Parathyroid

SESSION OBJECTIVES:

Use these session objectives to test your knowledge of the important concepts presented in this chapter and as study topics to return to prior to your exams.

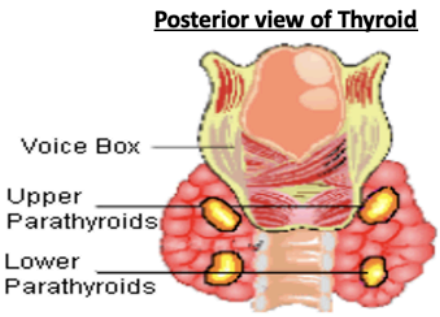
1. Describe pathology of normal parathyroid gland.
2. Compare pathology of normal parathyroid gland to that of parathyroid neoplasia.
3. Describe pathologic features of hyperparathyroidism.

OPTIONAL PRE-CLASS MATERIALS FOR THIS SESSION:

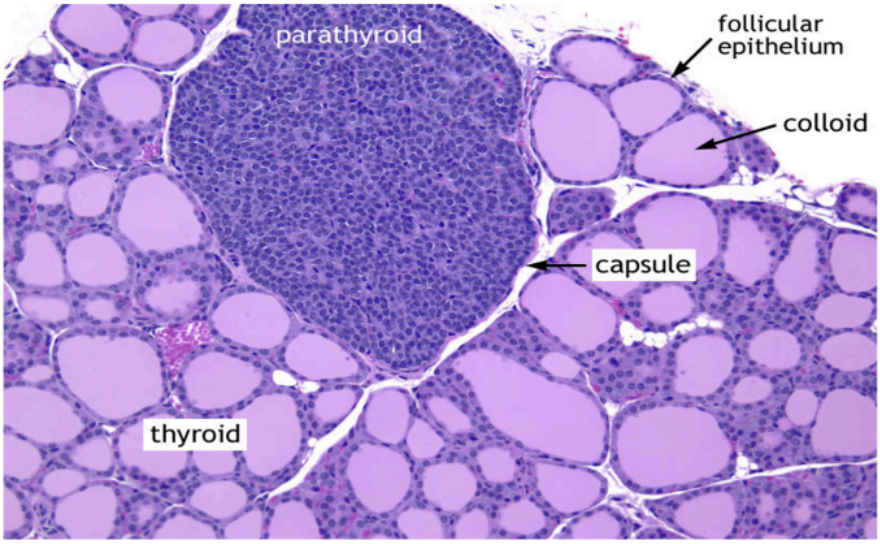
- Skim the **section titles**, **bolded terms,** and **image captions** from Robbin's & Kumar 11th edition, Chapter 18 to fill in any knowledge gaps you need.
- In class exercise: PathPresenter

OVERVIEW:

The parathyroid glands are small, typically four in number, and located behind the thyroid gland. Their primary function is to regulate calcium homeostasis by producing **parathyroid hormone (PTH)**, which increases blood calcium levels by acting on the bones, kidneys, and intestines. Understanding the normal structure of the parathyroid glands and their associated pathologies is critical for diagnosing and managing calcium imbalances.



