THYROID FLYER

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Feature – Autoimmunity & Related Conditions

Editorial

By Christopher McDermott

Welcome to our first Thyroid flyer issue for 2006.

The feature of this issue are the various autoimmune conditions. Some people with thyroid conditions also develop other autoimmune disorders. So we thought it an opportunity to provide some information on just some of those other autoimmune conditions.

The first few months of this year has been an eventful time within Thyroid Australia. If you have glanced to the top of this editorial you will note that I have taken up the reins as Editor from Alun Stevens, who is taking some time out. I have also taken some of the other Presidential duties – particularly with the committee of management – until our next Annual General Meeting at the end of this year. Alun has been President – as well as Treasurer and Company Secretary – for almost two years – as well as being the "webmaster" for Thyroid Australia since its inception. Most of you reading this would have visited our comprehensive website. Indeed, for many of you, your first contact with us was probably through our website.

I am pleased that Alun will continue to manage the website. I would like to thank him – on behalf of you all for the work he has contributed – and will continue to contribute - to the organisation. Alun's reason for taking time out is good news rather than bad; his company has become so successful that he must devote more and more of this spare time to his business interests. I wish him continuing success.

The other departure from Thyroid Australia which I must unfortunately announce is that of Robyn Koumourou. I first met Robyn at the official launch of Thyroid Australia at Melbourne Zoo back in 1999.

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Overview: Autoimmune Disorders

From time to time, physicians have recognized situations in which diseases occur together more often than chance alone would allow. In 1926, M. B. Schmidt, a physician in Germany, described two patients in whom both the adrenal and thyroid glands had failed. Since then, more than 125 patients with both disorders have been described, enough to make us realize that something more than an "accident of nature" makes this rare combination happen.

In several places on this website www.allthyroid.org we have commented on the relationship between Graves' disease and Hashimoto's disease, which tend to occur in the same families, sometimes in the same patients, and which seem to be different presentations of a single disease process.

There are other conditions that tend to occur in patients with Graves' disease and Hashimoto's disease and in their relatives as well. Some, like the prominent eyes of Graves' disease known as exophthalmos, have been well-studied and their relationship to thyroid problems carefully examined. Others, such as some of the associated skin disorders, are less well understood in regard to their relationship to the thyroid.

These articles are not about those bodily changes that occur due to high or low thyroid hormone levels. High hormone levels, for example, can raise your upper eyelids, make your skin soft and smooth, and cause your hair to become fine and delicate. The high hormone levels do not, however, cause your eyes to protrude, make the white patches of vitiligo appear on your skin, or produce the patchy baldness we call *alopecia areata.*

The latter problems are diseases in their own right. These are not, in general, serious problems about which thyroid patients should be concerned. Many, like *alopecia areata*, are not helped much by treatment, and tend to go away after a period of time. Others, like pernicious anemia or vitiligo, can be cured or controlled by appropriate treatment. Some, like Addison's disease, are so uncommon that even thyroid specialists rarely see a patient with this condition. Nevertheless, we believe there should be a place on this website www.allthyroid.org to which patients with Graves' disease or Hashimoto's disease can refer if they discover that they or one of their relatives has one of these problems.

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Volunteer News

The Thyroid Australia Board: We are currently wanting to expand the Thyroid Australia Committee and Board of Directors.

Your Story: If you have a story to tell and would like to share your personal experi-ences with thyroid disease, and/or related conditions, please send in your story to Thyroid Australia, via email or mail. We are always looking for member's stories to include in future editions of the *Thyroid Flyer*.

Hashimoto's Disease and Other Forms of Thyroiditis

Thyroiditis is the general term used to describe several different disorders in which the thyroid gland becomes inflamed. Most commonly, the inflammation takes the form of a chronic, progressive disease known as chronic lymphocytic thyroiditis or Hashimoto's disease (in honor of the Japanese physician who first described the microscopic changes in the thyroid tissue of patients with the condition in 1912). Patients with this form of thyroiditis sometimes exhibit so few symptoms that the disease may go unnoticed for many years, but eventually it may destroy so much thyroid tissue that hypothyroidism develops.

Lymphocytic thyroiditis may also occur as a self-limited condition which lasts 2-6 months, resolving spontaneously, and leaving most patients with normal thyroid function. When it occurs after pregnancy, it is termed postpartum thyroiditis. Another painless variant of lymphocytic thyroiditis may occur at other times and has been termed silent thyroiditis.

Subacute thyroiditis or DeQuervain's disease is another condition caused by thyroid inflammation, one that is distinct from those mentioned above. The disease often seems to follow the course of a viral infection. The thyroid gland is usually painful and looks quite different on microscopic examination.

Finally, very rarely the thyroid may become suddenly and dramatically inflamed with a bacterial infection. This condition is referred to as acute suppurative thyroiditis.

Chronic Lymphocytic Thyroiditis (Hashimoto's Disease)

Hashimoto's disease appears to be an inherited condition. As with Graves' disease, you probably must inherit a gene or set of genes to be able to develop this disorder. However, even though you may inherit this genetic tendency, you still may never actually develop the disease itself. Therefore, there must be other factors which cause this condition to develop. These other factors include being a woman, your age, and your body's immune system. Thus, women are affected about eight times more often than men, and although you may develop this form of thyroiditis in childhood or adolescence, it is most commonly diagnosed after the age of forty, for this is when affected patients usually become hypothyroid. Your body's immune system plays a role in the production of the thyroid inflammation and tissue destruction that occurs in chronic lymphocytic thyroiditis. Substances known as autoantibodies, made by white blood cells called lymphocytes, appear in your blood in this condition. Although we do not yet fully understand how or why these lymphocytes and antibodies work, the final result is damage to thyroid tissue. When enough tissue has been destroyed, your thyroid hormone production falls below normal, and symptoms of hypothyroidism appear.

