Thyroid Eye Disease

Thyroid eye disease, or thyroid orbitopathy, is an autoimmune condition in which your body's immune system is producing factors that cause enlargement of the tissues in the eye socket (also called the orbit), including the muscles that move the eye. This can result in bulging of the eyes, retraction of the eyelids (making them more wide open), double vision, decreased vision, and eye irritation. This is often associated with abnormalities in thyroid gland function (either too much [Graves' disease]) or too little [Hashimoto’s thyroiditis]).

The connection between the eyes and the thyroid is through the immune system. If the immune system attacks the thyroid gland, it may become overactive. Signs of an overactive thyroid include tremors, shakes, weight loss, rapid heartbeat or palpitations, nervousness, and sensitivity to heat. Less commonly the attack on the thyroid gland leads to low thyroid production or even normal thyroid levels. We may see antibodies in your blood that can be identified as attacking thyroid tissue.

The eye findings of thyroid eye disease may be independent of treatment of your thyroid abnormalities and may not go away or get better even if the thyroid is now “controlled.” These symptoms may also be present even when your thyroid has no apparent problems.

How does the eye become involved in thyroid eye disease?

How or why the immune system attacks the orbital tissues, particularly the eye muscles, is not fully understood. The result is enlargement of the muscles and the fat that normally cushions the eye and other important structures in the orbit. As the eye socket tissues get larger, 3 things can happen. The eyeball gets pushed forward, and may not go away or get better even if the thyroid is now “controlled,” the muscles themselves become stiff (the eye may not move normally), or the muscles may press on the optic nerve.

Figure 1. Model of the orbit. The outer bones have been removed to show the soft tissues in the orbit. Legend: SR = superior rectus muscle, MR = medial rectus muscle, LR = lateral rectus muscle, IR = inferior rectus muscle.

The eye muscles

There are 6 muscles that move the eye around. The most commonly involved eye muscles in order of most to least often involved include the inferior rectus (IR) responsible for the eye looking down, the medial rectus (MR) responsible for the eye looking in, the superior rectus (SR) responsible for the eye looking up, and the lateral rectus (LR) responsible for the eye looking out. These muscles originate behind the eye at the very back of the eye socket (see figure 1) and attach near the front part of the eyeball. The muscles cannot be seen on the surface as they are covered by a thin layer of tissue (the conjunctiva) but may become visible as the blood vessels over their front portion become very prominent.

In thyroid eye disease, the immune system stimulates support cells within the eye socket tissues, causing the eye socket fat and the eye muscles to enlarge. With eye muscle enlargement, the eyeball is pushed forward and the eyes appear more prominent and wide open. In addition, the muscles become stiff and the upper eyelid tends to pull up (retract) away from the colored portion of the eye, making the white part of the eye more visible (see figure 3). The eyes may become red due to difficulty closing as well as increased prominence of the blood vessels. The inferior rectus muscle (located beneath the eye) tends to be more often affected than others. When it becomes stiff, the eye cannot move up normally. This often results in double vision with one image seen on top of the other. If other muscles are involved, two images may be seen side by side or diagonally.
Figure 2. CT scan of the orbits showing the enlarged eye muscles in thyroid eye disease.

The optic nerve

The optic nerve transmits information from the eye to the brain. Damage to the optic nerve results in decreased vision. If the muscles get large enough, they may press on the optic nerve causing damage to the nerve. If the optic nerve is compressed, the patient may experience blurred, dark or dim vision. Fortunately, this only occurs in about 5% of the patients with thyroid eye disease and may be reversible if the pressure on the optic nerve is relieved in a timely fashion.

It is important for your physician to sort out whether or not there is any evidence of abnormal optic nerve function. This is detected by carefully checking vision, color vision, pupil reaction, visual fields (side vision test), the appearance of the optic nerve in the back of the eye, and possibly a picture of the optic nerve, which measures its thickness (OCT).

Symptoms of Thyroid Eye Disease:

Blurred or distorted vision
Dry eyes: eye surface discomfort, feeling like there is something in your eye (more air hits your eye causing dryness of the surface, also called exposure)
Redness
Eyelid retraction (the eyelid gets pulled away from the colored part of the eye, exposing more of the white part)
Eyelid or eye swelling
Double vision due to eye misalignment
Protrusion, or bulging of the eye
Pressure or mild pain behind the eye
Dimming or darkening of vision

Patients with thyroid eye disease often notice blurred or double vision. As the eye is pushed forward, it frequently results in irritation, redness, tearing and a gritty sensation. Patients may be aware of fullness within the eye socket and sometimes a mild irritation, light sensitivity, or ache behind the eye. The double vision is most frequently one image on top of the other or offset although it may be side to side. Double vision will often change depending on which direction you look, seeming worse when looking up and to the side. Orbital fullness, ache, or double vision is commonly worse in the morning or after a period of laying flat due to increased congestion within the eye socket.

