

# Notes

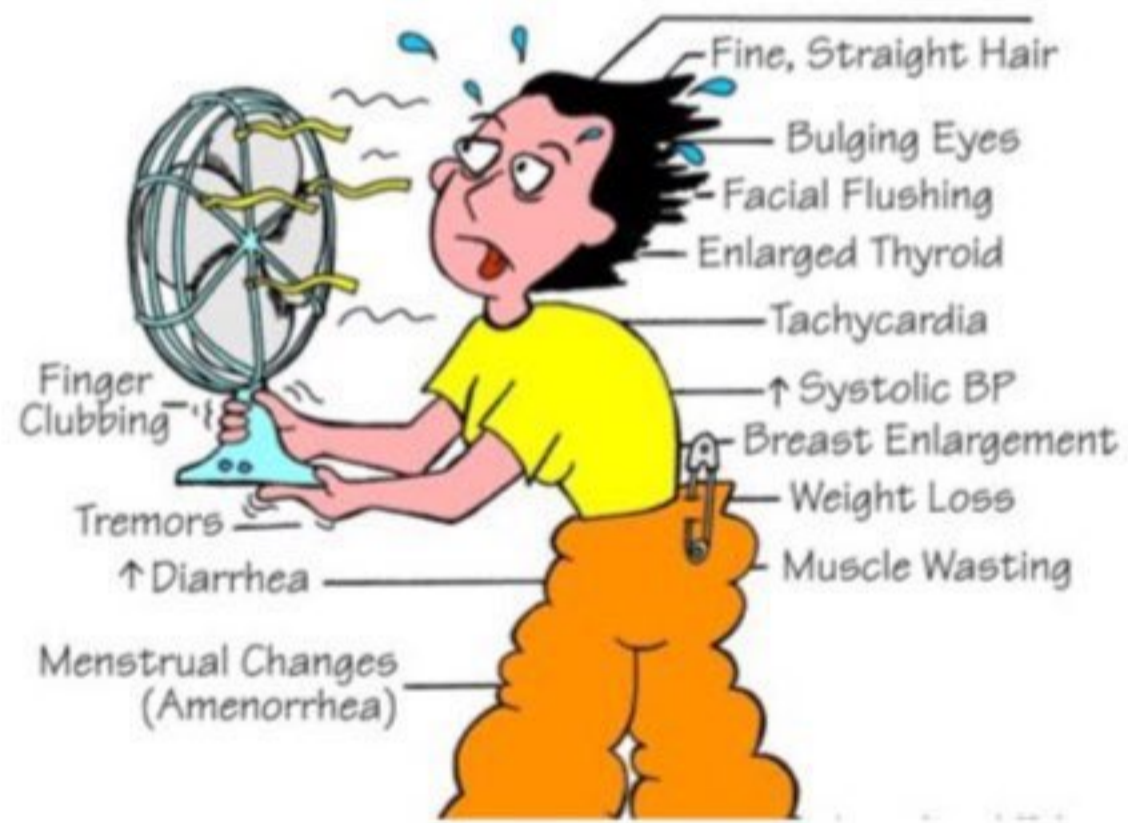
\* any disease in endocrine gland related to

\* parathyroid can't cause mass effect because it's too small to cause compression on other structure

abnormality in secretion of hormone (Hypothyroidism & Hyperthyroidism)  
mass effect: enlarged gland that cause compression on surrounding structure

In thyroid gland the enlargement of gland (goiter) regardless of cause + regardless of state of hormone.  $\Rightarrow$  No Relation Between mass effect & level of hormone production.

## Hyper-thyroidism



$\Rightarrow$  increase thyroid hormone  
 $\Rightarrow$   $\uparrow$  basal metabolic rate  
 $\Rightarrow$  increase appetite  $\Rightarrow$  increase breakdown of fat & Glucose.

$\Rightarrow$  increase heart rate [cause hypertension] increase body temperature.

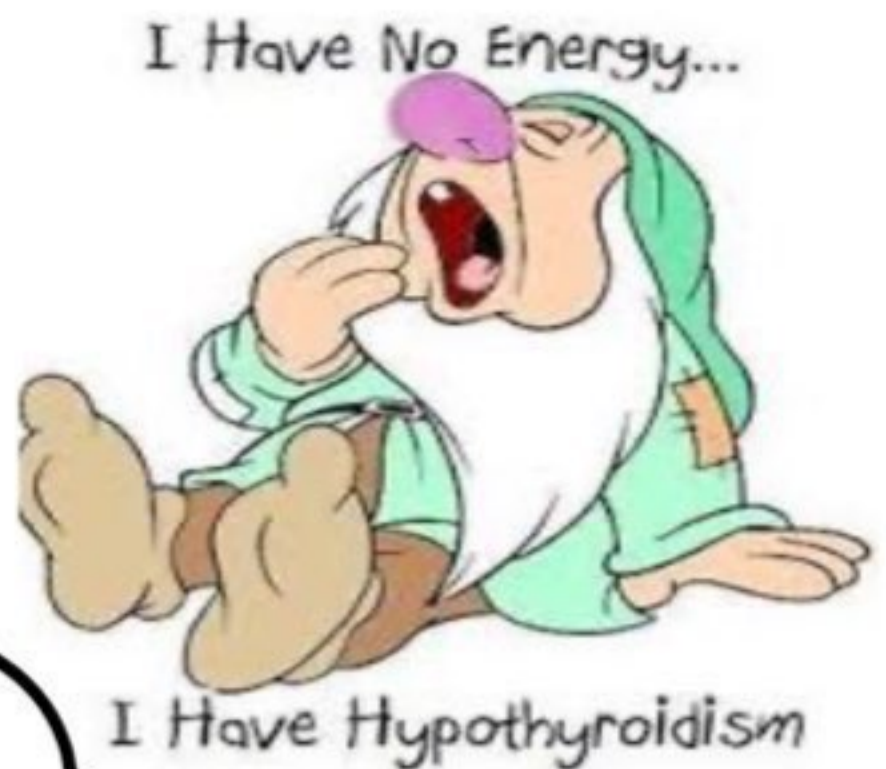
$\Rightarrow$  diarrhea, malabsorption ( $\uparrow$  intestinal motility)

$\Rightarrow$  heat tolerance & excessive sweating weight loss

$\Rightarrow$  50% develop proximal muscle weakness (thyroid myopathy)