The most sensitive test for hypothyroidism is a blood test that measures the level of the pituitary's thyroid stimulating hormone (TSH). When TSH tests are carried out on large numbers of people, we find that about 10 percent of women and 4 percent of men over the age of fifty have an elevated blood level of TSH. By age sixty, TSH is increased in as many as 16.9 percent of women and 8.2 percent of men. Put another way, at least one woman in six and one man in 12 will develop Hashimoto's disease in their lifetime. Each could potentially develop subsequent hypothyroidism and should be watched for signs of thyroid failure.

If you develop this condition, your thyroid inflammation will probably be so mild that at first you won't even know that anything is wrong. The first indication of a problem may be a goiter : You may develop a gradual painless enlargement of your thyroid gland. During this period, your thyroid gland is becoming infiltrated with lymphocytes, which start gradual thyroid destruction and scarring that may result in subsequent thyroid failure.

When hypothyroidism occurs, you probably will feel sluggish and run down, but the disease progresses slowly, so you may not realize that anything is wrong. Constipation, leg cramps, hair loss and mental dullness may appear, together with other symptoms and signs of thyroid failure. However, since chronic lymphocytic thyroiditis tends to be a progressive condition, your thyroid hormone level will probably continue to fall, causing your symptoms of hypothyroidism to worsen until your disease is recognized and treated.

Your physician can confirm the presence of hypothyroidism by means of a blood test that shows a low level of thyroid hormone (T4) and a high blood level of thyroid stimulating hormone (TSH). The elevated TSH level is the more important test, for it is more sensitive and proves that your thyroid, not your pituitary, has failed. Also, a blood test demonstrating the presence of antithyroid antibodies provides strong evidence of thyroiditis.

Since this condition may be progressive, lifelong follow-up is essential, but this usually amounts to no more than your physician examining your thyroid and testing your blood levels of T4 and TSH at your annual health checkup. As your thyroid gland's function declines, your thyroid hormone dosage may be increased appropriately. On the other hand, the dosage may actually decrease in some elderly persons.

The Overactive Thyroid: Hyperthyroidism Caused by Graves' Disease (Diffuse Toxic Goiter)

Introduction

The most common type of hyperthyroidism is produced by a generalized overactivity of the entire thyroid gland. This is called diffuse toxic goiter: diffuse because the entire gland is involved in the disease process, toxic because the patient appears hot and flushed, as if he or she were "toxic" due to an infection, and goiter because the overactivity enlarges the gland. Goiter is also known as Graves' disease, in honor of the Irish physician, Robert J. Graves, who was one of the first to describe this condition and who first noted the protrusion of the eyes that is sometimes associated with it. Between 1 and 2% of all people in the United States will develop Graves' disease. It is 3 to 4 times more common in women than in men, and typically begins between the ages of 20 and 40.

If you develop Graves' disease, your thyroid will begin to produce more and more thyroid hormone. As it does so, the gland will usually grow larger and will, in most cases, grow big enough to protrude noticeably in the front of your neck. You may notice the enlargement in your neck yourself, or you may not notice anything until a friend or your physician points it out. If the goiter is small, you may only sense the presence of a lump while swallowing. Typically, in this form of hyperthyroidism your thyroid gland is not tender, and it is not uncomfortable when you swallow.

As you develop hyperthyroidism, you may lose weight even though you seem to eat plenty of food. You may feel nervous and jumpy and may become quite irritable and quarrelsome. You are likely to perspire more than usual and dislike hot weather. Your skin may gradually become thin and delicate, and you may notice that you are losing some of the hair on your head. As your fingernails grow more rapidly, you may notice an irregularity of the nail margin, making it difficult for you to keep your fingernails clean. It is also possible that you could develop itchy hives on your skin.

Signs & Symptoms of Graves' disease

Muscle weakness, especially involving your upper arms and thighs, may make it difficult for you to carry heavy packages or to climb stairs. You may, in fact, experience such marked leg weakness that you cannot stand up from a squatting position without help. You may notice that your hands shake, and at times this tremor may become so severe that you can't even carry a cup of coffee without its rattling or spilling in its saucer. Your heartbeat may speed up from a normal rate of 70 or 80 to well over 100 beats per minute. Occasionally, without warning, your pulse may quicken abruptly, causing very rapid palpitations that last several minutes and then end as mysteriously and abruptly as they began. You are unlikely to have real diarrhea, but your bowel movements may become loose and more frequent.

If you are a woman, your menstrual cycle may change. Your flow may become much lighter and the interval between menstrual periods may lengthen. More rarely, your periods may become irregular, or may cease entirely, making it more difficult for you to become pregnant. If pregnancy does occur, there appears to be an increased likelihood that you will have a miscarriage. Women usually notice little change in their breasts, but if you are a man, your breasts may become slightly larger and may be tender.

Eye Involvement

One of the most puzzling and least understood aspects of Graves' disease is the way it may affect your eyes. Usually the change is simply an elevation of your upper eyelids that makes your eyes appear more prominent. Occasionally, however, swelling of the tissue behind your eyeballs may cause actual protrusion of the eyes known as exophthalmos or proptosis. Sometimes your eyes will feel dry or become red and irritated. A few patients have involvement of their eye muscles that may make them see double. In its most extreme (and very rare) form, the nerve to one or both of your eyes becomes inflamed and you may have trouble with your vision. This condition is known as optic neuropathy.

Elevation of the upper eyelids may be seen in anyone who has a high level of thyroid hormone, even someone who is taking thyroid hormone tablets in excess. The other things that can happen to your eyes in Graves' disease are unrelated to your blood level of thyroid hormone. If you are one of the people with Graves' disease who develops eye inflammation and protrusion, the eye problems probably will begin when you first become hyperthyroid. Quite often, however, eye problems and thyroid overactivity occur at different times, occasionally separated from one another by many years. Very rarely, a person may develop eye trouble as the only manifestation of Graves' disease.

