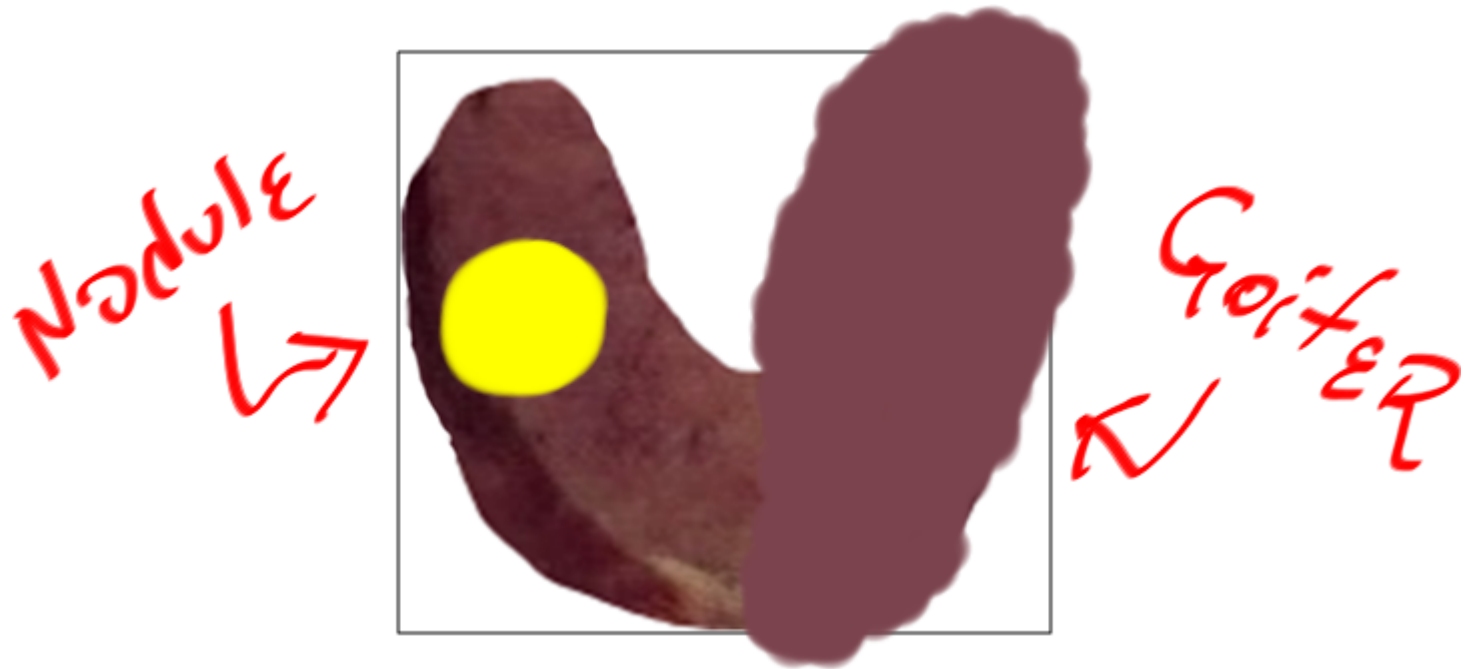


Goiter, Nodules and Tumors



Howard J. Sachs, MD

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Goiter = Diffuse Enlargement

Goiter = Euthyroid



Goiter = Diffuse Enlargement

Goiter = Euthyroid

Low T4
High TSH

High T4
Low TSH

High T4
High TSH

Hashimoto's
↓↓ Iodine

Graves
Thyroiditis

Pituitary
Adenoma



Goiter = Diffuse Enlargement

Goiter = Euthyroid

Step One:

- TSH
- Free T4

Low T4
High TSH

High T4
Low TSH

High T4
High TSH

Hashimoto's
↓↓ Iodine

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Diffuse (Simple) Nontoxic Goiter



Goiter = Diffuse Enlargement

What did I say about TSH?