**Primary causes**

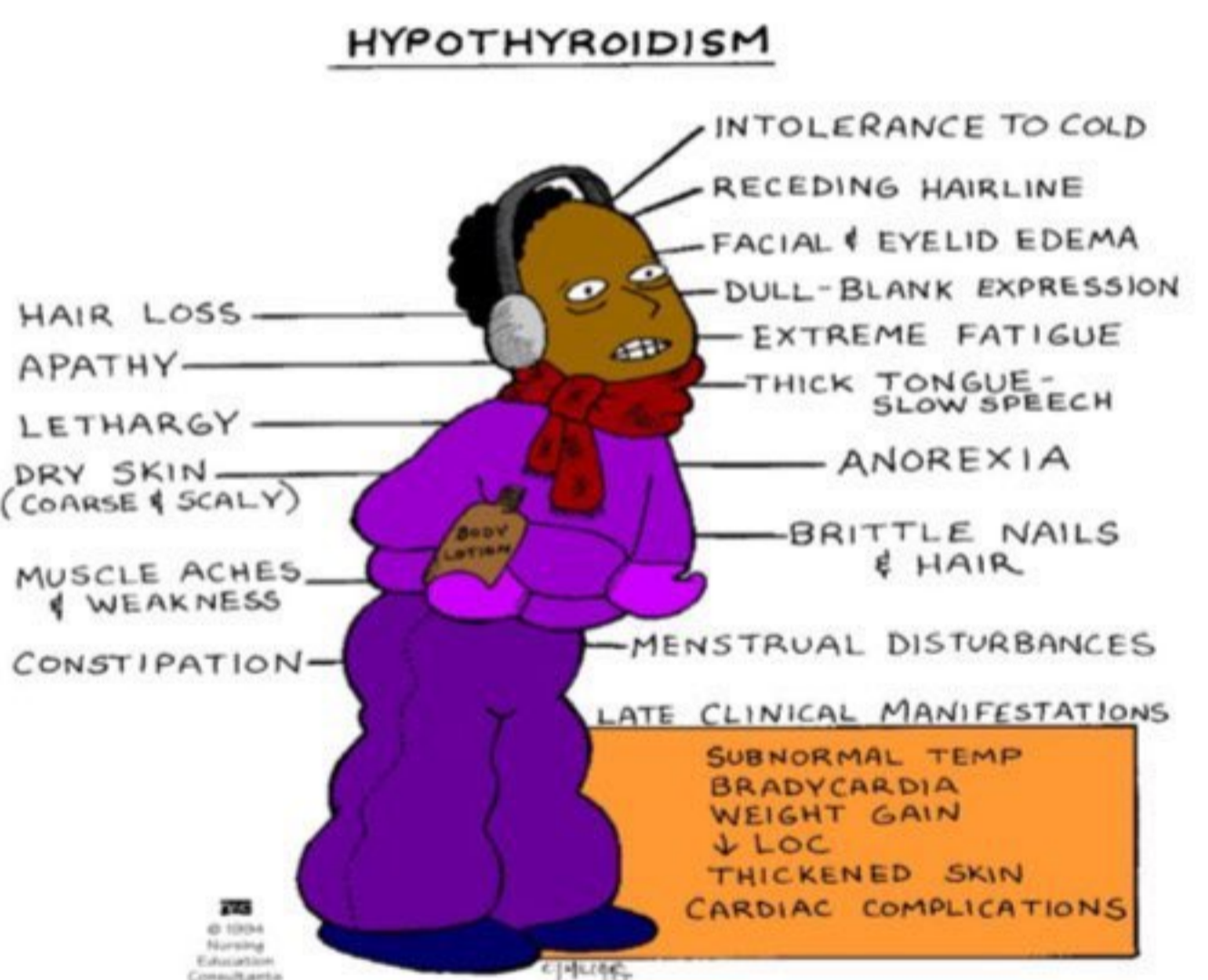


**Hypothyroidism**

- \* most common cause of Hypothyroidism is **deficiency of Iodine** [worldwide]
- \* most common cause of Hypothyroidism in developed countries is **Hashimoto**
- genetic  $\Rightarrow$  congenital biosynthetic defect [dysmormogenic goiter]

**secondary causes**

\* pituitary & Hypothalamic disorder



it causes two syndromes:

- **Cretinism**  $\Rightarrow$  Hypothyroidism in infancy & childhood.
- **Myxedema**  $\Rightarrow$  Hypothyroidism in older children & adults.

note: thyroid hormones are vital early in life for brain & body development  
 so many children have mental abnormalities.

**Cretinism**

- $\rightarrow$  **Endemic**: in dietary Iodine deficiency is endemic, including mountainous areas.
- $\rightarrow$  **sporadic**: caused by enzyme defect that interfere with thyroid hormone synthesis.



**Clinical Feature of Cretinism**

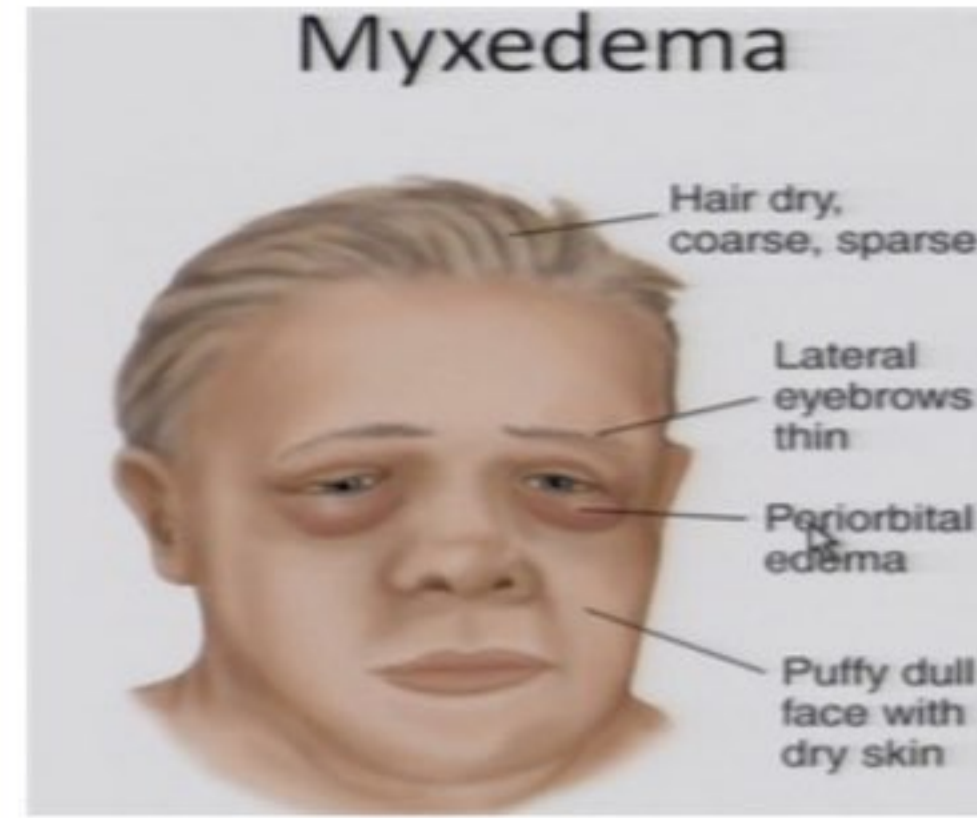
- $\rightarrow$  Impaired development of skeletal system (short stature)
- $\rightarrow$  Protuding tongue + umbilical hernia
- $\rightarrow$  Central nervous system problem with mental retardation
- $\rightarrow$  Coarse Facial Feature.

# Hypothyroidism

من قن اللاديه :-

- |  |  |
|--|--|
| <ul style="list-style-type: none"> <li>■ <u>Cretinism (child)</u></li> <li>■ Impaired CNS &amp; bone growth</li> <li>■ Mental retardation</li> <li>■ Short stature</li> <li>■ Coarse facial features</li> <li>■ Protruding tongue</li> <li>■ Umbilical hernia</li> </ul> | <ul style="list-style-type: none"> <li>■ <u>Myxedema (adult)</u></li> <li>■ Slow physical and mental activity</li> <li>■ Cold intolerance</li> <li>■ Over weight</li> <li>■ Low cardiac output</li> <li>■ Constipation and decreased sweating</li> <li>■ Cool pale thick skin</li> <li>□ Coarse or Broadening of Facial Feature</li> </ul> |
|--|--|

\* myxedema



- Enlargement of tongue
  - deeping of voice
  - Pericardial - effusions are common.
  - mucopolysaccharide - rich edematous
- Fluid accumulates in skin, subcutaneous tissue + number of visceral sites.

\* all thyroid diseases are more common in women ⚠

## Thyroiditis

Chronic lymphocytic thyroiditis  
[Hashimoto]

\* AutoImmune destruction of thyroid gland.

