express abundant calcium-sensing receptors.
- Proposed oxyphil products include PTH-related protein and calcitriol.
- These observations should not be equated with chief-cell PTH secretion.



Age affects the appearance of a normal gland

Stromal fat is a normal component that changes after childhood.

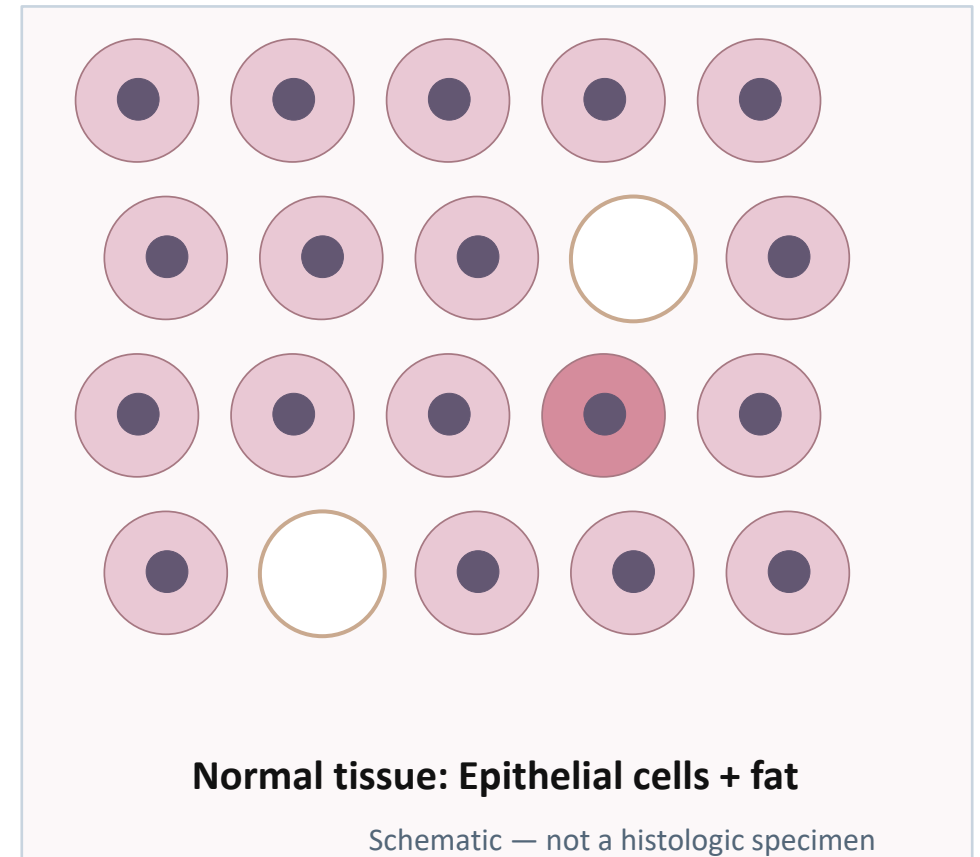
- Early-infant and childhood glands consist largely of solid sheets of chief cells.
- Adipose stromal tissue increases through young adulthood.
- Stromal fat reaches approximately 30% by about age 25, then plateaus.



Normal parathyroid architecture: A schematic

Assess epithelial cells together with interspersed adipose tissue.

- Chief cells form the predominant epithelial component.
- Oxyphil cells are admixed singly or in small groups.
- Adipose tissue becomes conspicuous in a typical adult gland.



Chief cells versus oxyphil cells

Cytoplasmic staining, organelles, and distribution distinguish normal cell populations.

Feature	Chief cells	Oxyphil cells
Relative abundance	Predominant	Scattered or small clusters
Cytoplasm	Light to dark pink	Strongly eosinophilic
Nucleus	Round, central, uniform	Similar; Cell is somewhat larger
Characteristic organelles	PTH-containing granules	Numerous mitochondria

Normal cell populations can coexist in the same gland.

PTH is the principal calcium-regulating output

Parathyroid secretion is regulated by circulating free (ionized) calcium.

- Low circulating ionized calcium stimulates PTH synthesis and secretion.
- PTH increases free calcium through coordinated effects on kidney, vitamin D, and bone.
- Rising calcium inhibits further PTH secretion.

