THYROID FLYER

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Inside

Nodules

Editorial

By **Alun Stevens**

This year has proved to be a difficult year for Thyroid Australia. I feel that it is important that I share some of the challenges with you as an introduction to the changes in services which the Board has decided on.

Thyroid Australia is primarily a voluntary organisation. We have received a few small grants, but our finances are reliant on membership fees and donations from individuals. We have managed to pay for one part time office manager over the last year, but our services depend on volunteers.

Our move into our own offices was a wonderful opportunity for us. It allowed us to centralise our operations and to provide a venue into which we could attract volunteer helpers. This did happen and we expanded our operations, but the increase in support has unfortunately not been sustained. We attracted a number of volunteers willing to help with office work, meetings, mail outs and the like. The problem is that we have not been able to attract anyone willing to take on the tasks of organising and managing these activities.

Administration has also proved to be a challenge. Our 800 odd members generate a good flow of requests, membership enquiries, renewals and general correspondence. The wider public demand for assistance generates a lot more and the demand has continued to grow. Despite the tight resources, we were just managing to keep up the service until our landlord required us to vacate our offices so that they could upgrade the facilities for disabled access. This prevented us doing any real administration for some two months.

The backlog this generated was extensive. Our resources were just able to keep up with normal demands. They were and are not sufficient to reduce this

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Thyroid Nodules and Thyroid Cancer

By **Emily J Mackenzie and Robin H Mortimer**

Thyroid nodules are very common, but have a relatively low risk of malignancy.

Thyroid nodules are common and of concern because of the risk of malignancy and hyperfunction. The incidence of papillary thyroid cancer appears to be increasing, both in Australia and worldwide.[1] While some of this increase is due to detection of small lesions by sensitive diagnostic tests such as ultrasonography, there also seems to be an increase in larger lesions. The cause of this is unknown.

Epidemiology

The reported prevalence of thyroid nodules varies with the method of screening. In autopsy series, up to 50% of clinically normal thyroid glands contain nodules. A lower prevalence is found by ultrasonography. For example, nodules were found in 27% of randomly selected people aged 19–50 years in an iodine-sufficient area of Finland; only 5% of these people had a clinically detectable abnormality.[2] Fifty per cent of people with clinically detected solitary nodules have additional nodules when studied further by ultrasonography.[3]

Nodules are more common in women and in areas of iodine deficiency. Exposure to ionising radiation in childhood and adolescence increases the risk of both nodules and thyroid carcinoma.

Reports of the risk of malignancy in thyroid nodules vary. An Italian study of more than 5000 consecutive patients with "cold" thyroid nodules reported an incidence of malignancy of 5.3% in those from iodine-sufficient areas. Risk factors for malignancy included male sex and age less than 30 years or greater than 60 years.[4]

Diagnosis

Thyroid nodules are usually found by the patient or a family member, or during a general physical examination, but increasingly they are incidentally discovered during neck imaging undertaken for other reasons, such as carotid duplex ultrasonography. It is important to determine whether the nodule is hyperfunctioning and whether it is malignant.

History is rarely helpful in differentiating benign and malignant nodules. An exception is a history of head and neck irradiation in childhood or teenage years. This is by far the greatest risk factor for thyroid malignancy, with a risk of carcinoma in nodules in this group of 35%–40%, in contrast to 5% in the general population.[5] However, such a

Abstract

- Thyroid nodules are common clinically (prevalence, about 5%) and even more common on ultrasound examination (about 25%).
- About 5% of thyroid nodules are malignant.
- Most thyroid cancers are well-differentiated papillary or follicular tumours with an excellent prognosis (10-year survival, 80%–95%).
- The incidence of papillary thyroid cancer appears to be increasing on the east coast of Australia.
- Fine-needle aspiration biopsy of the thyroid is the most cost-effective diagnostic tool.
- Recommended initial management of all follicular carcinomas and of papillary carcinomas > 1.0 cm is total thyroidectomy followed by radioiodine ablation.
- Most patients should be managed postoperatively with doses of thyroid hormone sufficient to suppress plasma levels of thyroid-stimulating hormone.

Continued Page 2

history is unusual in Australia, in contrast to countries such as the United States, where head and neck irradiation was used to treat enlarged tonsils, adenoids and acne in many children and adolescents. Another exception is a family history of medullary carcinoma of the thyroid, which should prompt consideration of familial medullary thyroid cancer or multiple endocrine neoplasia type 2, the latter being associated with hyperparathyroidism and phaeochromocytoma.

Physical examination is also rarely helpful in differentiating benign and malignant nodules, unless there is evidence of invasion of other structures in the neck (eg, hoarse voice) or enlarged regional lymph nodes, which suggest malignancy.

An algorithm for investigating thyroid nodules is shown in Box 1. This algorithm may be cut short in patients at high risk of cancer.

Biochemical assessment

Measurement of thyroid-stimulating hormone (TSH) level is helpful, as suppression suggests that a nodule is hyperfunctioning. Some of these patients will also have a raised level of serum free thyroxine (T4). Hyperfunctioning solitary nodules carry a low risk of malignancy (at least in adults). When TSH level is normal, assay of serum free T4 adds no further information. A raised TSH level in an elderly patient with a large, rapidly growing thyroid mass suggests thyroid lymphoma.

Serum calcitonin level should be measured if there is a family history of medullary carcinoma of the thyroid, as a raised level is both sensitive and specific for this cancer. However, this measurement is not cost-effective in the initial evaluation of nodules.

Thyroid imaging

Thyroid imaging should not be routine, but should be used to answer a specific clinical question.

Thyroid scanning using pertechnetate (^{99M}Tc) (and formerly radioiodine, ^{131}I) was traditionally used to screen thyroid nodules for malignancy. The finding of a hyperfunctioning or "hot" nodule (uptake of tracer within the nodule with suppression of uptake in the surrounding normal thyroid tissue) excludes malignancy in almost all patients. Current recommendations are that patients with TSH suppression should proceed to pertechnetate scanning to confirm that the nodule is hot. The patient can then be treated with therapeutic radioiodine or lobectomy. A non-functioning or "cold" nodule was thought to indicate increased risk of malignancy. Unfortunately, most nodules are cold, but perhaps only 5%–15% of these are malignant. Pertechnetate scans are thus not advocated for assessing a nodule in patients who are euthyroid or hypothyroid.

Thyroid ultrasonography is commonly performed. Although some ultrasonographic features, such as punctate calcification and irregular or blurred margins, suggest papillary carcinoma, routine ultrasonographic studies rarely aid clinical decision-making.[6] Despite early suggestions that nodules in multinodular goitres are less likely to be malignant, more recent studies show that the risk of malignancy in a dominant nodule in this condition is similar to that in a solitary nodule.[7]

Computed tomography is rarely necessary, but can be useful in delineating the extent of disease in patients with known thyroid cancer. It should be remembered that use of iodinated contrast medium reduces technetium uptake and may reduce the diagnostic utility of pertechnetate scanning.

Fine-needle aspiration biopsy

In the absence of TSH suppression, biopsy by fine-needle aspiration should be the first-line investigation for a solitary nodule or a dominant nodule in a multinodular goitre. An exception may be an "incidentaloma" (incidentally discovered small thyroid nodule) (Box 2).

