THYROID FLYER

Newsletter of Thyroid Australia — Volume 1 No 4 October 2000

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Feature - Postpartum Thyroid Conditions

Editorial

By **Megan Stevens**

Olympians

With the Sydney Olympics fresh in our memories, those of us who have felt the fatigue and muscle pain so common with thyroid disorders may wonder that there are fellow thyroid sufferers who manage to compete at the highest level of sport at the Olympics.

Carl Lewis

Who can forget the explosive power and grace of former Olympic great, Carl Lewis. He was diagnosed with Hashimoto's thyroiditis, five months before his fifth and final Olympics in Atlanta in 1996.

He described his feelings following his diagnosis, saying: "… no matter how hard I'm training, no matter how much emphasis I'm placing on proper nutrition and rest, my efforts are being sabotaged by a breakdown beyond my control.". Carl Lewis went on to win a gold medal in the long jump in Atlanta.

Gail Devers

On the team with Carl Lewis in Atlanta, and competing more recently in Sydney, was sprinter and hurdler Gail Devers. She was diagnosed with Graves' disease in 1989, while training for the Seoul Olympics. Because of the strict drug policies relating to Olympic athletes, she was not allowed to take beta-blockers to slow her heart rates.

Unfortunately for Gail, she exacerbated a hamstring injury during her finals 100m hurdle race in Sydney, and she failed to finish. She had previously won gold medals for the 100m sprint in Barcelona (1992) and in Atlanta (1996), as well as gold for the 4x100m relay.

Myriam Bédard

And as someone with hypothyroidism who has tried to learn to ski (and failed),

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After the Baby Arrives

Postpartum Thyroid Dysfunction

By **Robert Smallridge**

Postpartum thyroid disorders were described in small groups of women in the 1970's, and in much larger groups in the 1980's and 1990's. However, the earliest written description of *hyperthyroidism* occurring in a woman after childbirth was by Doctor C.H. Barry in 1825, and the first case of *hypothyroidism* by Sir H.B. Donkin in 1888.

Although Doctor H.E.W. Robertson, in 1948, is credited with recognizing that the postpartum condition predisposes women to thyroid problems, another quarter century passed before the medical community started to become aware of this association. Only in the past decade and a half has this association been recognized with regularity by doctors.

PPTD: And a Definition

Postpartum thyroid dysfunction (PPTD) encompasses several thyroid conditions that may occur (often for the first time) in a woman between 3 and 12 months after the birth of her baby. The majority of women have underlying autoimmune thyroid disease (which can be documented by detecting the thyroid peroxidase (TPO) antibody in their blood). Due to changes in a new mother's immune system during and after diabetes mellitus, or those who have had more than two miscarriages are at high risk for PPTD. The presence of goiter (enlarged thyroid) or a family history of thyroid disease also increases the risk.

Symptoms in Some

Women may experience a variety of symptoms in the first few months after delivery. Dramatic changes are occurring. First the young mother is recovering from the physical stresses of childbirth. The responsibilities in taking care of her newborn baby may cause anxiety and lack of sleep. Major changes in female hormone levels are occurring, too. Therefore, within the first three months postpartum, it is difficult to distinguish women with mild hyperthyroidism due to thyroiditis from women with no thyroid problem.

However, after the third month, a new mother should be feeling much better. If she continues to have symptoms such as heart palpitations, anxiety, or nervousness, then hyperthyroidism is a possible cause. More commonly, the hyperthyroid symptoms are mild, and it is the later occurrence of hypothyroidism (tiredness, weight gain, mental fatigue, depression) that should raise the suspicion of an underactive thyroid.

Diagnostic Strategies

At the present time, we still don't know the most effective way to identify women experiencing postpartum thyroid dysfunction. One approach would be to screen all women after delivery with a thyroid blood test, either a TPO antibody level or a TSH test. Since most women who develop symptoms are TPO antibody positive, then all women identified by this test could be counseled and followed carefully for the first year postpartum. The advantage of this test is that it identifies most women at risk. There are two disadvantages. First, considerable cost is incurred if we test all new mothers in order to identify the 5-10% of antibody positive women. Second, the TPO test does not tell which women are experiencing a change in thyroid gland function. That requires a TSH test.

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Editorial *from Page 1*

I have the greatest admiration for the winter Olympian, Canadian Myriam Bédard, who was diagnosed with hypothyroidism following the birth of her baby. She had previously won two gold medals in Biathlon (which combines cross country skiing with target shooting) in the 1994 Olympics in Lillehammer.

These people are a source of inspiration to us as they managed to overcome the difficulties, not only of performing so well at their chosen sports at the Olympics, but also did so with the added burden imposed by their thyroid conditions.