Eye disease is therefore one problem that occurs only in the type of hyperthyroidism that is caused by Graves' disease. Another condition unique to Graves' disease is a very rare skin disorder that appears on the front of your legs and rarely on top of your feet. This is called pretibial myxedema, and takes the form of a lumpy, reddish colored thickening of your skin. It is usually painless and not serious. As with the eye trouble in Graves' disease, pretibial myxedema may occur anytime. Its appearance does not necessarily coincide with the beginning of your thyroid problem, nor is its severity related to your blood level of thyroid hormone. One of the rarest manifestations of Graves' disease is thyroid acropachy, which causes the tissues around the base of the nails to become swollen, but not painful. Periodic paralysis is yet another condition seen in occasional patients with Graves' disease. This disorder causes sudden attacks of profound weakness of all of the muscles of the body. In susceptible patients, sugar or starchy foods appear to cause a lowering of the blood potassium level, which prevents normal muscle function. For unknown reasons, periodic paralysis is most often seen in Asian men with Graves' disease.

Causes of Graves' disease

Graves' disease seems to be caused by the interaction of a variety of different factors, including heredity, your body's immune system, your age, sex hormones, and stress. Some sort of genetic predisposition seems to be needed first, and can be thought of as an inherited tendency to develop hyperthyroidism. If you have this factor, you may develop Graves' disease at some time during your life, or you may not, but if you lack this genetic factor, you probably cannot develop this disorder.

This type of hyperthyroidism clearly runs in families. If you have Graves' disease, and if sensitive thyroid tests could be carried out on your relatives, they might show mild thyroid abnormalities in one of your parents and one of your grandparents, in some of your aunts, uncles, brothers, and sisters, and possibly in some of your children as well. Fortunately, few of these relatives will ever become sick enough from their thyroid problems to require treatment; but some of them should be checked occasionally in this regard by their family physician.

Studies in identical twins confirm the importance of genetics in Graves' disease and also show the ability of other factors to modify the disease. Usually, identical twins either both have Graves' disease or neither develops the problem. But since other factors influence the disease process, twins rarely experience the onset of hyperthyroidism at the same time, and the course of the disease in the twins may be quite different.

There appear to be many different factors that can "trigger off" Graves' disease in a person who has inherited a tendency to it. Many thyroid specialists believe that stress can play a role in starting the hyperthyroidism, for we have all seen patients in whom a stressful situation, such as a death in the family, has preceded the onset of this condition. Sex hormones are also important, for the disease is seven to nine times more common in women than in men, and not infrequently begins after a hormonal change such as pregnancy. Age also seems to have something to do with the onset of Graves' disease, since it is most likely to appear when you are between the ages of twenty and forty. Finally, your body's immune system appears to play a role in the production of this disorder.

By an unknown mechanism, substances called autoantibodies appear in your blood. These autoantibodies bind to the cells in your thyroid gland and stimulate the thyroid to overactivity by mimicking the effects of pituitary thyroid stimulating hormone (TSH). This causes the thyroid to enlarge and to make more thyroid hormone. Thus, instead of being under the control of your pituitary gland , which is the normal situation, your thyroid becomes controlled by these abnormal antibodies in your blood.

The immune disorder that characterizes Graves' disease usually develops spontaneously, but recent studies have shown that you could be at increased risk for the disease if your thyroid gland was inadvertently damaged by x-rays for cancer therapy in the past or if you are taking one of the new immune-altering drugs like interferon and interleukin.

Conclusion

In summary, a susceptible person develops Graves' disease because of one or more factors that trigger off thyroid overactivity. As thyroid function increases, more thyroid hormones are released into the blood stream, producing the symptoms of hyperthyroidism.

Eye Enlargement and Inflammation

Any hyperthyroid patient, no matter what causes their hyperthyroidism, may experience elevation of the upper eyelid anytime the blood level of thyroid hormone is above normal. For example, patients who are hyperthyroid because of too much thyroid hormone medication may have raised upper eyelids causing their eyes to appear enlarged or staring. In this situation, however, the eyes do not actually protrude.

If you have Graves' disease, you may develop protrusion and inflammation of your eyes without there being any evidence of infection. It is likely to begin about the time your thyroid becomes overactive, but it may precede your hyperthyroidism or occur years after your thyroid function has become normal. Very rarely, the eye disorder may occur without your having any obvious abnormality of thyroid function at any time in your life.

More serious eye problems may occur in patients with Graves' disease and (less commonly) Hashimoto's thyroiditis. The severity of these conditions is unrelated to the blood level of thyroid hormone. If the condition is mild, you may have only redness and irritation of your eyes. On the other hand, in those rare instances when the inflammation is more severe your eyes may protrude, you may have double vision, and your sight may be threatened.

It should be pointed out that the thyroid eye disease does not necessarily progress in an orderly fashion from mild to severe in any given patient. In fact, a rapid decrease in vision can occur due to pressure upon the optic nerve in a patient with only minimal swelling of the eyelids. For this reason, if you have Graves' disease and begin to show signs of eye trouble, you should have a complete eye examination. If your eye involvement is severe, your physician may refer you to an ophthalmologist (eye specialist), who will have at his/her disposal all of the equipment needed to evaluate the various eye problems that may occur in Graves' disease. Your vision can be accurately tested. The amount of eye protrusion can be accurately measured with an exophthalmometer. The cornea and other tissues of your eye can be examined by the use of a microscope-like instrument known as a slit lamp. Ultrasound pictures of your eye and eye socket (orbit) may be taken, using sound waves in a technique similar to radar. Alternatively, your physician may request special x-rays of your orbits done by computerized tomography (CT scan) or by a newer technique called Magnetic Resonance Imaging (MRI). These techniques will provide a clear picture of the inflamed tissues behind your eye.

Treatment of your eye condition will depend upon the kind of eye disease you have and whether it is getting worse. Mild inflammation may be treated simply by elevating the head of your bed at night and by lubricating your eyes with drops of "artificial tears."

