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# DYSTHYROID EYE DISEASE FROM THE INSIDE LOOKING OUT.

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**Abstract**

*The actual incidence of dysthyroid eye disease or thyroid ophthalmopathy in thyroid dysfunction is probably not known, as not all patients with thyroid disease are referred to endocrinologists or other hospital physicians, but are managed by their General Practitioner. Also endocrinologists tend to apply variable criteria for referral to an ophthalmologist. Some only consider ophthalmic opinion when there is obvious thyroid ophthalmopathy with serious visual disturbance, diplopia in the primary position or marked exophthalmos, and others will request ophthalmic advice when there is only minimal evidence of dysthyroid eye disease. It is possible therefore that some patients with thyroid eye disease may remain undiagnosed and untreated.*

*This paper discusses the author's own experience of thyrotoxicosis and thyroid ophthalmopathy particularly with regard to the understanding of the general disease process, the cause of some of the signs such as excessive blinking, lacrimation and adoption of abnormal head posture; and the importance of the right psychological approach to the management of some of the more unsightly cosmetic problems.*

## INTRODUCTION

Thyroid ophthalmopathy is a well known condition, characterised by exophthalmos, lid lag and lid retraction, peri-orbital and conjunctival oedema and mechanical restriction of eye movements. A characteristic sign is the abnormal head posture, usually a raised chin. The patient frequently complains of excessive lacrimation, red eyes and painful, uncomfortable eye movements. There is frequently diplopia, and visual disturbance which in some cases may lead to irretrievable visual loss due to optic nerve damage from compression by the muscles, or from raised intra-ocular pressure. The condition may be unilateral or bilateral, symmetric or asymmetric. Dysthyroid eye disease can occur with hyper or hypo thyroidism, and may present in patients with no sign of thyroid abnormality. It may occur at any time after the onset of the disease, and often presents when the patient is in an euthyroid state. Recurrence is said to be rare, but it is easy to find evidence of the disease several years after the active phase has passed, and the symptoms have resolved, due to the fibrosis of the formerly affected muscles and the tethering of one or both inferior recti.

## SIGNS AND SYMPTOMS OF THYROID DYSFUNCTION

I was given a diagnosis of thyrotoxicosis, some months after I had first started to experience

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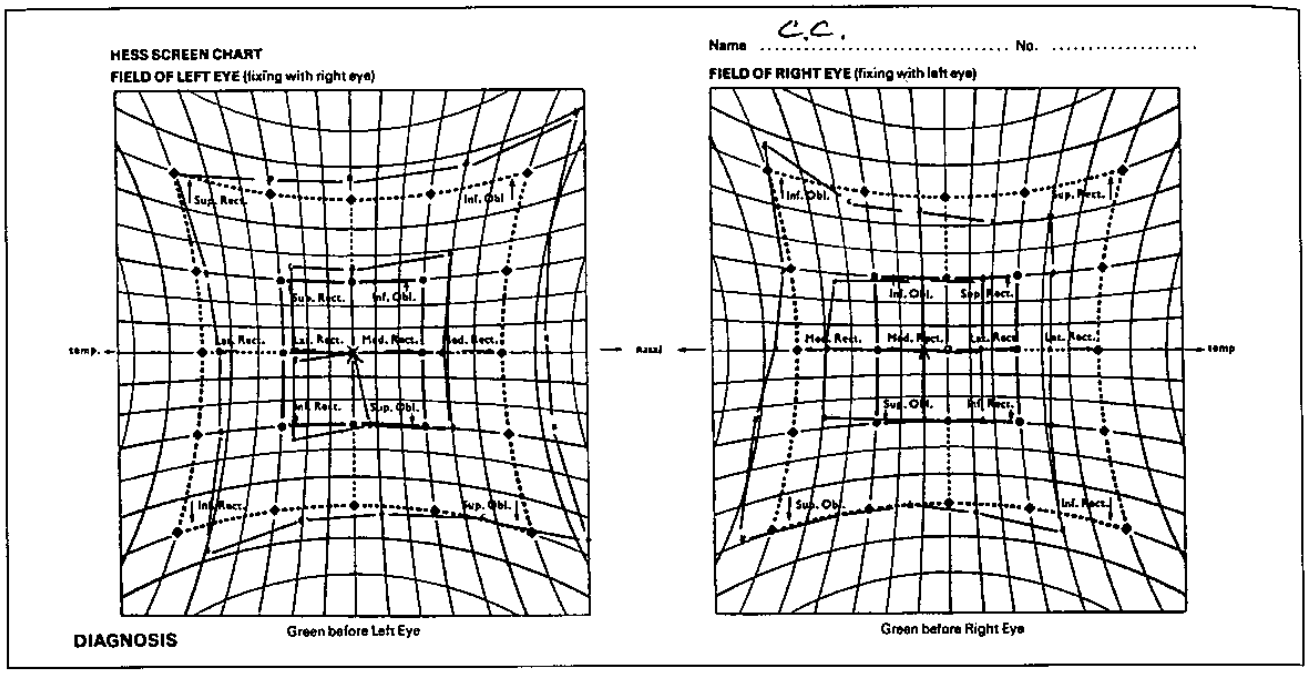