↓ Ionized calcium

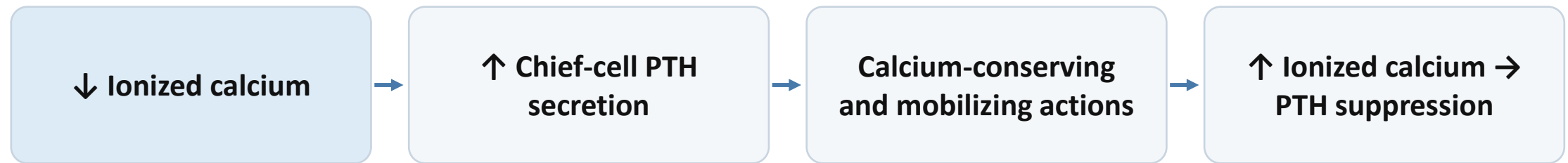
↑ PTH secretion

↑ Serum free calcium

Negative feedback ↓ PTH

The physiological negative-feedback loop

A fall in free calcium initiates a corrective endocrine response.



Homeostasis depends on the response of the glands to the free, not merely total, calcium level.

Why ionized calcium matters

PTH secretion is coupled to the biologically free fraction.

- The normal regulatory signal is blood ionized calcium.
- Hypocalcemia prompts secretion rather than suppression of PTH.
- Interpret any measured PTH concentration relative to the concurrent calcium level.

Ionized Ca^{2+} decreases

Chief cells release PTH

Ca^{2+} restores → PTH declines

The kidney conserves calcium under PTH

Renal handling is one direct route to restoring extracellular calcium.

- PTH enhances renal tubular reabsorption of calcium.
- Conservation limits urinary calcium loss from the filtered load.
- This action contributes to the rise in circulating free calcium.

PTH at renal tubule

↑ Calcium reabsorption

↑ Circulating calcium

PTH promotes phosphate excretion

The renal phosphate response differs from the calcium response.

- PTH increases urinary excretion of phosphate.
- Serum phosphate consequently tends to fall when this pathway is effective.
- Phosphate binds calcium: Changing phosphate availability affects free calcium.

PTH at renal tubule

↑ Phosphate excretion

↓ Serum phosphate

Renal effects of PTH are directionally opposite

Both changes contribute to calcium homeostasis.

Renal target	Response to PTH	System consequence
Calcium handling	Reabsorption increases	Conserves circulating calcium
Phosphate handling	Urinary excretion increases	Tends to lower phosphate
Kidney activation of vitamin D	Conversion increases	Supports intestinal calcium uptake

Do not assume that PTH increases renal retention of all minerals.

The kidney activates vitamin D in response to PTH

Indirect intestinal effects begin in the kidney.

- PTH increases conversion of vitamin D to its active dihydroxy form.
- Active vitamin D enhances gastrointestinal calcium absorption.
- Reduced kidney function can disrupt this endocrine linkage.

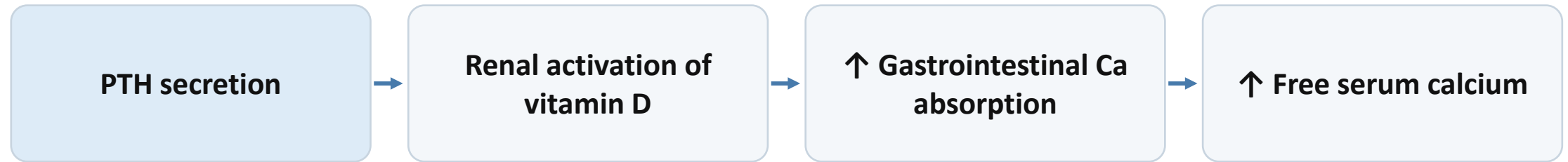
PTH stimulates kidney

↑ Active vitamin D

↑ Gut calcium uptake

The vitamin D arm links kidney and gut

This is an indirect route for PTH to increase circulating calcium.



In kidney failure, inadequate active vitamin D contributes to compensatory PTH secretion.

PTH increases bone resorption

Bone is a mineral reservoir in systemic calcium regulation.

- PTH promotes development of mature osteoclasts from precursor cells.
- Osteoclastic resorption releases mineral from bone.
- Persistent excess can damage cortical and trabecular skeletal architecture.

Persistent ↑ PTH

↑ Osteoclastic activity

Net bone resorption

Osteoclast differentiation links hormone to bone loss

The gland does not act on bone merely by changing renal calcium handling.