The success of a biopsy depends on the adequacy of the specimen and skill of the cytopathologist. In experienced hands, the false negative rate is less than 5%, and the false positive rate less than 1%. The procedure can be done in ambulatory patients without local anaesthesia. Discomfort is usually mild. Specimens can be taken from several parts of the nodule, and slides smeared immediately. Immediate staining of some slides allows evaluation of the adequacy of material while the patient is still present and further passes if material is inadequate. There is a small risk of bleeding, which settles quickly with ice packs. Ultrasound guidance is preferred by some practitioners and is needed for impalpable lesions that warrant fine-needle aspiration biopsy.

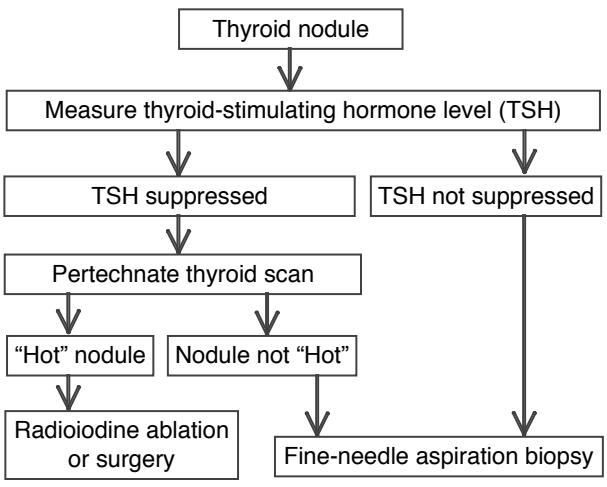
Larger thyroid nodules are often partially cystic, containing fresh or altered blood. Aspirates often contain large numbers of macrophages, but follicular cells may be sparse. A specimen is regarded as adequate for diagnosis if it contains six to 10 clusters of thyroid cells. The more cystic the lesion, the more likely the specimen is to be non-diagnostic. A further biopsy attempt under ultrasound guidance is warranted, yielding a diagnostic sample in 63% of nodules.[12] The remaining patients should not be considered to have benign disease, but even repeated aspiration may not obtain a sufficient sample in 5%–10%.[13]

The cytopathological report should be one of the following: insufficient for diagnosis; malignant; atypical; a follicular neoplasm; or benign. The presence of colloid and bland follicular cells suggests a benign lesion. The report should indicate adequacy of the specimen, as malignancy cannot be excluded if material is insufficient.

Papillary and often medullary carcinoma can be recognised on fine-needle aspiration biopsy (Box 3), but follicular adenomas and carcinomas have similar cytological appearance, with diagnosis of malignancy requiring demonstration of capsular or vascular invasion in paraffin sections. Follicular tumours are thus reported initially as "follicular neoplasms" and cannot be characterised as benign or malignant without histological examination. All follicular neoplasms should be excised unless there is clear evidence that the nodule is hyperfunctioning (see case report, Box 4).

Atypical cytology is found in 20% of specimens, and about a third of these lesions are found to be ma-

1: Algorithm for assessing a thyroid nodule



lignant when excised and examined histologically.

Indications for thyroid surgery thus include malignant or atypical cytology, follicular neoplasm, and failure to obtain a diagnostic sample. In the future, molecular genetic studies of aspirated material (eg, for the *RET*/*PTC* gene rearrangements often found in papillary cancer) may offer more precise diagnosis.

2: The "incidentaloma"

Incidental discovery of a small thyroid nodule is an increasing clinical problem. A small but significant proportion of these nodules are malignant, but there is persuasive evidence that they are rarely clinically significant. Occult thyroid cancers are commonly found at autopsy (mean, 36 cases per 1000 persons), while thyroid carcinoma has an incidence of 0.5–10 cases per 100 000 population,[8] implying that most thyroid cancers do not become clinically apparent.

Recent guidelines for management of incidentally found thyroid nodules do not advise needle biopsy unless diameter is > 1 cm.[9] However, many patients are concerned about the possibility of malignancy and request biopsy.

Benign findings are reassuring, as the subsequent risk of malignancy in that nodule is less than 1%. It is prudent to follow up patients with thyroid nodules that are benign on biopsy or have not undergone biopsy. In a small series of incidentally discovered lesions < 1 cm, no malignancies appeared after 5 years.[10] Although nodules that enlarge are not necessarily malignant,[11] most thyroid specialists would perform another biopsy.

Management

Benign thyroid nodules

Thyroxine suppressive therapy may be used to treat benign nodules. The rationale is that nodule cells may remain responsive to TSH. Indeed, two meta-analyses suggest that thyroxine treatment may retard growth and sometimes shrink nodules in 25% of patients. [14,15] Suppressive therapy should not be considered until all nodules have been shown to be benign by biopsy. Given the increased risk of cardiac arrhythmia and evidence that subclinical hyperthyroidism can lead to loss of bone mass in postmenopausal women,[16] TSH suppression should be used cautiously in older patients.

Well-differentiated thyroid cancer

Well-differentiated thyroid cancers arise from follicular cells and encompass both papillary and follicular carcinomas. Papillary carcinomas are the most common thyroid cancer. They are unencapsulated, often multicentric, and bilateral in a third of cases. The most common sites of metastasis are regional lymph nodes and, less commonly, the lungs.

Follicular carcinomas represent 5%–10% of thyroid cancers. They are encapsulated, and the feature of malignancy is invasion of the capsule. Prognosis depends on the degree of invasion and differentiation, with poorly differentiated tumours having poorer outcomes. A subtype, the Hürthle-cell tumour, with eosinophilic mitochondria-rich cells, also has a poorer outcome. Follicular carcinomas can metastasise to bone, causing lytic lesions.

Well-differentiated thyroid carcinoma has an excellent prognosis, despite the presence of lymph-node metastases in 5%–20% of patients and distant metastases in 10%–15%. Ten-year survival is 80%–95%. Factors that worsen prognosis include male sex, advanced age, large tumour, poor differentiation, local invasion and distant metastases.[8]

Management

Surgery: Initial management is surgical, with "total" thyroidectomy (postsurgical radioisotope scanning usually shows residual thyroid tissue) indicated for any malignancy with diameter > 1 cm. Thyroidectomy should be performed by an experienced surgeon, as this reduces complications, such as hypoparathyroidism and recurrent laryngeal nerve palsy. Although subtotal thyroidectomy has lower complication rates, thyroid remnant ablation with radioiodine is less successful when large amounts of thyroid tissue remain.

