THYROID FLYER

Newsletter of Thyroid Australia Ltd — Volume 5 No 2 July 2004

Thyroid Therapies

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Editorial

By **Gail Pascoe**

Welcome to this edition of Thyroid Flyer. In our last issue we told you of our move into our new office in Mt Waverley. We have made the big move, which involved the packing and relocation of nearly five years of history and files – this was no small task – and I extend my special thanks to all on the board who worked incredibly hard to bring this about. A special thank you for the extra help from the Stevens family and friends

Now with our office up and operational we need urgent help to keep it running and enable us to expand our services to our members. Our office opening hours vary from week to week, depending on the assistance available. Fortnightly, we are open two days and the alternate weeks three days. We need people to answer the phones and also perform administrative duties, such as receipting memberships, sending packs to new members, helping prepare the mailings of our Flyer – a broad variety of things that need helpers. Alun Stevens has set up a very simplified computer system for managing many of these tasks, so your computer skills don't need to be expert. If you can't manage the computer work, that's OK, taking messages, preparing the post, just stuffing envelopes & adding stamps is OK and is a task that has to be done. If you can assist us, even a half day a fortnight or month, please volunteer. It will greatly assist us help more people with Thyroid problems and it is personally greatly rewarding. We already have the services of a wonderful group of ladies that work in the office, so come and join in fun and become part of Thyroid Australia.

You may have heard about some changes to the storage of Oroxine and Eutroxsig by Sigma Pharmaceuticals.

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Taking care of thyroxine

By **Gregory W. Roberts**

Summary

Some of the pharmaceutical properties of thyroxine have important implications for the quality use of medicines. The stability of thyroxine tablets is limited and they may reach the expiry date before the bottle is finished. Administration should preferably be on an empty stomach and be consistent with respect to food and other drugs. The long half-life of thyroxine enables longer dosing intervals of up to a week if required. The two Australian brands of thyroxine are identical and patients can swap brands safely, but this should not be assumed for overseas brands.

Introduction

Thyroxine tablets are important in managing hypothyroidism, but treatment may be sub-optimal if they are used incorrectly. The tablets have pharmaceutical properties which can impair the patients management. Discussing the correct use and storage of the tablets is an important part of prescribing thyroxine.

Availability

Synthetic preparations of thyroxine contain the *laevo* isomer of thyroxine, usually as the sodium salt. There are two brands of thyroxine available in Australia, each as 50 microgram, 100 microgram and 200 microgram tablets (pack size 200) with five repeats on the Pharmaceutical Benefits Scheme. Parenteral preparations of thyroid hormone [*Preparations that are not administered by mouth*] have little use in Australia, outside of specialist centres.

The two Australian brands are marketed by Sigma and one of its subsidiaries. They are identical products so patients can swap them safely, but this assumption should **not** be extended to overseas brands.

Stability

Thyroxine is stable in dry air, but unstable in the presence of light, heat and humidity. In some cases overseas, thyroxine tablets have been unstable even at room temperature, and storage temperatures of 8°C to 15°C were required to maintain potency. In the USA, the Food and Drug Administration has determined that stability and potency problems with oral thyroxine preparations could potentially have adverse effects on health. It is therefore very important that thyroxine tablets should be kept in their original container and stored out of sunlight in a cool dry place.[1]

The expiry date for Australian manufactured thyroxine tablets is one year from the date of manufacture. There are 200 tablets in a bottle, so it is possible that patients on half tablet doses will not finish the bottle before the stock expires. The expiry date should be emphasised to the patient to ensure they do not continue taking a thyroxine preparation that may be waning in potency. However, stock with a shelf-life of 18 months will soon be available.This formulation will require refrigeration at all times.

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Taking Care of Thyroxine *from Page 1*

Absorption

Thyroxine is variably absorbed from the gut following oral administration. It has a bioavailability of 40–80%. Absorption may decrease with age.[1,2]

The extent of thyroxine absorption is increased in the fasting state and is influenced by the content of the gastrointestinal tract. Some substances bind the thyroxine, making it unavailable for diffusion across the gut wall. Concurrent administration with iron salts, antacids, calcium carbonate (including milk), sucralfate, cholestyramine and soy-based formulas may therefore decrease absorption of thyroxine.

Administration

Patients should be instructed to take thyroxine 30–60 minutes before breakfast in order to maximise absorption. If this is too difficult or threatens compliance, the patient may try taking the thyroxine last thing at night on an empty stomach. Patients who still decide to take their tablets with, rather than before, breakfast need to do this consistently, to avoid fluctuating thyroxine concentrations. Depending on the fibre and milk content of the meal, taking thyroxine with food may require a larger dose to maintain euthyroidism, because of the decreased bioavailability.

While most patients take a daily dose, the long half-life of thyroxine lends itself to longer dosing intervals, such as alternate daily dosing. Once-weekly dosing is also possible although a slightly larger dose than seven times the normal daily dose may be required. This regimen may be suitable for poorly compliant patients who require supervised dosing.[3]

For patients, particularly children, who cannot swallow tablets, the tablets may be crushed in 10–20 mL of water, breast milk or non-soybean formula. The resulting mixture should be used immediately and any remainder discarded.[2] Breast milk contains only 20–30% of the calcium concentration of cows milk, making the likelihood of decreased thyroxine bioavailability less likely. Nonetheless, if breast milk is used to deliver the thyroxine, it should be used consistently, in order to minimise any variation in absorption.

Onset and duration of action

The half-life of thyroxine in euthyroidism is 6–7 days. This is reduced to 3–4 days in hyperthyroidism and prolonged to 9–10 days in hypothyroidism. Thyroxine has a full therapeutic effect 3–4 weeks after starting treatment and will continue to have a therapeutic action for 1–3 weeks after treatment stops. In view of the long half-life, dose changes should only be made every 3–4 weeks. Despite undergoing both hepatic and renal clearance, there is no evidence that dose adjustment is required for patients with liver or kidney disease.[1,2]

Monitoring

The dosage is adjusted according to thyroxine and thyroid stimulating hormone plasma concentrations, which should always be interpreted in conjunction with each other and the patients condition.[4] Monitoring is suggested at six-weekly intervals when starting therapy until the patient has stabilised, then six monthly thereafter, or earlier if symptoms suggestive of hyper- or hypothyroidism occur.

Drug interactions

Most drug interactions are seen during shifts to and from the euthyroid state and rarely have any clinical significance during periods of thyroid stability. The hyperthyroid state increases clearance of some hepatically cleared drugs, notably propranolol, metoprolol and theophylline. Antacids, iron salts, calcium carbonate (milk), sucralfate, cholestyramine and soy-based formulas reduce the absorption of thyroxine.

Conclusion

There are significant stability, absorption and drug interaction issues surrounding the use of thyroxine. It is essential that prescribers and pharmacists convey this information to patients in order that therapeutic efficacy may be maximised.