Trophic Hormone: causes growth

Thyroid hormone 'dys-synthesis'

Global: Iodine Deficiency

Oxidation ($I^- \rightarrow I^2$)

Organification TGB - I^2)

MIT - Coupling - DIT



Hyperplastic



Diffuse Nontoxic Goiter



Colloidal

Key Point:
Growth is not uniform.
Different cells/follicles develop different
growth characteristics



Hyperplasia





Involution





Involution



Hyperplasia



Key Point:
Goitrous glands undergo perpetual
cycle of hyperplasia and involution





Involution



Hyperplasia

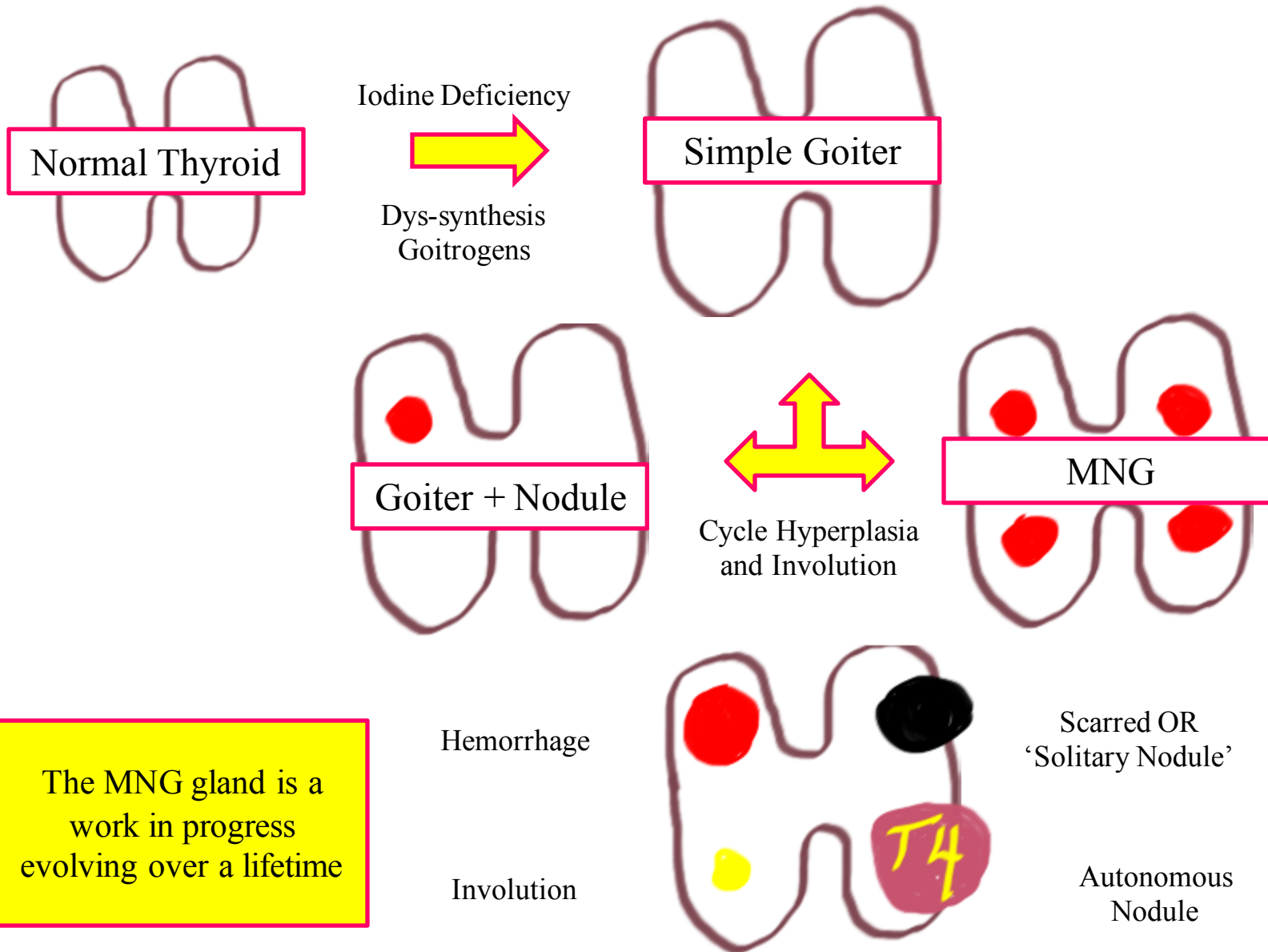


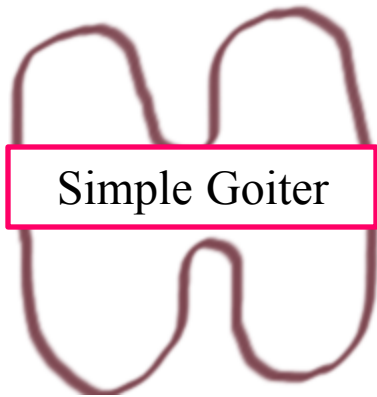
Key Point:

The goiter cycle is part of a continuum that leads MNG



So here comes the connection between Diffuse Goiter, MNG and Toxic MNG





Simple Goiter

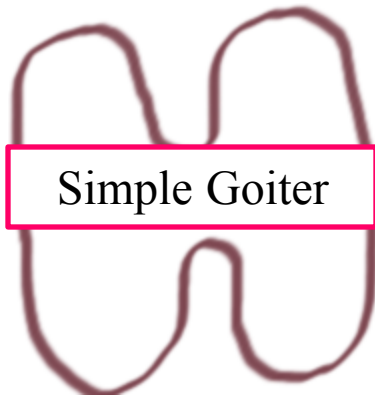
Loose Ends

Endemic (iodine deficiency)

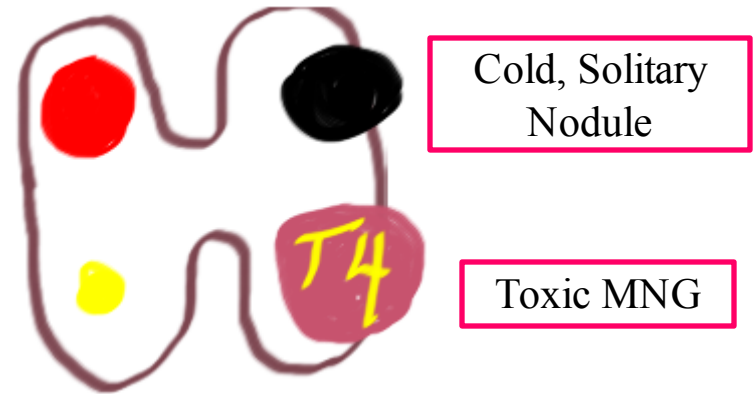
Sporadic (synthetic defect)

Compressive symptoms:

- Hoarseness
- SOB
- Dysphagia



MNG



Loose Ends

Endemic (iodine deficiency)
Sporadic (synthetic defect)

Compressive symptoms:

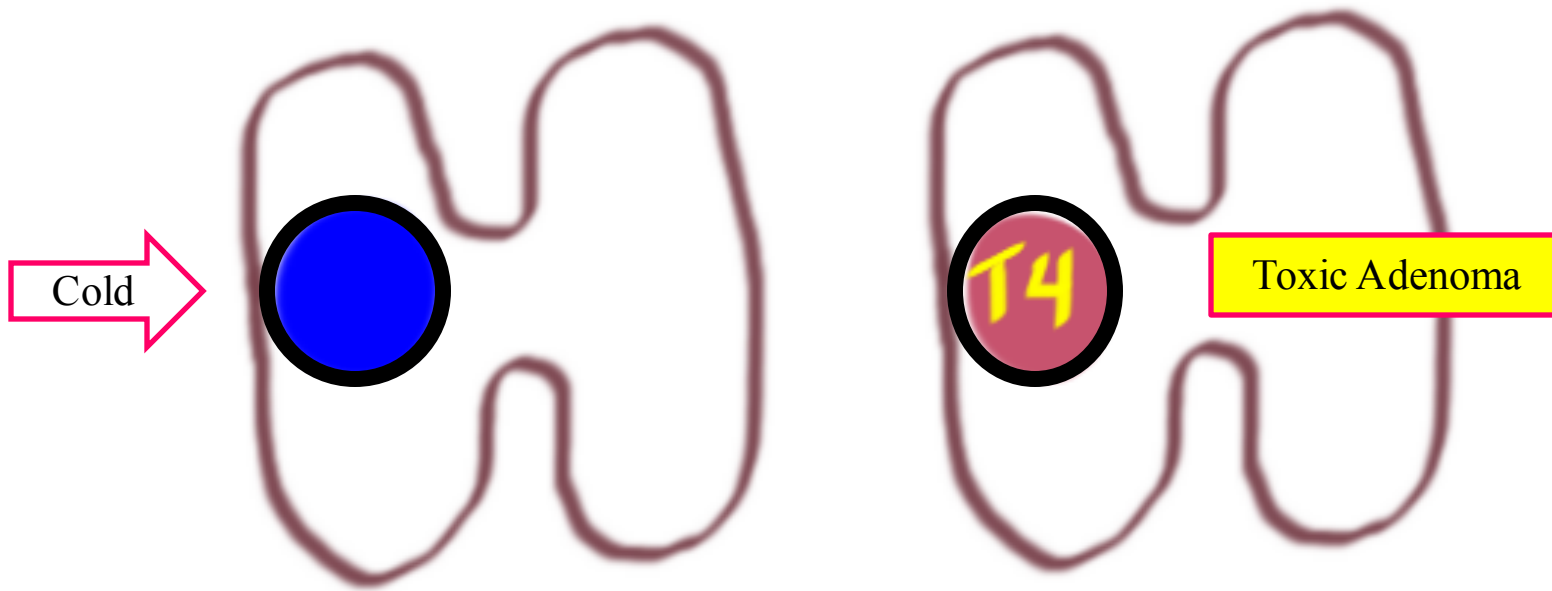
- Hoarseness
- SOB
- Dysphagia

Loose Ends

- Pathology: Scar, Ca^{+2} , Heme
- Compressive symptoms
- Hemorrhage – sudden growth
- Cold nodule – r/o malignancy
- Toxic MNG (benign) – treatment
I-131
PTU, MTZ

Free T4 normal; TSH normal or slightly elevated (unless hyperfunctioning nodule)

Follicular Adenoma



How does the adenoma compare with MNG?

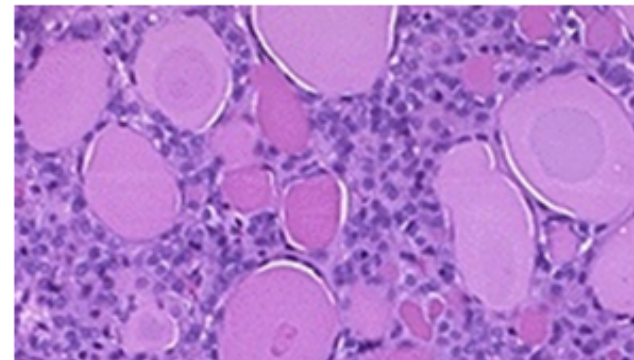
Solitary

Capsule

If cold, r/o malignancy

Follicular Adenoma [(non)toxic]

- Background
 - May or may not be functional (hot v cold on radionuclide scan)
- Pathogenesis
 - A/w **gain of function mutation** of the TSH receptor (and if hot, with autonomous function)
- Pathology
 - Discrete, **SOLITARY** mass derived from follicular epithelium
 - Solitary and **ENCAPSULATED**. **The capsule distinguishes from follicular carcinoma.**
 - Hurthle cells may be present