- PTH excess promotes maturation of osteoclast precursors.
- The resulting increase in osteoclastic activity mobilizes calcium.
- Excessive sustained resorption underlies characteristic skeletal lesions.

Osteoclast precursors

Mature osteoclasts

Resorb mineralized bone

PTH targets: Four complementary actions

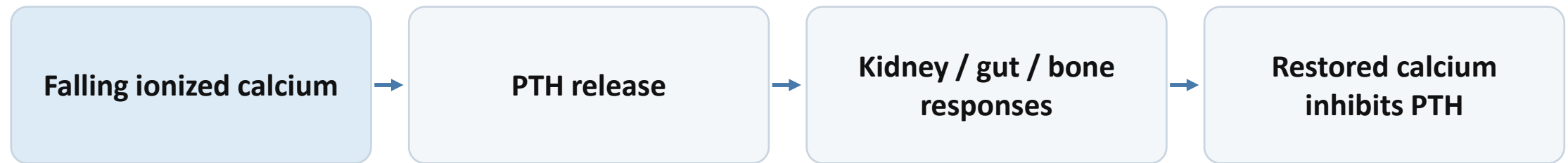
The physiological output can be summarized without collapsing distinct mechanisms.

Organ/pathway	PTH action	Principal result
Renal calcium	↑ tubular reabsorption	Less urinary Ca loss
Renal phosphate	↑ phosphate excretion	Lower serum phosphate
Vitamin D / intestine	↑ renal vitamin D activation	Greater gut Ca absorption
Bone	↑ osteoclast-mediated resorption	Ca released from bone

Combined response: Increased free serum calcium followed by feedback inhibition.

Restoring homeostasis requires feedback closure

A physiological response is self-limiting once the stimulus resolves.



Failure of suppression distinguishes autonomous hypersecretion from an appropriate response.

Interpret the calcium–PTH pair, not PTH alone

An “in-range” PTH can still be physiologically inappropriate.

- Under hypercalcemic conditions, normal negative feedback should suppress PTH.
- Persistently elevated or nonsuppressed PTH suggests a PTH-dependent process.
- Low or undetectable PTH during hypercalcemia suggests a nonparathyroid cause.

High calcium should ↓ PTH

Nonsuppressed PTH = inappropriate

Seek PTH-dependent cause

The broad functional states of parathyroid disease

The central classification concerns direction and appropriateness of secretion.

Functional state	PTH behavior	Typical consequence
Normal regulation	Feedback responsive	Stable calcium homeostasis
Primary hyperfunction	Autonomous secretion	Hypercalcemia
Secondary hyperfunction	Compensatory secretion	Underlying chronic low-calcium drive
Hypofunction	Insufficient PTH	Hypocalcemia

Tertiary hyperparathyroidism is acquired autonomy after prolonged secondary stimulation.

Parathyroid lesions often present biochemically

Endocrine output may dominate over local compression.

- Parathyroid tumors usually draw attention because of PTH excess.
- This differs from the frequent mass presentation of thyroid neoplasms.
- Morphologic assessment and biochemical interpretation are complementary.

PTH excess detected

Focused gland evaluation

Histology interpreted clinically

Hyperparathyroidism has three major categories

Classification asks whether excess secretion is autonomous or compensatory.

- Primary: Autonomous PTH production, commonly adenoma or hyperplasia.
- Secondary: Compensatory PTH increase in sustained low-calcium states.
- Tertiary: Autonomous secretion developing after prolonged secondary hyperfunction.

Primary: Autonomous

Secondary: Compensation

Tertiary: New autonomy

Primary versus secondary versus tertiary

This contrast prevents conflation of unrelated mechanisms.

Type	Driving mechanism	Representative setting
Primary	Intrinsic autonomous PTH excess	Adenoma / primary hyperplasia
Secondary	Response to chronic hypocalcemic drive	Renal failure / vitamin D deficiency
Tertiary	Acquired persistent autonomy	Long-standing secondary disease

Tertiary disease may produce hypercalcemia despite an initial compensatory origin.

Primary hyperparathyroidism is usually an adenoma

Parathyroid gland pathology commonly produces an altered hormonal set point.

- A solitary sporadic adenoma is the dominant lesion in primary hyperparathyroidism.
- Primary hyperplasia is less common and may affect multiple glands.
- Carcinoma is rare but requires different morphologic criteria.