But what of Australian Olympians? There must be some who have thyroid disorders. Thyroid Australia would welcome the stories of any Australian Olympians, or other elite sportsmen and -women, who have excelled despite their thyroid conditions. We need our own local heroes too!

Thyroid Function Tests

We publish here a provocative article by Denis O'Reilly, recently published in the British Medical Journal. He argues that thyroid function tests (TFT's) have taken on a position in medical practice which they do not deserve as they are not as reliable as they are assumed to be. The high accuracy with which hormone concentrations can be measured does not mean that the Reference Ranges against which these readings are judged have anything like the same accuracy or quantitative reliability. He argues that TFT's should be treated with more scepticism and that there should be a greater emphasis on symptoms. We applaud this stance. Our recommendation is that patients should have their active hormone levels within the Reference Ranges, but should also be free of symptoms.

TFT Result Record Sheet

Following our recommendation in our last newsletter for thyroid patients to record their thyroid function test results, we include with this newsletter a sheet on which to do so.

Members' Network

It's amazing what good ideas one can get from other thyroid support groups around the world. While reading the newsletter of the British Thyroid Foundation recently, I came across an idea of theirs, which I thought we might copy. The idea is this: Members write in looking for other members with similar conditions to contact them, using Thyroid Australia as a clearing house. So, if you would like to take part in this network, drop us a line. We will advertise your query in the newsletter, and forward any responses from members to you.

Our first request comes from Christopher, of Fitzroy VIC, who would like contact with others who are treating their hyperthyroidism on a long term basis with PTU or neomercazole in place of RAI treatment or surgery.

Prof Duncan Topliss

Our thanks to Prof Topliss for his excellent presentation on 23 September. His relaxed style and willingness to discuss issues (even controversial ones) candidly were greatly appreciated. Our thanks also for his time spent answering questions both before and after the meeting.

Medical Advisory Committee

It is with great pleasure and humility that we introduce to you the members of our Medical Advisory Committee. Their role is a fairly informal one, but we will draw upon them for advice both on the accuracy of articles we might want to publish, as well as helping with queries beyond our expertise. So far, the following eminent medical practitioners have agreed to serve on this committee:

- A/Prof Peter G Colman (Endocrinologist)
- Dr David Dammery (General Practitioner)
- Prof Creswell J Eastman (Endocrinologist)
- Dr Anthony J H Hall (Ophthalmologist)
- Mr William R Johnson (Endocrine Surgeon)
- Prof Jan R (Jim) Stockigt (Endocrinologist)
- A/Prof Duncan J Topliss (Endocrinologist)
- Dr Rosemary Wong (Endocrinologist)

We will be profiling the committee in this and upcoming editions of *Thyroid Flyer*.

Prof Colman has been Director of Diabetes and Endocrinology, Royal Melbourne Hospital since 1992. He graduated from Monash University in 1977; and did clinical and research training at Alfred and Royal Melbourne Hospitals and at the Joslin Diabetes Center in Boston.

He is responsible for inpatient and outpatient Endocrine Services at RMH, including the thyroid clinic. His major research interest is autoimmunity and how this relates to Graves' disease and type 1 diabetes. He has extensive experience working with the patient organisation, Diabetes Australia, and is keen to be part of aiding Thyroid Australia to develop its role in assisting people with thyroid disorders.

Dr Hall has a medical degree and an MD from the University of Melbourne. He trained in Ophthalmology at the Royal Melbourne Hospital and at the Eye and Ear Hospital and has undertaken further training at Moorfields Eye Hospital in London and at the University of California San Francisco. He is currently Director of the Department of Ophthalmology at the Royal Melbourne and an Ophthalmologist to the Ocular Immunology Clinic at the Eye and Ear and the Ophthalmology clinic at the Alfred Hospital. He has a special interest in inflammatory eye disease and medical ophthalmology.

After graduation from the University of Melbourne in 1985, **Dr Wong** completed training in Internal Medicine at the Royal Melbourne Hospital followed by training in Endocrinology at the Alfred Hospital. She subsequently spent five years at the National Institutes of Health (USA) investigating thyroid conditions using transgenic animal models. Since returning to Melbourne in 1997, she has been working as an Endocrinologist at the Western Hospital and at the Osteoporosis Clinic, RMH, and in private practice at Cabrini Hospital and in Box Hill. Her other areas of interest include osteoporosis and women's health.

On a 'sour' note

At our meeting at the Royal Women's Hospital on 26 August, we put out various thyroid books. Unfortunately one these books (*Understanding your thyroid problem*, by Mark Ragg (Gore & Osment, Sydney) 1993) went missing. This book belongs to one of the committee, and is used as a reference when people ring in for information, she now has to replace it. If anyone has the book, could you please return it.