\* Diffuse + symmetrical enlargement + painless mass + associated with Hypothyroidism [Hashimoto]

Subacute granulomatous [de Quervain]

\* viral infection خطيبا

- \* Fever + malasia
- virus
- \* self limited
- \* painful

Subacute lymphocytic thyroiditis

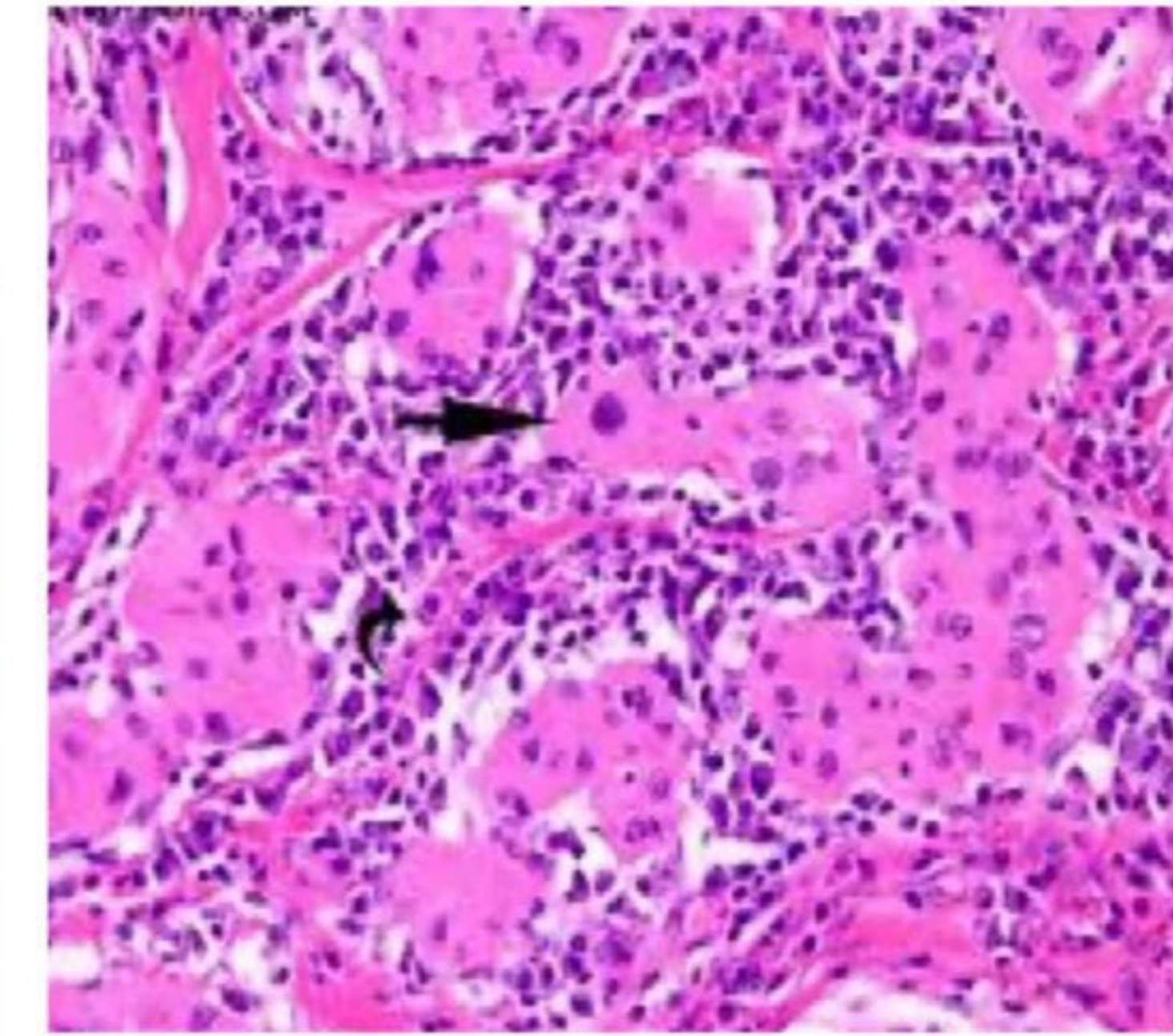
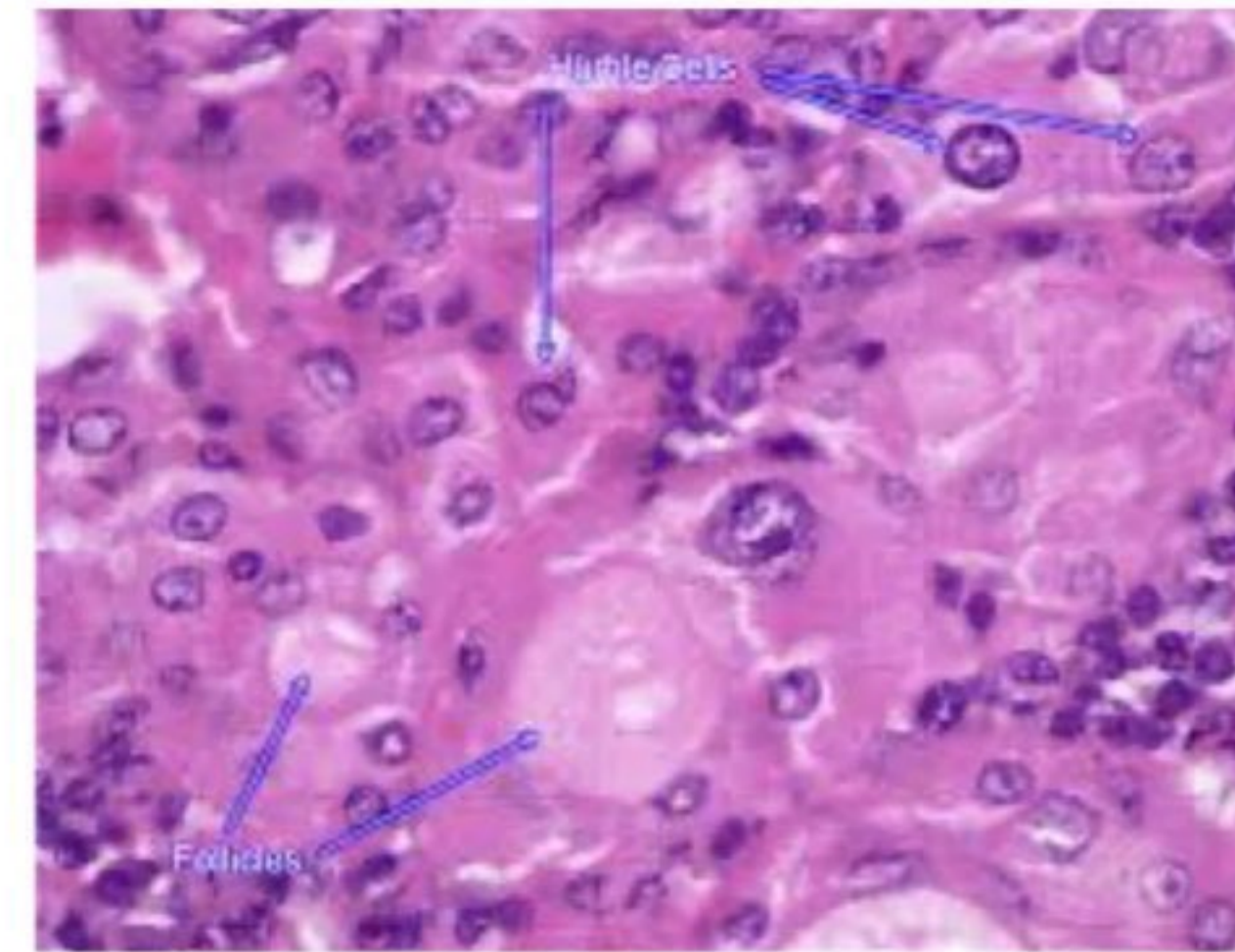
Riedel thyroiditis

\* Hashimoto more common in women than men. [between 45 - 65]  
\* it can occur in children + major cause of non-endemic goiter in children.

\* Hashimoto ⇒ painless enlargement  
 associated with some degree of Hypothyroidism

\* Patients with Hashimoto:

- ① have other autoimmune disease  
 At increased risk of development of
- ② B cell non-hodgkin lymphomas with thyroid gland.
- ③ Predisposition to Papillary Carcinomas.



↑ (Hurthle cells)  
 - is full of mitochondria

## Hashimoto thyroiditis



Kumar et al: Robbins Basic Pathology, 9e.  
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- \* Infiltration by lymphocyte (plasma cell)
- \* lymphoid aggregation with germinal center
- \* thyroid follicles are atrophic
- \* Hurthle cell ⇒ follicles lined by epithelial cell + abundant eosinophilic cytoplasm + large number of mitochondria.