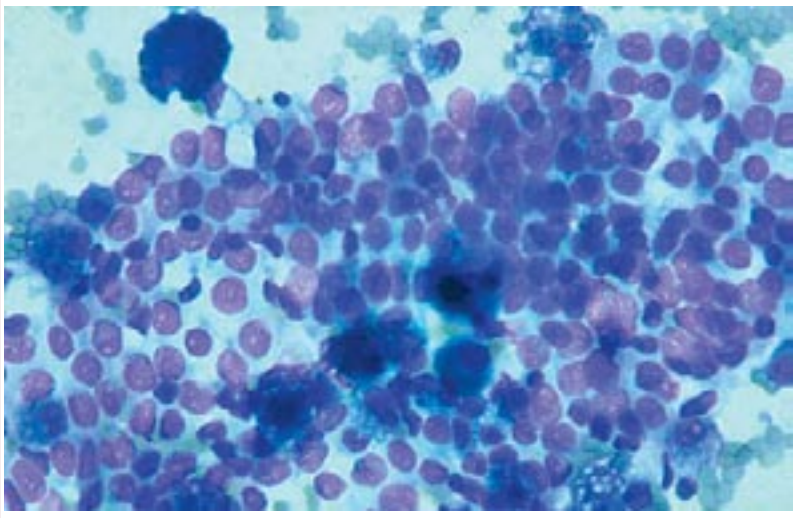
Remnant ablation: Retrospective studies indicate reduced recurrence rates and improved survival (at least in patients with follicular cancer or papillary cancer larger than 1 cm) when thyroid tissue remaining after surgery is ablated with orally administered radioiodine. This may not only destroy cancer cells in the neck and elsewhere, but also improve specificity of future radioiodine whole-body scans and serum thyroglobulin assays by removing normal thyroid cells.[17] Radioiodine doses of 4000 MBq are often used for postsurgical ablation in Australia, but lower doses are often adequate. Ablation is most effective when endogenous TSH levels are maximal, usually 3–4 weeks after total thyroidectomy. Admission to a specific isolation room is required for 2–3 days, after which radioiodine levels have usually fallen to levels that permit discharge. It is usual practice to perform a whole-body scan at the time of discharge, using the therapeutic radioiodine dose as the imaging agent. This usually demonstrates uptake in the thyroid bed, salivary glands, gastrointestinal tract and bladder. Uptake in other areas indicates metastatic disease (Box 5).

Follow-up

An algorithm for follow-up after treatment of differentiated thyroid cancer is shown in Box 6.

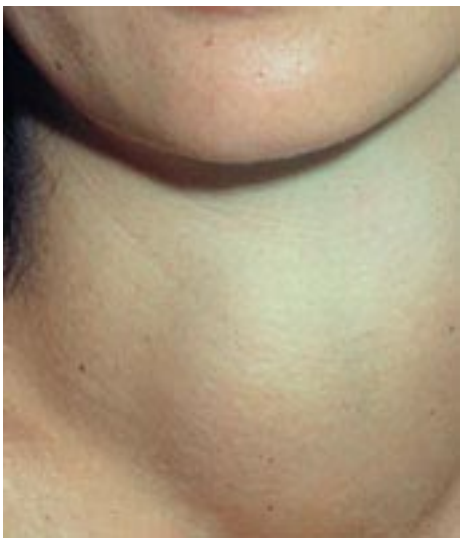
Clinical and biochemical follow-up: Clinical examination of the thyroid bed and regional lymph nodes may detect recurrence. Serum thyroglobulin levels are used to follow up patients treated by thyroidectomy and radioiodine ablation of the thyroid remnant. Tumour cells generally retain the ability to synthesise thyroglobulin in response to TSH stimulation. Unfortunately, a significantly elevated thyroglobulin level does not predict uptake of radioiodine and, conversely, tumour which concentrates radioiodine may produce insignificant amounts of thyroglobulin.[18] Recombinant human TSH (rhTSH) maximally stimulates thyroglobulin secretion by re-

3: Papillary thyroid carcinoma



Fine-needle aspirate from a papillary thyroid carcinoma, showing cells with crowded and overlapping nuclei with inclusions (haematoxylin and eosin stain).

4: Case report – managing a thyroid nodule



Presentation: A 38-year-old woman presented to her general practitioner after noticing a swelling on the left side of her neck which had gradually enlarged over the previous 18 months. She was otherwise well but concerned that she might have cancer. There was no relevant family history.

Examination: There was a 5 cm firm smooth mass on the right side of the larynx that moved with swallowing. No other abnormality was found.

Investigations: Thyroid function tests gave normal results. An ultrasound study confirmed a 48 mm solid mass in the right thyroid lobe. The remainder of the gland appeared normal. Fine-needle aspiration biopsy showed clumps of follicular cells but no colloid, consistent with a diagnosis of a follicular neoplasm.

Management: The patient was referred to a thyroid surgeon, who informed her that the nodule was probably benign but could be a follicular thyroid cancer. He recommended surgery and performed a right hemithyroidectomy. Frozen section of the 5 cm nodule showed a well-differentiated follicular lesion that was reported as an adenoma. However, examination of a paraffin section the following day showed capsular and vascular invasion by follicular cells. The diagnosis was revised to follicular cancer, and the surgeon advised removal of the left thyroid lobe, which was performed the following day.

The patient was reviewed by an endocrinologist, who recommended radioiodine ablation of remaining thyroid tissue. This was scheduled for a month later. In the interim, the patient took no thyroid hormone replacement therapy.

Plasma free thyroxine level fell to 2 pm/L (reference range [RR], 9–23 pmol/L) and plasma thyroid-stimulating hormone level rose to 94 mU/L (RR, 0.3–5 mU/L). After a negative pregnancy test, the patient was admitted to a radiation isolation room and given 4000 MBq of radioiodine.

Two days later, she underwent a whole-body gamma-camera scan, using the therapeutic radioiodine dose as the imaging agent. This showed a small amount of residual tissue in the thyroid bed and physiological uptake in salivary glands, stomach and bladder. There was no evidence of metastatic thyroid cancer.

The patient was discharged with thyroid hormone replacement therapy and returned home. To minimise exposure of her family to the remaining radioiodine, she avoided physical contact with her husband and children for the next week.

Course: The patient was reviewed every 3 months over the next year by the surgeon and endocrinologist. She remained clinically free of disease. Her thyroid function was also measured 3-monthly, and the dose of thyroxine adjusted to suppress plasma TSH levels. Serum thyroglobulin levels remained unmeasurable.

A year later, thyroxine was withdrawn for one month, and a whole-body radioiodine scan was performed. This showed physiological uptake and disappearance of the previously noted thyroid bed uptake.

Comment: *The patient had a large, well-differentiated follicular thyroid cancer, which has a good prognosis. However, there is a small risk of recurrent disease, and she will require long-term follow-up.*

sidual or recurrent thyroid cancer and, when given by injection, increases the sensitivity of serum thyroglobulin measurements in detecting recurrent disease. It is as yet unclear under what conditions rhTSH, an expensive drug, will be made available in Australia.

^{131}I whole-body scanning: Many centres repeat a whole-body scan 12 months after surgery and remnant ablation. We reserve this scanning for patients with measurable thyroglobulin levels and those with thyroglobulin autoantibodies, in whom thyroglobulin levels cannot be measured. TSH levels greater than 30 mIU/L are needed to maximise iodine uptake by residual or recurrent thyroid cancer. This is achieved by withholding thyroxine for 4 weeks, often with substitution of triiodothyronine for the first 2 weeks of the period to minimise symptoms of hypothyroidism. Alternatively, rhTSH injection allows whole body scanning without stopping thyroxine therapy. Care must be taken to ensure that no iodinated contrast medium has been administered in the 2 months before whole-body scan, as this reduces the sensitivity of the scan.