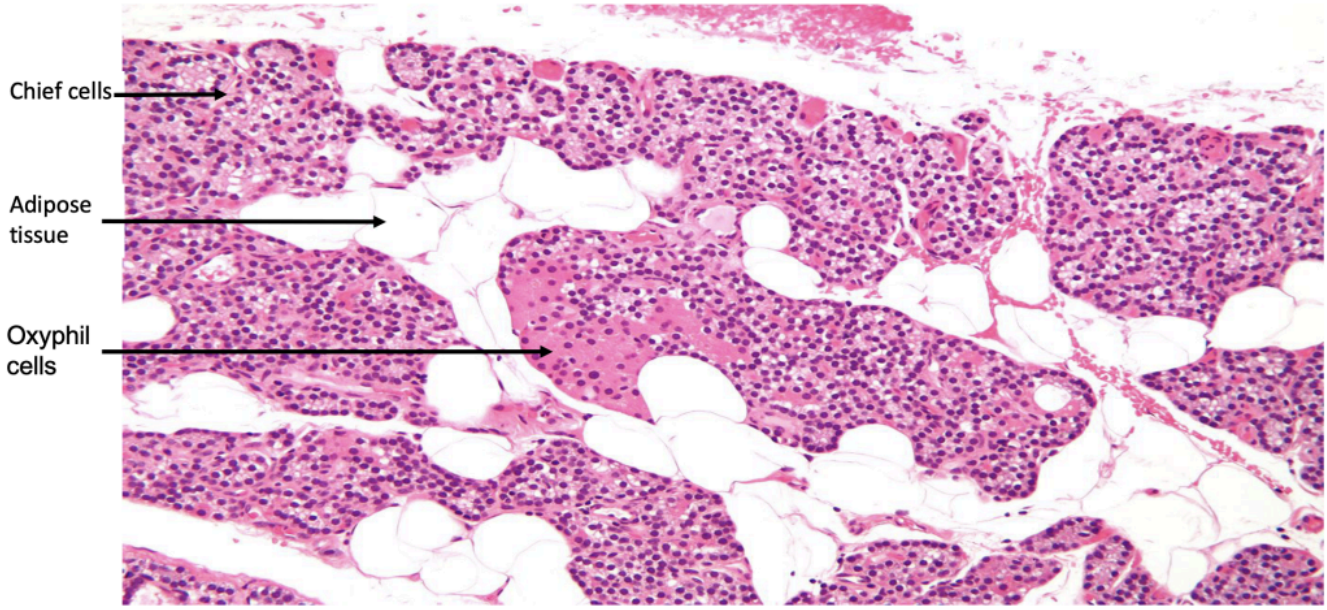
- 4th Pharyngeal Pouch - superior parathyroid
- 3rd Pharyngeal Pouch - inferior parathyroid, initially descends with thymus



NORMAL HISTOLOGY OF THE PARATHYROID GLANDS:

The parathyroid glands consist of two main cell types (in connective tissue):

- **Chief Cells**: These are the most abundant cells in the parathyroid glands and are responsible for producing PTH. Chief cells appear small and dark under the microscope.
- **Oxyphil Cells**: These are larger and less numerous than chief cells, with a more eosinophilic (pink) cytoplasm. Their function is not well understood, but they increase in number with age.
- **Adipose Tissue**: As individuals age, adipose tissue progressively infiltrates the parathyroid gland.



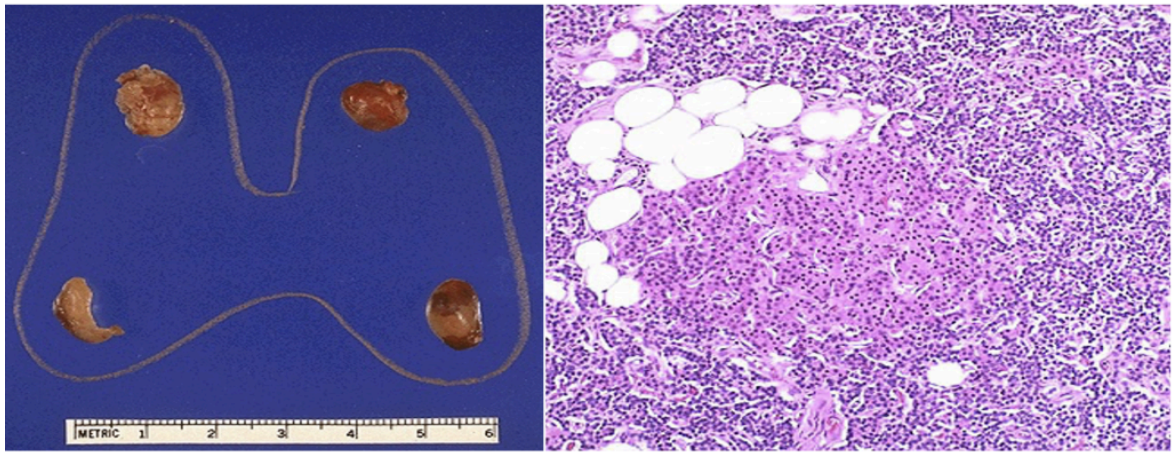
The normal architecture of the parathyroid glands is critical for the production and regulation of PTH, and any disruptions to this structure can lead to disorders of calcium metabolism.

