Management of **thyroid dysfunction** in **adults**

Key concepts

- Routine screening for thyroid dysfunction is not recommended unless there are symptoms and signs of thyroid disease
- TSH is the best initial test and an abnormal result will trigger laboratory reflex testing of additional thyroid function tests as indicated, in most laboratories
- Thyroid dysfunction (hypo- or hyperthyroid) can be classified as overt or subclinical and treatment is guided by TSH results and the clinical situation
- Levothyroxine is the treatment of choice for hypothyroidism
- Carbimazole is most often the initial choice of treatment for Graves' hyperthyroidism
- Other treatment options for hyperthyroidism include β-blockers, radioactive iodine and surgery
- Screening for thyroid dysfunction in pregnant women is not routinely recommended in New Zealand, however, testing should be considered if there are symptoms of thyroid disease or in women who are at increased risk of hypothyroidism

Diagnosis of thyroid dysfunction

Conditions that affect the thyroid gland are common, affecting 5% of women and 1% of men in New Zealand.[1] The incidence of thyroid dysfunction (particularly hypothyroidism) tends to increase with age.[2,3]

Clinical assessment and judgement should guide initial testing when a diagnosis of thyroid dysfunction is suspected. The common symptoms and signs are presented in Box 1. A family history of thyroid dysfunction may also increase clinical suspicion. There is a lack of evidence to support routine screening in asymptomatic people, therefore testing for thyroid dysfunction is not recommended unless there are symptoms and signs of thyroid disease.[4,5]

TSH can be used as the initial measure of thyroid function in most cases

In most situations serum thyroid stimulating hormone (TSH) can be used as the initial measure of thyroid function.[6] If further tests, such as serum free thyroxine (FT4), free triiodothyronine (FT3) or thyroid antibodies (see over page) are required following an abnormal TSH result, these may be added to the original request without the need for the patient to have a second blood test. Most laboratories do this automatically following an abnormal TSH result ("reflex" testing) or the additional tests may be added by the clinician. To assist the laboratory it is useful to include relevant clinical details and medications on the request form.

Box 1: Symptoms and signs of thyroid dysfunction (adapted from "Investigating Thyroid Function", bpac^{nz}, 2005)

	Hypothyroidism	Hyperthyroidism
High suspicion	Goitre Delayed reflexes	Goitre Thyroid bruit (secondary to increased blood flow) Lid lag Proptosis (bulging eyes)
Intermediate suspicion	Fatigue Weight gain/difficulty losing weight Cold intolerance Dry, rough, pale skin Constipation Facial swelling (oedema) Hoarseness	Fatigue Weight loss Heat intolerance/sweating Fine tremor Increased bowel movements Fast heart rate/palpitations Staring gaze
Low suspicion **Non-specific symptoms**	Coarse, dry hair Hair loss Muscle cramps/muscle aches Depression Irritability Memory loss Abnormal menstrual cycles Decreased libido	Nervousness Insomnia Breathlessness Light or absent menstrual periods Muscle weakness Warm moist skin Hair loss

In summary: the use of thyroid function tests for investigation

Asymptomatic patients:

- Do not test for thyroid dysfunction unless specifically indicated

Patients with symptoms or signs of thyroid dysfunction:

- Request TSH
- During a non-thyroidal illness (sick euthyroid syndrome), there may be transient changes in TSH, FT4 and FT3. If possible defer thyroid function testing until this illness has resolved.

Patients on medicines that can affect thyroid function:

- Amiodarone – patients on long-term therapy should have six monthly TSH tests
- Lithium – use TSH once a year to check thyroid function

See Page 28

Request both TSH and FT4:

- During pregnancy
- If there is suspected non-adherence to the thyroid replacement regimen
- When a patient is suspected of having pituitary failure (a low FT4 with an inappropriately normal TSH is usually seen)

Specific groups of people are at higher risk of developing hypothyroidism (Box 2) and some recommend screening these people every one to two years or if there are symptoms or signs of thyroid disease.