## Subacute granulomatous



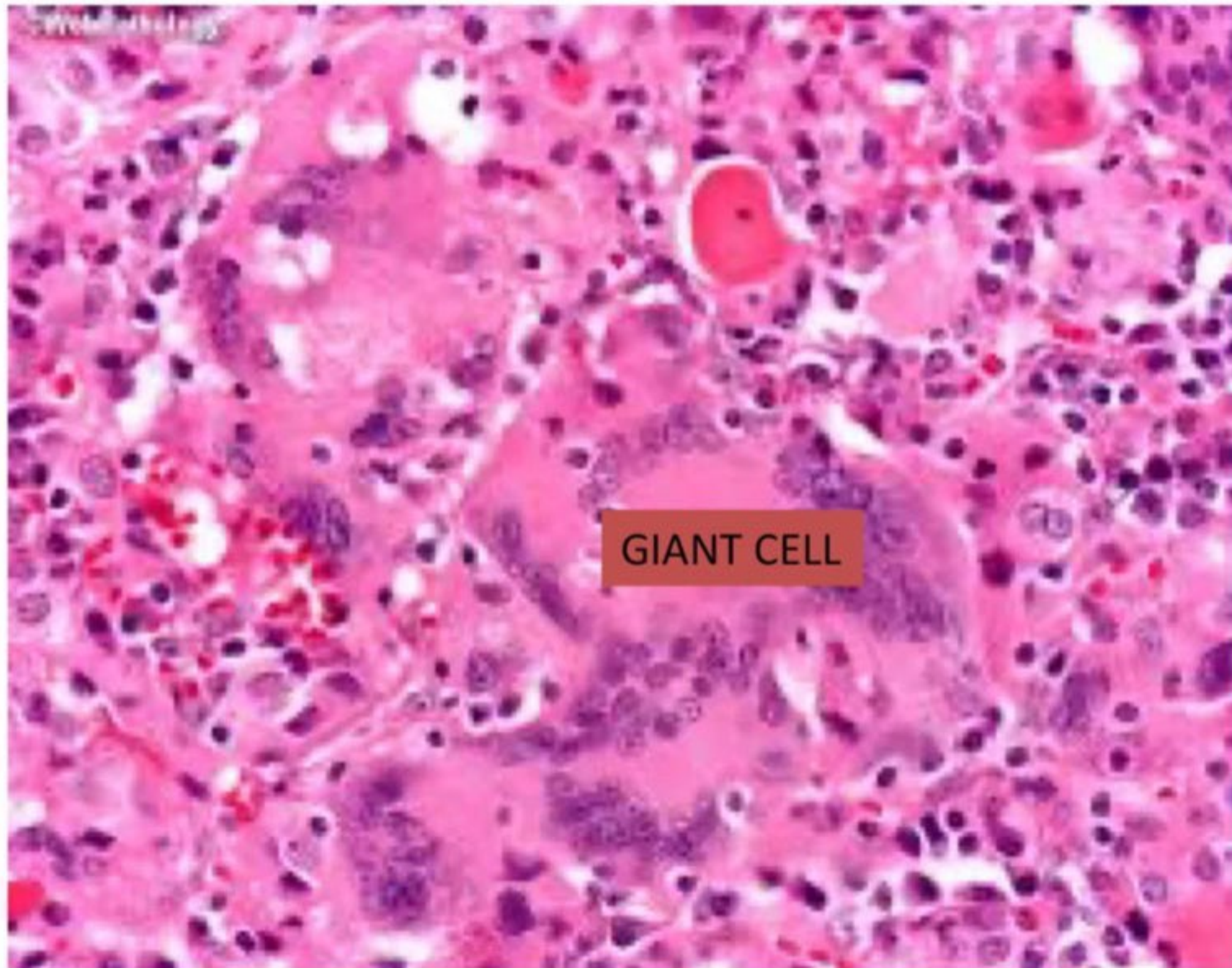
- ⇒ much less than Hashimoto
- ⇒ between (30-50)
- ⇒ Caused by viral infection + history of an upper respiratory infection just before onset of thyroiditis.



### Clinical Feature

- ⇒ [1] acute onset characterized by neck pain (with swallowing)
- ⇒ [2] Fever + malaise
- ⇒ [3] leukocyte count is increased
- ⇒ [4] self-limited ⇒ most of patient returning to a euthyroid state within 6-8 weeks.

## Subacute granulomatous thyroiditis



- \* No Hurthle cell
- \* granulomatous reaction with giant cell that contain fragments of colloid
- \* neutrophilic infiltrate which is replaced by lymphocyte + plasma cell + macrophage.

## Subacute lymphocytic thyroiditis

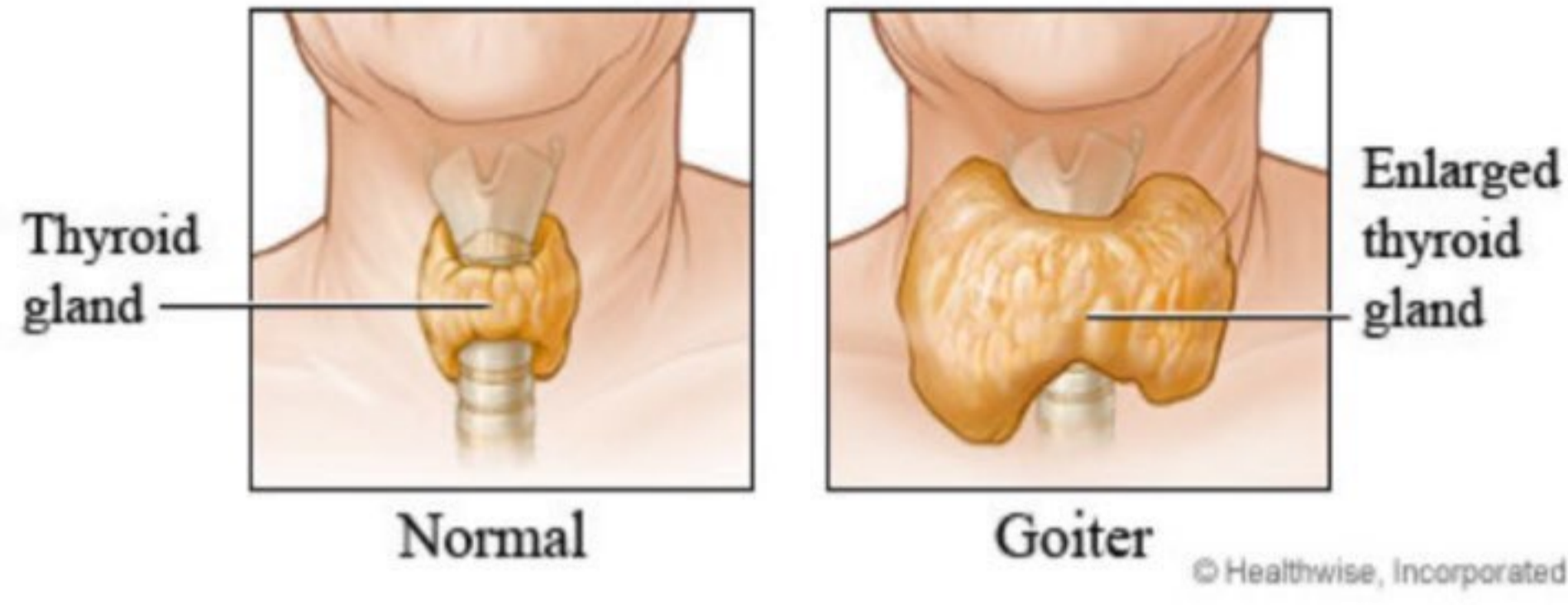
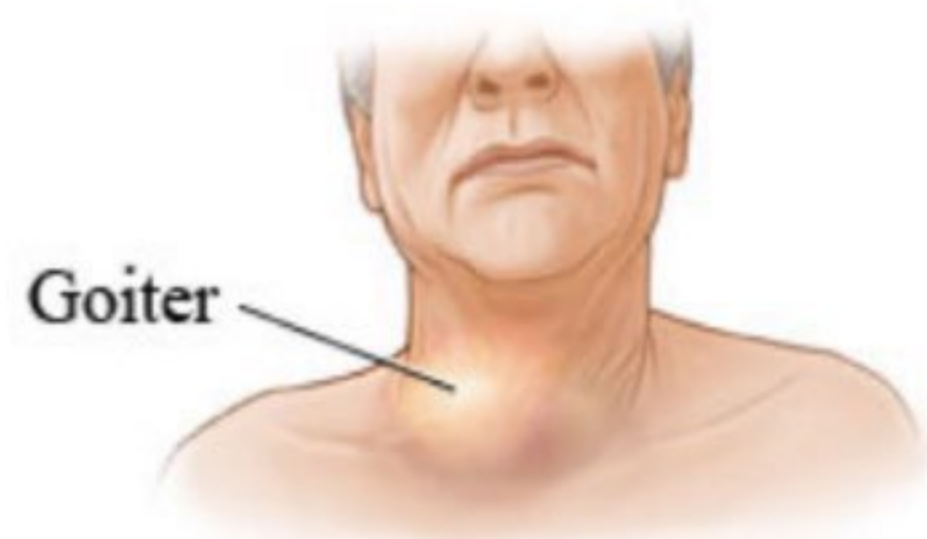
- ⇒ silent or painless
- ⇒ post partum thyroiditis ⚠
- ⇒ most likely to be autoimmune because of circulating anti-thyroid antibody
- ⇒ mostly affect middle-age women who present with painless neck + feature of hyperthyroidism.