Phone Calls

Please, no phone calls after 9:30pm. These late night phone calls can be quite taxing for our telephone contacts, as the adrenalin starts pumping before they answer the phone, and they wonder which of their loved ones is in trouble. It really is a relief to find that the person on the other end of the line is just looking for thyroid information. But, please, no phone calls after 9:30pm. Their nerves can't stand it!

Postpartum Thyroid Disorders *from Page 1*

Another screening approach would be to perform a TSH test in all women after pregnancy. Unfortunately, there is no single time after delivery that is ideal, since PPTD may develop over a span of almost a year. A normal TSh test on one occasion does not mean a woman will not develop a thyroid problem one, tow, or three months later.

Currently most physicians are approaching the diagnosis of PPTD differently. Essentially, they wait until a woman gets symptoms that are bothersome enough and last long enough to cause her to see a physician. The doctor then needs to suspect a thyroid problem and order the proper blood test (TSH). The advantage of this is that the cost of diagnosing PPTD is low. The disadvantages is that many women with milder symptoms would be missed since they may not seek medical care. Two studies, in fact, have shown that some women who are hypothyroid during or after pregnancy may not receive proper treatment for several years! One way to improve upon this diagnostic strategy is to increase patient and physician awareness of the symptoms and physical signs of PPTD so that affected women will have a TSH test earlier.

At present, the most reasonable approach seems to be one which focuses on women at highest risk for developing PPTD. Those with a history of thyroid or related immune problems themselves or in a close relative should be educated about the symptoms and signs of PPTD by their family physician or obstetrician. Ideally this subgroup of women should also have TSH or TPO antibody tests before and after pregnancy to treat recognized thyroid problems during pregnancy and to identify those who develop PPTD. The necessary research is now being done in several medical centers to find out if such testing is truly effective and economically feasible.

Treatment for Some

Not all women with PPTD develop symptoms. Hyperthyroidism due to thyroiditis is often mild and brief. Occasionally, a beta-blocking drug (such as propanolol or atenolol) is used to slow a fast pulse and calm agitation until thyroid function returns to normal. When hyperthyroidism from postpartum Graves' disease produces more severe and long-lasting symptoms, these women should see an endocrinologist for evaluation and therapy. The most common symptoms of PPTD are those associated with hypothyroidism. For those women who develop hypothyroidism, usually between four and eight months after delivery, treatment with thyroid hormone (L-thyroxine) should relieve their symptoms. Therapy should be discontinued after 6–12 months, and a TSH obtained 6–8 weeks later. By then, about 75% of women will have regained normal thyroid function and can remain off treatment. They should be counseled to have a TSH test *before* (if possible), *during*, and *after* any future pregnancies. Women who still have a high TSH test are likely to have permanent hypothyroidism. Thyroid hormone treatment needs to be restarted and adjusted until the TSH level is normal. For most women this treatment is life-long.

During the European Thyroid Association Congress in Milan, Dr Robert Smallridge took time out to address delegates of the Thyroid Federation International at a special seminar. Dr Smallridge is a member of the world renowned Mayo Clinic. We are indebted to Dr Smallridge for this comprehensive and clear overview of PPTD.

Robert C Smallridge, MD is Professor of Medicine and Chair, Endocrinology Division, Mayo Clinic Jacksonville Florida, USA.

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Thyroid function tests - time for a reassessment

By **Denis StJ O'Reilly**

In 1999, 890,000 measurements of thyroid stimulating hormone were performed by Scottish hospital laboratories - approximately one test for every six of Scotland's 5.1 million people.[1] This number does not include tests performed in the non-NHS laboratories or as part of the screening programme for congenital hypothyroidism. Although laboratory statistics are not collected nationally in England and Wales, the market in the United Kingdom (population 59 million) for thyroid stimulating hormone diagnostic tests is currently estimated at 9-10 million each year.

A remarkable downgrading of the clinical aspects of hypothyroidism and hyperthyroidism has paralleled the inexorable increase in the number of thyroid function tests performed over the past 20 years. This has led to chaos in the diagnosis of hypothyroidism. It has been stated that a diagnosis of clinical hypothyroidism can be made on the basis of biochemical measurements alone and that signs and symptoms are unnecessary.[2] Other authors protest, and maintain that biochemical tests can be misleading and that the diagnosis can be made on clinical grounds alone.[3] In hyperthyroidism, a suppressed thyroid stimulating hormone concentration is currently the cornerstone of biochemical diagnosis. No numerical value has been assigned to the serum concentration of thyroid stimulating hormone below which suppression is considered to occur. This value varies from centre to centre depending on the sensitivity of the local assay. Thus, to many non-specialists the diagnosis of hyperthyroidism is also confusing.

Methods

This review is based on my 20 years' postgraduate experience in providing biochemical thyroid function tests and treating patients with thyroid disorders. I have selected and highlighted some of the publications that have influenced my practice and call into question the increasing reliance on biochemical thyroid function tests in making a diagnosis.