Box 2: People who may be at increased risk of hypothyroidism (adapted from Vaidya, 2008)[5]
Those with other autoimmune disease, e.g. type 1 diabetes, Addison's disease, coeliac disease
Those with a genetic condition such as Down or Turner syndromes
Those who have had treatment with radioactive iodine therapy or surgery for hyperthyroidism
Those who have had radiotherapy to the neck for head and neck cancer
Those with a history of postpartum thyroiditis

"Thyroid antibodies" is a non specific term that encompasses the tests for thyroid peroxidase antibodies (TPO-Ab) and the less common anti-thyroglobulin antibodies. TPO antibodies were previously referred to as microsomal antibodies. TPO antibodies are a risk factor for autoimmune thyroid disorders. In subclinical disease, the presence of TPO-Ab increases the long-term risk of progression to clinically significant thyroid disease by approximately two-fold. Almost all people with autoimmune hypothyroidism and up to 80% of those with Graves disease have TPO antibodies, usually at high levels, although they are also found in a small number of people who are euthyroid.[4]

Thyroid dysfunction can be classified as overt or subclinical

Primary hypothyroidism

Overt hypothyroidism affects approximately 1–2% of women and 0.1% of men and is characterised by a TSH concentration above the normal reference range and a FT4 concentration below the reference range.[4, 6]

Untreated overt hypothyroidism can cause fatigue, weight gain, abnormal lipid profile, heart failure, and, in children, can retard growth and mental development.[7] Myxoedema coma is a rare complication of hypothyroidism most often

occurring in elderly people with undiagnosed disease or in patients who are poorly compliant with treatment.[4]

Subclinical hypothyroidism affects women more than men and occurs more frequently with increasing age – up to 10% of women over 60 years of age have elevated TSH levels.[4] It is characterised by a TSH concentration that is increased above the reference range but FT4 concentration within the normal range. Patients with subclinical hypothyroidism may develop overt hypothyroidism.

Hyperthyroidism

Overt hyperthyroidism affects 1.9% of women and 0.16% of men and is characterised by a TSH level lower than the reference range and FT4 and/or FT3 levels above the normal reference range.[8] Complications include Graves' opthalmopathy, thyrotoxic crisis, atrial fibrillation, loss of bone mass and congestive heart failure.[8]

Subclinical hyperthyroidism affects approximately 2% of adults and increases with advancing age with 3% of adults over 80 years of age being affected.[3] It is characterised by TSH lower than the reference range but FT4 and FT3 levels within the normal reference range.

Management of hypothyroidism

The most common cause of hypothyroidism is autoimmune thyroid disease (Hashimoto's thyroiditis and atrophic thyroiditis),[9, 10] although in many parts of the world iodine deficiency remains a major cause of hypothyroidism. Other causes include thyroidectomy, radioiodine ablation, drug induced hypothyroidism and congenital hypothyroidism.[5]

In most cases GPs diagnose and manage hypothyroidism. Replacement treatment with levothyroxine is appropriate for symptomatic patients with TSH above 10 mIU/L. However, the decision to treat may depend on the clinical situation, e.g. a lower threshold to treat in a young woman, particularly if she may become pregnant, than in a very elderly patient. It is good practice to request a second TSH to confirm the diagnosis, as treatment is usually life-long.[5]

Levothyroxine is used to treat hypothyroidism

Levothyroxine is a synthetic form of the natural hormone thyroxine (T4), and is the treatment of choice for hypothyroidism because it reliably relieves symptoms, stabilises thyroid function tests and is safe.[6] The body converts levothyroxine to liothyronine (T3) as necessary. The dose of levothyroxine is dependent on body weight and age. Most adults will achieve euthyroidism with a dose of approximately 1.6 mcg/kg/day.[5, 10] For example, in an adult weighing 60 kg the dose required would be approximately 100 mcg/day and for an adult weighing 80 kg, approximately 125 mcg/day.

Young, otherwise healthy patients can usually start with the expected full dose.[5, 6, 11] Long standing bradycardia due to hypothyroidism can mask substantial asymptomatic coronary artery disease.[5] Treatment with levothyroxine also carries a small risk of inducing cardiac arrhythmias, angina or myocardial infarction.[11] Therefore, for people older than 60 years and those with ischaemic heart disease, it is recommended that low initial doses are used, i.e. start on 12.5 mcg to 25 mcg daily with dose increases of 25 mcg, approximately every six weeks, as guided by TSH results, until euthyroidism is achieved.[5, 11]

Hypothyroid symptoms generally improve within two to three weeks, however, it can take several months before a patient feels back to normal health after biochemical correction of hypothyroidism.[5] Once the target TSH has been reached, a further TSH test in three to four months is often helpful to ensure the TSH is stable. Patients on long-term, stable replacement treatment usually require only an annual TSH, unless pregnant (see Page 31). If for any reason a dose adjustment takes place, TSH testing will be required after approximately six to eight weeks.