## Riedel thyroiditis

⚠ it's not malignant or premalignant but it mimics the malignancy (from doctor)

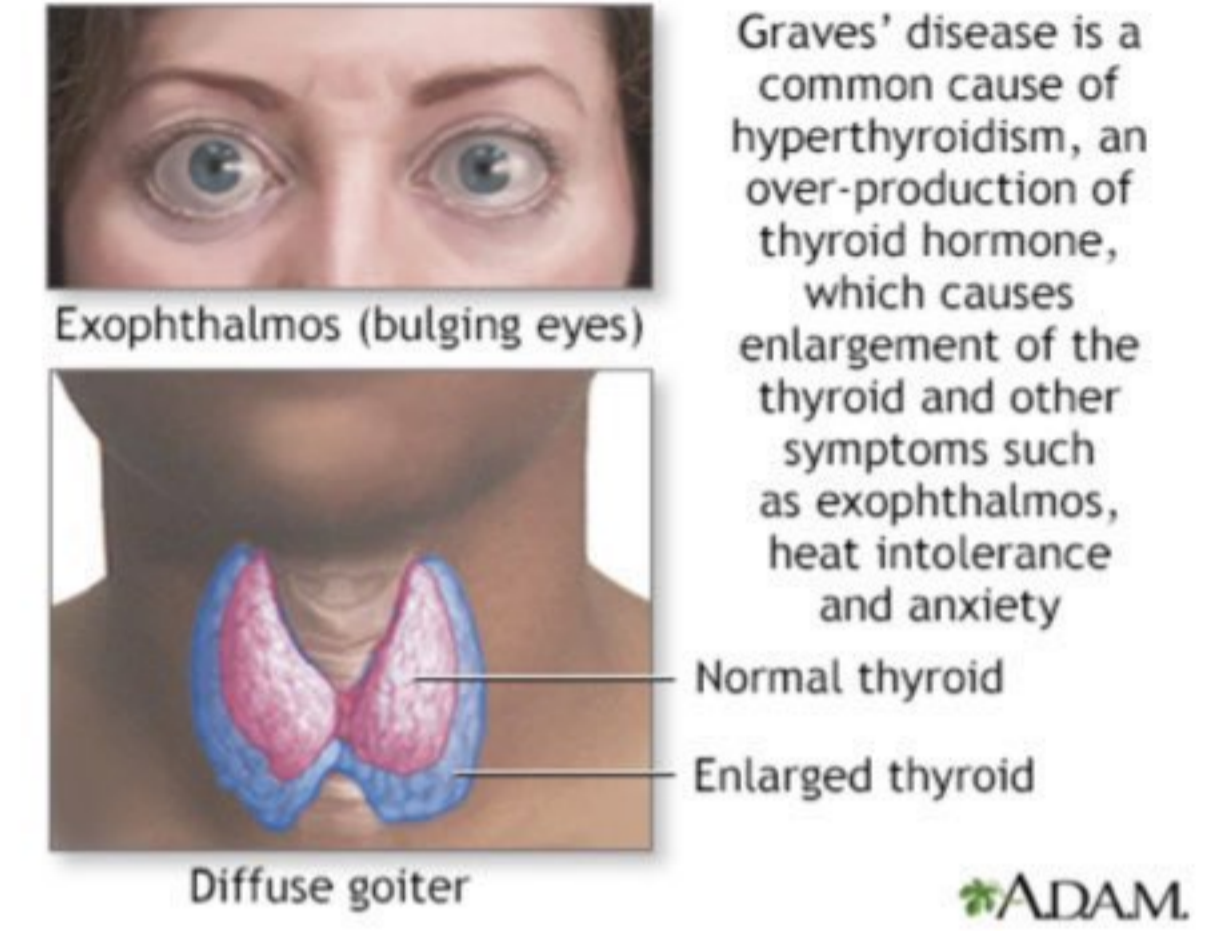
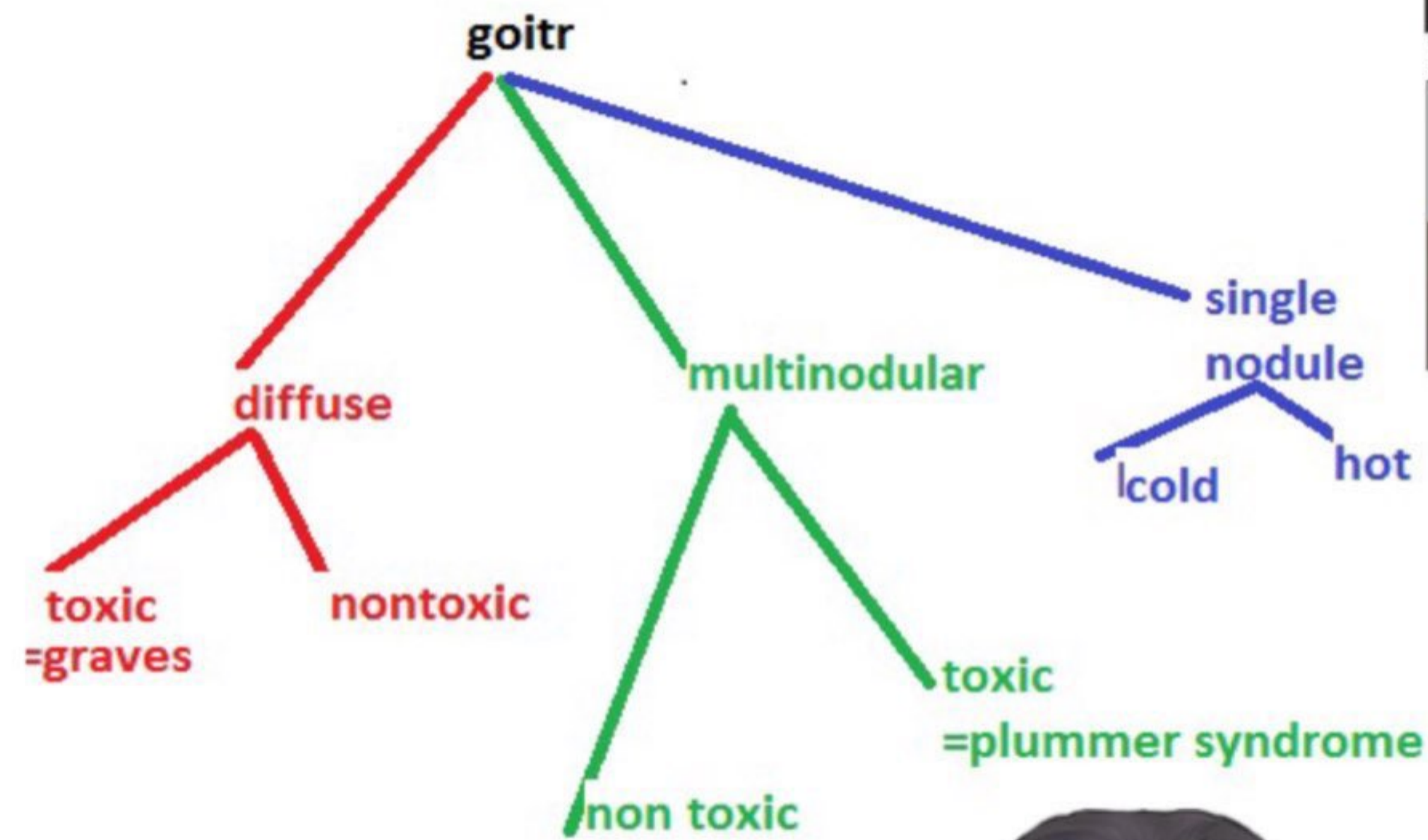
- ⇒ extensive fibrosis involving thyroid + adjacent structure simulating thyroid neoplasm ⚠
- ⇒ retroperitoneum fibrosis ⚠
- ⇒ presence of anti-thyroid antibodies in most patients suggests an autoimmune etiology.