Historical setting

The treatments currently used for hyperthyroidism and hypothyroidism were established by the beginning of the 1970s. Though the symptoms and signs of these disorders had been analysed and clinical scoring indices had been developed and validated in the 1960s, clinical diagnosis remained problematic.[4-8] The clinical diagnostic schemes for hypothyroidism were similar,[4-6] but there were considerable differences between diagnostic schemes for hyperthyroidism. For example, atrial fibrillation was considered by Wayne and Crooks to be one of the most powerful discriminating signs,[6 7] but it was not included by Gurney et al.[8] Age, on the other hand, was a major diagnostic factor according to Gurney et al,[8] but was not mentioned by Wayne or Crooks.[6 7] From knowledge of the pathophysiology of the hypothalamic-pituitary-thyroid axis available at that time, it was believed that measuring the concentration of serum thyroid stimulating hormone would simplify the diagnosis.

Summary Points

- There are no data on the relative importance of biochemical thyroid function tests and clinical symptoms and signs in assessing thyroid dysfunction
- Secretion of thyroid stimulating hormone is influenced by many factors other than the negative feedback inhibition by thyroxine or triiodothyronine
- Changes in thyroid stimulating hormone, thyroxine, and triiodothyronine concentrations during systemic illness are poorly understood
- Thyroid function tests cannot be interpreted in patients with systemic illness
- Since thyroid stimulating hormone concentrations are distributed logarithmically in the population, minor changes are unlikely to be clinically important
- The possibility of false positive and false negative results should be considered in interpreting thyroid stimulating hormone concentrations

Hypothyroidism

The publication of a reliable and practical assay for thyroid stimulating hormone was a landmark.[9] A normal range of <0.5-4.2 mU/l was established, based on measurements from 29 control subjects. One of the first applications of the assay was in patients who had undergone subtotal thyroidectomy for Graves' disease.[10] In 28 "unequivocally euthyroid" patients followed for three to 21 years, the mean concentration was 8.2 mU/l (range 1.3-34.0 mU/l). In four patients followed up for four to 12 years and in whom a therapeutic trial of thyroxine had shown no benefit, the thyroid stimulating hormone concentration range was 10.5-21.5 mU/l. These patients were considered to be unequivocally euthyroid by a group who had validated clinical indices for the diagnosis of hypoparathyroidism and hyperthyroidism.[5,7] They were used to show the superiority of thyroid stimulating hormone measurements in detecting hypothyroidism, and no suggestion was made that the normal range could be widened.

In 1973, the data on which the concept of subclinical hypothyroidism was based were published.[11] The reference range for thyroid stimulating hormone, established from measurement in 29 subjects,[10] was used to classify 22 euthyroid subjects as having subclinical hypothyroidism. In six of the 22 subjects given a therapeutic trial of thyroxine, treatment showed no benefit, and 10 had originally been recruited as normal controls.

Whickham survey

The Whickham survey was a further landmark.[12] All Whickham residents with a serum thyroid hormone concentration >6 mU/l were diagnosed as being hypothyroid, irrespective of their clinical status. This reinforced the view that the serum thyroid stimulating hormone concentration defined hypothyroidism.

The 20 year follow up study of the Whickham survey has yielded invaluable data on the natural history of thyroid disorders.[13] A main conclusion of the study, disseminated to most non-specialists in a review published in the BMJ, was that "thyroid stimulating hormone concentrations above 2 mU/l are associated with an increased risk of hypothyroidism."[2] Half of the population (male and female) fall into this category.[12] This conclusion was based on the change in the slope of the line obtained when the log of the serum thyroid stimulating hormone concentration was related to the

Relation between concentration and risk

The equation to describe the relation between the probability of developing hypothyroidism and the serum thyroid stimulating hormone concentration is[13]:

$\ln\{P/(1-P)\}=b_0+b_1\ln$ thyroid stimulating hormone+0.027age (+1.79 if antibody positive).

$b_0 = -5.02$, $b_1=0.30$ if thyroid stimulating hormone <2 mU/l

$b_0 = -6.38$, $b_1=1.97$ if thyroid stimulating hormone ≥2 mU/l

logit probability of developing hypothyroidism over a 20 year period in women (see box).[13] The probability of a 40 year old woman with a thyroid stimulating hormone of 2.1 mU/l developing hypothyroidism is low - at 1 in 50 over 20 years. In men, the probability is so low that an equivalent equation could not be derived.[13]

Clinical features ignored

The review also highlighted the fact that in making a diagnosis of clinical or overt hypothyroidism "symptoms are not considered a criterion by some authorities."[2] The review claimed great authority. It was pointed out that some of the data on which it was based had been collected for the consensus statement for good practice and audit measures in the management of hypothyroidism and hyperthyroidism published on behalf of the Royal College of Physicians of London and the Society for Endocrinology.[14] This publication makes no reference to the clinical manifestations or clinical diagnosis of hypothyroidism. Thus, the clinical features of hypothyroidism seem to have been relegated to the status of historical curiosities.