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2. Thomas J, editor. *Australian Prescription Products Guide 2003*. 32nd ed. Hawthorn: Australian Pharmaceutical Publishing Company Limited; 2003.
3. Grebe SK, Cooke RR, Ford HC, Fagerstrom JN, Cordwell DP, Lever NA, et al. Treatment of hypothyroidism with once weekly thyroxine. *J Clin Endocrinol Metab* 1997;82:870-5.
4. *Australian Medicines Handbook 2004*. Adelaide: Australian Medicines Handbook Pty Ltd; 2004.

Further reading

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Volunteering Is Fun

Come and join us.

A group of us has begun voluntary work in the Thyroid Australia Office. It is warm and friendly and we enjoy tea or coffee and a chat while we complete a variety of tasks from filing, preparing mailouts etc to more complex computer work. It is fun and really helps us help others.

You can choose what you would like to do. You can come for an hour or a whole day. Whatever you choose.

We look forward to hearing from you.

Diane J.

Robyn K.

Carol S.

Thyroxine Changes

By **Bronwyn Stevens**

There are two brands of thyroxine available in Australia – Oroxine and Eutroxsig – both manufactured by Sigma Pharmaceuticals. If you have had an Oroxine or Eutroxsig prescription filled since June, you will have discovered that these products now require refrigeration **at all times**. Up until now, these products could be kept in a cool, dry place below 30°C and protected from light. Oroxine and Eutroxsig are still the same; they are still manufactured in exactly the same way using exactly the same ingredients. The only thing that has changed is the storage requirement.

In June 2003 Sigma was instructed by the Therapeutic Goods Administration (TGA) to reduce the shelf lives of Oroxine and Eutroxsig from 24 months to 12 months (with refrigeration). This was in light of evidence that the potency of these products was reduced at the end of their shelf life. Sigma, following consultation with the TGA, has been able to extend the shelf life of these products to 18 months (with refrigeration). Furthermore, Sigma advises that these products are able to be kept un-refrigerated for up to 4 weeks (at below 25°C only). Oroxine and Eutroxsig bottles also now have 'child-resistant' caps to help keep little fingers out of your thyroxine (as it must be stored in an easily accessible place, your fridge).

Thyroid Australia is concerned that this change is yet another thing that will make it more difficult for patients to consistently take their required dose every day. We view it as a retrograde step in the treatment of hypothyroidism in Australia. It seems likely that the change to refrigeration could have a negative impact on compliance (the consistency and accuracy with which patients follows prescribed treatment regime), as well as being an additional hassle for hypothyroid patients.

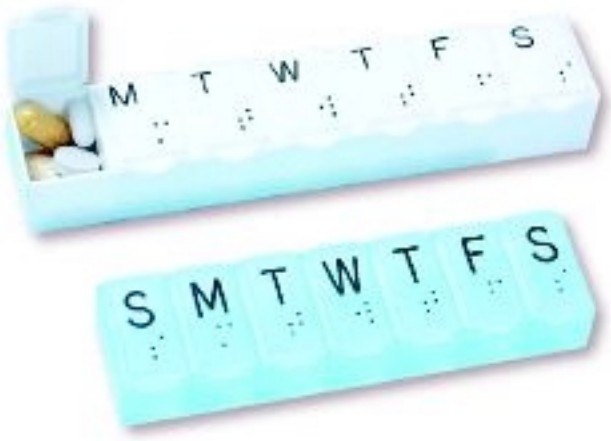
We have met with the Commercial Manager of Oroxine and Eutroxsig from Sigma since the introduction of refrigeration. Sigma is working with the TGA to improve the storage conditions for Oroxine and Eutroxsig, and is aware of the problems refrigeration is causing for patients.

A possible method to reduce the inconvenience surrounding the refrigeration of Oroxine and Eutroxsig is to put one week's worth of thyroxine in your usual place whilst keeping the rest in the fridge. We recommend using a seven-day dose pill box (as shown below). This method means you only have to go to the fridge once a week, and you are otherwise able to maintain the routine you are currently using. It also makes using alternate doses to obtain an intermediate dose easier (this is often required as small doses are unavailable in Australia).

This issue highlights the importance of not using Oroxine or Eutroxsig that is past its expiry date. Shelf lives are the length of time for which a product maintains its potency. After the expiry date the medication may not contain the stated amount of active ingredient (in this case, thyroxine). Often pharmacists place their label over the expiry date, making it very difficult for you to read. We suggest asking your pharmacist to try to not cover the expiry date when you get your prescription filled.

Thyroid Australia will continue lobbying for intermediate doses in Australia and will be seeking to have storage requirements and shelf lives restored to previous standards.

If your doctor prescribes a dose of Oroxine or Eutroxsig other than the available doses of 50, 100 or 200 micrograms (mcg), it can be difficult to achieve this dose. We have prepared a fact sheet on how to obtain intermediate doses (ie. 135mcg) by taking different doses on different days. This can be accessed via the Downloads page of our website (www.thyroid.org.au), but if you would like a copy mailed to you please send a stamped, self-addressed envelope with your request to Thyroid Australia.



Note: Eutroxsig is exactly the same as Oroxine – tablets in Eutroxsig bottles are identical to Oroxine tablets (and are even marked 'Oroxine'). The only difference is that Eutroxsig is cheaper.

Bronwyn Stevens is a final year Behavioural Neuroscience student at Monash University and Thyroid Australia's Volunteer Co-ordinator. ❀

Diagnosis and management of hyperthyroidism and hypothyroidism

By **Duncan J Topliss and Creswell J Eastman**

Despite the development of highly sensitive laboratory tests, clinical assessment and judgement remain paramount.

Thyroid dysfunction is common. In the United States, hypothyroidism is present in 4.6% of the population (clinical, 0.3%; and subclinical, 4.3%) and hyperthyroidism in 1.3% (clinical, 0.5%; and subclinical, 0.7%).[1] A long-term study in the United Kingdom found the incidence of hyperthyroidism was 0.8 per 1000 women annually, and hypothyroidism was 3.5 per 1000 women annually.[2]

Diagnosis of thyroid dysfunction

Thyroid function testing: Measurement of serum thyroid stimulating hormone (TSH), using second- or third-generation assays, is a sensitive index of primary thyroid disease.[3] Assay of free thyroxine (T_4) and triiodothyronine (T_3) reliably eliminates the difficulties in interpretation of total thyroid hormone levels caused by the common variations in serum thyroid hormone-binding protein, best exemplified by the oestrogen-induced rise in thyronine-binding globulin which increases total thyroid hormone levels. However, free hormone assays are still subject to in-vitro and in-vivo artefacts in severe non-thyroidal illness, severe disturbances of binding proteins, and heparin therapy.[4] Thus, if clinical assessment is not concordant with thyroid function results, additional tests and specialist opinion may be necessary. Interpretation of thyroid function results is summarised in Box 1.