There are currently several different brands of levothyroxine funded in New Zealand. The active ingredient, levothyroxine, is the same in all brands but some of the other tablet constituents differ and may affect absorption of levothyroxine. If a patient switches brands, TSH should be repeated six weeks later.

Liothyronine and whole thyroid extract

Liothyronine is a synthetic thyroid hormone which replaces T3. It is not funded on the Pharmaceutical Schedule in New Zealand, however, can be obtained via section 29.* Combined use of liothyronine and levothyroxine is promoted on some websites, however, there is no convincing evidence that such a regimen is better than levothyroxine alone, and it may even be harmful.[12]

Whole thyroid extract is produced from dried thyroid gland from domesticated animals (usually pigs). It contains both T3 and T4. There have been no clinical trials published to determine its effectiveness or safety.[5]

* Section 29 of the Medicines Act 1981 permits the sale or supply to medical practitioners of medicines that have not been approved, and requires the "person" who sells or supplies the medicine to notify the Director-General of Health of that sale or supply in writing, naming the medical practitioner and the patient, describing the medicine and the date and place of sale or supply.

Levothyroxine adverse effects and interactions

Adverse effects with the appropriate use of levothyroxine are rare, however, they may occur when excessive doses are taken.[6] Excessive doses may result in symptoms of hyperthyroidism such as fatigue, arrhythmias, sweating, tremor, heat intolerance, diarrhoea, muscle cramps and muscle weakness. These effects usually resolve with dose reduction or discontinuation.[4]

Calcium, iron, aluminium hydroxide (antacids) and cholestyramine reduce the absorption of levothyroxine, therefore these are best taken at least four hours apart from levothyroxine.[13] For maximum absorption, levothyroxine is best taken on an empty stomach before breakfast,[13] although if the patient forgets, the tablet should still be taken to encourage compliance. Levothyroxine has a long half-life of approximately seven days,[13] so in practice if a tablet is missed the patient will be unlikely to be aware of any noticeable change.[5]

Some anticonvulsants, e.g. phenytoin and carbamazepine, and oestrogen therapy, such as hormone replacement therapy, can increase levothyroxine requirements, therefore TSH should be rechecked six weeks after commencing treatment.[13] There are a number of other medications that may also affect the absorption of levothyroxine. For further information, refer to the medicine datasheet.

Use TSH for monitoring with levothyroxine

TSH is the most appropriate test when monitoring patients receiving levothyroxine for the treatment of hypothyroidism.[6] It should be measured no sooner than six to eight weeks after the start of treatment. If thyroid function needs to be assessed before this time, FT4 should be used, as TSH will not have plateaued at this stage. FT3 has little value in monitoring patients with primary hypothyroidism on replacement treatment as it may be affected by other factors such as illness.

The usual goal of treatment is for TSH to be within the reference range and symptoms to improve. Age and the presence of co-morbidities may guide the target TSH level

and the rate at which it is achieved, e.g. slower attainment of target TSH in elderly people and conversely more rapid in younger people.

Specialist referral may be required for some patients with hypothyroidism

It may be appropriate to refer patients for specialist care in the following circumstances:[4, 5]

- Patients who have TSH levels persistently above the normal reference range despite full doses of levothyroxine being taken. However, first check compliance and drug interactions and consider excluding coeliac disease (which may cause malabsorption) as there is some evidence that these two autoimmune conditions may co-exist.[5]
- Patients whose symptoms do not respond or worsen after treatment with levothyroxine
- Patients who are pregnant or postpartum
- Children aged less than 16 years
- Patients with co-morbidities, e.g. unstable ischaemic heart disease

If secondary hypothyroidism (from pituitary or hypothalamic disease) is suspected, then referral is always indicated.[4]

