PARATHYROID GLAND, ANATOM, HISTOLOGY, AND PHYSIOLOGY
(A SHORT REVIEW)

Fatimah K. Ibrahim Al- Mahdawi, Abed Sultan Hassan,
Wasan A.Wahab Alsiadi

Department of Medical Laboratory Technologies, Bilad Al-Rafidain University College.
(Received 8Februaryh 2020, Accepted 8 March 2020)

Keywords: Parathyroid, Physiology, Hormones.
Corresponding Author: DrFatimah@bauc14.edu.iq

ABSTRACT

Parathyroid glands take a special place in human physiology and anatomy, as they were the last major organs to be identified in humans. The average parathyroid gland measures about five to seven mm in length, and three to four mm in width and weighs about forty to sixty mg. The four parathyroid glands are derivative from the posterior endoderm of the 3rd and 4th pharyngeal pouches. Normally, there are four parathyroid glands (superior and inferior). It takes the bean-shaped or oval. The main function of the parathyroid gland is to release hormone called parathyroid hormone (PTH) consists of 84- amino acid, which is involved in calcium homeostasis.

Parathyroid Gland

The parathyroid glands take a special place in human physiology and anatomy, as they were the last major organs to be identified in humans. It consists of four masses, two posteriors inserted into each lateral thyroid gland mass \(^{(1)}\). Usually, the upper parathyroid glands are about 1 cm above the junction of the lower thyroid artery and the regular laryngeal nerve at the cricoid cartilage point.\(^{(2)}\) The lower parathyroid glands are most commonly hold on the posterior lateral part of the inferior thyroid pole, under the junction of the lower thyroid artery and the regular laryngeal nerve \(^{(3)}\). (Figure 1)
Figure 1: A dorsal view of the parathyroid glands. The Four parathyroids gland are embedded in the thyroid gland (Fox, 2006) (4).

Anatomy and Histology

The average parathyroid gland measures about five to seven mm in length, three to four mm in width and weighs about forty to sixty mg (5). The four parathyroid glands are derivative from the posterior endoderm of the 3rd and 4th pharyngeal pouches. In the 5th week of gestation they differentiate and lose their pharyngeal connections in the 7th week of gestation (6). The upper glands, occasionally mentioned as “parathyroid IV” for they ascend from the 4th pharyngeal pouch, descend from the base of the tongue with the higher pole of the thyroid gland, ultimately coming to middle along the dorsal boundaries of the thyroid (7). The upper parathyroid glands in the juxta-crico-thyroidal place posteriorly in 77%, behind the high pole of the thyroid gland in a subcapsular site in 22%, and behind the junction of the superior esophagus and inferior pharynx in the midline in 1% of people. Lower parathyroid glands were found directly nearby to or on the front or posterior-lateral surfaces of the inferior pole of the thyroid gland in 57%, intra-thymic in 41%, and along the carotid artery at or adjacent its bifurcation in 2% of people (8).

Normally, there are four parathyroid glands (superior and inferior). It takes the bean-shaped or oval (9). It naturally measures six mm $\times 4$ mm $\times 2$ mm and weighs forty mg to sixty mg (10). The chief cells are the component of parathyroid gland parenchyma (which also known as
the parathyroid gland primary) and oxyphil cells organized in trabeculae, through a stroma composed principally of adipose cells, (11) and divided into lobules by a thin fibrous capsule. The stromal fat round the parathyroid glands rises gradually with age up to thirty percent in age twenty-five. The percentage of fat is associated to the constitutional fat percentage, but it can decrease the individuals’ death; this means it is seventeen percent with wide variation (9). Microscopically, they are formed of chief cells, clear cells and oxyphilous cells. The “chief” cells are curved with homogenous and slightly acidophil cytoplasm, producing parathormone. When secretion is decreased or when at “rest” status, glycogen and cytoplasmic granules of lipids are accumulated, assuming characteristics of the so-called “clear cells” (12). “Oxyphilous” cells are greater, with acidophil cytoplasm because of its affinity to eosin, and appear in puberty, progressive increasing in number with age, they do not release parathormone and its function is still unclear (13). All cells are arranged on a rope-like arrangement, interrupted by tissue fat lobules (14). Oxyphil cells are observed also alone or in slight groups interspersed among chief cells. They are greater than chief cells and the rich cytoplasm is full with several large mitochondria (1).

Physiology

The main function of the parathyroid gland is to release hormone called parathyroid hormone (PTH) consists of 84- amino acid , which is involved in calcium homeostasis (15). PTH from the parathyroid gland increases Ca²⁺ in the blood by increasing the absorption of Ca²⁺ in the intestines and reabsorption in the kidneys (16), especially in distal tubules and maybe also in Henle loops and by stimulating the release of Ca²⁺ from bones (17) (18). Other functions of PTH, including stimulation of magnesium reabsorption by the loop of Henle and inhibition of phosphate reabsorption by the proximal tubule (18). The normal ranges of calcium in the human body 1000-1200g. Approximately 99% of body calcium participate in the skeleton; the other 1% is present in the intracellular and extracellular compartment. Although 99% of the total body calcium is resides in bone, calcium a critical cation in both the intracellular and extracellular compartment (19). Calcium plays a vital role in muscular contraction, nerve impulse transmission, hormone secretion, intercellular adhesion and blood coagulation (20). PTH rises calcium absorption from the bone through 2 effects on bones in causing absorption of phosphate and calcium. One is a fast phase that starts in minutes and rises gradually for several hours. this phase results from activation of the previously present bone cells (mostly the osteocytes) to promote
absorption of phosphate and calcium. The next phase a much slower one, needing several days or even weeks to become entirely developed; it results from proliferation of the osteoclasts, followed through significantly raised osteoclastic reabsorption of the bones itself, not only absorption of the calcium phosphate salts from the bone\textsuperscript{(18)}. An increased level of PTH causes the bones to release calcium and the kidneys to keep calcium that would otherwise be missing through the urine. Increase Ca\textsuperscript{2+} level in blood can then exert negative feedback inhibition on PTH \textsuperscript{(4)}. Figure 2

![Diagram of Parathyroid Hormone Regulation]

Figure 2: Actions and the control secretion of PTH (Fox, 2006)\textsuperscript{(4)}.