*Figure 1.*

anxiety attacks, hand tremors, fits of sweating, weight loss and metabolic disturbances. Whilst I do have a family history of thyroid dysfunction, I had attributed my initial symptoms to my age, stress, a recent orthopaedic operation and a possible reaction to my drug regime for rheumatoid arthritis. However, the onset of lid retraction and lid lag, and episodes of tachycardia at night convinced me that I had thyrotoxicosis. In referring me to the endocrinologist, my physician commented on the known relationship between thyroid dysfunction and rheumatoid arthritis in patients attending hospital for treatment of rheumatoid disease.

## MANAGEMENT

The management of thyroid dysfunction is to normalise the levels of thyroxine in the blood, with hyperthyroidism by inhibiting thyroid function with drugs, radio-active iodine or in some cases by partial or total ablation of the gland itself, and with hypofunction by introducing thyroxine artificially. It may take time to stabilise the condition and the patient may feel unwell for some long period. I was treated with carbimazole which rapidly rendered me euthyroid, although my symptoms of hyperactivity, metabolic disturbance, overemotionalism, and sweating attacks persisted until, unfortunately, I became hypothyroid. I became depressed, lethargic, oedematous, could barely summon the effort to walk, and found concentration difficult. Happily for me this state was shortlived, but it is important to note that in both thyroid states, not only did I feel most unwell, but I was aware that I was not functioning normally either physically or psychologically. My mental state in particular did not approach normality for some time after a stable euthyroid state had been achieved. This is, I am sure, a major contributory factor to the difficulties experienced with dysthyroid patients, who are time-consuming to manage and are often labelled neurotic and unco-operative.

## SIGNS AND SYMPTOMS OF DYSTHYROID EYE DISEASE

My eye disease presented initially during my hypothyroid state, some 4 months after the commencement of treatment for my thyroid disorder. I had become oedematous in appearance, and suddenly began to experience pain on eye movements and diplopia in downgaze. Recent studies[1] have suggested that there may well be an increase in cases of thyroid ophthalmopathy due to the rapidity with which patients

are rendered euthyroid with current treatment modalities, and although this might have been the cause of the onset of symptoms in my case, I have no personal clinical evidence of an increase in dysthyroid eye disease.

Because of the ocular symptoms associated with thyrotoxicosis, I found if very difficult, until I began to experience diplopia, to differentiate where the symptoms associated with the general disease stopped and where those due to thyroid ophthalmopathy started, and this dividing line is one which I suspect is not well appreciated by physicians, and is the reason that many cases with mild to moderate dysthyroid eye disease are not referred to ophthalmologists, particularly when diplopia is not present in the primary position.