Hyperthyroidism

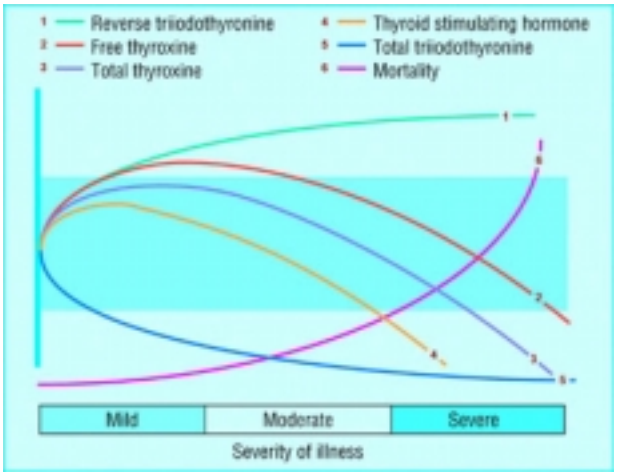
Assays capable of defining the lower end of the statistically derived reference range became available in the early 1980s. One evaluation of such an assay reported that all of 110 hyperthyroid patients studied had a thyroid stimulating hormone concentration <0.07 mU/l, and all 62 euthyroid control subjects had concentrations >0.07 mU/l.15 However, some clinically euthyroid subjects with abnormally low thyroid stimulating hormone concentrations were classified as having subclinical hyperthyroidism.[15] Assays can now detect thyroid stimulating hormone in serum at concentrations of 0.005 mU/l.16 At this low concentration, hyperthyroid patients were not distinguished from some euthyroid, though ill, patients.[16] The range of thyroid stimulating hormone concentrations in patients whose condition stabilised on thyroxine replacement treatment was <0.005 to >10.00 mU/l.16 It is therefore clear that measurement of the thyroid stimulating hormone concentration has failed to deliver what was expected of it.

Clinical aspects

During this period the clinical aspects of hyperthyroidism have also been downgraded. Most current undergraduate textbooks treat the clinical diagnosis of thyroid dysfunction by referring the student to lists. In the current edition of the Oxford Textbook of Medicine, this matter is dismissed in less than a line, and the reader is referred to unweighted lists of the symptoms and signs.[17] In the popular postgraduate textbook of Clinical Endocrinology, the biochemical diagnosis and assessment of hyperthyroidism are given before the clinical features.[18] Medical journals are now effectively devoid of references to the clinical features of hyperthyroidism. Though a symptom rating scale for the diagnosis of hyperthyroidism was described in 1988,[19] the clinical scoring systems for assessing hypothyroidism and hyperthyroidism are now rarely cited (table).

Citation frequency (in BIDS) of published papers on the clinical assessment of hypothyroidism and hyperthyroidism in relation to UK groups and worldwide,1987-97

First author	Number of citations: Worldwide	UK groups
Murray[4]	0	0
Billewicz[5]	20	2
Wayne[6]	9	1
Crooks[7]	0	0
Gurney[8]	6	0
Klein1[9]	28	0



The effects of illness on the concentrations of thyroid hormones: the shaded area represents the reference range for each method (reproduced with permission from Beckett and Wilkinson[20]

Non-thyroidal illness syndrome

We have recently become aware of the complexity of the effects of non-thyroidal illness on the hypothalamic-pituitary-thyroid axis and thyroid hormone metabolism. Figures like the one shown (taken from a recent review[20]) are frequently used to illustrate the nature of the changes that occur in serum thyroid hormone concentrations in the non-thyroidal illness syndrome. These figures have never been published with a numerical scale or error bars. The problem of interpreting free thyroxine was summarised by the author: "It is common to find that a sample obtained from a patient with non-thyroidal illness syndrome may have a raised free thyroxine by one method but a normal or low free thyroxine by another."[20] The equilibrium dialysis reference method used to profile free thyroxine in the figure is technically demanding and currently not established in the United Kingdom. As the original legend to the figure explains:

The profile for free thyroxine is that obtained using equilibrium dialysis and low sample dilution. The level of free thyroxine found using commercial methods will be heavily method dependent. A profile of free triiodothyronine is not included as some ultrafiltration methods suggest that normal or raised free triiodothyronine may be found in illness whilst equilibrium dialysis methods usually show diminished or normal concentrations.[20]

What free thyroxine and free triiodothyronine assays actually measure is controversial.[21] However, what is clear is that we cannot interpret thyroid function tests in systemically ill patients.