**Hyperparathyroidism**

Hyperparathyroidism HPT disease caused by over secretion of PTH \textsuperscript{(21)}. Inappropriately elevated parathyroid hormone level, classically associated with hypercalcemia\textsuperscript{(22)}. Hyperparathyroidism is classified to one primary and two secondary, or tertiary\textsuperscript{(6)}. Primary, hyperparathyroidism is a condition caused by over secretion of PTH with a concurrent alteration of the phosphor calcemic metabolism: commonly found an association between hypercalcemic status and raised PTH levels or not properly normal\textsuperscript{(21)}. Secondary, hyperparathyroidism as a results of chronic kidney failure, though sometimes associated with malabsorption of the intestines. The reabsorption of calcium by the reduced small intestine, there is phosphate
Retention and the usual effect of PTH on bones calcium release is lost cause an absence of 1,25-hydroxycholecalciferol, the serum PTH level rises in attempt to maintain calcium homeostasis, causing hyperplasia of the parathyroid gland \(^{16(23)}\).

### REFERENCES

1. Chen H, Senda T, Emura S, Kubo K. (2013) An update on the structure of the parathyroid gland. open Anat J.;5(1).

2. McHenry CR. (2001) The Parathyroid Glands and Hyperparathyroidism I. General Surgery Board Review Manual. Hosp Phys.;6:1–12.

3. Phitayakorn R, McHenry CR. (2006) Incidence and location of ectopic abnormal parathyroid glands. Am J Surg.;191(3):418–23.

4. Fox SL. (2006) Human Physiology 9th Edition. McGraw-Hill press, New York, USA;.
5. Yao K, Singer FR, Roth SI, Sassoon A, Ye C, Giuliano AE. (2004) Weight of normal parathyroid glands in patients with parathyroid adenomas. J Clin Endocrinol Metab.;89(7):3208–13.

6. Palestro CJ, Tomas MB, Tronco GG. (2005) Radionuclide imaging of the parathyroid glands. In: Seminars in nuclear medicine. Elsevier.; p. 266–76.

7. Mansberger Jr AR, Wei JP. (1993) Surgical embryology and anatomy of the thyroid and parathyroid glands. Surg Clin North Am.;73(4):727–46.

8. Wang C.A.(1976). The anatomic basis of parathyroid surgery. Ann Surg.;183(3):271.

9. Arrangoiz R, Cordera F, Caba D, Juárez MM, Moreno E, Luque E. (2017) Parathyroid embryology, anatomy, and pathophysiology of primary hyperparathyroidism. Int J Otolaryngol Head Neck Surg.;6(04):39.

10. Fancy T, Gallagher 3rd D, Hornig JD. (2010) Surgical anatomy of the thyroid and parathyroid glands. Otolaryngol Clin North Am.;43(2):221.

11. Bilezikian JP, Marcus R, Levine MA, Marcocci C, Silverberg SJ, Potts JT. (2014) The parathyroids: basic and clinical concepts. academic Press.

12. Stevens A, Lowe JS. (1995) Sistema muscular esquelético. Histol São Paulo, Manole.;14:226–48.

13. Young B, Heath JW. (2000) Histologia funcional. Rio Janeiro Guanabara.

14. Prospero JD de, Baptista PPR, Amary MFC, Santos PPC (2009) Parathyroid glands: structure, functions and pathology. Acta Ortopédica Bras.;17(2):53–7.

15. Ramasamy I. (2006) Recent advances in physiological calcium homeostasis. Clin Chem Lab Med.;44(3):237–73.

16. Al-Mahdawi FKI, Sultan AS, Al-Wandi NKMA. (2018) Evaluation parathyroid hormone function and some minerals in chronic renal failure. J Pharm Sci Res.;10(1):96–9.
17. Contributors W. (2012.) Human physiology. Blacksleet River.

18. Jackson JEH. (2006 ) TEXTBOOK OF MEDICAL PHYSIOLOGY/Arthur C. Guyton & John E. Hall/Copyright© by Elsevier Inc. Elsevier Inc.; 2006.

19. Blaine J, Chonchol M, Levi M. (2015) Renal control of calcium, phosphate, and magnesium homeostasis. Clin J Am Soc Nephrol;10(7):1257–72.

20. Hebert SC, Brown EM. (1996) The scent of an ion: calcium-sensing and its roles in health and disease. Curr Opin Nephrol Hypertens.;5(1):45–53.

21. Percivale A, Gnerre P, Damonte G, Buscaglia S, Monachesi M, Parodi L, et al. Primary hyperparathyroidism (2015) epidemiology, clinical features, diagnostic tools and current management. Ital J Med;330–45.

22. Wachtel H, Bartlett EK, Kelz RR, Cerullo I, Karakousis GC, Fraker D. L. (2014) Primary hyperparathyroidism with negative imaging: a significant clinical problem. Ann Surg.;260(3):474–82.

23. Neumann DR, Shin JJ. (2012) Imaging in Parathyroid Diseases. In: Diseases of the Parathyroid Glands. Springer;, p. 343–62.