Typically one gland

Circumscribed proliferation

Other glands often suppressed

Adenoma reflects clonal glandular growth

Sporadic adenomas arise through several established molecular mechanisms.

- CCND1 rearrangement can increase cyclin D1 expression.
- MEN1 tumor-suppressor abnormalities occur in a subset of sporadic tumors.
- The genetic mechanism and the biochemical phenotype are different levels of explanation.

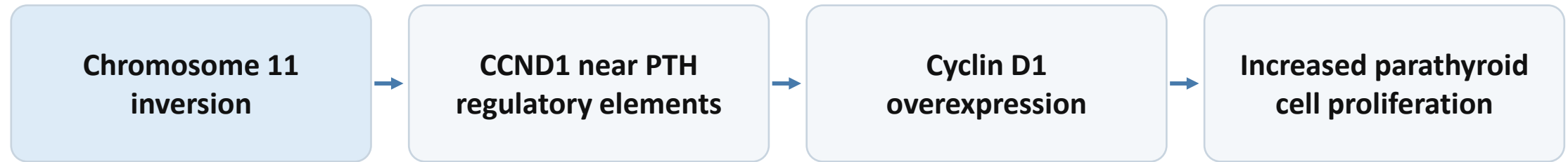
CCND1 pathway

MEN1 tumor suppressor

Excess cell proliferation

CCND1 dysregulation and parathyroid proliferation

A transcriptional rearrangement can enhance a cell-cycle regulator.



This is one established route, not the only cause of adenoma.

Parathyroid adenoma: Expected gross pattern

Anatomical isolation and neighboring tissue help interpretation.

- Typically solitary, circumscribed, soft, tan to reddish-brown nodule.
- It may be adjacent to the thyroid or at an ectopic site.
- Other parathyroid glands are commonly normal-sized or reduced in size.

Typically one gland

Circumscribed proliferation

Other glands often suppressed

Parathyroid adenoma: Microscopic pattern

The usual adenoma is a relatively uniform chief-cell proliferation.

- Sheets of polygonal cells with small central nuclei.
- Fat is inconspicuous relative to a typical adult normal gland.
- A compressed rim of residual normal parathyroid may remain at the periphery.



Endocrine atypia is not proof of carcinoma

Nuclear appearance alone can be misleading.

- Benign adenomas may contain bizarre or pleomorphic nuclei.
- Mitoses tend to be rare in typical adenomas.
- Demonstrated invasion or metastasis—not isolated cytologic atypia—defines carcinoma in the source.

Nuclear pleomorphism

May occur in adenoma

≠ carcinoma by itself

Parathyroid hyperplasia is generally multiglandular

Distribution is helpful, but not always definitive.

- Primary hyperplasia often affects all four glands, sometimes asymmetrically.
- Apparent sparing of one or two glands can mimic a solitary adenoma.
- Microscopically, chief-cell or water-clear-cell hyperplasia may be diffuse or nodular.

Multiple glands possible

Asymmetric enlargement

Diffuse or nodular cells

Adenoma and hyperplasia: Overlapping morphology

Assess the entire gland distribution rather than one microscopic field.

Feature	Adenoma	Hyperplasia
Gland distribution	Usually solitary	Often multiglandular
Stromal fat	Usually reduced	Usually reduced
Cell pattern	Uniform chief-cell sheets typical	Diffuse/multinodular chief cells typical
Practical pitfall	Peripheral residual rim may help	Asymmetry may simulate adenoma

There is no single microscopic feature that always resolves the distinction.

Carcinoma requires invasive behavior

Parathyroid cancer is not defined by nuclear pleomorphism alone.

- Tissue invasion and/or metastasis are the reliable defining features.
- Cells may resemble normal parathyroid chief cells.
- Gross lesions may be irregular gray-white masses rather than circumscribed nodules.

Irregular infiltrative border

Confirmed tissue invasion

Or distant metastasis

Atypical parathyroid neoplasm is an intermediate concern

Certain morphologic features are worrisome but insufficient for carcinoma.

- Fibrosis, cystic change, and macronucleoli may raise concern.
- Without unequivocal invasion or metastasis, the source places such lesions in an atypical category.
- Parafibromin deficiency can indicate underlying CDC73 alteration.

Worrisome changes

No proven invasion

Assess CDC73 / parafibromin

CDC73 and hereditary predisposition

Molecular findings can trigger familial evaluation.