I rapidly developed bilateral exophthalmos, left more than right, and retraction of the left upper lid, which was accompanied by acute discomfort on pursuit movements which worsened on saccades. My peri-orbital oedema increased, combined with conjunctival oedema and my eyes became red due to the dilated ciliary vessels over the insertion of the rectus muscles. My intra-ocular pressure on upgaze was raised to 27mmHg. Subjectively, there appeared to be fairly symmetric restriction of upgaze, with right hypotropia on dextro-elevation and left hypotropia on laevo-elevation, but the Hess chart (Fig. 1) showed that there was greater restriction of the right eye on up gaze. I had a right esotropia with diplopia on right gaze. There was an elevation of the chin as a compensatory head posture. The diplopia was only troublesome when I was driving a car, as the restriction of upgaze with diplopia made using the overhead mirror difficult, and the presence of right esotropia on right gaze made joining a motorway somewhat hazardous! However, provided I continued with my compensatory head posture, or turned my head to look to the right I remained relatively asymptomatic from the motility point of view.

## MANAGEMENT

The management of dysthyroid eye disease is clearly defined. There is no surgical intervention unless the sight is at risk, until the patient is euthyroid and any muscle imbalance has been stable over a three month period. In the short term Fresnel prisms and botulinum toxin therapy may help overcome diplopia in the primary position and increase the field of binocular single vision, and may even be all that is necessary in less severe cases. I was managing without prisms, but I did consider botulinum injections for my inferior recti in view of the possibility that it might inhibit the amount of fibrosis occurring at a later stage in the disease process.[2] However the risk of rendering my fairly symmetric condition asymmetric with botulinum injection was considered to be too high. Later, the response to the toxin injections was questioned.[3]

Whilst I was surprised at the speed at which I developed a compensatory head posture, and the manner in which my vertical fusional range increased, I was having some difficulty in coping with the other ocular symptoms, in particular the change in my appearance, mainly caused by the exophthalmos, peri-orbital oedema and lid retraction. Rapid eye movements were uncomfortable and produced excessive lacrimation, which in turn increased the blink rate. Blinking, unfortunately, did not stimulate my binocularity, but rather acted as a dissociating mechanism! I had red eyes and conjunctival oedema and a recurrent corneal erosion caused by my loss of Bell's phenomenon. Artificial tears helped the discomfort, but sleeping sitting up, using cold compresses or taking diuretics all failed to have any effect on the peri-orbital oedema. Guanethidine drops and lid exercises made absolutely no difference to the lid retraction. I felt miserable and realised why so many dysthryroid patients are unhappy and depressed and why some will consult plastic surgeons in an effort to improve the cosmesis. Once the more acute symptoms such as diplopia and loss of sight have been addressed, the patient has more time to consider their appearance. They want to feel well and look normal again. The clinician should be aware of this and once the patient is euthyroid, every effort should be made to explore the possibili-

ties of minor plastic surgery to the lids and periorbital tissue to improve the cosmetic appearance. To say, as some ophthalmologists do, that cosmetic surgery should not be undertaken for any reason shows a lack of understanding of, and sympathy for the patient. In my case, a Henderson procedure to the left upper lid normalised by appearance and reduced almost all my other ocular symptoms to within tolerance levels.

With time, the active disease phase passes and the oedema regresses, so that the appearance normalises although the mechanical restriction of eye movements persists. The patient however must be convinced that this will occur and it has been very helpful to be able to assure my patients that relative normality can be restored. They only have to look at their orthoptist!

COMMENT

One of my concerns has been that there are many patients with dysthyroid eye disease who do not have access to ophthalmic treatment, because their physicians are unaware of the necessity for the early differential diagnosis between dysthyroid eye disease and the ocular signs of thyroid dysfunction. Whilst patients with severe thyroid ophthalmopathy will always be referred, there is evidence that many less serious cases may never have reached the ophthalmic clinic, and may have forced to endure a considerable period of discomfort, and may even have suffered diplopia and irretrievable visual damage. It is imperative that patients with thyroid dysfunction with any signs or symptoms of dysthyroid eye disease are referred for management to the ophthalmology team, as this can only improve the management of the condition and early referral may result in earlier alleviation of symptoms, increased support of the patient and improved understanding of the ocular condition.

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