On the other hand, if you have a severe and rapidly progressive inflammatory condition with double vision or decreased vision, you may require special glasses or treatment with steroids. If your eye tissues continue to swell despite the use of steroid hormones, additional therapy is available. This may include x-ray treatments to the tissues behind the eye or surgery on the bony orbit (surgical decompression) to relieve the increased pressure behind your eye.

New research suggests that cigarette smokers are at greater risk for these troubles than non-smokers, so if you smoke and have just developed Graves' disease, stop smoking at once. Fortunately, serious eye problems are rare among thyroid patients. When they do occur, the treatment methods are excellent and are usually successful in improving the problem. Occasionally excessive drooping of the upper or lower eyelids may cause cosmetic problems, but plastic eye surgery can be very helpful for such patients.

Postpartum Thyroiditis

A woman's immune system is suppressed during pregnancy, but becomes more active following delivery of a baby. If you have a genetic tendency toward autoimmune thyroid problems, you may experience a painless inflammation of the thyroid as your immune system becomes more active in the months after delivery, even if you have no history of thyroid problems before or during pregnancy.

In its early stages, hyperthyroid symptoms may occur if excessive amounts of thyroid hormone leak into the bloodstream from your inflamed thyroid gland. Later on, when the thyroid's supply of hormones is exhausted, blood levels of these hormones often fall below normal and symptoms of hypothyroidism may appear.

Beta adrenergic blocking drugs like propranalol, atenolol, and metoprolol are usually enough to control the symptoms if you develop hyperthyroidism in the early weeks of this condition. If your thyroid fails after several months, supplementary thyroid hormone tablets can be given to maintain blood levels in the normal range.

Although complete recovery is common, about one-third of all women with postpartum thyroiditis progress to permanent hypothyroidism within three to four years, and require life-long treatment.

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Disorders of Other Endocrine Glands

Your thyroid is one of many endocrine glands, and autoimmune inflammations like those occurring in Hashimoto's thyroiditis may occur in these other glands too. When the inflammation leads to scarring and tissue damage, the glands may fail to produce enough hormones for your needs. The symptoms that result depend on the function of those hormones.

Your adrenal glands make cortisone and other steroid hormones, which are released into your blood stream daily and are especially important in your response to stressful situations. Adrenal failure (also called Addison's disease) is an uncommon condition, occurring in only one individual per 100,000 of the population. In most patients with Addison's disease, glandular damage is due to an immune attack on the tissues of the adrenal glands. If your adrenal glands fail, you will experience fatigue, loss of energy, weakness, and darkening of your skin, especially over your joints and inside your mouth. This condition is treated by replacing the hormones that the adrenals no longer make in sufficient amounts (cortisone and related steroid hormones).

Some women suffer from oophoritis, a painless autoimmune inflammation of their ovaries. In this condition, antibodies to ovarian tissue may be found in the bloodstram, and inflammation and scarring have been demonstrated in the ovarian tissues of affected individuals. Though rare, oophoritis is a condition your physician will consider if you experience early menopause.

Autoimmune damage to your parathyroid glands may lead to calcium deficiency (hypocalcemia). Symptoms of this condition include mood changes, numbness and tingling around your mouth and in your fingers and toes, muscle cramps, and, very rarely, convulsive seizures. Though associated with autoimmune disorders, it is actually a very rare cause of low calcium levels among thyroid patients. The more common cause of hypocalcemia is accidental damage to the parathyroid glands after thyroid surgery. If you develop hypoparathyroidism, your physician will likely prescribe calcium and Vitamin D tablets to eliminate your symptoms by adjusting your doses of these nutrients to bring your calcium into the normal range.

Even the pituitary, the master gland of the endocrine system, may suffer immune damage. This rare disorder (termed hypophysitis because "hypophysis" is another name for the pituitary) occurs most often in women during or just after pregnancy. In the thirty patients described in one report, slightly more than half experienced headaches, 32% lost part of their vision (the pituitary is located very near the optic nerves), and most experienced fatigue and weakness as other glands like the adrenals and the thyroid which depend on the pituitary for stimulation begin to fail. Treatment involves replacing the hormones that are lost when pituitary function declines.

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Arthritis

Some patients with Graves' or Hashimoto's disease also have a tendency to certain kinds of tendon and joint inflammation. Painful tendonitis and bursitis of the shoulder, for example, was reported in 6.7 percent of patients but occurs in only about 1.7 percent of the general population.

Rheumatoid arthritis is a more serious disease, in which there is a symmetrical inflammation of many joints of the body, most typically the knuckles, wrists, and elbows. It is also characterized by joint stiffness that is most severe in the morning. Severe rheumatoid arthritis appears to be only slightly more common among patients with thyroid dysfunction than in the general population. If you have hyper- or hypothyroidism you may notice mild morning joint pain and stiffness. If so, like patients with rheumatoid arthritis, you can benefit from treatment with heat, aspirin, and related drugs. On the other hand, some hypothyroid patients have joint pain and stiffness that improves when they are treated with thyroid medication. ✪

Anemia

Anemia is a disorder characterized by a decrease in the number of red blood cells that carry oxygen to various body tissues. If you have hypothyroidism, you may also have an associated mild anemia as one manifestation of the general slowing of your body functions that occurs in your condition. The anemia usually causes no symptoms and corrects itself when your hypothyroidism is treated. It is not a separate disease, but is due instead to the low thyroid hormone level.

A more serious type of anemia, known as pernicious anemia, is a separate disease that tends to occur in older patients who have or have had Graves' disease or Hashimoto's thyroiditis, and their relatives. This kind of anemia is caused by a deficiency of Vitamin B12.

Under normal circumstances, cells lining your stomach make a substance known as intrinsic factor that enables your body to absorb Vitamin B12 from food. Some individuals lose the ability to absorb Vitamin B12 due to failure of the cells that make intrinsic factor. The damage seems to be caused by a self-destructive process involving the body's immune system, similar to what occurs in Addison's and Hashimoto's diseases.