# Goiter



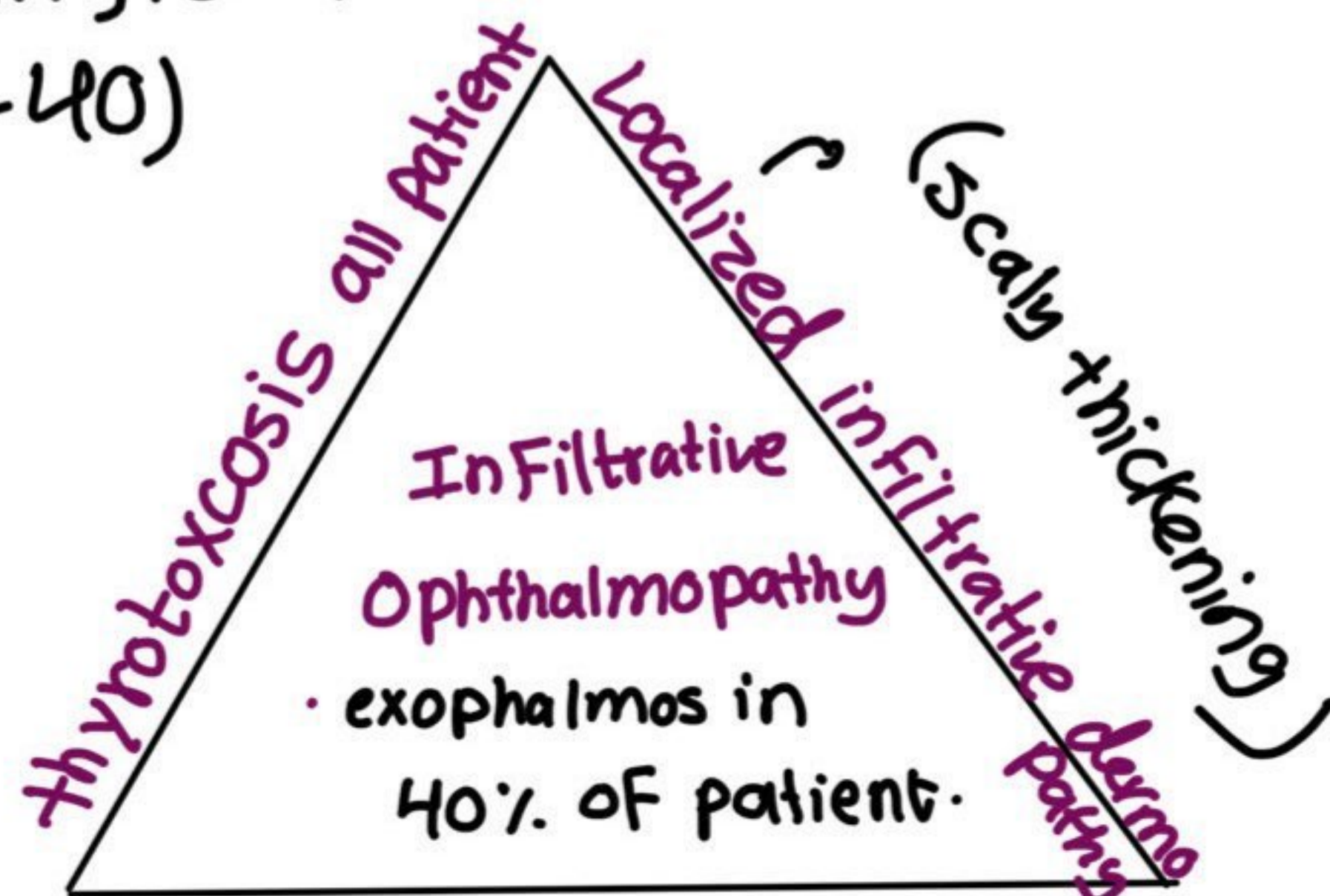
Notes

- \* Non toxic = non Function
- \* toxic = Secretory
- diffuse + toxic + hot  $\Rightarrow$  Grave's
- diffuse + nontoxic  $\Rightarrow$  Hypothyroidism
- diffuse + nontoxic + normal  $\Rightarrow$  simple Goiter