Antibody testing: The most common cause of thyroid dysfunction in Australia is autoimmunity. The most sensitive in-vitro index for this is measurement of thyroid peroxidase antibody level,[1] but this can occasionally give false negative results (eg, in juvenile autoimmune thyroiditis). In primary hypothyroidism, a raised level of thyroid peroxidase antibody is evidence for autoimmune chronic lymphocytic thyroiditis.

In hyperthyroidism, the role of routine antibody testing is less clear. Diagnosis of Graves' disease is usually possible clinically, and measurement of thyroid peroxidase antibody or TSH-receptor antibody may not contribute to diagnosis or management. However, measurement of TSH-receptor antibody has a role when the cause of hyperthyroidism is obscure, in assessing the risk of neonatal hyperthyroidism in pregnant women with a history of Graves' disease, and in assessing the risk of relapse after a course of antithyroid drugs in Graves' disease.

Radionuclide scanning: Radionuclide thyroid scanning is not routinely necessary for diagnosing Graves' disease or toxic multinodular goitre, but is useful when the cause of hyperthyroidism is not apparent (eg, when no goitre is palpable, when neck pain or tenderness suggests subacute thyroiditis, or when a solitary "hot" nodule is suspected).

Hyperthyroidism

The terms hyperthyroidism and thyrotoxicosis are used interchangeably, irrespective of whether the disorder is caused by endogenous overproduction or excessive ingestion of thyroid hormones. Causes of hyperthyroidism are shown in Box 2. Most common in Australia are Graves' disease and toxic multinodular goitre. Management of hyperthyroidism depends on the cause (particularly whether it is self-limiting, as in subacute thyroiditis), the size and nature of the goitre, intercurrent illnesses or medication and, especially in Graves' disease, patient preference.

Graves' disease

Graves' disease is caused by stimulation of the thyroid by antibodies which bind to TSH receptors and mimic the effect of prolonged TSH stimulation. These TSH-receptor antibodies result from abnormal immunoregulation permitting generation and expansion of clone(s) of antibody-producing cells in genetically predisposed individuals with specific HLA-D subtypes.[5] Spontaneous exacerbations and remissions of Graves' disease can occur. The environmental triggers are still not well characterised, but postpartum exacerbation is common, and such a history should be sought routinely when Graves' disease is diagnosed. Excess iodine can precipitate active Graves' disease by providing more substrate for hormone synthesis and possibly also by disturbing immune function. Persistence or recurrence of Graves' disease is more likely when there is a previous history of recurrent disease, in the pres-

1: Interpretation of thyroid function results *

	High T_4	Normal T_4	High T_4
High TSH	*In vivo* or *in vitro* artefact Pituitary hyperthyroidism [TSHoma] Thyroid hormone resistance	Mild thyroid failure (primary) (also termed subclinical hypothyroidism and diminished thyroid reserve)	Primary hypothyroidism
Normal TSH	As above Sampling within 6h of thyroxine dose	Normal (in patients taking thyroxine, TSH>3 may indicate subtle underreplacement)	Pituitary or hypothalamic hypothyroidism. Severe non-thyroidal illness
Low TSH	Hyperthyroidism (for this diagnosis, TSH must be suppressed rather than just low)	Subclinical hypoerthyroidism Subtle thyroxine overreplacement Thyroid autonomy (multinodular goitre or autonomous functioning thyroid nodule) Non-thyroidal illness	Pituitary or hypothalamic hypothyroidism Severe non-thyroidal illness

T_4 = serum free thyroxine. TSH = thyroid stimulating hormone.
* The Health Insurance Commission restricts financial reimbursement to serum TSH alone, except in specified clinical circumstances, when additional measurements of free T_4 and free triiodothyronine (T_3) are sanctioned. We suggest that practitioners routinely request "thyroid function tests" and provide clear clinical information to enable the laboratory to perform additional tests if justified. The information should include the suspected condition (especially if hypopituitarism) and medications (eg, thyroxine, carbimazole or propylthiouracil, amiodarone or phenytoin).

ence of a large goitre, when T_3 excess persists despite control of T_4 with thionamide therapy, and when TSH-receptor antibody persists during thionamide therapy. In addition to hyperthyroidism, other autoimmune manifestations of Graves' disease are Graves' ophthalmopathy (Box 3) and Graves' dermopathy (pretibial myxoedema). Autoimmune thyroid disease is associated with some other, less common, autoimmune diseases, including pernicious anaemia and Addison's disease.

2: Causes of Hyperthyroidism

- Graves' disease
- Multinodular goitre
- Autonomously functioning single thyroid nodule (adenoma)
- Thyroiditis (subacute, postpartum, lymphocytic)
- Factitious hyperthyroidism (thyroid hormone ingestion)
- Functioning thyroid carcinoma (follicular carcinoma)
- hCG-mediated (hyperemesis gravidarum, trophoblastic disease)
- Fetal and neonatal hyperthyroidism (TSH-receptor-antibody-mediated)
- Struma ovarii
- TSH-secreting pituitary tumour
- Partial (pituitary-selective) thyroid hormone resistance

hCG = human chorionic gonadotropin.
TSH = thyroid stimulating hormone.

Management of Graves' disease

The three modalities of therapy for Graves' disease are:

- blocking synthesis of thyroid hormone with antithyroid drugs;
- subtotal or "near-total" thyroidectomy; and
- destruction of the thyroid by radioactive iodine (radio-iodine ablation).

While each modality is satisfactory in rendering the patient euthyroid,[6] none is ideal, as all have a risk of adverse effects and none but total thyroidectomy eliminates the risk of recurrence. Although total thyroidectomy virtually eliminates this risk, it is at the expense of a certain requirement for thyroid hormone replacement. Selecting treatment for an individual depends on many factors, not least being patient choice and physician bias. In the United States, radioiodine is the preferred primary modality, but, in Europe and Australia, antithyroid drug therapy is preferred for patients with a first episode of Graves' hyperthyroidism, ahead of radioiodine and, lastly, surgery.[7]

Antithyroid drugs: Most patients with Graves' disease require short-term (several months) treatment with an antithyroid drug (thionamide) before consideration of longer-term or definitive therapy. Prolonged thionamide therapy (12–18 months in a first episode[8]) has the advantage of avoiding surgery with its inherent risks and destruction of the thyroid by radioiodine, and seems to give the best chance of sustained remission. Nonetheless, the risk of relapse is greater than 50%.

Thionamide dose must be individualised, depending on the initial severity of disease and response, but an initial divided dose of 10–30 mg daily of carbimazole is usually satisfactory. Response should be assessed after 2–4 weeks and periodically thereafter, with a minimum eventual frequency of every third month. Initial high doses should be progressively reduced to once-daily maintenance doses of 2.5–10 mg/day (Box 4).

Occasionally, in very active Graves' disease, thionamide therapy can lower serum free T_4 level below normal, while free T_3 level remains raised, and the patient remains hyperthyroid. Thionamide dose should thus not be reduced on the basis of serum free T_4 level alone. Clinical assessment remains paramount, guided by full thyroid function testing.