- CDC73 encodes parafibromin and is altered in many parathyroid carcinomas.
- Germline CDC73 causes hyperparathyroidism–jaw tumor syndrome.
- Carcinoma and parafibromin-deficient neoplasms warrant assessment for germline CDC73.

CDC73 abnormality

Parafibromin deficiency

Hereditary assessment

CASR dysfunction changes the calcium set point

Receptor impairment can mimic a parathyroid hyperfunction pattern.

- Familial hypocalciuric hypercalcemia often reflects heterozygous CASR loss of function.
- Reduced extracellular-calcium sensitivity permits nonsuppressed PTH despite elevated calcium.
- The text notes that many affected people lack typical end-organ effects of hyperparathyroidism.

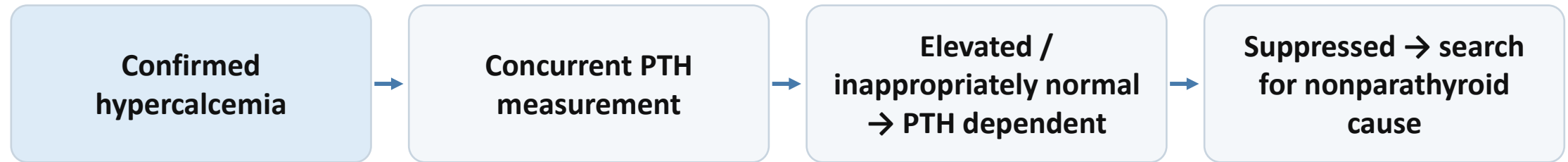
Defective Ca²⁺ sensing

PTH not appropriately suppressed

Hypercalcemia / low urine Ca

A biochemical approach to hypercalcemia

Measure intact PTH in relation to elevated serum calcium.



Family history and urine calcium may be relevant when familial hypocalciuric hypercalcemia is considered.

Why hypercalcemia has different PTH responses

Biochemical patterns point toward distinct mechanisms.

Context	Expected PTH pattern	Interpretation
Primary hyperparathyroidism	High or inappropriately normal	PTH-dependent hypercalcemia
Malignancy-associated hypercalcemia	Low / suppressed	PTH-independent hypercalcemia
Familial hypocalciuric hypercalcemia	Not appropriately suppressed	Altered CASR calcium sensing

PTH-related peptide can mimic PTH effects without elevating true PTH.

High PTH injures bone when excess persists

Physiologic mobilization becomes pathology under sustained stimulation.

- Increased osteoclastic activity drives loss of bone mineral.
- Cortical surfaces, including subperiosteal and endosteal areas, are particularly affected.
- Prolonged severe disease can produce fibrous replacement and reactive lesions.

Chronic PTH excess

Subperiosteal/endosteal resorption

Cortical bone loss

Dissecting osteitis is a distinctive bone pattern

Osteoclasts can burrow into trabecular bone.

- Osteoclasts tunnel longitudinally within trabeculae.
- The resulting resorption has a described “railroad-track” appearance.
- Adjacent marrow spaces can be replaced by fibrovascular tissue.

Trabecular tunneling

Resorption tracks

Fibrovascular replacement

Brown tumors are reactive, not primary bone cancer

Bone fragility and repair create a mass-like lesion.

- Bone loss predisposes to microfractures and hemorrhage.
- Macrophages and reparative fibrous tissue accumulate around the injury.
- Vascularity, hemorrhage, and hemosiderin explain the brown appearance.

Microfracture + hemorrhage

Macrophage / fibrous repair

Brown reactive mass

Osteitis fibrosa cystica signals severe disease

Several skeletal processes converge in an advanced phenotype.

- Excessive osteoclast activity produces resorption.
- Peritrabecular fibrosis replaces normal supporting spaces.
- Brown tumors and cystic degeneration can accompany the changes.

Osteoclastic resorption

Peritrabecular fibrosis

Brown tumors / cysts

Renal stones reflect the hypercalcemic milieu

Increased calcium availability can injure the urinary tract.

- PTH-associated hypercalcemia favors nephrolithiasis.
- Stones can produce pain, obstruction, and secondary renal dysfunction.
- Urine calcium excretion may rise despite PTH-mediated renal calcium conservation.

Hypercalcemia

Urinary stone formation

Pain / obstruction

Nephrocalcinosis is not the same as nephrolithiasis

Distinguish precipitated deposits within renal tissues from formed stones.