Vitamin B12 is an important ingredient in the manufacturing of red blood cells, and if levels of this vitamin fall, anemia may result. Vitamin B12 is also important in nourishing your nervous system, so if you develop pernicious anemia, you also may experience numbness and tingling of your hands and feet, loss of balance, and even leg weakness. It is not clear how many patients who have thyroid functional problems also develop pernicious anemia. Some studies have suggested that as many as 5 percent of patients with Graves' disease and 10 percent of those who have Hashimoto's disease may develop this condition.

Since pernicious anemia tends to develop in later years, it is probably even more common in older patients with either condition. Therefore, it seems appropriate to measure the blood level of Vitamin B in every patient over the age of sixty who has ever had Graves' disease or Hashimoto's Thyroiditis. Doctors do this because pernicious anemia is both common and treatable. If your blood level of Vitamin B12 appears low or borderline low, another test, known as a Schilling test, can be performed. This test demonstrates whether you have difficulty absorbing Vitamin Bl2 from your food. If you do have pernicious anemia, it can be easily treated.

On the basis of new research, your physician may choose to treat you initially with tablets of B12 to see if you are able to absorb enough of the vitamin to restore your blood level to normal and thus cure the condition. However, since your body's ability to absorb B12 tends to decrease with time, you will probably need treatment with a monthly intramuscular injection of Vitamin B12 as you grow older.

Platelet disorders are also more common in this group of thyroid patients than they are in the general population. Normally you have about 2.5 million platelets in every teaspoonful of your blood. Despite their small size, they play a major role in helping your blood to clot normally. Some thyroid patients experience easy bruising due to a decrease in the number or function of their platelets. The bruising can become much worse if you take aspirin, or one of the non-steroidal anti-inflammatory drugs such as ibuprofen (Advil or Motrin) or Naprosyn. If that is your situation, your physician may choose to order a platelet count or check your platelet function with a "bleeding time" test, which tells how long it takes your blood to clot. He or she may also recommend that you take an alternative pain medication such as acetaminophen (Tylenol) which will not worsen your bleeding tendency.

Very rarely, immune processes may destroy large numbers of platelets producing thrombocytopenic purpura. The word purpura refers to red or blue bruises which appear on the skin in this condition, especially on the legs. Tiny purplish-red spots known as petechiae that represent smaller areas of bleeding within the skin are also commonly present in this condition. If you develop this type of rash, your physician is likely consider it an emergency and order an immediate platelet count because of the risk of more serious bleeding elsewhere. If thrombocytopenic purpura proves to be your problem, treatment is usually helpful, and often includes steroid medication.

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Robin's
"T.E.D." (Thyroid Eye Disease) Story

I was told when first diagnosed that my Graves and my T.E.D. would run 2 different courses so I've always thought of them separately, so here are my experiences with TED so far.

In the beginning

I started having soreness in my left eye when using my computer back in Jan 2004, over 2 years ago and 6 months before the onset of full blown Graves Disease started. My right eye never really got swollen and sore until about September 2004. By 2005 my eyes were pretty bad…. left eye 25mm and right 24mm. They stuck out about 5-6mm more than normal. I had to wear sun glasses all the time, found it difficult to watch TV, go to the movies or look at a computer monitor for very long. Wind in my eyes created havoc causing my eyes to stream with tears... my Graves Disease was and still is a hassle but most of the time with medication I could forget it and move on with my lot but T.E.D. was with me constantly! I also had double vision when reading. The only good news was my primary viewing long distance field of vision was mainly intact, my upward gaze was no good, but I could still play bad golf, bowl and do such things as long as it wasn't too windy.

What to do?

I studied up on the situation and talked to as many people as I could find. It became apparent that it was now too late to consider any drug or Radio Therapy that may or may not have helped the eye muscles swelling. So that left me with the surgery option. I ruled out minor surgery to close my eye lids further over my bulging eyes as I was concerned this may last only temporarily and further drag the process on. I had advice from 3 eye specialists and they all classed my eyes as moderate to severe T.E.D. and all recommended orbital decompression surgery to let my swollen eye muscles settle further back into my head. They all pointed out that whilst it was a long and major operation that we were lucky in Victoria to have a very experienced and expert orbital surgeon available that they were confident in. They all said orbital decompression would definitely get rid of my bulging eyes and I should lose most of my eye soreness and pain. However I was most concerned about increased double vision.... 2 eye specialists thought that after the operation my double vision would be similar or worse whilst the other thought I had a good chance of losing it. However, the eye specialists also recommended another highly experienced double vision eye (strabismus) specialist for me to work with post op. So that made me feel a lot better about that risk aspect of orbital decompression surgery.

Making a Surgical decision

So I eventually concluded that the most likely out come would be, I would lose my bulging eyes, have less dryness and pain , but still likely have double vision and it may be worse but through squint surgery and/or special prism lenses that I would be able to get on top of the double vision problem with in 4 - 8 months.

I decided after some agonizing that the surgical route end picture looked better than the current one so I booked surgery for Jan 25th 2006!

Operation and recovery

The surgeon did both eyes together which took about 4hrs. I woke up with no pain and past the fingers up eye test. I had no numbness in my face or scalp and it was almost like I had never been operated on. The most discomfort I had was about 6 hrs after the operation when I became quite nauseous and vomited, which wasn't very pleasant. After that I only ate soup and toast for the next day whilst I still felt slightly nauseous. I only had pain when I moved my head and eyes. Whilst I lay still I was quite comfortable with no headaches or anything. I had no loss of feeling to speak of, just a little bit around the eyes. It was difficult to see where the surgeon had made the incisions so all in all with my eyes back in my head I was really pleased with the result and very thankful for the surgeon's skill. I was in hospital 3 nights, the swelling was worse on the 3rd day but went down quite rapidly after that. I was a good patient and held cold compresses on my eyes nearly all the time. My double vision was pretty bad after the operation but got a little better by the time I went home. My upward gaze was still pretty poor but as most of my friends are less than 7 ft tall, that is not a real problem! I took it easy when I got home but was moving around comfortably after a week. I am semi-retired but I guess if I was working I probably would have gone back to work about two weeks after the operation.