Combined thionamide and thyroxine therapy (block–replace regimen) is useful for patients with unstable hyperthyroidism, in whom small variations in thionamide dose cause major fluctuations in thyroid function, but does not increase the likelihood of long-term remission.[9]

Beta-blocker drugs are useful adjuncts for rapid symptomatic relief in hyperthyroidism. Standard doses reduce heart rate, sweating and tremor, but do not influence hypermetabolism or hormone levels. Non-selective £]-blockers have generally been preferred for their better effect on tremor.

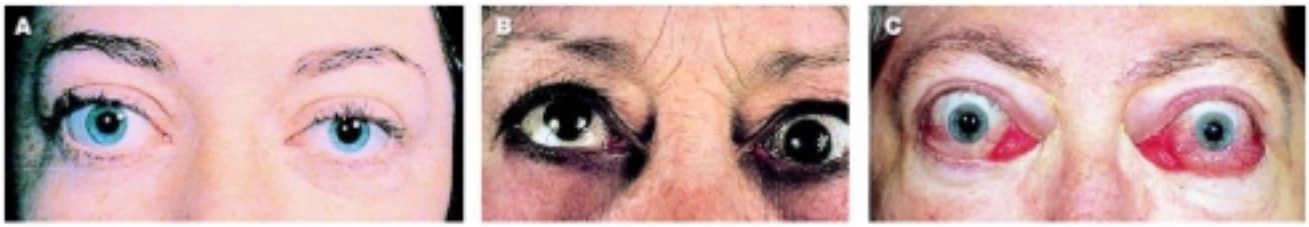
All patients should be warned about the rare but serious complication of thionamide therapy, agranulocytosis, with instructions to suspend therapy while obtaining a white cell count if fever, sore throat, or other sepsis develops. We do not recommend routine blood testing for this side effect, as it is rare and of abrupt onset.

Radioiodine ablation: Ablative therapy with radioiodine is recommended for patients with recurrent hyperthyroidism or hyperthyroidism that persists after a prolonged course of antithyroid drugs. Reassuringly, several large, long-term studies have shown no increased risk of thyroid cancer, leukaemia, other malignancies, reproductive abnormalities or congenital abnormalities in the offspring of treated patients.[10] It is thus the default option for definitive therapy in adolescents and adults. More caution is recommended in children because of the known greater risk of inducing thyroid nodules and carcinoma from external irradiation and other radionuclide exposure in childhood.

Radioiodine therapy does not achieve euthyroidism immediately, necessitating low-dose thionamide therapy for several months in many patients. Occasionally, early (usually transient) hypothyroidism occurs, with low serum free T_4 levels without TSH elevation, as TSH is often suppressed for weeks to months after hyperthyroidism.

The likelihood of control of hyperthyroidism after a single radioiodine treatment varies with the dose, continuing immunological stimulus of the thyroid, and thyroid responsiveness to the radioiodine. Overall, control is achieved initially in about 75% of cases. Eventual control through repeated doses, given after a minimum of six months, is virtually assured, but often at the expense of permanent hypothyroidism.

3: Graves' Ophthalmopathy



A: Asymmetrical proptosis, more marked in the right eye. Although unilateral proptosis sometimes occurs in Graves' disease, further imaging may be needed to exclude diagnoses such as orbital tumour.
B: Patient attempting to look up, showing tethering of the left eye by swollen inferior external ocular muscles, slight proptosis and lid retraction (with sclera visible above the iris of the left eye) and some infiltrative swelling above the inner canthus of each eye.
C: Marked bilateral proptosis with lid retraction and conjunctival injection indicating active inflammatory ophthalmopathy.

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The probability of developing hypothyroidism increases with the dose of radioiodine and the passage of time, so that long-term follow-up and patient education is required in all cases.

Thyroidectomy: Thyroidectomy is generally undertaken only after euthyroidism has been attained with thionamide therapy. The high rate of relapse after subtotal thyroidectomy has led many surgeons to recommend complete thyroidectomy (termed total or near-total thyroidectomy), but this inevitably necessitates permanent thyroid hormone replacement therapy. Thyroidectomy offers rapid control without radiation exposure and reliably removes a substantial goitre. It becomes the therapeutic choice when malignancy is suspected or a large goitre is causing local compression. As with all surgery, the individual skill of the surgeon is paramount, and there should be no role for the occasional exponent.

Toxic multinodular goitre

Toxic multinodular goitre is almost always preceded by long-standing multinodular goitre and probably, in most patients, by episodes of subclinical hyperthyroidism. Hyperthyroidism is often precipitated by exposure to excessive iodine from adventitious sources, such as medications and radiocontrast media used in imaging procedures (see list under *Iodine-induced hyperthyroidism*).[11] The goitre may compress or obstruct the trachea. Plain radiography of the trachea may reveal this, but computed tomography is better, provided no contrast is administered. Magnetic resonance imaging best demonstrates the extent of the goitre but is not directly available to the general practitioner. Oesophageal symptoms occasionally require assessment by barium swallow. Venous obstruction, which is more common than oesophageal obstruction, is assessed clinically (Box 5).

The initial choice of therapy for toxic multinodular goitre is a thionamide to render the patient euthyroid. Spontaneous remission is not part of the natural history of this condition and, unless there has been a specific precipitating episode of iodine exposure, ablative therapy is indicated. In the absence of obstruction, radioiodine is the treatment of choice. Higher doses, and sometimes multiple treatments, may be necessary for a satisfactory outcome. If obstruction is present then thyroidectomy is indicated. Total thyroidectomy also obviates the risk of regrowth of the goitre.

4: Case report — hyperthyroidism

Presentation: A 25-year-old woman presented with a 6-month history of tremor, heat intolerance, irritability and weight loss of 4 kg. She had a small, diffuse, non-tender goitre (less than twice normal size), a pulse rate of 104 bpm, and slightly tremulous, warm, moist hands but no proximal myopathy. She had no personal or family history of thyroid disease. Fertility was not a current issue.

Investigations: Serum free thyroxine (T4) level was 34 pmol/L (reference range [RR], 9.3–23.8 pmol/L); free triiodothyronine (T3) level was 12 pmol/L (RR, 1.8–6.0 pmol/L); thyroid stimulating hormone (TSH) level was < 0.03 mU/L (RR, 0.4–4.7 mU/L); and thyroid peroxidase antibody level was 250 U/mL (RR, < 35 U/mL).

Comment*: Radionuclide thyroid scanning will not assist management in the absence of a reasonable need to exclude subacute thyroiditis and in the presence of goitre.*

Management: Graves' disease was diagnosed, and carbimazole (10 mg twice daily) prescribed.

At 3-week review, the patient was feeling better, and free T4 level was 20 pmol/L; free T3, 6.5 pmol/L; and TSH, still < 0.03 mU/L. The carbimazole dose was reduced to 15 mg/day, and a further 3-week review scheduled.

