

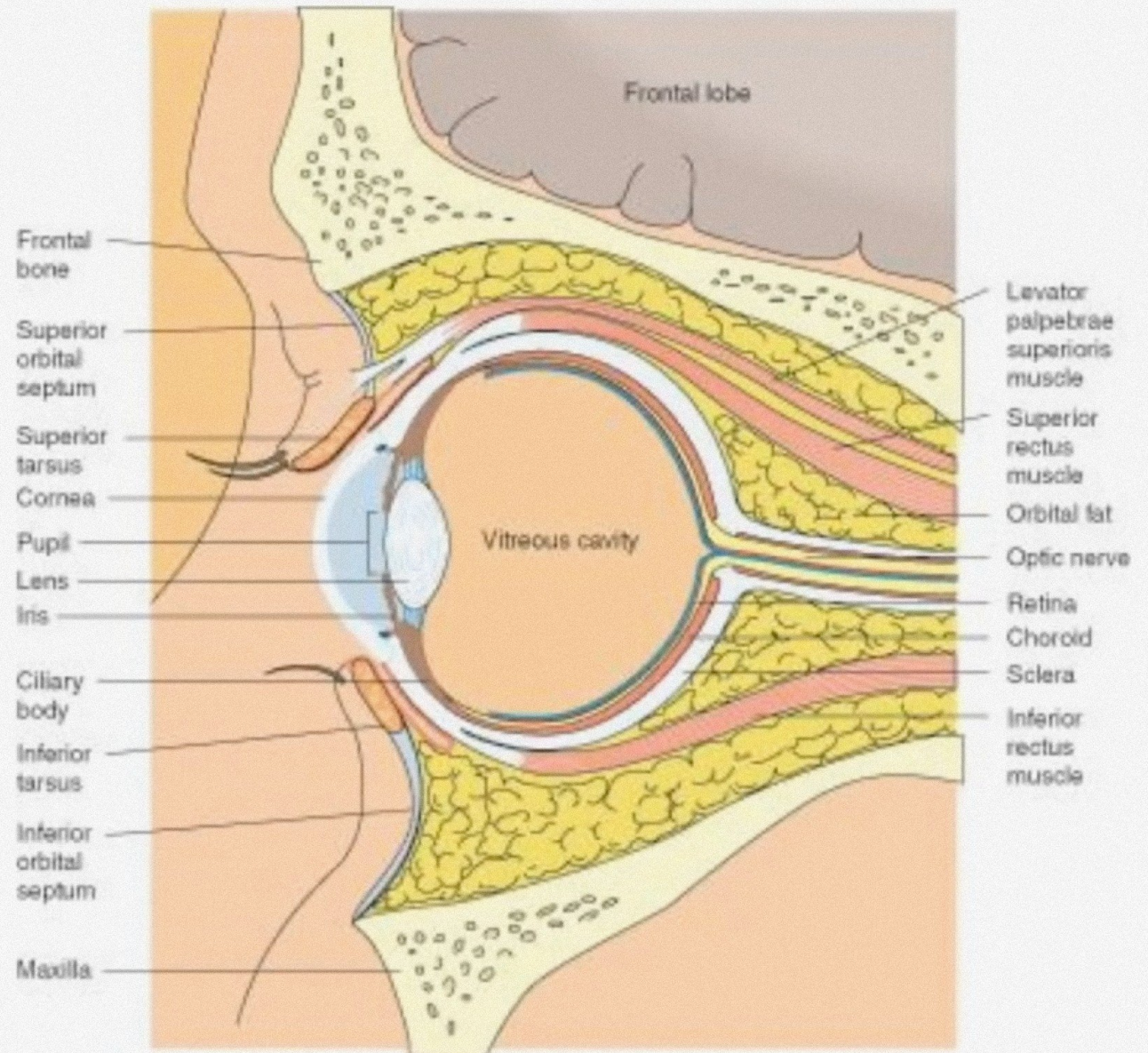
# Thyroid Eye Disease (TED)

aka Graves' Ophthalmopathy (GO), Thyroid Associated Orbitopathy (TAO)