- Nephrocalcinosis refers to calcium deposition in renal interstitium and tubules.
- Nephrolithiasis refers to concretions in the collecting system.
- Both may occur during clinically significant hypercalcemia.

Nephrolithiasis = stones

Nephrocalcinosis = deposits

Both may injure kidney

Multisystem effects of primary PTH excess

The phenotype is broader than bone and kidney disease.

- Gastrointestinal symptoms may include constipation, nausea, and pancreatitis.
- Neuropsychiatric symptoms include lethargy and mood disturbance.
- Weakness, fatigue, and valvular calcification are other reported manifestations.

Bone / urinary tract

Gut / muscle / nerves

Cardiovascular effects

Incidental hypercalcemia is often the first clue

Laboratory recognition has changed clinical presentation.

- Primary hyperparathyroidism commonly presents without striking symptoms.
- Severe brown tumors and osteitis fibrosa cystica are consequently less common in diagnosed patients.
- Malignancy remains an important competing cause of symptomatic hypercalcemia.

Incidentally high calcium

PTH–calcium interpretation

Potentially silent pathology

Secondary hyperparathyroidism is compensatory

A persistently low calcium drive stimulates intact glands.

- Renal insufficiency is a common cause.
- Vitamin D deficiency, inadequate calcium intake, and malabsorption are other examples.
- Unlike primary disease, the initiating problem is usually extrinsic to parathyroid tissue.

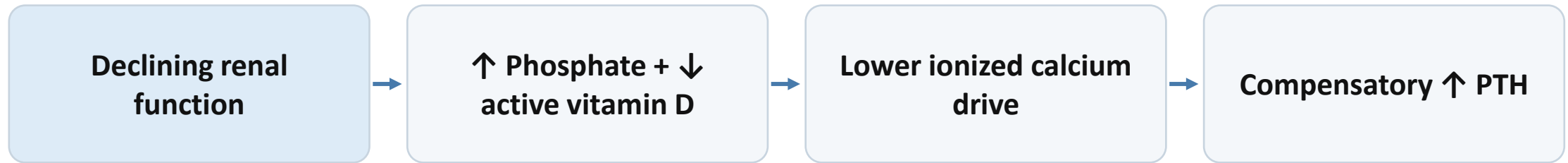
Chronic low-calcium drive

Compensatory ↑ PTH

Glandular hyperplasia

Renal failure drives secondary PTH secretion

Phosphate retention and impaired vitamin D activation converge.



Vitamin D deficiency also removes a suppressive influence on parathyroid growth and secretion.

Secondary hyperplasia changes gland morphology

Chronic stimulation is visible at the tissue level.

- Parathyroid glands become hypercellular and may enlarge asymmetrically.
- Chief-cell and water-clear-cell proliferation may be diffuse or multinodular.
- Stromal fat decreases; Adenoma and hyperplasia may overlap histologically.

Multiple glands possible

Asymmetric enlargement

Diffuse or nodular cells

Tertiary hyperparathyroidism follows chronic drive

The gland may eventually become autonomous.

- A minority of patients with long-standing secondary hyperparathyroidism develop persistent autonomous PTH production.
- Hypercalcemia can then supervene.
- The classification depends on disease history, not only one histologic field.

Long-standing secondary state

Autonomous PTH secretion

Hypercalcemia possible

PTH deficiency produces hypocalcemia

Loss of normal parathyroid output is a different pathophysiologic problem.

- Insufficient PTH prevents the usual corrective response to low calcium.
- The resulting hypocalcemia increases neuromuscular excitability.
- Common clinical context is inadvertent gland loss during neck surgery.

Inadequate PTH

Hypocalcemia

Tetany / systemic effects

Postoperative hypoparathyroidism

Anatomic proximity creates the risk.

- Accidental parathyroid removal during thyroid or neck surgery can produce PTH deficiency.
- Extensive removal during parathyroid surgery may have the same result.
- Embryologic and anatomic variation complicates gland preservation.

Thyroid or neck surgery

Gland removal or excess resection

Low PTH / low calcium

Autoimmune and genetic causes of low PTH

Not all parathyroid failure is acquired by surgery.

- APS-1, caused by AIRE variants, may include candidiasis, hypoparathyroidism, and adrenal insufficiency.
- Activating CASR variants suppress PTH through excessive calcium sensing.
- PTH processing defects and impaired parathyroid development are rarer mechanisms.