Double vision

My double vision continued to improve and after a couple of weeks I had single vision most of the time in my long distance primary viewing area over 5 meters. However it seemed unlikely that I would get rid of my short range double vision so I made an appointment with the double vision (strabismus) specialist for a week later. The strabismus specialist ruled out squint surgery as he said that due to the swollen and changeable nature of my eye muscles that getting my eye muscles correctly aligned and them staying there only had about a 50% likelihood of success with just one operation. So after much testing he stuck a temporary plastic prism lens onto the inside of my left spectacle lens. As he pointed out I really needed to be wearing glasses all the time anyhow so it didn't really add any difficulties to my way of life.

The prism on my left lens works well and I now have single vision all the time and I am feeling really good with less sore eyes and discomfort than previous. My eyes don't look quite what they did once but that's not all that important to me at my stage in life.

Next move

I go back to see the Strabismus Specialist in two months and he will then either organize for the temporary prism to become permanent or make any changes which may be necessary. I see my Orbital Surgeon in another three weeks time, so I will let everybody know how it is all going in a month or two.

12 Weeks after Operation

Well things have settled down and I've been back to see all the specialists. The Strabismus specialist is still not keen on doing squint surgery on me so I'll stay with prisms for the time being and probably for good, they are working well for me but I will replace the stick on Fennel prisms with ground lenses which will be clearer. I'm told the lens will be a bit thicker but not to much that it would look odd.

Eye lid surgery

My orbital decompression surgeon said I can get my eye lids lowered a little which will further reduce sun glare and wind on my exposed eye whites plus further improve my appearance. So I've decided to get this done 22nd April. I'm told the operation will take 1 to 1 1/2 hrs and I will be in hospital over night with my eyes fully bandaged. Swelling should decrease in a few days and if I was employed I would be back to work in about 2 weeks. Any how I will let you now if that's the way it works out and how it settles in. At this point I'm glad I had the orbital decompression done, am feeling really good and am looking forward to getting the eye lids lowered.

Below is my appraisal of the changers that have occurred from before Orbital Decompression Surgery till now (12 weeks after surgery).

My Eyes Improvement Rating after 12 weeks @ 12 / 4 / 06

('10' being extent of how bad it got before operation)

Bulging eyes was 10, now 1. Very big improvement. Eyes were 24 and 25mm, now they are about normal at 18mm.

Eye appearance was 10, now 4. Much improved and quite acceptable to me, but my eyes still don't look quite right, mainly caused by my eyes pointing a little down, left more than right which causes my lids to stay up.

Dry sore eyes were 10, now 5. The first 8 weeks varied a lot day to day, some days my eyes felt a lot better and other days very little difference, but the last 4 weeks has seen a marked improvement. But of course I still have the disease, I still use drops and I still use slip over or clip on polarized lens when around sun and wind. However before surgery I had to use shade welding 5 specs just to watch a large screen plasma TV, now I can get away with just my normal specs, so that shows a fair bit of improvement. My surgeon says the further lid surgery to drop my eye lids a little will further reduce the whites of my eyes that are exposed and improve dryness.

Eye vision without specs was 10, now 20. Double vision is worse. (before op. D/V was only short range and peripheral where as now with out prisms in my specs I would have double vision over my long range as well. Although to be fair I think if I had not gone to using prisms my long range vision would have eventually returned to normal, but I'm sure my short vision would not have recovered. So I think I made the right decision going to prisms. My upward gaze is still restricted but my sideways gaze has improved.

Eye vision with prism lens specs was 10, now 5. Prism specs have improved my vision but it's now essential that I must wear prism specs all the time. After several weeks of wearing prisms my eyes became dependent on them! Even with my prism specs I get a little double vision when reading if my eyes are tired. My eyes get tired quickly when I'm reading for more than 30 minutes with out a break. I read for 2 - 4 hours at times but I tend to be often closing one eye and be shifting my vision around when I read for prolonged periods. Overall I would judge with my prism specs things are pretty good. I enjoy my boating, bowling and with my golf I see the small moving ball reasonably well in the distance (usually amongst the trees!).

Strong pain behind left eye was 10, now 0. I did get modest to strong pain pre op. behind left eye in the mornings maybe once or twice a week, it never lasted all that long but it is now gone so that's a plus.

F**acial numbness** was 0, now 1. I am very pleased with the surgeons skill that I only have a very small numb area on my lower left eye lid which doesn't bother me at all and will disappear over time. Maybe I was a tad lucky as well.

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"THANKS EVERY ONE FOR THERE SUPPORT AND WELL WISHES, THE WONDERFUL DOCTORS AND MEDICAL CARERS AND A SPECIAL THANKS TO MY WIFE WHOSE LOVE AND SUPPORT HAS MEANT SO MUCH TO ME DURING THE HARD TIMES."

I'll let you all know how my T.E.D. is going in a few months

Cheers, Robin

✪

A personal note from Robyn Koumourou

I wanted to write this letter and send a huge thank you to my fellow colleagues at Thyroid Australia. I have recently had to resign from the Board. I have been ill over the last few months with problems other than my thyroid. My health issues have forced me to take a back step and remain at home to rest and recover.

I would like to thank my fellow board members, extended committee and the volunteers I have worked closely with for their friendship and support over the last 6 ½ years. Your tireless effort and dedication to Thyroid Australia and sufferers throughout this country has been remarkable. I have thoroughly enjoyed working with such passionate and committed people, who truly care for others and desire for them to have a better life. As volunteers we have given so much of our spare time to this very important cause. Hopefully, one day our voices will be heard and positive changes will occur in Australia in the treatment of thyroid sufferers, and more support given to organizations like ours.

Thank you also to the many people I have spoken with on the phone. Your stories, struggles and triumphs have been inspiring and invaluable in the search for understanding and knowledge. Over the next few years I hope to continue in the area of research and hopefully will complete a second book covering all aspects of thyroid disease. I also want to express my appreciation to Thyroid Australia and its members in the support of my first publication, 'Running on Empty'. I hope this has been a blessing for many.