Well differentiated normal appearing follicles

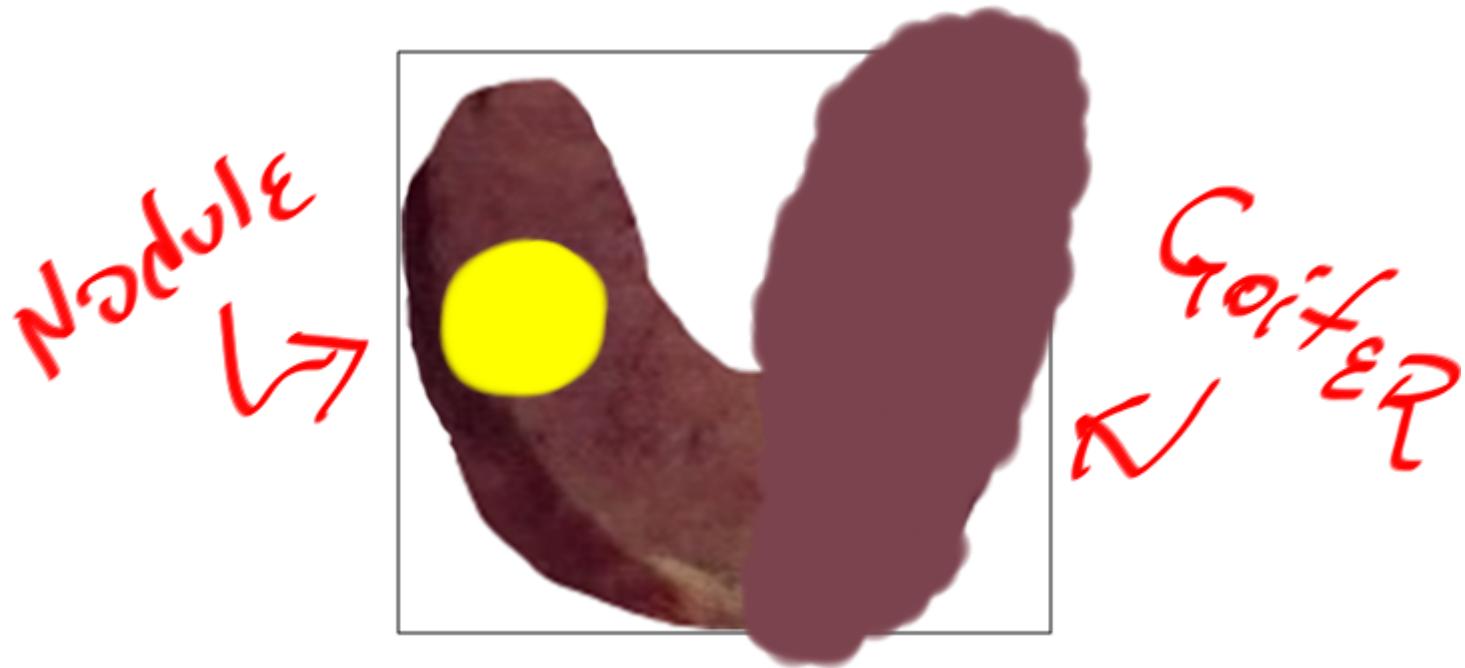
Follicular Adenoma [(non)toxic]

- Clinical
 - Solitary nodule
 - Functioning or not (based on free T4)
 - S/s hyperthyroidism
- Diagnostics
 - Lab: If toxic, low TSH/hi T4
 - Imaging (only if functioning): radionuclide hot if toxic
 - FNA (if euthyroid): cytology - indeterminate
- Treatment
 - Surgery (cold), Radiation (hot)



FNA does not tell us about
Capsular Invasion

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