The sensitivity of scanning depends on the dose of ^{131}I administered, but higher doses may reduce subsequent radioiodine uptake by tumour cells ("thyroid stunning"). Scans done after therapeutic doses of radioiodine (4000–6000 MBq) are the most sensitive.

Other imaging: Where there is evidence of metastatic disease (eg, raised thyroglobulin level) but no abnormal ^{131}I uptake is detected on whole-body scan, ultrasonography or computed tomography may locate a tumour mass. Positron emission tomography using fluoro-18-deoxyglucose may detect lesions not seen on radioiodine scanning, and evidence is emerging that this is a negative prognostic indicator.[19] Use of this technique has become common overseas and is increasing in Australia.

Treatment of metastatic disease

In metastatic disease, the tumour should be debulked by an experienced surgeon where practical, and radioiodine administered either after surgery or as the primary treatment. Thyroxine suppressive therapy must be resumed after radioiodine therapy and can slow the growth of unresectable disease.

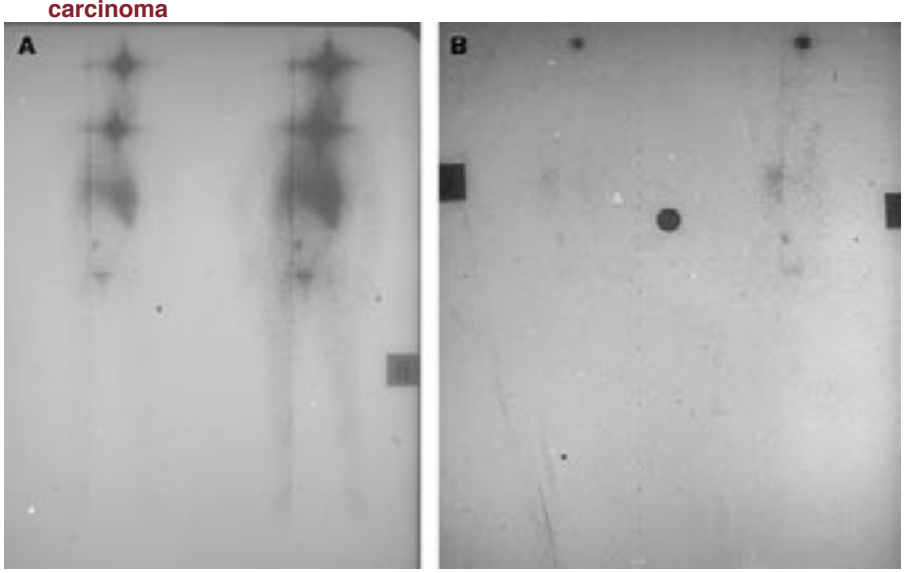
Medullary thyroid carcinoma

This tumour arises from the neuroendocrine C-cells of the thyroid and manufactures calcitonin, with the preoperative level correlating with the burden of disease. Fifty per cent of patients have lymph node metastases at presentation. Primary management is surgical. Thyroxine is needed as replacement rather than suppressive therapy, as the tumour cells are not under the influence of TSH. Prognosis is less favourable than for well-differentiated cancers, but, even so, survival is 86% and 78% at 5 and 10 years, respectively. Patients with distant metastases have only 36% 5-year survival.

Optimal management of metastatic disease is controversial and ranges from observation to aggressive surgery and chemoradiotherapy. Survival benefit with aggressive therapy is unclear.[20]

Medullary thyroid carcinoma represents just 5%–10% of thyroid carcinomas but is familial in up to 20% of cases, associated with the *RET* proto-oncogene. No mutation can be identified in 2% of affected families, while a germline mutation is discovered in 5%–6% of those with no family history.[21] As implications for other family members are significant, most experts recommend genetic testing after appropriate counselling of all patients presenting with this carcinoma. Current guidelines advocate thyroidectomy in early childhood for known carriers of *RET* mutations. Patients must then be screened periodically for other features of the syndrome, such as hyperparathyroidism, pituitary disease (multiple endocrine neoplasia [MEN] type 1), and phaeochromocytoma (MEN2).

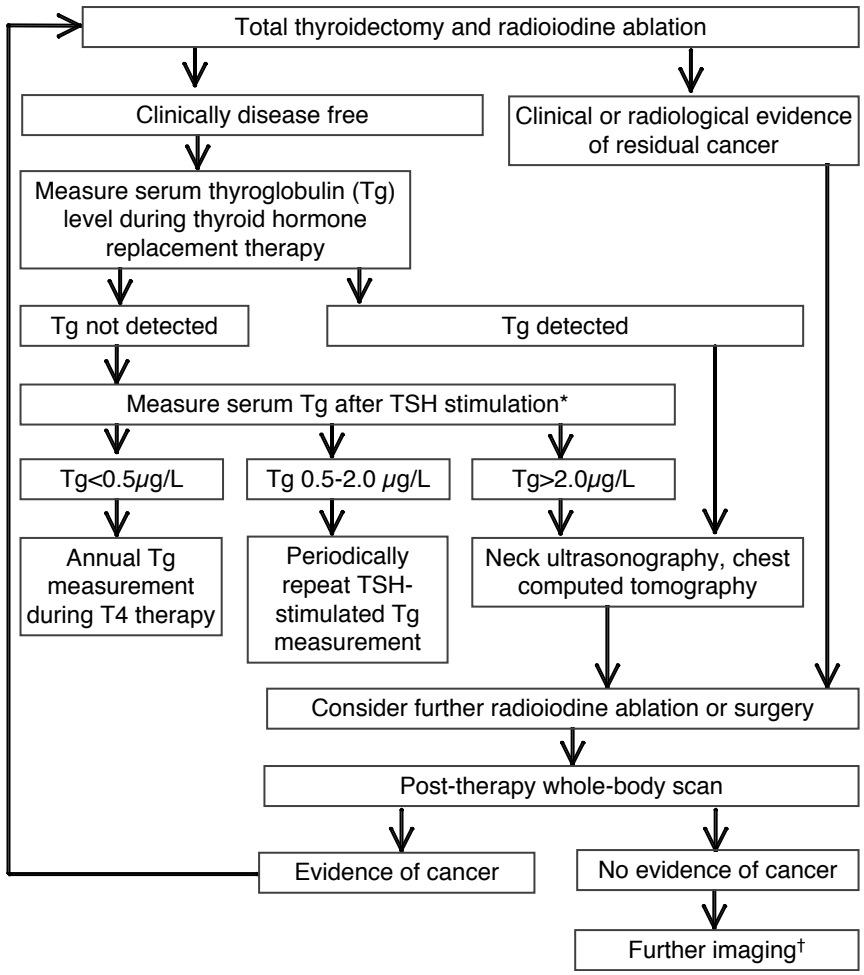
5: Radioiodine whole-body scan of a patient with metastatic follicular carcinoma



A. Scan (anterior views) after thyroidectomy, showing residual disease in the neck and evidence of metastases in the skull and pelvis. The liver is visualised as the cancer synthesises radiolabelled thyroid hormone that is metabolised in that organ. The bowel is visualised as the radiolabelled hormone is excreted in the bile, while the bladder is visualised as free radioiodine is excreted by the kidneys.