Comment: *There was a clinical and biochemical response, but free T3 level was still high. Dose reduction by 25%–50% was necessary to avoid overshoot to hypothyroidism.*

At 6-week review, the patient was feeling much better, and her pulse rate was 76 bpm. Free T4 level was 16 pmol/L; free T3, 4.8 pmol/L; and TSH, 0.03 mU/L. The carbimazole dose was reduced to 10 mg/day as a single dose. The options for longer-term therapy were now discussed in detail with the patient.

Comment: Serum TSH can remain suppressed for weeks to months after euthyroidism is restored and therefore cannot be used to guide acute antithyroid drug therapy. Carbimazole is effective in a once-daily dose for maintenance, assisting compliance. When patients are euthyroid and well, the longer-term options for therapy should be discussed.

The patient elected to have a thionamide course. The dose was reduced and maintained at 5 mg/day in subsequent visits, with thyroid function monitoring every 2–3 months. After 18 months therapy was ceased.

Comment*: Thyroid function should then be checked at decreasing frequencies indefinitely (eg, at 1 month, then after a further 2 months, 3 months, 6 months, and then annually) or if symptoms develop.*

Three years later, the patient wished to get pregnant and enquired about possible thyroid problems. She was clinically euthyroid.

Comment*: Thyroid function should be checked, not only to exclude subclinical hyperthyroidism, but also hypothyroidism, which is part of the natural history of Graves' disease and can have a subtle adverse effect on fertility and fetal development. If the patient is euthyroid without prior ablative therapy, then assay of TSH-receptor antibodies is unnecessary, but may be useful in predicting the risk of neonatal hyperthyroidism. Thyroid function should be monitored, especially in the first trimester and particularly post partum, and the patient should be reminded about postpartum recurrence.*

Autonomously functioning thyroid nodule

A single hyperfunctioning nodule, usually an adenoma, is an uncommon cause of hyperthyroidism. If a single nodule is detected on clinical or ultrasound examination in a hyperthyroid patient, the diagnosis should be confirmed by a radionuclide scan. Radioiodine is the preferred treatment after control of thyroid function by a short course of thionamide. The risk of radiation-induced hypothyroidism is small in this condition, in contrast to Graves' hyperthyroidism, as the hyperfunctioning nodule suppresses the rest of the thyroid.[12] Lobectomy is still an acceptable choice.

Subacute thyroiditis

Subacute thyroiditis is a self-limiting, postviral condition, which can vary from a mild disorder to a debilitating disease lasting up to a year. Thyroid pain and tenderness are usual but not ubiquitous. Because the hyperthyroidism results from leakage of preformed hormone from damaged thyroid cells rather than increased production, the circulating T_3/T_4 ratio is lower than in Graves' disease and toxic multinodular goitre. Thyroid radionuclide uptake is reduced or absent. This finding is often considered diagnostic of subacute thyroiditis, but is also seen in patients ingesting large quantities of iodine or taking suppressive doses of thyroxine. The conditions can be differentiated by serum thyroglobulin level, which is usually raised in subacute thyroiditis but low in the others. Antithyroid antibodies can appear transiently at modest levels.

Antithyroid drug therapy is inappropriate and ineffective. Beta-blockers, aspirin, and non-steroidal anti-inflammatory drugs provide symptomatic relief. Glucocorticoids, such as prednisolone (20 mg per day), provide symptomatic improvement in those who do not respond well to anti-inflammatory drugs. The hyperthyroid phase is characteristically followed by a hypothyroid phase (not usually requiring treatment), then eventual return to euthyroidism. Further episodes can occur.

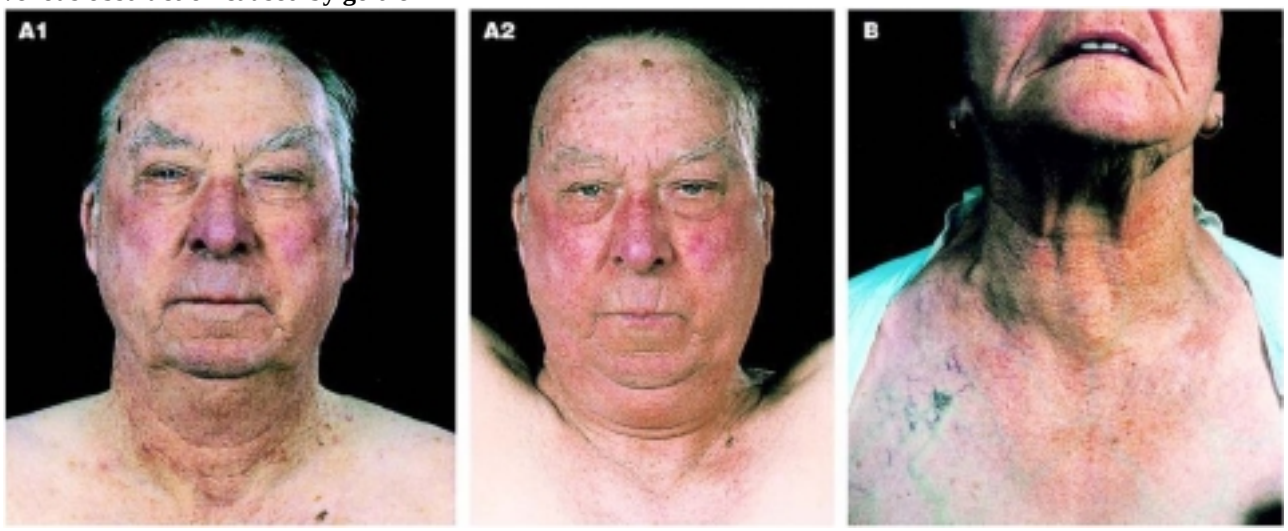
Postpartum hyperthyroidism

Hyperthyroidism in the postpartum period is usually autoimmune, caused by either Graves' disease or thyroiditis. It differs from postviral thyroiditis, although it may follow a similar transient course, and may recur after subsequent pregnancies and progress to established hypothyroidism.

Iodine-induced hyperthyroidism

Iodine can induce hyperthyroidism in patients with an underlying autonomously functioning thyroid gland (caused, for ex-

5: Venous obstruction caused by goitre



A: Pemberton's sign in a man with a goitre extending through the thoracic inlet and obstructing venous drainage. (**A1**) At rest, he has moderate puffiness of the face. (**A2**) After raising the arms briefly to further obstruct venous drainage, swelling increases and there is marked suffusion of the face. **B**: Woman with an obstructive, predominantly retrosternal, multinodular goitre, showing dilated anterior chest veins caused by increased collateral venous flow.

ample, by a nodule, multinodular goitre or Graves' disease). It is characterised by suppressed serum TSH level with normal circulating thyroid hormone levels, and is most commonly found in patients with longstanding multinodular goitre.[13] A history should be sought of recent exposure to iodine from radiocontrast media and pharmaceuticals such as amiodarone, herbal and vitamin preparations, kelp and Cellasene (a preparation marketed as a treatment for cellulite).

As radiocontrast media are clear precipitants, computed tomography should be performed without contrast in the investigation of euthyroid goitre and undiagnosed neck mass.