# Anatomy Review





# Pathogenesis

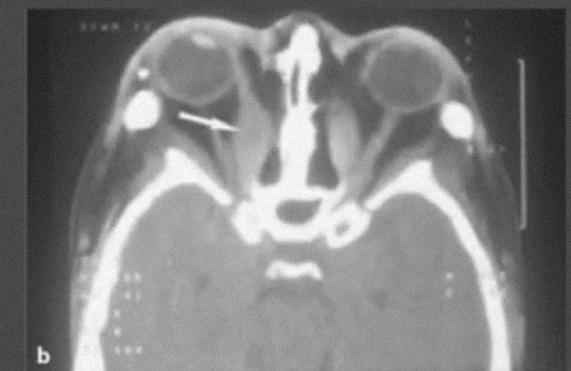
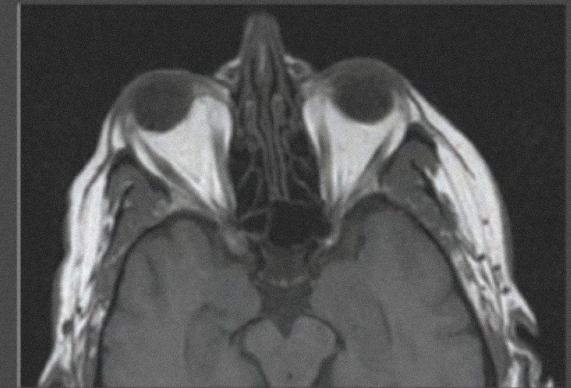
- Antibodies directed against receptors – most often the thyroid stimulating hormone receptor (TSHR) – present in the thyroid cells may also target TSH receptors on the surface of the cells behind the eyes, namely fibroblasts and adipocytes
    - Thyroid stimulating immunoglobulins (TSI)
    - Thyroid stimulating hormone receptor binding inhibitory immunoglobulins (TBII)
  - These TSH receptor antibodies and cytokines from activated T cells lead to fibroblast activation and secretion of hydrophilic glycosaminoglycans, which accumulate in the extraocular muscles and retroocular tissue along with excess fluid, leading to swelling and increased pressure
-



# Pathogenesis

- Antibodies directed against receptors – most often the thyroid stimulating hormone receptor (TSHR) – present in the thyroid cells may also target TSH receptors on the surface of the cells behind the eyes, namely fibroblasts and adipocytes
  - Thyroid stimulating immunoglobulins (TSI)
  - Thyroid stimulating hormone receptor binding inhibitory immunoglobulins (TBII)
- These TSH receptor antibodies and cytokines from activated T cells lead to fibroblast activation and secretion of hydrophilic glycosaminoglycans, which accumulate in the extraocular muscles and retroocular tissue along with excess fluid, leading to swelling and increased pressure

Normal extraocular muscles



Enlarged extraocular muscles (TED)





## Symptoms

- **Proptosis** (exophthalmos)
- **Lid retraction**
- **Periorbital edema**
- **Tearing** (often made worse by cold, wind, or bright light exposure)
- A gritty or foreign object sensation in the eyes
- Blurring of vision
- Eye or retroocular discomfort or pain
- Restriction of eye movements
- Diplopia (double vision)
- Color vision desaturation
- Occasionally loss of vision



# Diagnosis

Typically diagnosed clinically by the combination of:

- Characteristic ocular abnormalities, and
- Hyperthyroidism labs (commonly low TSH; high free T4 & T3; TSH receptor antibodies)

But...important to rule-out nonspecific signs of thyroid hormone excess, such as stare and lid lag (without associated proptosis)

Imaging: can help with differential diagnosis but often used to assess risk of optic nerve compression by enlarging extraocular muscles and retroocular tissues