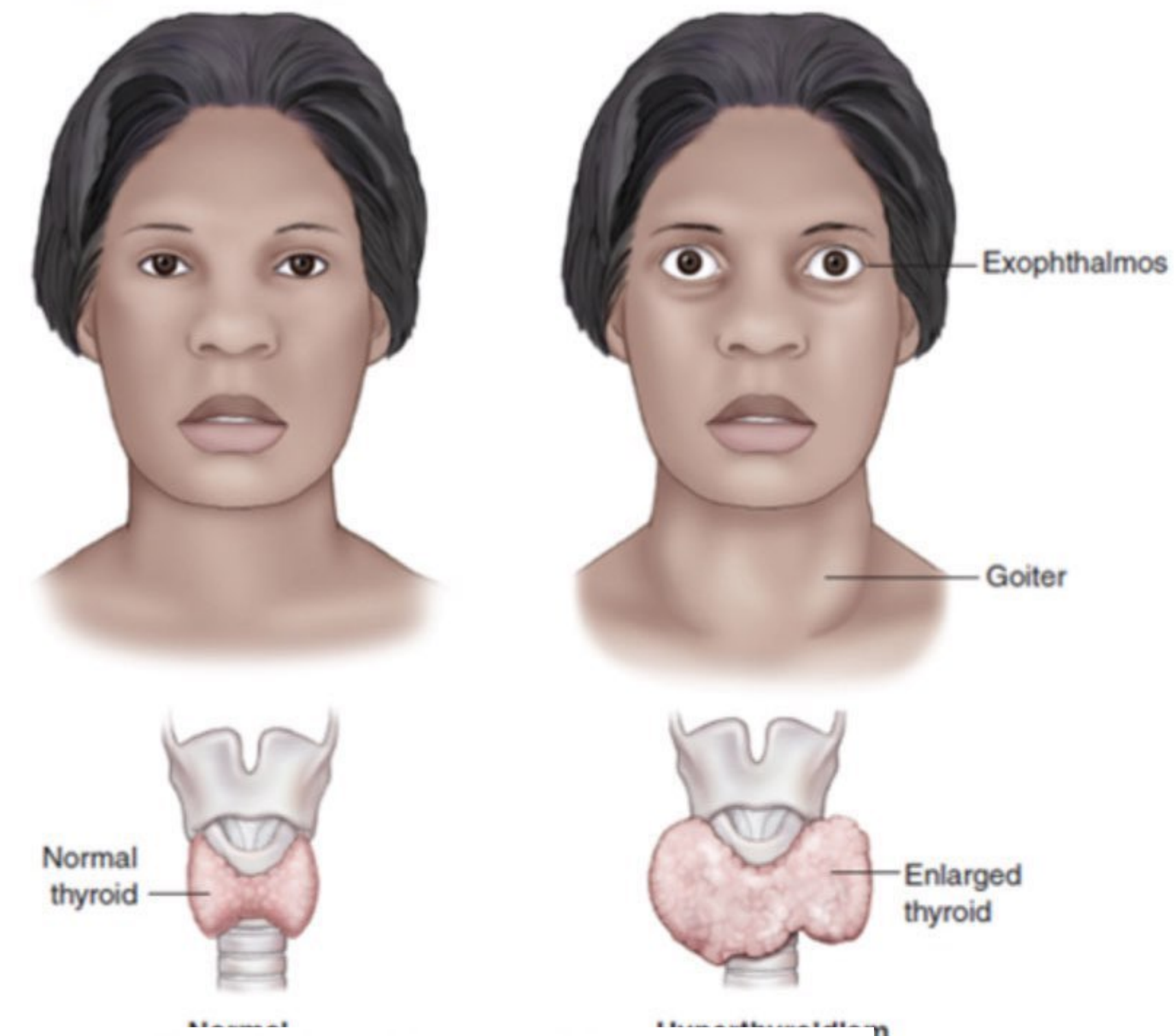


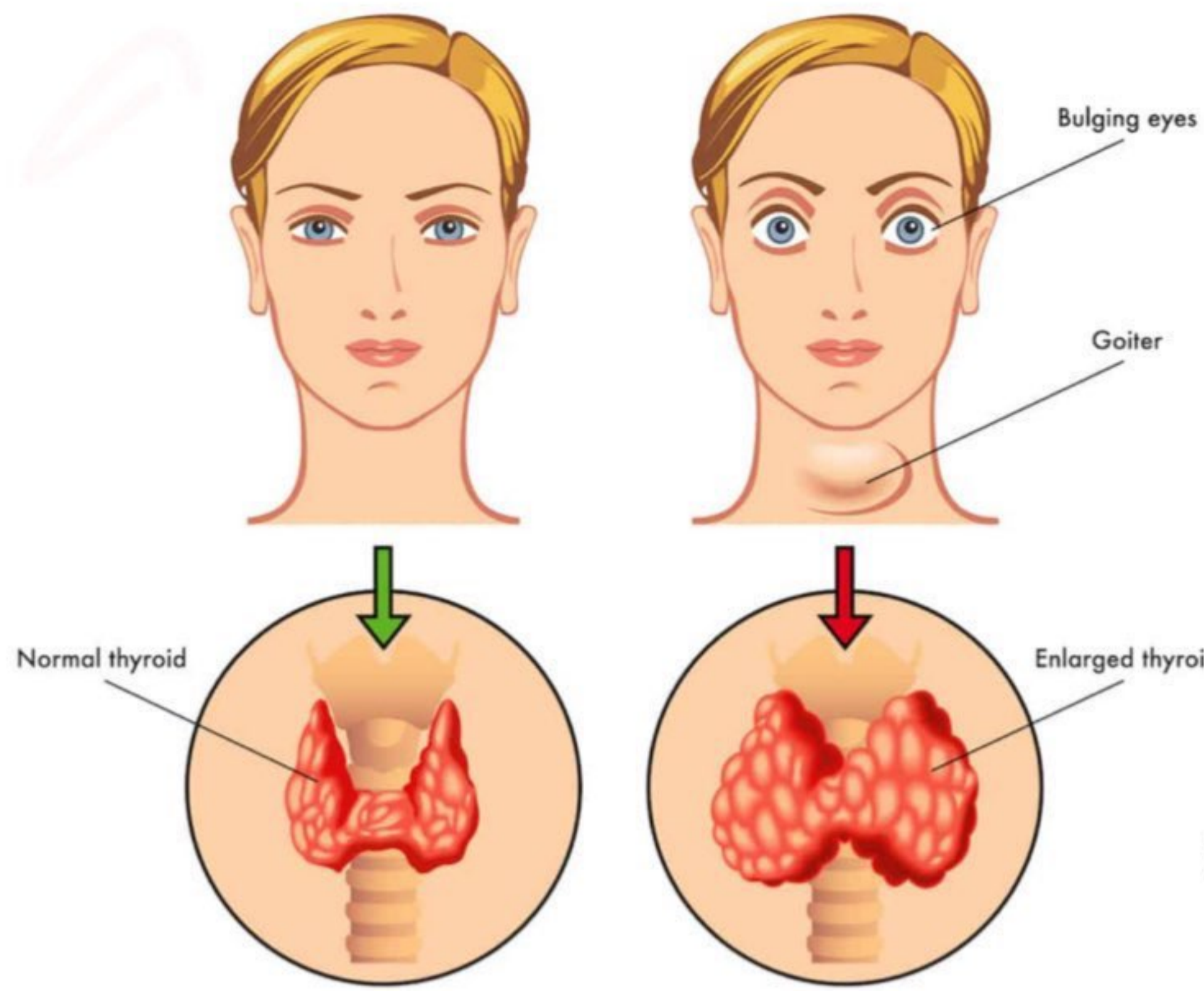
## \* Graves disease

① most common cause of endogenous Hyperthyroidism with a peak in women (20-40)



Hashimoto & Graves both AutoImmune but Hashimoto  $\Rightarrow$  its antibody destruct thyroid while Graves their antibody don't destruction they increase  $\uparrow T_3, \uparrow T_4$ .





exophthalmos is a result of increased volume of Retro-orbital connective tissue by } → interfering with function of extraocular muscles.

- ① T cell with inflammatory adema
- ② Accumulation of glycosaminoglycans
- ③ Increased numbers of adipocytes (Fatty infiltration)

⚠️ \* Even you treat Hyperthyroidism Exophthalmos may persist. + may result in Corneal injury.



Pretibial myxedema



\* localized infiltrative dermatopathy [ pretibial myxedema]



Gross: **Diffuse Symmetrical** enlargement of the thyroid gland with intact capsule,



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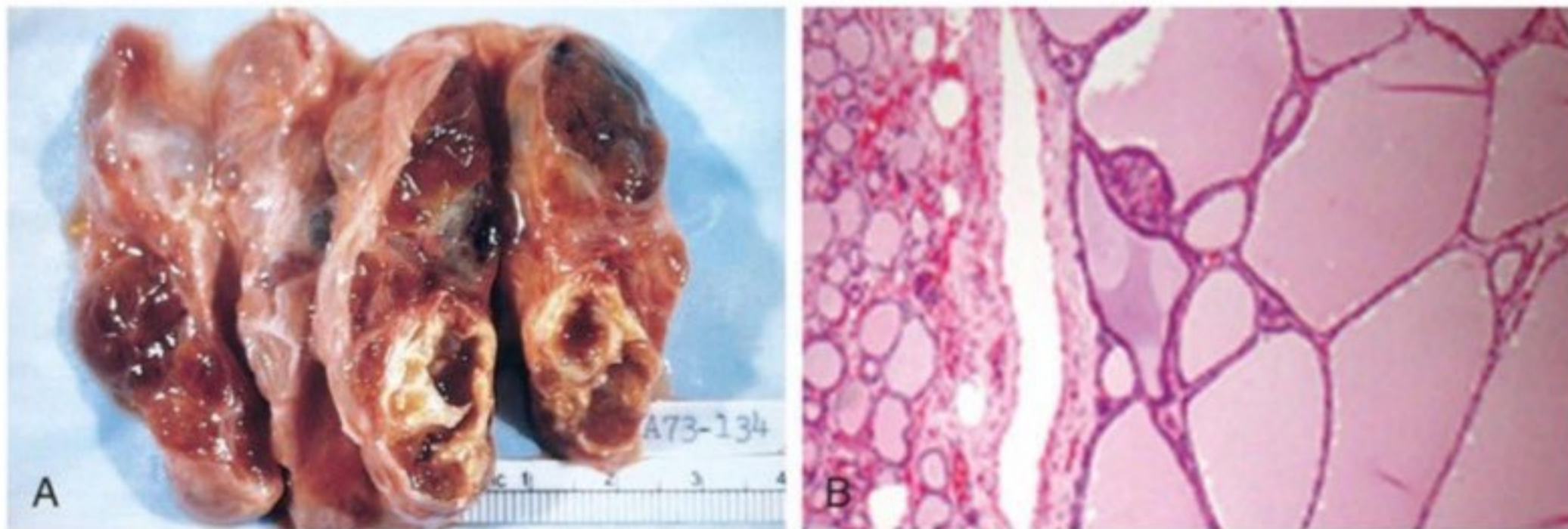
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⇒ Simple Goiter  
enlargement of gland  
Because of Iodine deficiency  
[ hormonal normal) or low decrease  
 $T_3 + T_4 = \uparrow TSH$  to compensate  
Hyperplasia of Follicular cells