B. Repeat scan 6 months after radioiodine ablation, showing some radioiodine uptake in the skull and pelvis, but much less than at the time of treatment. Liver and bladder are again visualised.

6: Algorithm for follow-up after treatment of differentiated thyroid cancer.



T4=thyroxine. TSH=thyroid stimulating hormone

* Achieved by discontinuing thyroid replacement therapy for 1 month. Recombinant TSH has a potential role.

† To detect non-iodine avid disease

Anaplastic thyroid carcinoma

About 5% of thyroid cancers are anaplastic. In contrast to other forms of thyroid cancer, anaplastic thyroid carcinoma is one of the most aggressive tumours in humans, and death occurs at a mean of 6 months after diagnosis. An aggressive combination of chemotherapy, external beam radiotherapy and surgery is required to reduce strangulation, but the tumour is often widely metastatic and almost never curable, and therapy is aimed at palliation.[22]

Primary lymphoma of the thyroid

This entity, usually a high-grade B-cell lymphoma, tends to affect middle-aged and older patients. All patients with thyroid lymphoma have a goitre, and most report rapid enlargement.[23] Almost without exception there is evidence of lymphocytic infiltration of the gland in keeping with pre-existing Hashimoto's thyroiditis, and close to half of all patients are found to be hypothyroid. Treatment is excision of the thyroid followed by combination chemotherapy. About 50% of patients are cured, while the remainder relapse, often with generalised lymphoma.

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Hypothyroid subjects treated with L-thyroxine

Dr. Mary Samuels and colleagues at the Oregon Health and Science University in Portland, Oregon compared quality of life, mood, and memory in treated hypothyroid and control subjects. They tested for general health, mood, and did a battery of cognitive tests targeted to different aspects of memory, including working memory, long-term memory, and motor memory. They found no significant difference between the groups in any of the cognitive measures, though quality of life seemed to be somewhat less in the individuals on thyroid hormone treatment. TSHs were slightly higher in this group (2.56 compared to 1.81) and they suggested that the change in quality of life might be due to under-replacement of thyroid hormone in the patients taking this treatment.

Extract from *New Research: Reports from the Endocrine Society Annual Meeting*, New Orleans, LA, June 16-19, 2004.

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What is a Normal TSH?

At What TSH Level Should We Treat For Hypothyroidism?

Dr. Stephanie Lee of Boston Medical Center reviewed this topic which is of extreme importance to thyroid patients and the doctors that care for them. Speaking at the Endocrine Society annual meeting in June, she pointed out the normal range of TSH of 0.5-5.0 mIU/ml is outdated and wrong. Surveys of populations of individuals who have no personal or family history of thyroid disease, goiter, thyroid antibodies, or thyroid related conditions suggest that a TSH above 3.5 miu/ml is probably not normal and indicates hypothyroidism. However, treatment has not objectively been shown to benefit these individuals.

On the other hand, if such an individual has a positive test for TPO antibodies, high cholesterol or other lipid levels, symptoms consistent with hypothyroidism, or a strong family history of thyroid disease, then it may be reasonable to treat. It is also possible that thyroid hormone may benefit individuals with depression, especially if they are given lithium to treat the depression. Regardless of the decision to treat immediately, they need to be watched if they antibody test is positive, for their hypothyroidism will increase with time as shown by **Dr. M.P. Vanderpump** and colleagues who followed thyroid hormone levels in a large segment of the population of the town of Whickham in northern England over many years.

We conclude that the decision to treat borderline TSH levels between 3.5 and 5.0 miu/ml should still be the decision of the physician and the result of a careful discussion between the physician and patient, but that a TSH level of 5.0 mIU/ml seems to be the most appropriate time to begin thyroid therapy for most individuals.

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Michelangelo painted a picture of ill health

By **Jill Margo**

It is not at all surprising that when they look in the shaving mirror in the morning, most men wouldn't notice if they had a goitre. After all, the goitre on the neck of God painted on the ceiling of the Sistine Chapel was only noticed late last year – almost 500 years after Michelangelo painted it.

In the artist's image of *The Separation of Light from Darkness*, God's throat is fully exposed and reveals an over-enlarged thyroid gland, or goitre.

It was noticed by Swedish doctors, who have now written about it in the *Journal of the Royal Society of Medicine*. They say it is too clear-cut to be an accidental feature.

"Michelangelo was a perfectionist in his art, he was obsessed by anatomy, and he was no doubt familiar with the appearance of a goitre. A native of Tuscany, he spent his youth where goitres were a common sight."

He would also have seen the diseased glands in their anatomical context while dissecting corpses in his own dissecting room at the Church of Santo Spirito in Florence.

In men, a healthy thyroid gland is the size of a medium plum and is shaped a bit like a butterfly. It sits in the front of the throat just below the Adam's apple.

It produces hormones that regulate many metabolic processes and keep the machinery of the body running evenly. But, when diseased, it can become enlarged or lumpy.

When excessively enlarged, it can grow to the size of a grapefruit. Usually before it gets that big, men notice it because their collars no longer close. Although they are not gaining weight elsewhere, they notice their necks are getting broader.

A swollen goitre can also cause swallowing or breathing difficult and change the sound of the voice, by putting pressure on the small nerves that control the voice box.

Bruce Robinson, an endocrinologist and professor of Medicine at the University of Sydney, says some men eventually notice a goitre because the bodily changes can be very marked.

If the gland is overactive, it produces too much thyroid hormone and puts the man's life into fast forward. He feels revved up, anxious and speedy. He has palpitations, loses weight and knows he is in overdrive.

If it is underactive, it produces too little hormone and he feels sluggish. Everything is slowed, he gains weight, he feels constipated, he's lethargic, tired and complains about the cold.

He says many men don't notice a goitre developing because it can grow very slowly and as they see it every morning in the mirror, they get used to it. Also, if the are overweight, it can remain hidden in the fat of their necks.

If, however, it grows suddenly, in a matter of weeks or months, it could be malignant. There has been an increase in thyroid cancer in Australia which no one can explain. The good news is that the cure rate for this cancer remains good.

Although women develop thyroid disease at 10 times the rate of men, men tend to leave their goitre longer.

"If men have a goitre they need to see someone about it and, if necessary, have a biopsy to make sure it is benign. Men go into denial much more than women, and before you know it, they present with a cancer, 4 cm in diameter, which didn't need to be there," says Robinson.

He says some goitres are caused by a single lump or by several nodules within the gland. In parts of the world where people are iodine deficient, communities can develop multi-nodular goitres.

They become iodine deficient because the soil in which foods are grown is iodine depleted.

In Australia, mountainous areas along the Great Dividing Range and regions around New England in NSW have been traditionally low in iodine. Tasmania was classically deficient too and for many years iodine was added to bread. This is no longer done.

Robinson says Australian milk tanks used to be washed out with compounds containing iodine and, as a result, people got it incidentally. But this is no longer used either and figures now show the Australian population is becoming more deficient than it used to be.

"You actually only need a teaspoonful of iodine in your whole lifetime and the consequences of not having it can be devastating. A deficiency in pregnancy, for example, can mean the baby is born a cretin."