In the meantime, may support of Thyroid Australia continue, so they can continue to support you!

Kind regards,

Robyn Koumourou

THYROID AUSTRALIA LTD

SUPPORT FOR THOSE WITH THYROID CONDITIONS, THEIR FAMILIES AND FRIENDS

Scientific Review

By Alun Stevens MSc FIAA

Whose Thyroid Hormone Treatment is it Anyway?

It is under this heading that AP Weetman, a well known and respected British endocrinologist, reflected and commented in the March 2006 edition of *Clinical Endocrinology* on the current state of the debate over hypothyroidism treatment. The article provides a good illustration of the views of the medical profession in response to increasing demands from patients.

The article, it would seem, was prompted by advocacy by various groups in the UK against the current norms of clinical practice in this area and in particular by the lodgement of a petition by a group of UK patients with a Member of Parliament and the UK General Medical Council as a 'formal complaint against the clinical practice of the majority of the medical profession with regard to the diagnosis and management of hypothyroidism on four counts:

1. Over-reliance on thyroid blood tests and a total lack of reliance on signs, symptoms, history of the patient and a clinical appraisal.
2. The emotional abuse and blatant disregard by the majority of general practitioners and endocrinologists over the suffering experienced by untreated/incorrectly treated thyroid patients and their lack of compassion over the fate of these patients.
3. Stubbornness of general practitioners and endocrinologists to treat patients suffering from hypothyroidism with a level of medication that returns the patient to optimum health. In addition the unwillingness to prescribe alternate thyroid treatment for patients on individual grounds... such as Armour thyroid.
4. The ongoing reluctance to encourage debate or further research on hypothyroidism.'

The themes of the petition are common themes across the world amongst some patients and patient support groups and reflect common approaches by the medical professions in many countries including Australia. They also reflect the growth in movements for alternative therapies many of whom promote their particular approach by representing the medical establishment as ignorant and uncaring.

Weetman's response, rather than answering these challenges is a good example of the approach and attitude of the medical profession that has actually led to these complaints. He gives two reasons for rising patient dissatisfaction and mistrust.

His first is simply that we live in a post modern society characterised by the world view that objective facts are unimportant and reality has many meanings. In other words, patients feel justified to reject and ignore objective facts as presented by the scientific medical profession if these facts clash with the patient's subjective reality.

He couples this post modern approach with his view that patients presenting with multiple ongoing symptoms despite normal thyroid function tests generally have functional somatoform disorders – that is psychological conditions that produce physical symptoms. In other words these people think they have a problem so they develop symptoms and because they have the symptoms, they must have the condition so it is the tests that must be wrong or ineffective. This attitude that patients with persistent symptoms must be psychologically disturbed is undoubtedly one cause of the claims for emotional abuse, stubbornness and indifference.

The primary failure of Weetman's argument though is that it assumes that the results of thyroid function tests are applied as objective scientific facts in clinical practice. Modern hormone assays are extremely accurate. Test results are factual, but they are objective scientific facts only to the extent that they show the concentration of certain hormones at a point in time. Simply knowing the concentrations is never sufficient. It is the meaning of these concentrations that is always the issue and the interpretations are not facts, they are opinions and are frequently not objective.

The interpretation of thyroid function tests is particularly problematic because the tests are statistically based. They reflect the averages in the non-thyroid afflicted population. Their proper interpretation can therefore only ever be probabilistic. To be scientifically objective, one can only ever say that a result has a low, medium or high probability of being normal. No result is ever a certainty. Despite this, virtually all doctors will state categorically that a result is either normal or not. In many instances, this is not a problem, but in many others it is.

For instance, a TSH reading above 5.0 has such a low probability of being normal that a doctor can safely say it is categorically abnormal. He or she would only be wrong a couple of times in a lifetime of practice.

A TSH reading of 1.5 however is much more problematic. For someone not on thyroid medication, it has a high probability of being normal. Most people have a reading near to this and it is a long way from the upper levels recorded in the normal population. The result cannot be interpreted as categorically normal. The high probability of normality would support the view that presenting symptoms are probably due to something other than a thyroid problem. Ascribing the symptoms to thyroid problems would only be correct very occasionally. But these occasions will occur and should be kept in mind.

For someone taking thyroxine replacement, the situation is different and more problematic. A TSH reading of 1.5 has about a 60% probability of being within the normal range for the individual presenting. This is because individuals have much narrower personal ranges than the overall population range. 60% is only slightly better than an even money bet so is definitely not capable of objectively sustaining a categoric interpretation that the result is normal or that the thyroxine dose is correct. If the patient has no symptoms, it would be safe and sensible to conclude that the result is normal. But if the patient has persistent symptoms of hypothyroidism, it would be quite unsafe

to conclude that the result is normal because this interpretation will be objectively wrong in close to 50% of cases.

The scientifically inappropriate interpretation of test results as categorically normal when they are not leads doctors to ignore patients' views and the symptoms and is undoubtedly a cause for the first three complaints in the petition. The problem is not a post modern clash of feelings versus facts. The problem is a lack of appropriate methodologies for interpreting the objective facts.

Weetman acknowledges this in his second reason for patient dissatisfaction which he states as 'any innate sense of disbelief (by patients) in science has been heightened by the lack of consensus amongst endocrinologists, particularly with regard to the diagnosis of subclinical hypothyroidism, its importance, and the need for treatment.' He goes further by stating, 'The difficulty we face as clinicians in formulating guidelines, or more commonly in judging how to apply them to our own practice, stems from our individual perceptions of benefit and risk.' This is an acknowledgement that medical methodologies are not fully robust or objective. The clash is not between patients' perceptions and objective medical facts. It is between patients' perceptions and doctors' perceptions.