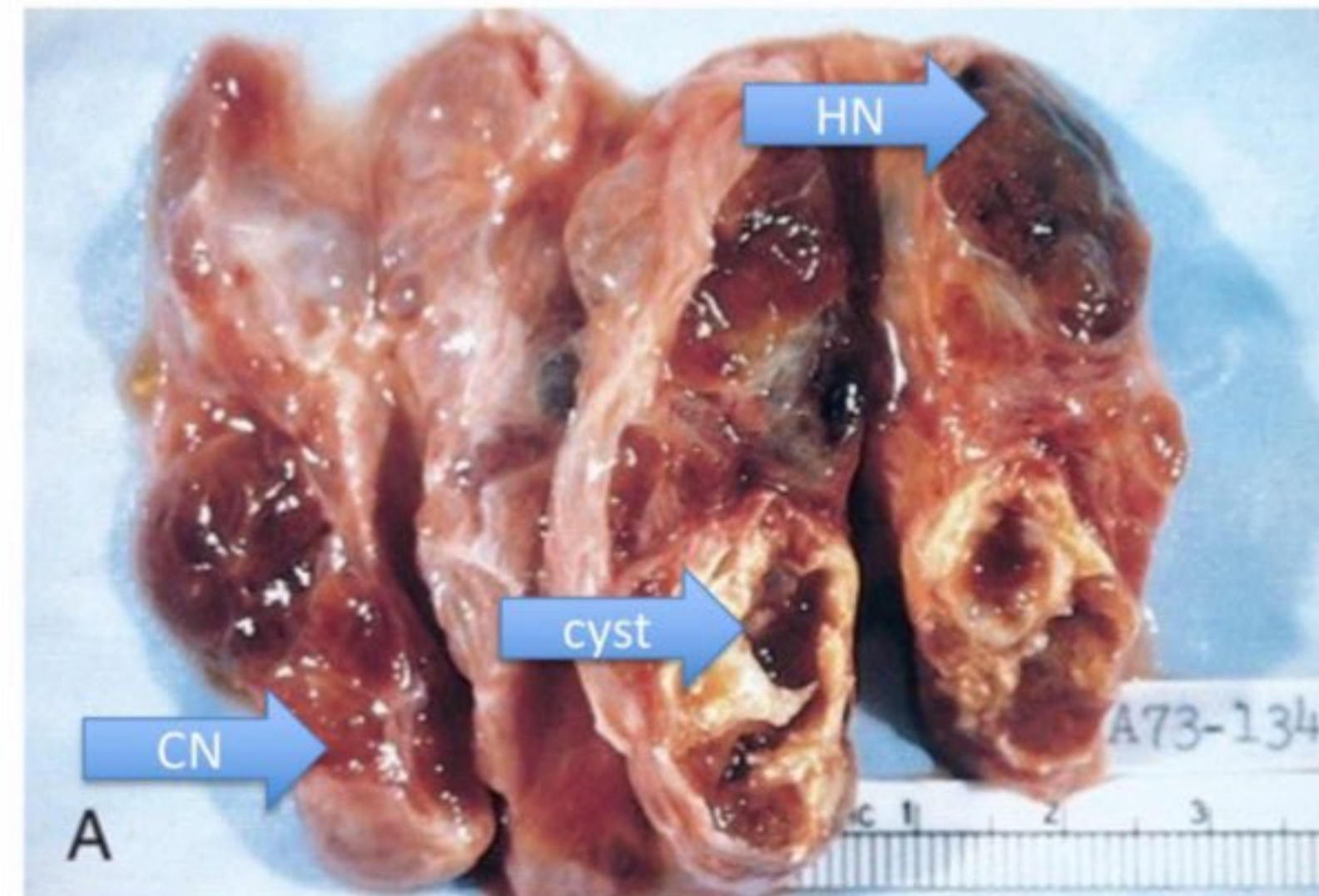
Multinodular goiter: thyroid shows several nodules, some are hemorrhagic (HN), others contain colloid (CN) and some become cystic.

## Macroscopic appearance

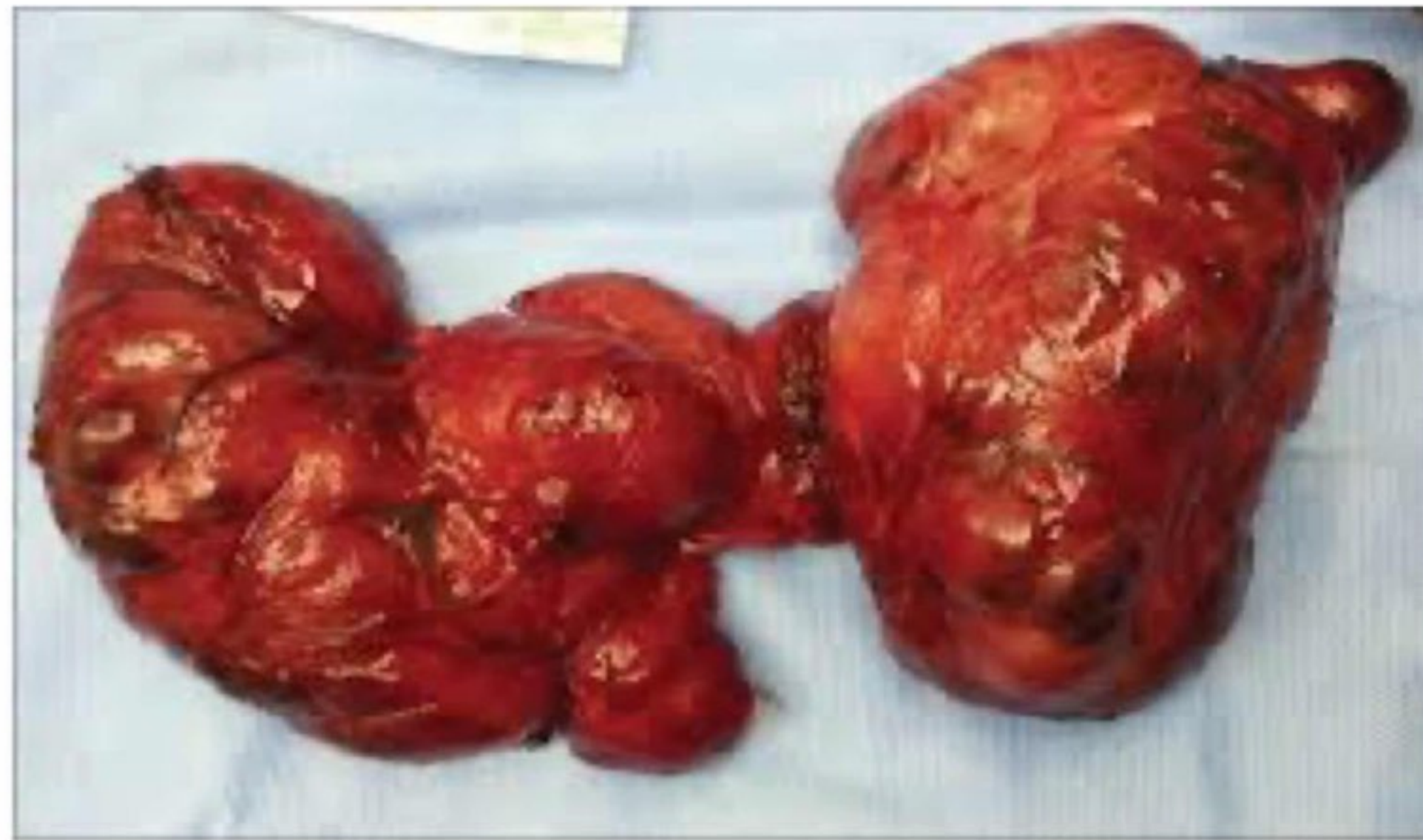
- Multinodular goiters **cause multilobulated, asymmetrically** enlarged glands. Old lesions often show fibrosis, hemorrhage, calcification



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# multinodular goiter



\* hormonally normal, silent (Not hyperthyroidism)  
\* 10% of patient may manifest with thyrotoxicosis due to development of autonomous nodules producing hormone independent of TSH stimulation.  
So, we called it [toxic multinodular goiter or **Plummer syndrome**]

## Clinical Features :

- a. The dominant features are *mass effects* of the goiter
- b. may cause airway obstruction, dysphagia, and compression of large vessels in the neck and upper thorax
- c. The incidence of malignancy in long-standing multinodular goiters is low (less than 5%) **but not zero** and concern for malignancy arises with goiters that demonstrate sudden changes in size or associated symptoms ( hoarseness)