He says it is very difficult to overdose on iodine from natural sources and recommends that when you buy salt in the supermarket, you buy the iodised version, because it contains iodine, in miniscule amounts.

Fish and other seafoods are high in iodine which explains why goitre is so uncommon in places like Japan.

However, food such as cabbage, broccoli, cauliflower and soy products can interfere with the manufacture of thyroid hormone. Drugs, such as lithium, can do this too.

Because it is in the food chain, goitre is not restricted to humans. Herds of animals get it, sometimes with severe economic consequences.

The Swedish researchers suggest Michelangelo saw it in animals too and they note that in modern times, after iodised salt was made available by legislation in Italy, the prevalence of goitre in the Tuscan Apennines close to Florence, where Michelangelo grew up, fell from 60 per cent to 8 per cent.

They suggest Michelangelo had a goitre and that he actually made God in his own goitrous image.

In a satirical poem, written to a friend while struggling with the unwanted task of painting the Sistine ceiling, he described himself as being afflicted with a goitre, illustrating this statement with a caricature in the margin showing his goitrous profile.

The researchers say "our interpretation of this portrayal is that Michelangelo 'signed' his massive achievement – a backbreaking fresco of almost 588 square metres – by incorporating himself as an embodiment of the Supreme Creator in the final panel to be painted, *The Separation of Light from Darkness"*.

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Member's Story
Hypothyroidism & Coeliac Disease

I am wondering how many people who seem to have continuing symptoms of hypothyroidism, also have coeliac disease and unaware of it.

I was diagnosed with hypothyroidism in December 2000, at the age of 35. (My TSH was 25). Although I was continually tired, I had not realized there was anything 'wrong' with me. Once I found out the symptoms of hypothyroidism, I realized I had had this condition for a long time and had adjusted my lifestyle gradually in order to cope. (After all, nearly everyone of my acquaintance say they're tired). At the time of diagnosis I was actually feeling far better than a couple of years previously with a sleepless baby (now 7 years old). My second child was 10 months at the time I was diagnosed, and a far better sleeper. In addition to feeling tired, I had always had poor concentration, constipation, and 'aches and pains' which I had had investigated in my early 20s, with no resolution, and had accepted as a part of life. I was tired but coping, and enjoying parenthood.

I had an initial huge improvement to the thyroxine (100mcg) and hoped for continuing improvements (to do some of the things I had always wanted, and never had sufficient motivation). I did not get the continuing improved energy levels I hoped for. Over the next 3 years, with the support of my GP, I tried varying dosages of thyroxine, and a combination of T4/T3, finally settling on a dosage of 150mcg of thyroxine, in which my TSH was 0.6, and my T4 and T3 within the normal range. I accepted this was as good as it was going to get.

Over this time, my elder daughter (now 7) had some developmental difficulties. As soon as I was diagnosed with hypothyroidism in December 2000, this daughter also had a blood test for hypothyroidism. It was negative. In July 2003 she again had a test and the TSH was 5.23. She (like me) did not have antibodies for thyroid disease, and her pediatrician suggested the test be repeated several months later. In the meantime, my GP suggested she also be tested for a range of conditions including coeliac disease, as this may also cause developmental problems, although it is not common. My (very reluctant) daughter again had blood tests in December 2003. Her TSH was now normal but she tested positive for coeliac disease. This was confirmed by biopsy in February 2004. (She will continue to have her TSH monitored). Her energy levels and concentration have improved significantly on her gluten free diet.

After my daughter's positive blood test for coeliac disease, my husband, younger daughter and I were also tested. My husband and younger daughter were OK, but my blood tests results were very high. I also had coeliac disease confirmed by biopsy in February. The improvements in my health are not proceeding as rapidly as my daughters, although my energy is improving and I have a comfortable stomach. (The ferritin was below range but is now normal). However, the gastroenterologist said he thought it would be 1-2 years before I "know what normal is". Thus far it has only been six months. My small intestine needs to heal, and I will then absorb more nutrients from my diet.

I have learnt that many symptoms of hypothyroidism are also symptoms of coeliac disease. I wonder how many members of Thyroid Australia with continuing symptoms of hypothyroidism have had a blood test for coeliac disease. Unfortunately, I don't think a lot of GPs are aware of the wide variety of symptoms of coeliac disease. There appears to be the belief that a trial of gluten free food to test for improvements is worthwhile before a blood test. This is not true for everyone – I did not get any improvements within the first 3 weeks of my gluten free diet. I also did not have the gut pain or diarrhoea that some GPs seem to believe always accompanies coeliac disease, although I have always had a bloated stomach which I thought was normal. However, the pathology report stated that I had near total villous atrophy on the biopsies taken. In fact, a gluten free diet prior to a blood test can give a false negative result.

I think everyone with continuing symptoms of hypothyroidism, or people who are wondering if they are 'normal yet' should have a blood test for coeliac disease. There are possibly significant consequences (including an increased risk of cancer) for those people with coeliac disease who continue to have gluten in their diets, in addition to lowered quality of life.

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Scientific Review

By **Alun Stevens MSc FIAA**

T_3/T_4 Combination not more effective than T_4 alone

The use of T_3 together with T_4 instead of T_4 alone has been a matter of contention for some time. The promoters of T_3 are firm believers that therapy without T_3 is ineffective and inappropriate in all. The medical establishment are equally firm believers that the use of T_3 for long term thyroid hormone replacement is unnecessary, inappropriate and is potentially dangerous. So where does that leave the people who need thyroid hormone replacement to live and the right amount of the right stuff in order to feel well?

Thyroid Australia began covering this debate in *Thyroid Flyer* Volume 2 Number 4 in October 2001 when we devoted the whole edition to the topic of T_3. There was peer reviewed evidence that only treatment with T_3 and T_4 ensured euthyroidism in all tissue in the rat.[1] There was also evidence from Bunevicius and others in the *New England Journal of Medicine*[2] that combination therapy produced better quality of life outcomes. Individual case reports as presented in *Thyroid Flyer* also supported the case that some people at least had better outcomes with combination therapy.

The Bunevicius study[2] unfortunately had some problems of methodology and design so it was not clear whether the findings were generally applicable. A number of other groups around the world have now sought to verify the findings. One of the best studies was carried out in Western Australia by John Walsh and others[3] and was reported in *Thyroid Flyer* Volume 5 Number 1 of March 2004. This study found that there was no improvements across the experimental group when taking combination therapy as compared to T_4 alone. The experimental design was significantly better than the Bunevicius study[2], but the authors noted some shortcomings in that a fixed ratio for substituting T_3 for T_4 was used and the T_3 was given in a single dose in the mornings. Within the experimental group there were people who improved, but there we also people who did not change and some who had worse outcomes. This left the situation still unresolved, but it was clear that the use of T_3 was not working for everyone.

In *Thyroid Flyer* Volume 5 Number 3 of October 2004, we reported further studies into the use of T_3. In the first report, Henneman and others [4] reported that slow release T_3 was better at maintaining stable T_3 levels than normal T_3. The most interesting result from this study however was that the most stable and sustainable levels of T_3 were obtained using T_4 alone. This suggested that the issue for most people who don't feel well on T_4 alone might be their dose being too low.

