Hypertsm	
symptoms	-Thyrotoxicosis -inc appetite & weight loss! -hypercalcemia -hypocholesterolemia -hyperglycemia
Thyrotoxicosis	-is inc blood free thyroids **Primary** -commonest -by hypertsm -dec TSH -etiology (C-R): graves, multinodular goiter, toxic thyroid tumor, iodine Induced, early hashimoto **Secondary** -inc TSH **Nonthyroidal** -de quervain (granulomatous thyroiditis “painful”) -lymphocytic thyroiditis (“painless”) -struma ovarii (ovary thyroid teratoma) -factitious (exogenous - meds)

Graves	Is	Ai
	Pathog	Ig mimics TSH (keeping the gland stimulated 24/7)
	Ig types	**Thyroid stimulating Ig** -IgG & LATS (long acting thyroid stimulation) -using ACE **Thyroid growth stimulating Ig** -acts on TSH receptors on thyroid and promotes growth **TSH inh Ig** -prevents TSH from binding to thyroid -causes hypotsm (patient might have multiple types of the above, explaining the episodes of hypotsm & hypertsm)
	Sympt	-audible bruit (cuz of inc in blood flow to the gland) -exophthalmos (cuz of T-cells activation) -pretibial edema -audible bruit
	Diagnosis	-high thyroids -low TSH -Ai Ig detecting -hot RUS
	L/M	-scalloped colloid (the colloid “follicle” seems like a beach shell, where its sides are white, due to inc of colloid intake by follicular cells, cuz they’re super active) -hyperplastic columnar follicular cells -lymphocytosis

Hypotsm	
etiology	**Primary** -Ai (hashimoto) -iodine def -post-ablative (post-treatment - thyroidectomy…) -cong. (mostly: dysgenesis which is mutation in PAX, FOX, TSH-receptors) **Secondary** -mostly TSH def related than TRH (PG failures > HT failures)
If in	**Infants: Cretinism** -caused by: thyroid agenesis (malformation), dyshormonogenetic (unable to produce hormones due to enz problems) **Adults: Myxedema** -caused by: hishimoto -non-pitting edemas: Glycosaminoglycans, hyaluronic -deeper voice & enlarged tongue -femals amenorrhea (menstrual cycle disturbance)
Hashimoto	**Is:** AI infl destructing thyroid causing thyroid failure **Ag targeted:** thyroglobulin & peroxidase **Serum:** high TSH **Risk:** genetic mutations (especially familial) **Starter:** as hypertsm (follicles content release) then hypotsm **Nodules:** might be present **L/M: -**replacement of follicular cells with mononuclears & fibrosis -Infiltration of huge number of lymphocytes & plasma cells, And the presence of germinal center (mimic a lymph node) -atrophic follicles (shrunken), lined by hurthle/oxyphil cells (eosinophilic, granular cells with lots of mitochondria) **Diagnosis:** TSH, thyroids & Ai Ig detection

Goiter	
Info	-is the most common symptom of thyroid disorders -if present with normal thyroid called “euthyroidism”
Investigation (in order)	-TSH & T4 measurement -T3 measurement -RUS (if cold use fine needle aspiration)

Thyroiditis	
Types	**Acute & painful** -inf -de Quervain **Little infl & thyroid dysfun** -lymphocytic -fibrotic (Riedel thyroiditis)
Subacute granuloma thyroiditis	**L/M:** giant cells **Etiology:** viral inf (ass with upper resp inf) **Symptoms:** painful & hypertsm (cuz follicles expel their contents) **Feature:** most common cause of thyroid pain, self-limiting, permenant hypotsm is uncommon.
Riedel thyroiditis	is chronic infl with exagg fibrosis that may extend to resp system & cause breathing problems