Treatment of subclinical hypothyroidism

For patients with **TSH less than 10 mIU/L**, treatment with levothyroxine may be considered if symptoms of hypothyroidism develop. Treatment may also be considered in patients with a rising TSH or in those who have goitre. If treatment is initiated then it should be for a sufficient length of time, e.g. three months, to assess whether there is symptomatic benefit.[4, 11] Patients not treated with levothyroxine should be monitored using TSH every 6–12 months or if symptoms develop.[4, 11]

A common cause of subclinical hypothyroidism is autoimmune Hashimoto's thyroiditis and many of these patients subsequently develop overt hypothyroidism

In summary: the use of thyroid function tests for monitoring patients on levothyroxine

Men and non-pregnant women:

- Wait at least six weeks to test TSH after any adjustment of the dose of levothyroxine
- Monitor stable patients annually with TSH only

Women planning pregnancy:

- Check TSH of women with past TSH elevation or positive thyroid antibodies (whether or not on treatment)

Pregnant women:

- Check TSH and FT4 early in pregnancy, four weeks later, four to six weeks after any change in the dose of levothyroxine, and at least once each trimester

Postpartum:

- The levothyroxine dose can be reduced to the usual (pre-pregnancy) maintenance dose postpartum with TSH checked six weeks later

(approximately 5% per year), especially if thyroid antibodies are strongly positive. For patients with strongly positive thyroid antibodies and TSH persistently above 7 mIU/L, levothyroxine therapy is sometimes commenced.

For patients with **TSH persistently greater than 10 mIU/L** (i.e. TSH ≥ 10mIU/L on repeated testing at least three months apart), treatment with levothyroxine should be considered depending on the clinical situation.

Management of hyperthyroidism

Common causes of hyperthyroidism are Graves' disease and toxic nodular goitre. Graves' disease generally appears in people aged 20 to 40 years, whereas the prevalence of toxic nodular goitre increases with age. Thyroiditis is another important cause of hyperthyroidism which commonly occurs in women who are postpartum,[19] as well as in people with viral-type symptoms and neck pain, referred to as "subacute thyroiditis".

Amiodarone and lithium can cause thyroid dysfunction

Amiodarone

Amiodarone can cause thyroid dysfunction (either hyper- or hypothyroidism) in 14–18% of patients due to its high iodine content (75 mg organic iodine per 200 mg tablet)[14] and its direct toxic effect on the thyroid.[15] Although treatment with amiodarone causes an initial rise in TSH because of the effect of the excess iodine, levels return to within the normal range after three months. Amiodarone inhibits the peripheral conversion of T4 to T3 and therefore during treatment FT4 is usually increased and FT3 normal or decreased.[15,16]

Recommendations for monitoring thyroid function in patients on amiodarone vary, but the best marker of amiodarone-induced thyroid dysfunction appears to be TSH. In the majority of laboratories, TSH results that are outside the normal reference range will trigger reflex testing of FT4 and if TSH is low, FT3. TSH testing is therefore recommended at baseline and then six monthly for patients taking amiodarone. Amiodarone has a long half-life so monitoring is required up to 12 months after cessation of treatment.[15]

Clinical monitoring for symptoms and signs of thyroid dysfunction is also required as often amiodarone induced hyperthyroidism can develop rapidly.[16] If new signs of arrhythmia appear, consider hyperthyroidism as the potential cause.[17] Patients with multinodular goitre are at increased risk of developing amiodarone-induced hyperthyroidism.

Pre-existing Hashimoto's thyroditis and/or the presence of TPO antibodies increase the risk of developing hypothyroidism during treatment with amiodarone therefore some experts recommend testing for TPO antibodies before amiodarone is initiated.[16]


Previous guidance on monitoring amiodarone has recommended that both TSH and FT4 are tested. It is now standard practice to monitor only TSH, as abnormal results will trigger reflex testing.

Lithium

Lithium-associated hypothyroidism is common and can appear abruptly even after long-term treatment. Females and people with positive TPO antibodies are at increased risk of this.[18]

Lithium-associated hyperthyroidism is rare and occurs mainly after long-term use.[18]

It is recommended that for monitoring patients on lithium, TSH and FT4 are tested at baseline, then TSH only at three months and annually thereafter. Patients should also be monitored for signs of thyroid dysfunction and should have thyroid function tests earlier if symptoms develop.