The second study reported was that of Siegmund and others[5] who also tested outcomes of T_4 therapy alone against combination T_3/T_4 therapy. In this study, the T_3 dose was not fixed, but was based on the T_4 dose. T_3 was used to replace part of the T_4 dose. The subjects showed no difference in results of tests for psychological state, cognitive function or physiologic function. This study too had some shortcomings in that the sample was small and there was no clear determination that the starting T_4 dose was optimal. Nonetheless, this study also demonstrated that there was no general efficacy of T_3 therapy.

We can now report on three more studies that have been carried out since 2004. The first is a study by Appelhof and his associates that considered the effects of two different T_3/T_4 combination dose regimes against a T_4 only regime. The study group was 141 people (120 women) with an average age of 48 all of whom suffered from autoimmune hypothyroidism. All had been taking T_4 for at least 6 months and had normal TSH levels. They were assigned randomly to one of three groups. The first group received the same T_4 medication as prior to the study. The second received a combination of T_4 and T_3 in the ratio 5:1 and the third received the combination in the ratio 10:1 with the dose related to their starting T_4 dose. The subjects were not aware of the group to which they had been assigned.

The groups were evaluated at the end of 5, 10 and 15 weeks. The assessments included TSH, Free T_4 and a range of standard questionnaires to assess mood, fatigue, quality of life and cognitive function. The 5 and 10 week assessments were for management. After the 5 week assessment those whose TSH was out of range had their doses adjusted to normalise the TSH. The experimental result was based on the 15 week results which showed that all groups showed improvements in the tests for quality of life, mood, fatigue and cognitive function and that the improvements were statistically the same for all groups.

The 5:1 ratio group lost 1.8 kg in weight on average whereas the loss for the 10:1 ratio group was 0.5 kg and for the T_4 only group the loss was only 0.1 kg.

The subjects were also asked for their subjective assessment of the study therapy in comparison to their original therapy. 30% of the T_4 only group preferred the study therapy (which was unchanged from the original therapy), 40% of the 10:1 ratio group and 50% of the 5:1 ratio group preferred the study therapy. There was an increasing preference for the higher T_3 dose, but only half of the higher T_3 dose group preferred it to T_4 alone and there was clearly a strong placebo effect.

This study was the best designed of any of the studies we have reported and the study size was such that the results are statistically credible. The result shows that there was no quantifiable difference in physical outcomes between the groups and the majority of participants preferred the initial T_4 only regime. But it also showed that a significant minority of participants felt better with combination therapy and the percentage who did was greater for the higher T_3 dose.

The second study was carried out by Escobar-Morreale and associates.[7] This study group was much smaller comprising 28 women of average age 48 years. 23 had autoimmune hypothyroidism and the others had been treated with radioiodine for hyperthyroidism. The women had all been taking 100mcg of T_4 for at least a year and had normal TSH levels. The women were randomly assigned to receive either 100 mcg T_4 or &5 mcg T_4 plus 5mcg T_3 for a period of 8 weeks. The medication of the two groups was then switched for a period of 8 weeks. After that all participants

received 87.5mcg T_4 plus 7.5mcg T_3 for 8 weeks. Tests for TSH, Free T_4 and questionnaires for mood and cognition were completed at the end of each 8 week period and the women were asked their preferences at the end of the study.

46% preferred the 75:5 combination regime, 23% preferred the 87.5:7.5 combination, 8% the T_4 regime and 23% had no preference. The questionnaire scores for mood, cognitive performance, quality of life and hypothyroid symptoms were very similar with a few exceptions - eg hypothyroid symptom scores were better with the 87.5:7.5 combination than T_4 alone. Interestingly TSH was lower and Free T_4 higher for T_4 alone than the preferred 75:5 combination. The authors concluded that the 75:5 combination was preferred by nearly 50%, but there were few subjective, biochemical or other differences at the end of the three treatment periods.

This study is the first to show a clear preference by the participants for a combination therapy, but the results cannot be regarded as indicative because the small number of participants make it statistically unreliable.

The third study was carried out by Saravanan, Dayan and others.[8] The study subjects were 697 people aged between 18 and 75 recruited from family practices in the UK. They had all taken 100mcg of T_4 or more for at least 3 months and had normal TSH levels. The subjects were randomly assigned to two groups. The first continued with their existing T_4 therapy. The other replaced 50mcg of T_4 with 10 mcg of T_3. The two groups were evaluated at the start and after 3 and 12 months.

The evaluations consisted of tests for TSH, Free T_4, Free T_3 and neuromuscular symptoms, a physical examination and questionnaires for mood, depression, anxiety, hypothyroid symptoms cognition and general health. The T_4 alone group showed no change in TSH and Free T_4 over the period (as would be expected). The combination therapy group showed a drop in Free T_4 and rise in TSH at the 3 month point compared to their starting position. The levels present at 3 months persisted at 12 months.

The questionnaires at 3 months showed improved scores in both groups on all the factors with slightly greater improvements for those taking combination therapy. There was also a similar improvement in neuromuscular symptoms in both groups. Body weight did not change. At 12 months, the questionnaires showed that the improvements at 3 months were no longer present with no difference between the groups. The combination therapy therefore produced a slight improvement on some measures, but these improvements were not sustained.

The deterioration in thyroid hormone levels of the T_3/T_4 combination group unfortunately compromises the findings. It is a pity that this anomaly was not corrected at some point, because restored levels may have resulted in the combination group reporting a sustained improvement.

The results of these recent studies when coupled with the previous studies leave us in the position that:

- The medical establishment has invested some effort to answer this question. Good on them.
- Most of the studies have been flawed in some way thereby making it impossible to be fully confident of their results. It is frustrating that this many studies have been undertaken when only one of them was actually capable of definitively answering the question being studied.
- No equivalent studies have been reported by the groups promoting the universal use of T_3 therapy. Why not?
- Despite the flaws, these studies show unequivocally that T4 therapy is preferred by the majority of patients and produces outcomes at least as good as combination therapy.
- Combination therapy is not a panacea that will improve outcomes for most hypothyroid patients.
- There is solid evidence that combination therapy improves outcomes for some people, but the studies provide no information on how to identify these people.

The situation for people needing thyroid hormone replacement therapy is that the majority of them will be best served by using synthetic T_4 alone providing the dose is high enough. It is cheaper, more easily managed and produces better outcomes. The important point being that the dose must be high enough. A minority of people will be best served by a combination therapy. For those in this group, a dose ratio of 5:1 would appear to be the best choice and the T_3 should be a slow release form taken more than once per day.

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TELEPHONE SUPPORT VOLUNTEERS

These Thyroid Australia members have agreed to support others with thyroid conditions in their times of need. They follow our guidelines in an effort to provide reliable information to callers. Telephone Support Volunteers are generally not medically trained, but they will provide a personal viewpoint based on their own experience. The names of the Volunteers are given on the left according to the conditions with which they can assist. Their phone numbers, and their contact times are given on the right.
Please be considerate about when you ring. No calls on Sundays please. Our Volunteers also have family and business responsibilities and might not be available when you call. They will return your call if you leave a message when requested to do so.

