**Thyroid Pathology**

The thyroid gland is located in a fairly accessible and visible part of the body. We will approach thyroid pathology in a systematic fashion, looking at:

I.**Anatomy**

* Understanding the anatomical relations helps you to work out the clinical presentations of thyroid enlargement (goitre).

**II. Function**

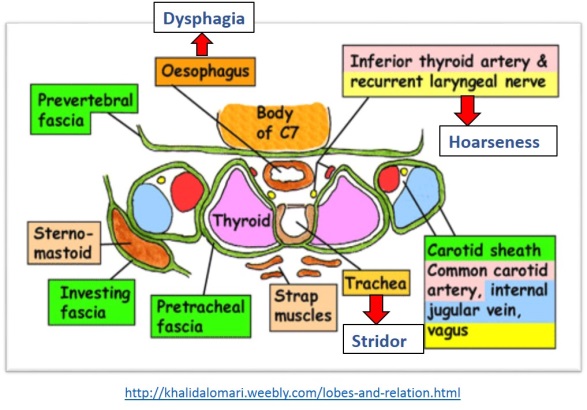
* The function of the thyroid gland relates to the type of parenchymal cells it contains
* It is also important to appreciate that the thyroid gland is an ***endocrine***organ, and therefore part of a system of regulatory mechanisms

**III. Clinicopathologic Correlates**

* This section helps you correlate the clinical manifestations of thyroid pathology with specific disease entities
* Build on this framework by reading about the specific *pathogenesis*, *clinical features* and *morphologic features* of each condition

**Anatomy**

The relations of the thyroid gland are particularly important. Think about thyroid enlargement 🡪 what gets compressed? This gives rise to the clinical presentation.



Reference websites for thyroid anatomy:

1.<http://khalidalomari.weebly.com/lobes-and-relation.html>

2.<http://fitsweb.uchc.edu/student/selectives/Luzietti/Thyroid_anatomy.htm> (You can peel away the layers of the neck)

**Function**

The main functions of the thyroid gland are brought about by the TWO main parenchymal cell types:

**1. Follicular cells –>** Thyroid hormones T3 (tri-iodothyronine) and T4 (tetra-iodothyronine/thyroxine)

* Regulate basal metabolic rate (think about the clinical signs and symptoms of *hyper/hypo thyroidism*)
* Growth and development, especially of the central nervous system (*read about Cretinism – hypothyroidism in infancy or childhood)*
* The free (unbound) hormones are the metabolically active forms

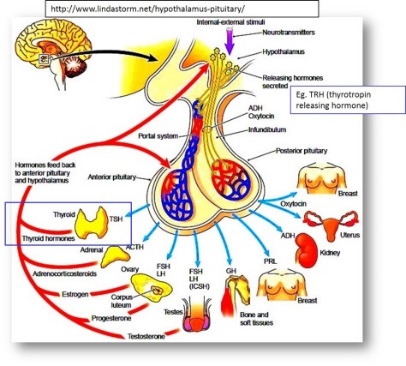
More on T3 and T4 production: <http://fitsweb.uchc.edu/student/selectives/Luzietti/Thyroid_hormones.htm>

**2. Parafollicular C cells** –> Calcitonin

* Calcium metabolism – maintains calcium homeostasis (generally, calcitonin opposes the effects of Parathormone)

**Regulation of T3 and T4 production**

As an endocrine organ, the thyroid gland is subject to secondary (pituitary – TSH) and tertiary (hypothalamus – TRH) control mechanisms that all endocrine organs are subject to. This is the ***hypothalamo-pituitary axis.***



Mindmap - Thyroid anatomy and function: <http://blog.nus.edu.sg/pathotest2/chapter-thyroid-pathology/ii-function/>

**Clinicopathologic Correlates**

Here are TWO main clinical manifestations of thyroid disease:

**1. Enlargement** (non-neoplastic or neoplastic)

* Determined by history, clinical examination and imaging

**2. Abnormal function**(hyper or hypothyroidism)

* Assessed by blood investigations of various hormone levels (free T3 Free T4, TSH)

Remember, these two can co-exist (e.g. Graves disease – think about what the clinical presentation is).

- The **Aetiology** of thyroid conditions is widely variable, but the few that are more common and important are as featured in your lecture notes:

1. **Congenital** conditions (e.g. hypoplasia, ectopic thyroid)
2. **Hyperplasia** (simple or nodular hyperplasia due to decreased iodine availability)
3. **Immune related** (autoimmune, or other mechanisms of thyroiditis)
4. **Neoplasms**

In clinicopathologic correlation, we would ask some questions. E.g. What condition is more likely to cause diffuse enlargement? Which causes a solitary nodule?

Mindmap - Clinicopathologic correlation:

<http://blog.nus.edu.sg/pathotest2/chapter-thyroid-pathology/iii-clinicopathologic-correlates/>

**Talking POTS and slides**

<http://blog.nus.edu.sg/pathotest2/chapter-thyroid-pathology/thyroid-iv-talking-pots-and-slides/>

**Quiz**

<http://blog.nus.edu.sg/pathotest2/chapter-thyroid-pathology/v-thyroid-quiz/>