The antiarrhythmic drug amiodarone has a 37% iodine content, with high tissue penetration and a half-life of months. The elemental iodine load of about 9 mg per day from a 200 mg tablet can precipitate hyperthyroidism if there is pre-existing goitre (type I amiodarone-induced hyperthyroidism), while the drug itself causes thyroiditis in 5%–10% of users, typically after about 2 years of therapy (type II amiodarone-induced hyperthyroidism, the most common form in Australia[14]). Although type I may respond to high-dose thionamide therapy, and type II to glucocorticoid therapy, these conditions can be severe and treatment-resistant, and thyroidectomy may be needed.

Subclinical hyperthyroidism

"Subclinical" hyperthyroidism is a poor term for this condition, as the definition is biochemical rather than clinical, and clinical consequences are possible. It is characterised by chronically suppressed TSH levels but normal serum free T_4 and T_3 levels. The condition must be distinguished from transient TSH suppression in acute illness or after drug therapy (eg, high-dose glucocorticoids). It occurs commonly with multinodular goitre, a hyperfunctioning single thyroid nodule and thyroxine overreplacement. Subclinical hyperthyroidism is associated with an increased risk of atrial arrhythmia in those aged over 60 years[15] and loss of bone mineral density in postmenopausal women.[16] Antithyroid therapy should be considered in patients with subclinical hyperthyroidism at increased risk of complications, and reduction of thyroxine dose in those taking replacement therapy. Subclinical hyperthyroidism is intentionally produced by TSH-suppressive thyroxine therapy for differentiated thyroid cancer, but it is unnecessary to continue this therapy lifelong if likely eradication of carcinoma has been demonstrated.

Factitious hyperthyroidism

This is the deliberate, usually surreptitious, excessive ingestion of thyroid hormone.[17] It is often part of a wider psychiatric condition and may be refractory to medical advice. It may be severe, with weight loss, weakness, cardiac tachyarrhythmia and failure. When due to thyroxine ingestion, serum free T_4 level is elevated disproportionately to T_3 level, but, when due to liothyronine (T_3) ingestion, serum free T_4 level is suppressed. Thyroid radionuclide uptake and serum thyroglobulin are suppressed. No treatment strategy is known to be particularly effective, but non-judgemental explanation of the medical consequences and gradual dose reduction with psychological supportive therapy, if accepted, is recommended.

Rare forms of hyperthyroidism

TSH-secreting pituitary tumours constitute less than 1% of all pituitary tumours. Thyroid stimulation results in hyperthyroidism with a diffuse goitre, clinically similar to Graves' disease. The presence of high serum free T_4 and T_3 levels with an unsuppressed serum TSH level suggests the diagnosis.[18] Other clinical conditions that need to be distinguished include the partial (pituitary) resistance form of inherited thyroid hormone resistance syndrome,[19] and anomalous laboratory TSH results caused by heterophil antibodies (common antibodies that interfere with assays using mouse monoclonal antibodies; they are either generated directly against mouse antigens after exposure to mice or cross-react with these antigens).

Hypothyroidism

The most common cause of hypothyroidism is primary failure of the thyroid gland. While secondary hypothyroidism from pituitary or hypothalamic dysfunction is rare, it is vital to identify the site of dysfunction at the outset. As serum TSH levels rise logarithmically in response to declining thyroid hormone levels, the distinction between primary and secondary hypothyroidism is usually obvious. The situation can, however, be confounded by impaired pituitary response in concomitant severe non-thyroidal illness, occasionally in ageing, and in profound prolonged hypothyroidism. A modest TSH elevation (up to 15–20 mU/L) in the recovery phase from the severe sick euthyroid state (eg, after acute trauma, burns or infection), or on cessation of dopamine infusion,[20] can cause diagnostic confusion.

Continued Page 8

Hypothyroidism covers a wide spectrum of clinical and biochemical disease, from clinically inapparent disease to myxoedema coma (Box 6). The causes of hypothyroidism are shown in Box 7.

Primary hypothyroidism

In Australia, the most common cause of hypothyroidism is autoimmune chronic lymphocytic thyroiditis. It is more common in women than men (ratio, five to one) and is most common in the fifth and sixth decades, when the prevalence of antithyroid antibodies in women is 10%. Classic Hashimoto's disease, with an enlarged, firm, bosselated thyroid gland, is less common than the atrophic form. Both are characterised by the presence of serum antithyroid antibodies. Whereas levels of thyroglobulin and microsomal antibodies have conventionally both been measured, the sensitivity and specificity of thyroid peroxidase antibody assays make this the only test now required.

Around the world, iodine deficiency still remains the predominant cause of hypothyroidism. Mild iodine deficiency is re-emerging in Australia (possibly related to decreased consumption of iodised salt), but apparently not yet to a level to cause hypothyroidism.[21] Public health measures to address this issue are likely to be needed, including monitoring of iodine intake in the population. More widespread use of iodised salt, both domestically and in manufactured food, may be sufficient to deal with the problem.

Congenital hypothyroidism

Congenital hypothyroidism has an incidence of about one in 4000 births, with thyroid agenesis and ectopia the main causes. Exposure of premature infants to iodine-containing antiseptics can result in transient hypothyroidism.[22] Routine screening of all neonates by heel-prick blood sampling enables detection and treatment by the 10th day after birth. Appropriate thyroxine therapy, progressively adjusted by body weight, results in normal intellectual development.

Subclinical hypothyroidism

Also known as mild thyroid failure and diminished thyroid reserve, "subclinical" hypothyroidism is defined biochemically by a raised serum TSH level in the presence of a serum free T_4 level within the reference range. Because the T_4 set point varies between individuals, it is possible for a serum free T_4 level in the normal range for the population to be too low in a given individual. The risk of progression to frank hypothyroidism increases with increasing TSH levels (odds ratio for women, 8), significant titres of circulating autoantibody (odds ratio, 8) and even more with both (odds ratio, 38).[2]

In general, treatment is recommended if serum TSH level is more than 10 mU/L or progressively rising, or if thyroid antibodies or dyslipidaemia are present[23] (see case report,Box 8).

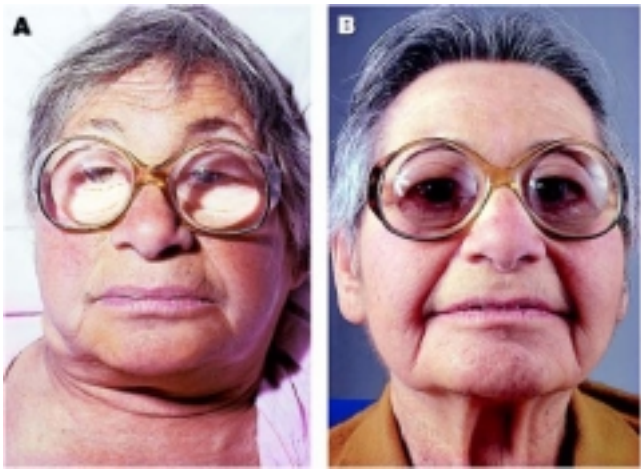
Management of hypothyroidism

Replacement thyroxine is the cornerstone of therapy for hypothyroidism. A dose of 1.6 µg per kg body weight daily is the average required in adults. The principal determinant of dose is lean body mass, so patients in old age may need as little as 50 µg/day. In patients with ischaemic heart disease, a low initial thyroxine dose is recommended (12.5–50 µg/day) to avoid exacerbating angina, but in some patients thyroxine replacement is impossible until coronary artery bypass surgery has been performed, with extreme attention to drug and fluid therapy.

