

PARATHYROID GLANDS

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Primary Disciplinary Field(s): Endocrinology, Physiology, Anatomy

1. Core Definition and Anatomy

The parathyroid glands constitute two pairs of small, ovoid, endocrine organs, typically located adjacent to or sometimes embedded within the posterior capsule of the larger **thyroid gland** in the neck. Historically, these glands were often overlooked due to their diminutive size and close association with the thyroid, leading to accidental removal during thyroid surgery, which resulted in severe clinical consequences that helped elucidate their crucial physiological function. Functionally, they operate independently of the thyroid, serving as the body's primary regulators of **calcium metabolism** and maintaining strict calcium-phosphorus balance necessary for cellular function, neurotransmission, and musculoskeletal integrity.

These glands usually number four in total: two superior parathyroid glands and two inferior parathyroid glands. Their precise anatomical location is highly variable, though they are generally found between the posterior aspect of the thyroid lobes and the fascial sheath encasing the thyroid gland. They are supplied by the inferior thyroid arteries, ensuring a robust blood supply essential for sensing minute changes in blood calcium concentration. The chief cells within the parathyroid glands are the specific cell type responsible for the synthesis and secretion of their signature product, parathyroid hormone.

The regulatory role of the parathyroid glands is fundamentally critical because calcium is not merely a component of bone structure; it acts as a ubiquitous second messenger in virtually all physiological systems. It is instrumental in muscle contraction, blood clotting cascades, enzyme activation, and, most importantly, determining the electrochemical excitability of nervous tissues. Therefore, the ability of these small glands to precisely fine-tune serum calcium levels underscores their profound systemic importance, preventing both debilitating nervous hyperexcitability and dangerous systemic depression.

2. Parathyroid Hormone (PTH): Structure and Function

The principal hormone secreted by the parathyroid glands is **Parathyroid Hormone (PTH)**, also known as parathormone. PTH is an 84-amino acid polypeptide hormone whose release is exquisitely sensitive to circulating ionized calcium concentrations. Unlike many endocrine systems that rely on pituitary or hypothalamic trophic signals, the parathyroid glands function autonomously, utilizing calcium-sensing receptors (CaSRs) located on the chief cell membrane to directly monitor and respond to plasma calcium levels. When blood calcium levels drop below a narrow physiological set point (hypocalcemia), PTH secretion is immediately stimulated.

PTH exerts its effect via three primary target organs: the skeletal system (bone), the kidneys, and the gastrointestinal tract (indirectly). In bone, PTH promotes the rapid mobilization of calcium reserves from the skeletal matrix into the bloodstream. This is achieved by increasing the activity and differentiation of **osteoclasts**, the bone-resorbing cells. While excessive, chronic PTH exposure can lead to skeletal demineralization, acute, pulsatile secretion is integral to maintaining moment-to-moment calcium stability.

In the kidneys, PTH has a dual role essential for mineral retention and excretion. First, it stimulates the reabsorption of calcium from the filtrate back into the blood in the distal tubules, minimizing urinary calcium loss. Second, and equally important, PTH promotes the excretion of phosphate (phosphorus), which counteracts the effects of calcium release and helps maintain the desired calcium-to-phosphorus ratio in the plasma. This reciprocal regulation is key to preventing the precipitation of calcium phosphate salts within soft tissues.

Furthermore, PTH plays a critical indirect role in intestinal calcium absorption. PTH stimulates the renal conversion of the inactive vitamin D precursor (25-hydroxyvitamin D) into its most active form, 1,25-dihydroxyvitamin D (calcitriol). This **calcitriol** then travels to the intestine, where it dramatically enhances the efficiency of dietary calcium uptake, ensuring a long-term supply of the mineral necessary for homeostasis. Thus, PTH is not just a reactive hormone but a comprehensive orchestrator of the entire calcium supply chain.

3. The Calcium-Phosphorus Balance Mechanism

The core function of the parathyroid glands is to manage the delicate equilibrium between calcium and phosphorus. This balance is critical because the product of calcium and phosphorus concentrations (x) must remain stable to prevent complications like ectopic calcification. The regulation is highly reciprocal: when parathormone levels are elevated, calcium levels rise, and, simultaneously, phosphorus levels fall due to increased renal excretion promoted by PTH.

Conversely, when PTH secretion is suppressed (often by high calcium levels), the effect reverses. Calcium reabsorption by the kidneys decreases, the mobilization of calcium from bone slows down, and phosphate excretion is reduced, causing phosphorus levels to rise. This complex feedback loop ensures that the body can quickly adjust serum calcium without causing dangerous fluctuations in phosphate concentrations, which could otherwise lead to systemic metabolic distress.

This dynamic equilibrium is essential because phosphorus, like calcium, is a necessary element, critical for ATP production, nucleic acid structure, and cell membrane integrity. However, excess phosphorus can bind with calcium in the blood, effectively reducing the amount of free, ionized calcium available for biological processes. By promoting phosphaturia, PTH ensures that the calcium it mobilizes remains biologically available and functional.