The management of hyperthyroidism depends of the cause and severity of disease, patient's age, goitre size, concurrent medication or co-morbidities and, especially in Graves' disease (where there may be a choice of treatment), patient preference.[9] Anti-thyroid medicines, radioactive iodine and surgery are the main options for treatment of persistent hyperthyroidism. β-blockers, e.g. propranolol, may be used as a treatment adjunct to control symptoms such as tremor and tachycardia.[8,20]

Patients who are systemically unwell or who have severe symptoms and signs of hyperthyroidism, e.g. fever, agitation, heart failure, confusion or coma, may require hospital admission.[8]

Carbimazole is often used for the first episode of Graves' disease

Anti-thyroid drugs, such carbimazole (a thionamide), are normally used for the first episode of Graves' disease. Thionamides, however, are not indicated for thyroiditis where there is no excessive production of thyroid hormones.[19]

Carbimazole decreases thyroid hormone synthesis by interfering with the organification (oxidation and binding) of iodine.[20,21] Treatment with carbimazole may be started in primary care. In patients where the diagnosis is uncertain, referral to an endocrinologist is recommended.

Carbimazole is usually given at a dose of 15 to 40 mg daily until the patient becomes euthyroid, usually after four to eight weeks. The dose is then gradually reduced to a maintenance dose of 5 to 15 mg.[22] A block and replace regimen has been used where high doses of a thionamide are used in combination with levothyroxine. However, there is no clear benefit to this method[19] and it is not suitable in pregnancy.[22]

Prolonged use for 12 to 18 months provides the best chance of sustained remission in Graves' disease.[19] However, relapse occurs in approximately 50% of patients.[9] Relapse is more likely in patients who smoke, who have large goitres or who have suppressed TSH levels at the end of therapy.[19,23]

Monitoring patients on thionamides

It is recommended that thyroid function be monitored every four weeks using FT4 and TSH to adjust the dose until the TSH normalises and clinical symptoms have improved. The patient can then be monitored every two months using TSH only.

N.B. some patients can have T3 toxicosis where monitoring of TSH, FT4 and FT3 is necessary – advice from an endocrinologist is recommended.

Adverse effects of thionamides

Minor adverse effects such as rash, fever and gastrointestinal disturbances occur in up to 5% of patients taking thionamides and can often be treated symptomatically without the need to discontinue treatment. Bone marrow suppression resulting in agranulocytosis is a rare but serious adverse effect of thionamides occurring in 0.1 to 0.5% of patients taking these medicines (see sidebar over page).[20,21]

β-blockers provide rapid relief of adrenergic symptoms

β-blockers, such as propranolol, provide rapid relief of adrenergic symptoms, e.g. tachycardia, tremor, heat intolerance and anxiety. They can be initiated in most patients, as soon as a diagnosis of hyperthyroidism is made, to provide symptomatic relief while waiting for test results. β-blockers can be continued until the patient becomes euthyroid. They are also used to provide symptomatic relief in patients with thyroiditis where thionamides are not appropriate.[9]

Other treatments – radioactive iodine and surgery

Relapses of hyperthyroidism due to Graves' disease are usually treated with radioactive iodine, or occasionally surgery, as repeat courses of drugs rarely lead to

Risk of agranulocytosis with thionamides

All patients on thionamides should be warned about the rare but serious complication of agranulocytosis. Patients should be instructed to stop their anti-thyroid medication and consult a doctor if fever, sore throat or other infection develops. Patients should have an urgent white blood cell count performed, looking for evidence of neutropenia.[22] Because the onset of agranulocytosis is abrupt, and the occurrence is rare, routine full blood counts are not recommended for patients on thionamides.[9]

Best Practice Tip: When writing a prescription for a thionamide, include instructions for the patient to report fever, sore throat or infection that will be printed on the medication label. Some patient management systems will allow for this information to be stored in "preferred medication prescription instructions" so it does not have to be typed in on an individual basis.

remission.[7] These options may also be an appropriate first choice treatment for toxic nodular goitre because remission is rare when it is treated with anti-thyroid drugs.[9, 19] In some countries, such as the United States, radioactive iodine is used as a first-line treatment for both Graves' disease and toxic multinodular goitre.[20]