As thyroxine has a half-life of 1 week, once-daily administration is fully adequate to maintain stable levels. It should be taken on an empty stomach, separately from other drugs.[24] Dose should not be adjusted until after a minimum of three to five half-lives to allow a steady state to be attained. In primary hypothyroidism, normalisation of serum TSH level is the best biochemical marker of adequate therapy. Occasionally, symptoms of hypothyroidism appear to persist when TSH level is at the upper end of the reference range, and dose adjustment to achieve the mean normal TSH level of 1–2 mU/L[1] is recommended. In pregnancy, the dose may need to be increased. Maintaining a normal TSH level is important for fetal development.[25]

Combined thyroxine and liothyronine (T_3) therapy is being promoted, particularly on some Internet sites, for patients whose symptoms persist despite apparently adequate thyroxine therapy. There are few published data to support this practice,[26] and a recent Australian study showed no benefit.[27] Thus, it should be discouraged

6: Severe primary hypothyroidism



A: A patient with unrecognised severe primary hypothyroidism who became severely obtunded after surgery for fractured neck of femur. Marked myxoedema is evident.
B: Several months later, after therapy including thyroid hormone replacement.

7: Causes of hypothyroidism

Autoimmune lymphocytic thyroiditis
- Atrophic thyroiditis
- Classic Hashimoto's disease

Post-ablative therapy
- Radioiodine (RAI) therapy
- Thyroidectomy

Transient
- Subacute thyroiditis
- Postpartum thyroiditis
- Early post-ablative therapy (RAI, subtotal thyroidectomy)

Drug-induced
- Thionamide
- Lithium
- Amiodarone
- Interferon
- Drugs that interfere with thyroxine absorption in treated hypothyroidism (iron salts, cholestyramine, sucralfate)

Iodine-associated
- Iodine-deficiency disease
- Iodine-induced

Infiltrative
- Reidel's thyroiditis
- Scleroderma
- Amyloid disease
- Haemochromatosis

Neonatal/congenital
- Thyroid agenesis/ectopia
- Genetic disorders of TSH, TSH receptor, thyroid peroxidase, thyroglobulin, pendrin
- Transplacental passage of blocking TSH-receptor antibody

Secondary
- Pituitary or hypothalamic disease

Other
- Thyroid hormone resistance

TSH = thyroid stimulating hormone

in the absence of evidence of benefit, with thyroxine alone remaining standard therapy.

In patients with hypothalamopituitary hypothyroidism, serum TSH level is not a valid biochemical indicator of adequate replacement, and it is usual to adjust the thyroxine dose to achieve an approximate mean normal serum level of free T_4.

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8: Case report — subclinical hypothyroidism

Presentation: A 52-year-old woman presented with tiredness, increasing over the previous 6 months. She was perimenopausal but did not have severe vasomotor symptoms. She was not depressed, and there were no significant physical findings.

Investigations: Routine laboratory tests excluded anaemia and iron deficiency, but serum free thyroxine (T_4) level was 11.9 pmol/L (reference range [RR], 9.3–23.8 pmol/L); and thyroid stimulating hormone (TSH) level was 8.2 mU/L (RR, 0.3–4.7 mU/L). On review of specific symptoms, the patient felt her skin was a little dry and her mental concentration had deteriorated. She had chronic constipation.

Comment*: This is a common problem. Symptoms are mild, non-specific and shared by many in the population who are not hypothyroid.*

Repeat thyroid function tests confirmed earlier results, with free T_4, 12.2 pmol/L; and TSH, 7.6 mU/L. Thyroid peroxidase antibodies were present at 1200 U/mL (RR, < 35 U/mL). Serum lipid tests showed cholesterol, 5.7 mmol/L (RR, < 5.5 mmol/L); high density lipoprotein, 1.2 mmol/L (RR, > 1.0 mmol/L); and triglycerides, 1.8 mmol/L (RR, < 2.0 mmol/L).

Comment*: The patient has subclinical hypothyroidism (also known as mild thyroid failure or diminished thyroid reserve). In the presence of circulating antithyroid antibodies (thyroid peroxidase or thyroid microsomal antibodies), the risk of progression to frank hypothyroidism is 5% per year. Hypothyroidism may be contributing to the hyperlipidaemia and to accelerated coronary disease.*

The patient was convinced that hypothyroidism was the cause of her symptoms. She had read on an Internet site about severe problems from hypothyroidism and that combined thyroxine and liothyronine (T_4/ T_3) therapy is best.

Comment*: The worth of therapy in these circumstances is unresolved. In its favour are a possible reduction in symptoms, early prevention of progression to frank hypothyroidism, and possible prevention of accelerated vascular disease.*

Against giving therapy is that symptoms very often do not change, and compliance is therefore poor, lipid profile generally does not improve when TSH is < 10 mU/L, there is no good evidence that vascular disease risk is reduced, and therapy increases the risk of subclinical hyperthyroidism and loss of bone mineral density in postmenopausal woman.

General recommendations for asymptomatic patients are:

- *Patients with TSH < 10 mU/L and no antithyroid antibodies should be monitored, with therapy started if TSH level increases above 10 mU/L.*
- *Patients with TSH 5–10 mU/L and antithyroid antibodies present could be treated, depending on patient preference. The best therapy is thyroxine alone, adjusted for a TSH level of 1–2 mU/L. Patients who choose not to be treated should have annual monitoring.*

The patient elected to have treatment (thyroxine, 100 µg/day). After 4 months, her thyroid function results were normal (free T_4, 15.8 pmol/L; TSH, 1.9 mU/L), but she felt no different. She elected to discontinue treatment, but, as she was positive for thyroid peroxidase antibodies, her GP recommended annual thyroid function testing.

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Volunteer News

By **Bronwyn Stevens**

As you have probably read, we now have an office! Until now Thyroid Australia has been running out of Board members' houses, making it difficult to coordinate volunteers. Now that all information and contacts are passing through a central point and we have a meeting place, this is changing. This means we can not only help volunteers more, it also means we need more volunteers to help us!

If you have called Thyroid Australia recently, your call was probably answered by our answering machine. This is because we are desperately in need of administration volunteers. We currently have fewer than ten volunteers who help with administration duties and, as they have lives to lead outside Thyroid Australia, they simply cannot be in the office everyday. They are also having trouble coping with the over 100 work items that Thyroid Australia needs to process each week.