AIRE / CASR / PTH / GCM2

Distinct molecular defects

Deficient PTH action

CASR gain versus loss of function

Opposite receptor defects shift the endocrine calcium set point.

CASR change	Sensing behavior	Associated pattern
Gain of function	Senses calcium too strongly	PTH suppressed; Hypocalcemia
Heterozygous loss of function	Senses calcium insufficiently	Nonsuppressed PTH; FHH
Severe biallelic loss	Very impaired calcium sensing	Severe neonatal hyperparathyroidism

Biochemical direction follows the receptor defect rather than a simple change in cell number.

Hypocalcemia manifests as tetany

Reduced calcium alters neuromuscular excitability.

- Paresthesias, circumoral numbness, and carpopedal spasm are typical.
- Severe episodes may lead to laryngospasm or seizures.
- Duration and severity determine the broader clinical phenotype.

Tingling / paresthesia

Carpopedal spasm

Seizures / laryngospasm

Chvostek and Trousseau signs

Clinical elicitation demonstrates latent neuromuscular irritability.

- Chvostek sign: Facial muscle contraction after tapping over the facial nerve.
- Trousseau sign: Carpal spasm during prolonged inflation of a blood-pressure cuff.
- Neither sign substitutes for biochemical assessment of calcium status.

Facial nerve tapping

Cuff-provoked carpal spasm

Correlate with serum Ca

Chronic hypocalcemia affects multiple tissues

Sustained deficiency produces findings beyond tetany.

- Basal ganglia calcification and cataracts are described consequences.
- Electrocardiography may show prolongation of the QT interval.
- Early-life disease may disrupt dental development and eruption.

Basal ganglia / lens

Cardiac QT prolongation

Developing teeth

Low calcium: Deficient PTH or resistant target?

Both patterns can produce hypocalcemia, but the hormone response differs.

Disorder	PTH concentration	Pathophysiology
Hypoparathyroidism	Low / insufficient	Inadequate glandular secretion
Pseudohypoparathyroidism	Normal or high	End-organ resistance to PTH
Appropriate response to low Ca	Increases	Preserved gland feedback

Elevated PTH does not guarantee effective downstream hormone action.

Case: Persistent hypercalcemia

A patient has incidentally detected elevated calcium. Concurrent PTH is not suppressed.

- Why is the measured PTH physiologically inappropriate?
- Which broad causes of PTH-dependent hypercalcemia should be considered?

Normal feedback should suppress PTH in hypercalcemia. Consider primary parathyroid hyperfunction and, in the appropriate context, altered calcium sensing such as familial hypocalciuric hypercalcemia.

Case: Renal impairment and elevated PTH

A patient with chronic renal insufficiency has raised phosphate and elevated PTH.

- What biochemical pathways stimulate the glands?
- What tissue change may develop in the parathyroids?

Phosphate retention plus inadequate active vitamin D contribute to a hypocalcemic drive and compensatory PTH. Chronic stimulation produces gland hypercellularity, often multiglandular.

Case: Symptoms after thyroid surgery

A patient develops circumoral tingling and carpopedal spasm after thyroid surgery.

- What endocrine complication should be suspected?
- Why does low calcium cause these clinical findings?

Consider postoperative hypoparathyroidism. Inadequate PTH produces hypocalcemia with neuromuscular irritability; Confirm biochemically.

Integrated interpretation: PTH and calcium

A framework for connecting physiology with pathology.

Calcium state	PTH pattern	Principal consideration
High	Nonsuppressed	Primary/autonomous PTH process or abnormal sensing
High	Suppressed	Nonparathyroid hypercalcemia
Low	Inadequate / low	Hypoparathyroidism
Low	Appropriately high	Compensatory drive or target resistance

Interpret with phosphate, renal function, vitamin D context, and clinical history.

Core distinctions for pathology practice

Morphology and function must be interpreted in the same clinical frame.

- Chief cells secrete PTH; Oxyphil cells are mitochondria rich.
- PTH raises calcium by coordinated renal, vitamin D—intestinal, and skeletal pathways.
- Adenoma is usually solitary; Hyperplasia is often multiglandular.
- Unequivocal invasion or metastasis—not nuclear atypia alone—identifies carcinoma.

References

Robbins, Cotran & Kumar: Pathologic Basis of Disease.

11th edition. Elsevier; 2026.

Endocrine system: parathyroid anatomy, physiology, and disorders.

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