Upper lid elevation, particularly when looking down, is very characteristic of thyroid eye disease. The eyes frequently bulge forward and the blood vessels on the inner and outer edges of the eye surface tend to become dilated. The lids often don't close completely at night. The pupils may not react normally and the eyes may be limited in their movement. The stiffness of the eye socket may cause the pressure inside the eye to be high, particularly while looking in one direction.

Sometimes patients will only be aware of symptoms related to thyroid overaction (nervousness, tremors, rapid or irregular heart beat, increased sweating and intolerance to heat, weight loss, and diarrhea) or underaction (fatigue, weight gain, constipation, thickening of the skin). These symptoms may precede eye symptoms by months or even years.
Figure 3. The appearance of the eyes and eyelids in a patient with thyroid eye disease. Note the eyelid retraction (upper lid elevation) and protrusion of the eyes (proptosis), eyelid swelling, and redness of the eyes.

**Prognosis:**
Thyroid eye disease, like other autoimmune diseases, often comes and goes on its own. There is frequently only one inflammatory episode that comes on over a short period of time and lasts for about 1 to 2 years. This is called the “active phase”. The effects on your eyes may persist for years or even permanently, especially if untreated. This is referred to as the “inactive phase”. Smoking will exacerbate the severity of eye involvement in thyroid eye disease and thus, quitting tobacco use is recommended. Even when the inflammation resolves, the eyes usually do not go back to what they looked like beforehand. There may be some reduction of the prominence of the eye and the eyelid may come down a little, but patients may still require treatment to reach a comfortable state. If eye movements are significantly involved and there is double vision, this will often not return to normal. Fortunately, permanent damage to vision from optic nerve involvement is rare (5%) and often reversible.

**Treatment:**
Treatment is aimed at improving the symptoms and severity of orbital involvement to prevent long-term problems. In all severities of eye involvement, avoiding tobacco smoking and controlling the thyroid function with medications or other methods is recommended to decrease severity of eye involvement. Treatment is most effective early in the active phase (see figure 4).

**Mild thyroid eye disease**
In patients with mild involvement, irritation and foreign body sensation from surface dryness may improve with artificial tears (preferably preservative free if using more than 4 times daily) and the use of lubricating ophthalmic ointment at night. If the eyelids are not closing completely, they may be taped closed at night. Humidifiers and moisture chamber goggles/glasses may also be used to reduce evaporation of tears and improve dry eye symptoms. Your doctor may place small silicone plugs into the tear drainage holes in your eyelids to keep tears on the surface of your eye. Non-steroidal anti-inflammatory medications (such as diclofenac, ibuprofen, naproxen, etc.) may be used to manage pain or discomfort from orbital inflammation. Depending on where you live, your doctor may recommend selenium supplementation as well.

**Moderate to severe thyroid eye disease**
With increased prominence of the eye and opening of the eyelids, symptoms of exposure of the eye surface may be too severe to be treated with lubricating drops and ointment. Double vision from misalignment of the eyes may lead to significant decrease in quality of life. Treatment of these signs and symptoms will depend on whether the patient is in the active or inactive phase. Surgery for treating thyroid eye disease is usually performed in a specific order, preferably in the inactive phase.

Double vision may be addressed with wearing temporary prism glasses, which shift the images to allow single vision. If the images are too far apart to address with prisms, covering one eye may be necessary to relieve double vision. Either eye may be covered.

Steroid treatment is used to treat the inflammation in the active phase in moderate to severe eye involvement from thyroid eye disease. Reasons to get steroid treatment include severe double vision, severe exposure of the eye surface from prominence of the eye with inability to close the eye, or if the optic nerve becomes involved. The most commonly used method is high dose intravenous (IV) steroid infused once a week for 12 weeks in a row. During this treatment, side effects need to be monitored and treated.

An alternative treatment to steroids includes low dose radiation (X-ray therapy) to the orbit. This treatment has been shown in some studies to be effective in the active phase, but some doctors may prefer other treatments.

Medications that suppress the immune system other than steroids may also be used, but these have not been definitely proven to work and are given on a case-by-case basis to people who are unable to tolerate steroid treatment.