Radioactive iodine is an effective treatment with 80–90% of patients becoming euthyroid after a single dose.[19] There is a risk of worsening hyperthyroidism shortly after treatment due to pre-formed thyroid hormone leaking from the damaged thyroid.[19] Patients may be prescribed anti-thyroid medicines that are taken before or shortly after treatment with radioactive iodine in an attempt to prevent this. Although there is a small risk that this may increase treatment failure.[19,24]

The risk of permanent hypothyroidism increases with the dose of radioactive iodine and with time.[9] Most patients will eventually develop hypothyroidism after treatment with radioactive iodine and therefore lifelong annual monitoring of TSH is recommended.[9,19]

Surgery is an appropriate choice for patients with a goitre causing local compression[9] and for selected patients with Graves' thyrotoxicosis, for example in a patient with very large goitre and patients with severe ophthalmopathy (which may be exacerbated by radioiodine).[19,20]

Subclinical hyperthyroidism

Common causes of subclinical hyperthyroidism include excessive levothyroxine replacement, autonomously functioning multi-nodular goitre and subclinical Graves' disease. These patients are at increased risk of developing atrial fibrillation and possibly osteoporosis. Further investigation and treatment should be considered for patients with an undetectable TSH on repeated testing.

Management of thyroid dysfunction in pregnancy

Hypothyroidism in pregnancy

Screening for thyroid dysfunction in women planning pregnancy, and those who are pregnant, is not routinely recommended in New Zealand. However, it is known that subclinical hypothyroidism may be associated with ovulatory dysfunction and infertility. Undetected subclinical hypothyroidism during pregnancy may be associated with adverse outcomes such as hypertension, pre-eclampsia, premature delivery and a risk of cognitive impairment in the infant.[4,25]

Thyroid testing should be considered if there are symptoms of thyroid disease or in women who are at increased risk of hypothyroidism, such as those with type 1 diabetes, a personal or family history of thyroid disease or those with goitre.[4]

TSH may be temporarily suppressed during the first trimester of pregnancy, due to the thyroid stimulating effect of hCG. FT4 levels tend to fall slowly in the second half of pregnancy.

In women with previous mildly elevated TSH who are considering pregnancy, TSH should be checked. If TSH is elevated, thyroid function should be restored to within the reference limit prior to conception if possible.

In hypothyroid pregnant women receiving treatment, the goal should be normalisation of both TSH and FT4. The majority of women receiving levothyroxine need a dose increase during pregnancy, usually during the first trimester. A "pro-active" dose increase of 30% has been recommended once pregnancy is confirmed.[26] This is most easily done by asking the woman to double her maintenance daily dose of levothyroxine on two days each week. Dose requirements stabilise by 20 weeks and then fall back to non-pregnant levels in a short time after delivery. FT4 should be maintained above the 10th percentile of the range (about 11–13 pmol/L) from week six to week 20. There is strong observational evidence that this approach allows optimal foetal neurological development. TSH and FT4 should be checked early in pregnancy then every six to eight weeks during pregnancy and at the start of trimesters two and three. More frequent re-testing is sometimes indicated, e.g. four weeks after adjustment of levothyroxine dose.

Hyperthyroidism in pregnancy

Pregnant women with hyperthyroidism may be at increased risk of foetal loss, pre-eclampsia, heart failure, premature labour and having a low birth-weight infant.[26]

Thionamides are the preferred treatment choice in pregnancy. It is appropriate to use the lowest possible dose needed to control symptoms and achieve euthyroidism. In the last trimester many women can cease their anti-thyroid medication. Aiming for a FT4 in the upper third of the normal reference range for non-pregnant women may minimise the risk of foetal hypothyroidism.[26]

Of the thionamides, propylthiouracil is preferred (but is only available via the Exceptional Circumstances scheme*) as carbimazole has been associated with rare teratogenic effects. Propylthiouracil has rarely been associated with significant liver toxicity and some guidelines recommend changing from propylthiouracil back to carbimazole after the first trimester. A block and replace regimen is not suitable in pregnancy because thionamides cross the placenta in excess of levothyroxine and may result in foetal hypothyroidism and goitre.[26]

Graves' thyrotoxicosis frequently relapses postpartum. Monitoring of TSH at six weeks postpartum and if symptoms recur is appropriate.