PATHOLOGY OF THE PARATHYROIDS:

The parathyroid glands play a crucial role in maintaining calcium homeostasis by secreting **parathyroid hormone (PTH)**. When the regulation of PTH is disrupted, it can lead to **hyperparathyroidism**, a condition

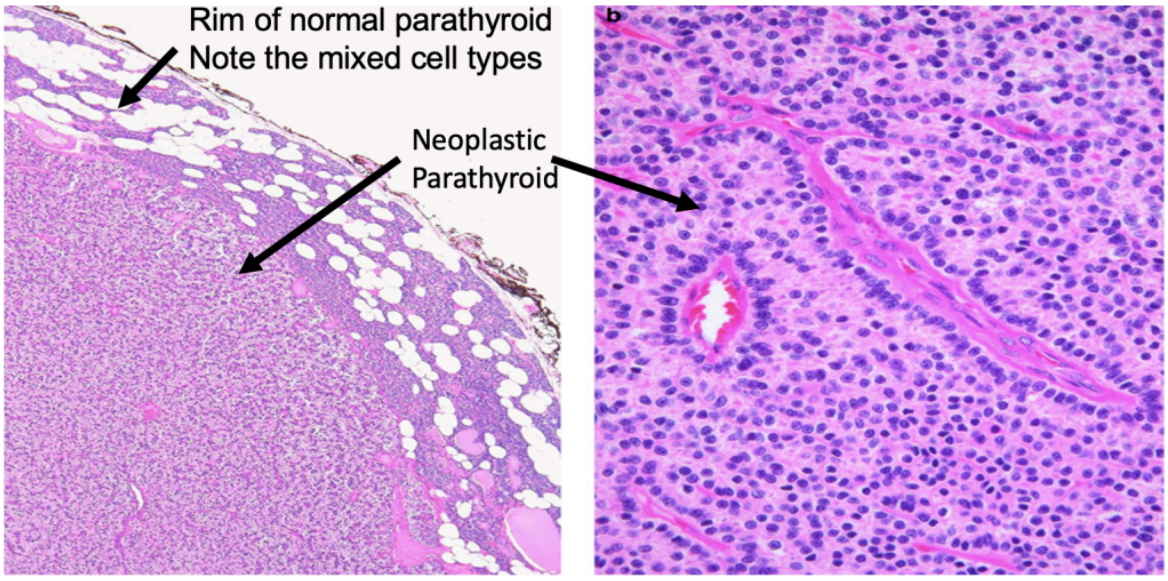
characterized by excessive secretion of PTH. This overproduction of PTH leads to elevated calcium levels in the blood (hypercalcemia) and causes various systemic symptoms, ranging from kidney stones to bone pain and neuropsychiatric disturbances. There are three main types of hyperparathyroidism:

- **Primary Hyperparathyroidism**: This occurs due to overproduction of PTH from the parathyroid glands themselves, commonly caused by a parathyroid adenoma or, less frequently, parathyroid hyperplasia.
  - **Parathyroid Hyperplasia**: This condition involves an increase in the number of cells in all four glands (left image below). Histology shows an expansion of both chief and oxyphil cells with minimal adipose tissue (right image below). Treatment often involves surgical removal of three and a half glands, with the remaining portion implanted in a different location (e.g., the arm).



Increased number of Oxyphil and Chief cells with not much adipose tissue

  - **Parathyroid Adenoma**: A benign tumor affecting one parathyroid gland, resulting in excessive PTH production. Histologically, the adenoma consists of monotonous neoplastic cells, frequently with a rim of normal parathyroid tissue and decreased adipose tissue (image below).



Monotonous, neoplastic parathyroid cells

- **Secondary Hyperparathyroidism**:
  - This condition results from chronic stimulation of the parathyroid glands due to **hypocalcemia**, typically caused by **chronic kidney disease**. The failing kidneys are unable to convert vitamin D to its active form or adequately excrete phosphate, leading to hypocalcemia and compensatory overproduction of PTH.
- **Tertiary Hyperparathyroidism**:
  - This develops after long-standing secondary hyperparathyroidism, when the parathyroid glands become autonomously hyperactive, producing PTH even when calcium levels are normal or elevated.

This Chapter's PDF

LINK

- Note: The interactive features of this chapter are not reproducible in this PDF format.