Current status of thyroid function tests

Our understanding of the complexity of the cerebral-hypothalamic-pituitary-thyroid axis and the mechanism of thyroid hormone action has grown enormously. Current knowledge indicates that the cardiac effects of thyroid hormones, which are clinically very important, are mediated via the [alpha]$_1$ thyroid hormone receptor independent of the [beta] receptors, which are the dominant regulators of thyroid stimulating hormone secretion.[22]

False positive and negative results

Overlap between the statistically derived normal and abnormal ranges is accepted in diagnostic tests, giving rise to false positive and false negative results. These concepts have not been applied to measurements of thyroid stimulating hormone. Rather than accepting that the test can be fallible, we transfer the problem to the patient. In patients with systemic disease, the non-thyroidal illness syndrome is invoked to explain the anomalous results, and healthy subjects are diagnosed as having subclinical hy-

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Over To You

This section of the newsletter has proved to be very popular. We are keen to have more members' stories. Please consider helping us to help other readers by jotting down **your** story and sending it to us. If you find the prospect daunting. please call us and we will help. The stories will be published anonymously unless you ask to be named. So, Over To You!

The views expressed in this section are not necessarily those of Thyroid Australia. Check all treatment options with your doctor.

Hashimoto and Me

My perception of living with undiagnosed postpartum thyroiditis

"You're probably still depressed, but we'll do a blood test anyway."

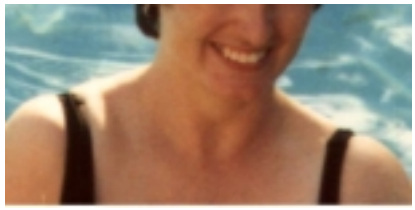
That was how my GP informed me in November 1994 that my exhaustion could be due to something other than depression. When I rang to get the test result, I was told that there was nothing to worry about, but my TSH level was marginally up (5.9 mIU/L - Ref. range: 0.5-4.0 mIU/L). I needed another test to see whether I had thyroid autoantibodies, which I did (Positive thyroid microsomes. Titre 6400). I had Hashimoto's thyroiditis. What did this mean for me? Nothing much, but I needed to take thyroxine for the rest of my life. When I next saw my GP, I could get a prescription for my medication. I asked him if I could collect the script that evening, so that I could start taking the tablets immediately. What a relief! There was actually something wrong with me. Just knowing it could be remedied gave me an emotional lift.

I was at that stage unaware that I had displayed symptoms of hypothyroidism over many years:

- My husband complained that I wanted too many blankets on our bed, and that he was getting too hot. My baths and showers were also always hotter than he could stand.
- In June 1989 I had a D & C, to try to find the reason for my heavy and irregular periods. I had another such procedure in August 1994. No gynaecological problems were ever found. No thyroid tests were ever suggested.
- In 1991 my daughter, then in Grade 3, had to write about her mother. Her friend wrote: "My mother likes being a mum." My daughter wrote: "My mother likes to sleep."
- I had even asked my GP about the pins and needles in my fingers. He replied that it was carpal tunnel syndrome, and that I should not worry about it. Pins and needles on waking in the morning can be symptomatic of hypothyroidism.

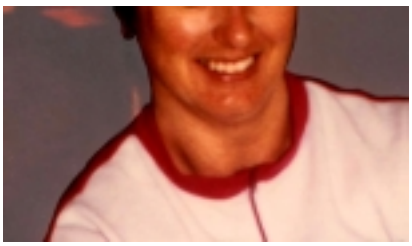
But depression had been my major affliction. I suffered two nervous breakdowns, and consulted four psychiatrists between 1986 and November 1994.

My first nervous breakdown occurred in August 1986 when my son was ten months old. I had been feeling down for



The first signs of a goitre are visible three months after my son's birth

some months, was suffering from sinusitis, feeling rotten and had to care for two small children. I longed to curl up in bed, but I couldn't. I needed mothering, but I was the mother. Unfortunately I cracked. My GP suggested that I should see a psychiatrist, who said: "You're tense and



My goitre is now quite noticeable, nine months after my son's birth and about a month before my nervous breakdown.

anxious, and you need to relax more." He didn't ask to see me again.

This breakdown savaged my self-confidence. It took me years to learn to trust myself again.

Thyroid problems commonly present between 3 and 6 months after a baby is born. About 5-9% of women, especially those with thyroid antibodies present during pregnancy, develop some thyroid problems after giving birth. My thyroid function, however, was never tested.

Recently, while going through some old photographs, I came upon some of me taken shortly after my son's birth and about a month before my breakdown. It seems to my untrained eye that I had quite a distinct goitre at that time which was never picked up.