## Assessment of Disease Activity/Severity

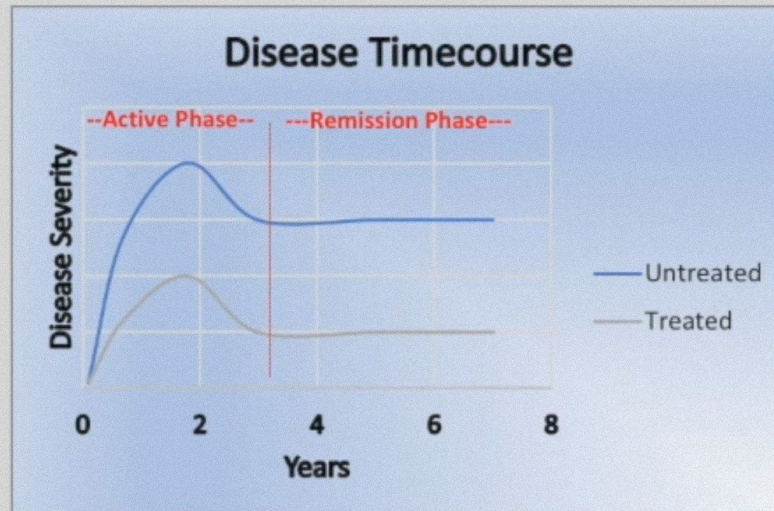
- Clinical Activity Score (CAS): **Scores  $\geq 3/7$**  (first time patient assessment) or  **$\geq 4/10$**  (for subsequent assessments) correlate with active disease and likelihood of response to corticosteroids (add 1 point for each finding)
- Symptoms
  - Pain or pressure in a periorbital or retroorbital distribution
  - Pain with upward, downward, or lateral eye movement
- Signs
  - Swelling of the eyelids
  - Redness of the eyelids
  - Conjunctival injection (redness of the conjunctiva)
  - Chemosis (edema of the conjunctiva)
  - Swelling of the caruncle (fleshy body at medial angle of eye)

-----end here for first assessment-----
- Changes (included as subsequent assessments)
  - Increase in measured proptosis  $\geq 2$  mm over 1-3 months
  - Decrease in eye movement limit of  $> 5^\circ$  over 1-3 months
  - Decrease in visual acuity  $\geq 1$  Snellen chart lines over 1-3 months



# Treatment

- Can be divided into two phases: treatment of the **active** (inflammatory) eye disease phase and treatment of the **stable** (post-inflammatory) remission phase





# Active Phase Treatment

## Hyperthyroidism reversal

- Thionamides (methimazole or propylthiouracil)
- Surgery (total thyroidectomy = gold standard)
- Radioactive iodine (can cause development or worsening of TED) with concurrent glucocorticoids for those with risk factors (smoking or high T3)
- Does not directly improve TED other than to help correct for lid lag and stare
- Hypothyroidism can cause fluid retention and worsen disease course, so thyroid function monitoring is critical

## Cessation of smoking

- Reduces the severity, duration of activity, degree of scarring, and risk of optic nerve involvement & increases likelihood of response to anti-inflammatory therapy

## Local measures to reduce ocular surface irritation

- Artificial tears**
- Sunglasses
- Sleeping with elevated head position

## Treatment of inflammation and swelling in periorbital tissues

- Corticosteroids
  - Biologics or external orbital radiation if
- Orbital decompression surgery

## Treatment of double vision

- Occlusion therapy
- Prisms



# Remission Phase Treatment

Orbital  
Decompression  
(proptosis)

Strabismus  
Surgery (double  
vision)

Eyelid Surgery (lid  
retraction,  
periorbital edema)





# Conclusion

Describe

Describe how the orbital anatomy is relevant to the development of TED and the “bulging eye”

Discover

Discover the pathogenesis of TED

Discover

Discover the symptomatology of TED

Discover

Discover the clinical approach to evaluating and treating for TED