Hypothyroidism
Kay Laurel Madeline
Michele Rick

Hashimoto's Thyroiditis (HT)
Gail Kay
Liz Rick

Hyperthyroidism
Christopher Jenny
Karen Maria

Graves' Disease (GD)
Christopher Jane Karen
Jenny Maria

Thyroid Hormone Replacement
Kay Gail
Liz

Antithyroid Medication
Christopher Jenny Karen

Congenital Hypothyroidism
Julie

Thyroid Eye Disease (TED)
Jane Karen
Margaret Maria

Eye Surgery
Margaret

Psychological Concerns
Geri

Post Partum Thyroid Problems
Susan

Radioactive Iodine (RAI)
Sue

Thyroid Cancer
Geri Diane (Follicular)

Thyroid Surgery
Jane

Name	Phone Number	Monday - Friday	Saturday
Christopher	(03) 9417 1720	7.00pm - 9.00pm	10.00am - 8.00pm
Diane	(03) 9560 5857	10.00am - 4.00pm	
Gail	(03) 9850 3854	6.00pm - 8.30pm (Mon-Wed only)	
Geri	(03) 9822 1942	10.00am - 4.00pm	
Jane	(03) 9597 0426	10.00am - 4.00pm	12.00pm - 4.00pm
Jenny	(03) 5689 1251	10.00am - 8.00pm	
Julie	(03) 9764 2426	8.00pm - 9.30pm	10.00am - 8.00pm
Karen	(03) 9380 8505	10.00am - 4.00pm (Mon-Thur)	10.00am - 4.00pm
Kay	(03) 9560 4688	3.00pm - 8.00pm	3.00pm - 8.00pm
Laurel	(03) 5255 1436	10.00am - 4.00pm	
Liz	(03) 9370 1568	7.00pm - 9.00pm	10.00am - 8.00pm
Madeline	(03) 5144 4470	1.00pm - 5.00pm	
Margaret	(03) 9741 2524 or (03) 5158 0308 if unavailable at above number	7.00pm - 9.00pm	7.00pm - 9.00pm
Maria	(02) 9832 3958	6.00pm - 9.00pm	1.00pm - 5.00pm
Michele	(03) 5821 6681	10.00am - 4.00pm	12.00pm - 4.00pm
Rick	(07) 3808 4961	7.00pm - 9.00pm (Mon-Wed)	
Sue	(02) 6259 5342	7.00pm - 9.00pm (Mon&Tues)	10.00am-4.00pm
Susan	(02) 6278 3348	7.00pm - 9.00pm	7.00pm - 9.00pm

We would like to expand our list of Telephone Support Volunteers and need more members willing to share their experiences. We provide all Volunteers with a pack of reliable information as well as access to the articles on the Publications List. New Support Volunteers are asked to attend a short workshop to ensure that everyone works off the same knowledge base, and to allow them to gain some insights from others who have been performing this task already. Suitable arrangements will be made for those members from outside of Melbourne.

If any member would like to help us as a Telephone Support Volunteer, please leave a message for our Telephone Contact Coordinator on (03) 9888 2588 (10am to 3pm, most weekdays) or send an e-mail to support@thyroid.org.au

Meetings and Support Groups

Adelaide
First Thursady of Each Month
11.00am to 2.30pm
Support Group Meetings
Tea Tree Gully Community Health Centre
77 Smart Road
Modbury SA

South Gippsland
Fourth Monday of Each Month
10.30am
Support Group Meetings
Foster Community Health Centre
93 Station Rd
Foster VIC

Gold Coast / Tweed Heads
Dates not yet announced
Contact office for more information

Brisbane
Dates not yet announced
Contact office for more information

Inner Melbourne
21 February 2006
10.30am to 12.30 pm
Support Group Meeting
Cost $5.00
North Carlton Railway Station Neighbourhood House
20 Solly Ave
Princes Hill VIC

Perth
4 February 2006 - 10.30am
18 March 2006 - 10.30am
29 April 2006 - 10.30am
Support Group Meetings
Salavation Army Hall
565 Walter Road East (c/- Wicks St)
Morley WA

Melbourne
2 April 2006 - 2.00pm to 5.00pm
Lecture: Introduction to Thyroid Matters
Royal Women's Hospital
Grattan Street
Carlton VIC

Editorial *from Page 1*

backlog at the same time. Although we are working through our correspondence and other demands, we have not been able to reduce the backlog. Members may have first hand experience of these delays. I apologise for the delays, but unfortunately without extra resources, there is little that can be done. So please bear with the office staff and volunteers as they try to meet your requirements.

In looking forward to 2006, we face the prospect of little improvement in resourcing. The Board has therefore had to consider changes to our services and operations to reduce the work load and to improve our finances so that we can extend the employment of an office manager.

The major change we will be making to our operation from 2006 is that we will be restricting our support services to members. Our telephone support services will from 2006 only be for members and contact details will only be provided to members. Non-members who contact Thyroid Australia will still receive basic information, but ongoing support and access to our extensive library will only be provided to those who join Thyroid Australia as members.

The web site will also be redeveloped so that only basic information is provided to the general public. We will also provide details of meetings and support groups. The full site with its extensive links, articles and newsletters will require a password which will only be provided to members. Members will be told of their user name and password before we implement the changes to the site. Our email support service will also be restricted to members only.

These changes will make no difference to the services delivered to members, but they will reduce the administration work load and will hopefully encourage more people to become members and thereby provide the financial support we require.

We are currently in the process of recruiting a part time office manager to provide the 'backbone' to our administration service. We are endeavouring to have this position filled by the time we re-open for 2006.

We are still keen to hear from anyone who can assist in the office especially those who are willing to organise some of our primary services. You don't need to be an expert in thyroid matters. You also don't need to be an expert organiser or management guru. We only need a few hours per week. Please call or email if you can help.

We also interested in attracting some more telephone volunteers who are prepared to talk to others about their own experiences. Once again, you don't need to be a thyroid expert. The main thing that callers need is to talk to someone who understands and being able to talk about your own experience is the most important requirement. We will supply any extra information you need. Please call or email if you can help.

The difficulties in the office have not deterred our hard working support group convenors continuing to help people in their local communities. We now have groups in Perth, Brisbane, Gold Coast, Adelaide, Horsham, Gippsland and Inner Melbourne. The convenors and their local committees need to be congratulated for their efforts and I would like to thank them all on your behalf.

Attendances at our annual information day in October were a bit down on previous years, but the focus on mothers and babies attracted a vigorous audience. The speaking panel of Prof Duncan Topliss, Dr Christine Rodda, Dr Richard Arnott and Dr Robert Hanner provided an excellent overview of thyroid matters for mothers, babies and everyone else too. I would like to officially thank them for once again giving their time to talk to us. We will be making conference packs available for purchase in the new year for those who were not able to attend.

Our offices will be closed from 10 December 2005 until 6 February 2006.

I trust that you all have a safe and enjoyable Christmas break and a prosperous 2006. ❀

Please copy or detach and mail to the address below.

Request for Membership Application Form

Date: .....

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

Please send me a Membership Application Form and general information.

I have been diagnosed with the following thyroid condition (please specify): .....

Please send this information to:

Title: ..... Name: .....

Address:.....

☐

P☐

Signature: .....

Disclaimer

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