Shortly after this we moved from Melbourne to Brisbane, where the temperature agreed with me more. However, I felt alone and isolated in the new city. The psychiatrist I saw was terrific. He helped me out of my own private hell, that pit full of dragons which seemed only to have a downwards spiral. I asked him whether my depressive state could have a hormonal component and he referred me to an endocrinologist, who, without doing any tests, declared that I was fine.

In March 1989 I had an operation. Six days later I visited a friend. I didn't feel great, but the coffee went down well. After a while I found I could hardly move. My friend took me to see my GP. He examined me, took my temperature and blood pressure, and declared that I was suffering from 'psychogenic shock'. No other tests were ordered. I was stunned. They lent us a wheelchair to get me back to her car. I have since been told me that operations can trigger thyroid problems. (Surgery can cause a decrease in serum Total T3 and Free T3.)[1]

We were transferred back to Melbourne in October 1990. Even though we had lived there before, I still felt below par, but knowing my 'history' of 'depression', I once again visited a friendly psychiatrist. He helped me, and even suggested I attend a stress management course. But my enjoyment of life was marginal, even though I had a great husband, great kids, a lovely home, and no financial worries. And my sister and her two sons were coming to stay for eight months in 1992.

My sister was recovering from lymphoma, and the chemotherapy and radiation treatment. It was not a good visit, even though she ostensibly made a good recovery. I was tired and irritable all the time.

Then in May 1993, five months after she left us, but before I could really re-establish a good relationship with her, she died of leukaemia.

I was devastated, and asked my GP if I could see another psychiatrist, whom I found to be a wonderful help. But in August that year I had what can only be described as another nervous breakdown, amidst the chaos of grief.

I had by now gained fourteen kilos in two and a half years. Okay, people with hypothyroidism apparently only put on about four kilos a year, but I had been through rather a difficult time.

Then friends in Sydney invited us to share Christmas 1993 with them. We had a wonderful, relaxing holiday, despite my increasing nervousness in the car. I was not comfortable in the car because, I think, my reaction times were now that much slower than my husband's and I would startle him with late reactions. When we visited Darling Harbour I could hardly walk. My memories of the Powerhouse Museum are of me standing in a corner, exhausted and crying. Driving back to Melbourne through the Sydney bushfires in January 1994 didn't affect me much, as I was asleep in the car most of the time. My husband visited the wineries on his own.

I retreated within myself. I had, however, recently started my Master's degree. I completed two semesters, then found I couldn't cope as I was too exhausted, so I took a break. I also felt a lump in my throat when swallowing. The ENT specialist told me it was 'globus hystericus' (a lump in the throat caused by hysteria), and that I should try not to swallow so often. Whether I actually had a goitre then, I do not know, but it certainly seemed to go away after I started treatment with thyroxine.

Then in November 1994 I was diagnosed with Hashimoto's thyroiditis. Life was looking brighter. In May 1995 I had another thyroid test. My GP expressed concern that my TSH level was a little low (0.3 mIU/L), but he kept my daily dose at 100 µg. In August when I went to see him with a rotten cold, he suggested that I was running the long-term risk of developing osteoporosis by taking too much thyroxine and cut my daily dose to 75 µg.[2] He did so without ordering another thyroid test.

Two weeks later I collapsed. Once again I could hardly walk. For 6 weeks I spent almost 16 hours a day in bed. The physician to whom I was referred ordered all sorts of tests, including those for thyroid function. My TSH level was now 9.72 mIU/L. High, but not excessive. Two months later, after my daily dose had been doubled to 150 µg, my TSH level was 0.21 mIU/L. I don't know what other tests were ordered, but none was positive. The physician was convinced that my problem lay with my 'psychological state'. He concluded my difficulties were a direct result of stress caused by my much enjoyed Master's course. The GP concurred.

I no longer consult that GP or that physician. I feel that they treated me condescendingly, and that their assessments were incorrect. But I think I understand why they latched on to my 'psychological state' as a reason for my symptoms. Firstly, the GP did not want to admit that he had, in my opinion, erroneously lowered my dose; and secondly, I had this wonderfully long history of 'depression' and 'anxiety', as documented by my numerous visits to psychiatrists and other medical experts, which they could use as a hook on which to hang their hats.

At this stage, however, I insisted on seeing an endocrinologist, who has kept my daily thyroxine dose at 150 µg.

So, where did this leave me? My faith and trust in doctors was severely dented. I also felt frustrated and angry that my condition had taken so long to be diagnosed, when I had concurrently presented with a number of symptoms over the years: heavy menstrual bleeding, depression, tiredness, weight gain, a lump in my throat, haemorrhoids caused by constipation, pins and needles in my fingers, difficulty in walking, and sensitivity to cold.