We would like to employ a part-time paid staff member, but we do not have the funds to do so. Membership fees and donations go towards providing support and information to those suffering from thyroid conditions; we simply don't have enough money left to employ anyone. So as you can see, we are also desperately in need of fundraising volunteers, especially someone to organise activities. We have a number of ideas for raising money, but we lack people to put these ideas into action.

If you feel you are able to help with administration, fundraising, or any of the positions listed below, please contact me via the Thyroid Australia office for more detailed information about what these roles entail. If you feel that you can help Thyroid Australia in a way not described, please also contact me with your ideas. Remember, you can take on informal roles as well as a formal position. You can even take on multiple formal or informal volunteer opportunities. However, bear in mind that you have to live your life as well. Don't feel obligated to help in ways that you feel you cannot, but if you can help, please call us. We need you.

You won't be dumped in the corner and be expected to work out what to do by yourself. The volunteers we have already are a cheery bunch who will be able to show you the ropes. We have detailed instructions for all the jobs so you won't have any problems getting into the tasks. We all know what it is like living with a chronic condition so we are flexible.

If you are not too sure, why not pop in on us and see what goes on. I am sure that you will be pleasantly surprised while we would welcome the company. We are close to the Mount Waverley station and the bus route along Waverley Road and we have ample off road parking. Just call ahead to make sure someone is in the office.

If you are already generously donating your time to us, THANK YOU. Your assistance is invaluable and greatly appreciated.

Formal Positions

We have prepared guidelines for people filling these roles. This is to help them and to ensure the reliability of information provided to our clients. Taking on any of these positions also provides you with bonus advantages on top of those provided by your membership. These include free entry to all Thyroid Australia meetings, free articles from the publications list and a free Ulysses Butterfly pin.

- Administration Work at the Office - 333 Waverley Rd, Mount Waverley, Victoria.
  - Work as regularly as you choose (one day or half a day per week, fortnight or month).
- Coordinating the Distribution of Promotional Material
  - Mailing publicity material to those who wish to distribute it in their areas.
- Emergency Telephone Support
  - Providing support to people with your thyroid condition (none of your details are publicly available).
- Fundraising
  - Organising fundraising activities, applying for grants and maximising Thyroid Australia's potential.
- Liaising with Key Organisations
  - Keeping up to date with affiliated organisations' activities.
- Running Regular Coffee Mornings for Members at the Office
  - Organising and running a regular small coffee morning at the office for members to attend.
- Telephone Support
  - Providing support to people with your thyroid condition (your contact details are publicly available).
- Turning Your Members Contact List (Area) into a Support Group
  - You don't need to organise large or frequent meetings, and we help you to get set up.

Informal Roles

These roles require much less commitment than the formal volunteer positions. Most of them can be done in your own time and in your own home, and you are free to resign from any them whenever you need. They are ideal for people who do not have a fixed schedule. Taking on one of these roles doesn't qualify you for any formal benefits, but your help will be appreciated immensely.

- Distributing Publicity Material
  - Distributing general Thyroid Australia publicity material (brochures and posters) in your local area.
- Emergency Administration Work at the Office
  - Helping with general administration in the office on a non-regular basis.
- Helping at Mass Mailouts
  - Helping send out the newsletter from the office.
- Helping at Meetings
  - When you help at a meeting, entry is free.
- Liaising with Other Organisations
  - Keeping up to date with the activities of an organisation of your choice.
- Preparing Material for Publication in the Newsletter
  - Researching and/or writing an article on a topic of your choice for the newsletter.
- Preparing a Thyroid Australian Information Pamphlet
  - Preparing Australian pamphlets on thyroid topics. We will provide all the relevant information.
- Transcribing Audio Tapes of Seminars
  - Transcribing normal audio tapes (not Dictaphone tapes) of the speakers' presentations.
- Translation of Material
  - Translating Thyroid Australian Information Pamphlets into any language other than English.

Bronwyn Stevens is a final year Behavioural Neuroscience student at Monash University and Thyroid Australia's Volunteer Co-ordinator. ❀

Editorial *from Page 1*

These products are now required to be kept under refrigeration. The Therapeutic Goods Administration (TGA) required the change because the strength of the thyroxine could not be guaranteed over a long shelf life. We recommend asking your pharmacist to ensure that the expiry date on your thyroxine is visible. One effect we expect in the long term will be the reduction in the number of tablets per bottle as the shelf life isn't long enough. The consequence of this is likely to be increased charges for thyroxine. More information on this is contained in this edition. Want to do something about it? Write to the Therapeutic Goods Administration, PO Box 100, Woden ACT 2606 or your local federal parliamentarian.

We are now in planning our SEMINAR for 2004. It will be held at Monash University on Sunday 7th November, 2004. The Seminar involves a very large amount of work for our committee and this year will be our major event to raise funds for the organization. The fee this year will be $50 per person – part of which will be a tax deductible donation of $25.00. This is more than in previous years, but will include the cost of a catered lunch and pre-prepared seminar notes by the speakers:

Professor Jack Wall (Endocrinologist) will speak on Autoimmunity as related to Graves, Hashimoto's and Thyroid Eye disease.

Dr Richard Arnott (Endocrinologist) will speak on the treatment and management of Thyroid illnesses

Dr Maryanne Papalia (Endocrinologist) will speak on Insulin Resistance, Type 2 Diabetes and Hypothyroidism.

Cathy Thesing (Dietician) will speak on the weight management issues related to people with a slow metabolism and Type 2 diabetes.

This year it is essential that you book your place (closing date is the 22nd October) – so we can get you entry ticket to you before the day.

We look forward to seeing on the 7th November.

Good Health to you all,
Gail M. Mgt. ❀

Thyroid Australia Ulysses Butterfly Pins

Show your support for Thyroid Australia by wearing a Ulysses butterfly pin.

Beautifully made in pewter and enamel by craftsmen in Ballarat.

Suitable for men and women.

Shown full size. Centre section is bright blue.

Cost: $7.00 (includes $2 postage and packaging).

Send us your name, address, and a cheque or money order for $7.00 (please, no cash or credit card payments) and we will get a pin out to you as soon as possible.

A butterfly pin will make it easier for members participating in the Members' Contact Lists to recognise each other when meeting up for a coffee …

✂

Please copy or detach and mail to the address below.

Request for Membership Application Form and Information

Date: .....

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

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

Please send me a Membership Application Form and information about the following (please tick relevant boxes):

- ❑ General thyroid information
- ❑ Thyroid function tests
- ❑ Hypothyroidism (underactive thyroid)
- ❑ Hyperthyroidism (overactive thyroid)
- ❑ Thyroid eye disease
- ❑ Thyroid cancer
- ❑ Thyroid nodules
- ❑ Paediatric issues
- ❑ Fertility and pregnancy
- ❑ Other (please specify) .....
- ❑ Publications List & Order Form

Please send this information to:

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

Address: .....

.....

Phone: .....

Signature: .....

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