Radioactive iodine is contraindicated in pregnancy and for six months pre-conception.[26]

* Propylthiouracil is not listed on the Pharmaceutical Schedule, but can be made available for patients meeting specific criteria, where there is no suitable alternative.

ACKNOWLEDGEMENT Thank you to **Dr Penny Hunt**, Consultant Endocrinologist, Canterbury DHB, Senior Lecturer, Christchurch School of Medicine, University of Otago, Christchurch for expert guidance in developing this article.

References:

1. Ministry of Health. A Portrait of Health. Key Results of the 2006/07 New Zealand Health Survey Ministry of Health: Wellington; 2008.
2. Flynn RWV, MacDonald TM, Morris AD, et al. The thyroid epidemiology, audit and research study: thyroid dysfunction in the general population. J Clin Endocrinol Metab 2004;89(8);3879-84.
3. Gibbons V, Conaglen JV, Lillis S, et al. Epidemiology of thyroid disease in Hamilton (New Zealand) general practice. Aust NZ J Pub Health. 2008;32(5);421-3.
4. Clinical Knowledge Summaries. Hypothyroidism. 2007. Available from: www.cks.nhs.uk (Accessed Nov, 2010).
5. Vaidya B, Pearce S. Management of hypothyroidism in adults. BMJ 2008;337:a801.
6. McDermott M. In the clinic. Hypothyroidism. Ann Intern Med 2009;151(11):plTC6 2-16.
7. National Prescribing Service. Management of common thyroid diseases. MeRec Bulletin 2002;12(3):9-12.
8. Clinical Knowledge Summaries. Hyperthyroidism. 2008. Available from: www.cks.nhs.uk (Accessed Nov, 2010).
9. Topliss D, Eastman C. 5: Diagnosis and management of hyperthyroidism and hypothyroidism. Med J Aust 2004;180(4):186-94.
10. Davoren P. Modern management of thyroid replacement therapy. Australian Prescriber 2008;31(6):159-61.
11. Ross DS. Treatment of hypothyroidism. UpToDate 2010. Available from: www.uptodate.com (Accessed Nov, 2010).
12. Royal College of Physicians. The diagnosis and management of primary hypothyroidism. 2008. Available from: www.rcplondon.ac.uk (Accessed Nov, 2010).
13. Boucher & Muir New Zealand Limited t/a Goldshield Healthcare (New Zealand). Levothyroxine. Medicine Safety datasheet. 2009. Available from: www.medsafe.govt.nz (Accessed Nov, 2010).
14. Sanofi-Aventis New Zealand Limited. Cordarone X. Medicine Safety datasheet. 2008. Available from: www.medsafe.govt.nz (Accessed Nov, 2010).
15. Ross DS. Amiodarone and thyroid dysfunction. UpToDate 2010. Available from: www.uptodate.com (Accessed Nov, 2010).
16. Eskes S, Wiersinga W. Amiodarone and thyroid. Best Pract Res Clin Endocrinol Metab 2009;23(6):735-51.
17. Medsafe. Keep an eye on amiodarone patients. Prescriber Update 2005;26(1):5-6.
18. Lazarus J. Lithium and thyroid. Best Pract Res Clin Endocrinol Metab 2009;23(6):723-33.
19. Pearce E. Diagnosis and management of thyrotoxicosis. BMJ 2006;332(7554):1369.
20. Reid J, Wheeler S. Hyperthyroidism: diagnosis and treatment. Am Fam Physician 2005;72(4):623-30.
21. Ross DS. Pharmacology and toxicity of thionamides. UpToDate 2010. Available from: www.uptodate.com (Accessed Nov, 2010).
22. British National Formulary. London: BMJ Group and RPS Publishing; 2010.
23. Ross DS. Thionamides in the treatment of Graves' disease. UpToDate 2010. Available from: www.uptodate.com (Accessed Nov, 2010).
24. Walter M, Briel M, Christ-Crain M, et al. Effects of antithyroid drugs on radioiodine treatment: systematic review and meta-analysis of randomised controlled trials. BMJ 2007;334(7592):514.
25. Ross DS. Overview of thyroid disease in pregnancy. UpToDate 2010. Available from: www.uptodate.com (Accessed Nov, 2010).
26. Marx H, Amin P, Lazarus J. Hyperthyroidism and pregnancy. BMJ 2008;336(7645):663.