Despite his unsupported and somewhat arrogant attack on patients' perceptions, Weetman's final recommendations to doctors are sensible and heartening. He says, 'Communication lies at the heart of managing patients whose health problems cannot be explained and the focus should be on the patient's concerns, the relief of symptoms and the avoidance of alienation. Finally, we should retain our own sense of perspective, scepticism and humility.' The adoption of these principles by the medical profession would go a long way to providing better outcomes for patients because it is *their* 'Thyroid Hormone Treatment'.

AP Weetman, 'Whose Thyroid Hormone Treatment is it Anyway?', *Clin Endocrinol*. 2006;64(3):231-233.

Editorial from page 1

Full of enthusiasm and drive, Robyn told us at that launch she was so committed to helping others she intended to write a book – which she promptly did. "Running on Empty" was published in 2004 and has continued to sell well ever since. At the same time, Robyn was a dedicated telephone volunteer, helped in the office and was an active board member – not to mention looking after her family of John and the two girls. No doubt many of you have had personal contact with Robyn and you will join me in wishing her well. Robyn is having a well-earned rest and recuperation.

The very good news is that we welcome our new office manager to Thyroid Australia. Brenda Stocks started in February this year and works part-time four days a week. She has had a baptism of fire with us still struggling with the backlog of admin work left over from the closure of the office for renovations in late 2005. We welcome Brenda. She has been a godsend already. With a couple of new volunteers recently started, we now have a really effective office operation. Though – if you want to help in the office – we can still do with your assistance. Ring Brenda if you are interested.

We are now starting to make headway with the backlog. We have sometimes been slow in banking cheques, responding to requests for information, and such things. Thank you all for you patience as we get on top of this situation.

The other good recent news is of our meeting in Brisbane at the end of April. Alun Stevens presented his introductory talk on thyroid conditions and their treatments. Over 160 people attended this meeting and, on behalf of all those who attended, I thank Alum for his contribution. We look forward to a continuing and active group in Brisbane.

Lyrics of as a song provided by Christopher McDermott about someone with an autoimmune condition. Please note we have not referred the remedies taken by Lillian to our medical advisory committee and cannot endorse her actions—nor recommend her fate!

Lillian's story

Lil was a girl, she was a beauty, she lived in a house of ill-reputee;

She drank deep of the demon rum, and she smoked hashish and opium.

Chorus:

Oh, de boom boom, de boom boom, de boom boom boom

She was young and she was fair, she had masses of golden hair;

Folks they came from miles to see Lil in her deshabille.

Day by day that girl grew thinner from insufficient protein in her,

Till at last the day came when she had to cover up her abdomen.

She took sunbakes in the sun, she took Scott's Emulsion.

She took liver, she took yeast, but still her clientele decreased.

She consulted a physician who prescribed for her condition;

She had, as the doctors say, pernicious anaem-i-a.

As Lil lay there in her dishonour she felt the hand of the Lord upon her.

She said, "O Lord, I will repent, but that must cost you fifty cents."

Lil she died and went to Hell, to live with Delilah and Jezabel;

Now she's working from five to nine as Satan's favourite concubine!

Thank You
To
The City of Monash
For their generosity in
Providing us with office
accommodation

CITY OF
MONASH

Meetings and Support Groups

Gold Coast / Tweed Heads

Dates not yet announced

Contact office for more information

Brisbane North Side

Chermside Library 2pm-4pm

Jun 3rd / Aug 5th / Sept 16th

[Oct 14th Seminar (Bookings essential)]

Brisbane South Side

Sunnybank Hills Library 2pm-4pm

May 13th / Oct 21st / Nov 25th

Perth

10th June and 22nd July - 10.30am

Contact office for more information

Support Group Meetings

Salvation Army Hall

565 Walter Road East (c/- Wicks St)

Morley WA

Inner Melbourne

Dates not yet announced

Contact office for more information

Support Group Meeting

Nth Carlton Railway Station

Neighbourhood House

20 Solly Ave

Princess Hill VIC

South Gippsland

Fourth Monday of Each Month 10.30am

Support Group Meetings

Foster Community Health Centre

93 Station Rd

Foster VIC

Melbourne

12th Nov - 2pm to 5pm

Public Meeting & AGM

Royal Children's Hospital

The Murdoch Institute, 10th Floor

Thyroid Australia Inner Melbourne Support Group

On 24 May 2005 Thyroid Australia held it's first support group for people in the Melbourne metropolitan area. It was held in the North Carlton Railway Station Neighbourhood House at 20 Solly Avenue, North Carlton.

Although there were only ten people attending (all women) it was a huge success. At the end of the meeting they asked, "When are you holding the next one? Next month?" So they were certainly very pleased with the way things went and obviously gained something by attending.

There was no guest speaker as such, but each woman was able, in some cases for the first time, to be able to tell her story and how having a thyroid condition had affected her life and the lives of her family and friends.

We covered the full gamit of illness, from thyroid cancer, hypothyroidism, Hashimoto's, goitres, thyroidectomies, hair loss, Graves disease, infertility, miscarriage, depression, looking at blood test results and weight gain.

The meeting ran over time with everyone getting in small groups to discuss things in more detail and exchange information on good doctors worth visiting.

The next meeting was held in the evening on 12th July. It attracted a fewer number of people, but some had come from long distances in order to attend. It followed the same format as the first day meeting and again, the response from people was heartfelt. They were pleased to tell their stories and be heard by sympathetic ears. Holding an evening meeting made it possible for those that work during the day, to attend.

The last meeting we held was on 26 July and we had a full house. Robyn Koumourou did an excellent job as guest speaker, her topic, "Thyroid gland disorders".

For my part, all the hard work and many hours spent in getting everything organised was well worth it. Karen

Please copy or detach and mail to the address below.

Request for Membership Application Form and Information

I am interested in learning more about my thyroid condition and about Thyroid Australia.

Please send this information to:

Title: ..... Name: …….....

Address: .....

.....Phone:.....

Date: .....Signature:…….....

Disclaimer

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Thyroid Australia Ltd
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