If medical treatments fail to improve symptoms, surgery may be necessary in the active phase. Eyelid surgery may be required to help partially close the lids to treat exposure of the eye surface. Orbital decompression surgery during the active phase may also be required to create more room in the eye socket for the expanding tissues, but this is usually reserved for more severe eye involvement with optic nerve compression causing damage to the vision. This is usually done by thinning or removing one or more of the bony walls of the orbit. Since the optic nerve is usually compressed at the very back of the orbit, removing the backmost part of the inner wall of the orbit is most critical to relieving the pressure on the optic nerve. This may be done directly through the soft tissues or skin around the eye, or through the nose and sinuses. To further reduce the eye bulge, the bottom, outer wall, or even the top of the orbit may be removed or thinned. Orbital fat may
also be removed to allow the eye to sit further back in the eye socket. One of the side effects of surgical decompression is that it may affect eye movements, potentially producing or worsening double vision.

**Surgical treatment in the inactive phase**

If orbital decompression surgery is necessary, it is usually done first, before addressing double vision or the eyelids. Reasons to do orbital decompression surgery in the inactive phase include significant proptosis (prominence of the eye) causing severe discomfort from exposure of the eye surface or episodes of the eyelids getting stuck behind the eye (known as globe subluxation).

When double vision cannot be corrected with prisms, eye muscle surgery may be necessary. In most cases, physicians measure the alignment of the eyes and choose to wait until the double vision/alignment is stable for a few months before performing eye muscles surgery. If we operate on a patient who is undergoing progressive change, we may correct them now but have things change within the next few months. Occasionally, multiple muscle operations are necessary. It is sometimes not possible to completely remove double vision, but the goal is to remove double vision looking straight ahead and in reading position, as these are the most common positions used.

After the double vision is addressed, eyelid surgery may be performed. Retraction of the upper eyelid can be fixed by releasing the muscles that raise the upper eyelid to allow the upper lid to sit at a more normal position. The lower eyelid can be fixed by releasing the tissues that pull the eyelid down and by placing a spacer material to prop the eyelid up in a higher position (sources may include acellular material derived from a human or porcine source, a piece of the ear cartilage, or a piece of tissue from the roof of the mouth (hard palate)). Often eyelid closure can be improved by permanently sewing the outer corner of the eyelids shut (tarsorrhaphy).

**Frequently asked questions**

*The doctors tell me they fixed my thyroid and that it is now normal. Why are my eyes acting up?*

In Graves’ disease the thyroid gland is stimulated by the immune system to release too much hormone. This excess hormone results in nervousness, palpitations, weight loss, diarrhea, tremors, and a feeling of being hot all the time. Treatment is aimed at limiting the thyroid gland’s ability to make thyroid hormone. This may be done with medications, surgery, or radioactive iodine, usually resulting in normalization of thyroid production (occasionally requiring thyroid replacement). This does not, however, affect the primary autoimmune process and the immune system may continue to target other tissues in the body. Orbital symptoms may even worsen following treatment with radioactive iodine. The eye and orbit changes must be treated separately as outlined above.

*The steroids made my eyes much more comfortable. Can’t I just continue taking them?*

Steroid therapy may be effective in reducing the inflammatory phase of thyroid eye disease and partially shrinking the swelling in the eye socket. Steroid side effects are very common with long term continued treatment and may be irreversible and severe. If there are still problems with eye movements (double vision), exposure problems (irritation and foreign body sensation), or decreased vision, then other treatments such as surgery should be considered.

*Why can’t you fix my eyelids now?*

Eye muscle surgery on the muscles that move the eye up or down may change the eyelid position. We do not want to do eyelid surgery until we have done any possible orbital or muscle surgery that might affect the final position of the eyelid.

*Can’t you just put my eyes back?*

We can reduce the bulging of your eyes by doing orbital decompression surgery. If you already have tight muscles, decompressing the orbit may produce double vision. This is usually treatable with eye muscle surgery but if you don’t have double vision now and your central vision is normal, we may be able to deal with the bulged appearance with lid surgery alone without the risks of having more involved orbital surgery.

*Why do you want to operate on my "good" eye?*

The eye muscles work in a balanced fashion to produce eye movements. In thyroid eye disease, muscles become incapable of moving normally due to enlargement and scarring of the muscle. Operating on only the tight muscles will sometimes not be effective enough to make the eyes straight. By operating on muscles in the other eye, we can achieve the balance necessary to allow you to have as much single vision as possible.

**Additional Online Resources**

- [http://patient.info/health/thyroid-eye-disease-leaflet](http://patient.info/health/thyroid-eye-disease-leaflet)
- [http://www.british-thyroid-association.org/info-for-patients/](http://www.british-thyroid-association.org/info-for-patients/)
- [http://www.thyroid.org/thyroid-information/](http://www.thyroid.org/thyroid-information/)