4. Regulation of Nervous and Muscular Excitability

One of the most immediate and profound consequences of parathyroid gland dysfunction relates directly to the control of **nervous system excitability**. Calcium ions play a critical role in stabilizing nerve cell membranes. Specifically, calcium stabilizes the voltage-gated sodium channels responsible for generating action potentials. When blood calcium levels fall (hypocalcemia), the extracellular calcium concentration decreases, leading to a destabilization of these channels.

The result of this destabilization is that the nervous tissue becomes hypersensitive and irritable; it requires a much smaller stimulus to reach the threshold for firing an action potential. If the parathyroid glands fail to produce sufficient hormone (hypoparathyroidism), the resulting drop in serum calcium causes the peripheral nerves to fire spontaneously and continuously.

This increased irritability manifests clinically in several ways, beginning with subtle signs such as tingling sensations (paresthesia) and progressing to involuntary muscle activity. Early symptoms include muscle twitching, especially in the face and hands, and reflexes that become noticeably **hypersensitive**. The patient may experience the Chvostek sign (facial twitching upon tapping the facial nerve) and the Trousseau sign (carpal spasm induced by temporary occlusion of blood flow to the arm).

In cases of severe, uncorrected parathormone deficiency and profound hypocalcemia, the patient becomes afflicted with generalized **tetany**. Tetany is characterized by sustained, painful muscle cramps and spasms, leading to rigidity, particularly in the limbs. If the deficiency affects laryngeal and respiratory muscles, it can result in life-threatening laryngospasm and convulsions, often accompanied by significant **emotional lability** and altered mental status due to central nervous system involvement.

5. Role in Bone Metabolism and Homeostasis

Beyond its rapid regulatory role in serum calcium, PTH is a primary determinant of long-term bone remodeling and homeostasis. Bone tissue is constantly being broken down by osteoclasts and rebuilt by osteoblasts. PTH influences the activity ratio between these two cell types. Chronic elevation of PTH, as seen in primary hyperparathyroidism, leads to a net loss of bone mass as osteoclast activity dominates.

The mechanism by which PTH indirectly controls the amount of calcium deposited in bone involves receptor activator of nuclear factor kappa-B ligand (RANKL). PTH stimulates osteoblasts (bone-forming cells) to produce RANKL. RANKL acts locally to signal precursors to differentiate into mature, active osteoclasts, which then resorb the bone matrix, releasing stored calcium and phosphate into the circulation.

The skeletal effects of PTH are particularly significant in conditions of chronic kidney disease (secondary hyperparathyroidism), where impaired vitamin D activation and phosphate retention lead to persistent hypocalcemia, causing massive, unrelenting PTH release. This prolonged hormonal signal drives severe bone disease, known as renal osteodystrophy, illustrating the necessity of precise parathyroid function for skeletal health throughout the lifespan.

6. Clinical Significance: Hyperparathyroidism

Hyperparathyroidism is a condition marked by the overproduction of PTH, usually stemming from an adenoma (benign tumor) in one of the glands (primary hyperparathyroidism). This excess PTH leads to chronic **hypercalcemia** (high blood calcium) and hypophosphatemia. The clinical presentation is often summarized by the mnemonic "stones, bones, abdominal groans, and psychic moans."

Specific complications include the formation of kidney stones (renal calculi) due to excessive calcium excretion, bone demineralization leading to osteoporosis, fractures, and cystic bone lesions (osteitis fibrosa cystica). Systemically, patients experience gastrointestinal symptoms (nausea, constipation, ulcers), and neuromuscular symptoms (fatigue, weakness), alongside significant psychological and cognitive disturbances (the "psychic moans").

Treatment for primary hyperparathyroidism often involves parathyroidectomy--surgical removal of the overactive gland or glands. This intervention is curative and is aimed at normalizing serum calcium levels to prevent irreversible damage to the kidneys and skeletal system. Management requires careful monitoring, especially post-surgery, when patients may temporarily experience severe hypocalcemia ("hungry bone syndrome") as calcium rapidly redeposits into depleted skeletal stores.

7. Clinical Significance: Hypoparathyroidism

Hypoparathyroidism, the insufficient production or secretion of PTH, is most commonly an iatrogenic consequence following thyroid or neck surgery when the small parathyroid glands are accidentally damaged or removed. Less frequently, it may result from autoimmune destruction or genetic disorders. This condition leads directly to hypocalcemia and hyperphosphatemia.

The hallmark clinical feature of hypoparathyroidism is the neuromuscular hyperexcitability detailed earlier, ranging from paresthesia to frank tetany and convulsions. Chronic hypocalcemia can also cause non-acute complications, including the calcification of the basal ganglia in the brain, cataracts, and dental abnormalities if onset occurs during childhood development.

Management of hypoparathyroidism focuses on restoring calcium homeostasis. This typically involves lifelong supplementation with high doses of oral calcium and activated vitamin D (calcitriol)

to enhance intestinal absorption, effectively bypassing the need for endogenous PTH to activate vitamin D. Achieving stability requires meticulous titration of dosage to prevent both hypocalcemic crises and the long-term risk of hypercalcemia-related renal damage.

8. Further Reading

[Parathyroid Glands \(Wikipedia\)](#)

[Physiology, Parathyroid Hormone \(StatPearls - NCBI\)](#)

[Calcium Homeostasis \(Wikipedia\)](#)

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