I have now been on a stable dose of thyroxine since September 1995. My thyroid function levels are fine (my TSH is still low, and my T4 and T3 levels are at the upper end of normal). Physically I feel better than I have for years, but mentally there were scars. Hashimoto's thyroiditis may not be life-threatening (except in extreme cases), but it certainly affects one's quality of life. I feel that I was not as actively involved in my own activities, those of my husband, or those of my children as I would have liked to have been. We cancelled holidays and social outings because I was just too tired. The effect of the condition on my relationships was immense: my husband buried himself in his work; my daughter became rather aloof (or maybe that's just being a teenager); my son was convinced that I had never wanted him as I was always so tired and distant; my sister died not knowing I was ill; and my parents and my brother didn't quite know what to make of me - it seemed as if they felt that my personality had taken a turn for the worse for good. It is also difficult to sustain friendships when you are feeling perpetually tired and irritable. So I am quite terrified of becoming ill again. I try very hard to control my condition, instead of having it controlling me.

Like all Hashimoto's thyroiditis sufferers, I will never know when the condition first struck - maybe in my teens - but probably after I had my children. I reckon I had had it for at least nine years before I was diagnosed. I understand that the effects of undiagnosed hypothyroidism come and go, and that it is essentially difficult to diagnose, but it would have been helpful to know sooner. My family and I would then have been spared a great deal of anxiety, stress, medical treatment, and expense. I have at times felt that my husband and children were robbed of an effective wife and mother for many years.

I believe in obtaining reliable information about my condition so that I can learn how to live with Hashimoto, my autoimmune disease. I believe that awareness of thyroid conditions should be raised amongst the general public and doctors alike. It would be good if people could be diagnosed earlier, so that they do not have to wander about in the medical wilderness for years, as I feel I have had to.

(PS I submitted my Master's thesis in June 2000, and am now awaiting the result. It took me six and a half years to complete a four year course. My thyroid condition contributed in no small measure to the length of time it took. Submitting brought with it a great sense of relief - and a feeling that this difficult period of my life was now behind me.)

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2. It is debateable whether thyroid hormone treatment can lead to osteoporosis. A study presented at the World Congress on Osteoporosis by Dr Martin Stenstrom of the University of Gothenburg suggests that thyroxine treatment does not affect bone mineral density. (W A Thomasson, 'Thyroid supplementation does not increase osteoporosis risk', http://www.pslgroup.com/dg/1D62E2.htm, Accessed 24 July 2000.)

pothyroidism or hyperthyroidism.[11,15] The distribution of the serum thyroid stimulating hormone concentration in the population is logarithmic.[13] Thus, minor deviations from the statistically derived reference range are unlikely to be clinically meaningful.[11]

Confusion

Studies in 1580 inpatients[23] and in 630 patients admitted as medical emergencies[24] found that thyroid function tests performed as screening tests yielded abnormal results in 33% and 20% of patients respectively. In both studies, the biochemical tests suggested thyroid disease incorrectly (that is, they gave false positive results) in nine cases out of 10. Thus, indiscriminate use of thyroid function tests is more likely to confuse than to help.

We do not know how important the thyroid function tests are for making a diagnosis of thyroid dysfunction. It is a matter of personal judgment. Experience has shown that thyroid function tests, like all the signs and symptoms associated with hypothyroidism and hyperthyroidism, are not totally reliable. As it becomes clear that biochemical assessments cannot deliver the diagnostic accuracy expected of them, the fact that the clinical aspects of assessing thyroid dysfunction are being sidelined is a cause for concern. Doing more biochemical tests will lead to further confusion, not the hoped for clarity. The information obtained from thyroid function tests, despite its quantitative numerical appearances, is "soft." How soft has yet to be established.

I thank Dr David Lyon for mathematical help, Dr Ann Wales for obtaining the citation data given in the table, and Drs G H Beastall and H G Gray for constructive comments and discussion.

Competing interests: None declared.

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(Accepted 3 November 1999)

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Originally published Br Med J 2000;320:1332-4. Published with permission from the BMJ Publishing Group.

UPCOMING MEETINGS

Morwell Community Health Centre
14 October
(Workshop)

Royal Melbourne Hospital
18 November
Dr Anthony Hall will discuss Thyroid Eye Disease. The venue is the Ewing Lecture Theatre, 5th floor Clinical Sciences Building. Enter via the Melbourne Private Hospital on Royal Parade.

Both meetings will begin at 2.00 pm and will finish at 5.00 pm

Please diaries these dates.

We have received acceptances from Dr Meredith Taylor to discuss weight management and Dr Heather McCormack to discuss the mental aspects of thyroid dis-ease and coping with a chronic condi-tion. We also have indications from other doctors that they are willing to speak, but have not yet finalised the topics. These meetings will take place in the new year and details will be published in the next *Thyroid Flyer.*

Next issue of the *Thyroid Flyer*

The next issue of the *Thyroid Flyer* will be published in January 2001. Articles or letters for publication should be sent to The Editor by 15 December 2000.

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