Current choice of treatment for hypo- and hyperthyroidism

Anthony Weetman MD, DSc, FRCP, FMedSci

Thyroid dysfunction is a common disorder and most cases of hypothyroidism are managed in general practice. Our Drug review discusses the currently available drug treatments for hypo- and hyperthyroidism, followed by sources of further information in Resources.

Disorders of thyroid function are extremely common – the prevalence of thyrotoxicosis in women is 1–2 per cent, similar to that of spontaneous and iatrogenic hypothyroidism combined. These disorders are 5–10 times less common in men and can present at all ages.

Biochemical diagnosis of thyroid dysfunction has been greatly facilitated by the introduction of sensitive thyroid-stimulating hormone (TSH) and free-thyroid hormone assays. However, it must be appreciated that these tests do not identify the cause of the problem, which is important in selecting the appropriate treatment.

By far the most common causes of hypothyroidism are autoimmune thyroiditis and thyroid destruction secondary to radioiodine and surgery (see Table 1). Most patients with autoimmune thyroiditis have positive thyroid peroxidase (TPO) antibodies but these can occur in other disorders, including transient destructive thyroiditis appearing most typically in the postpartum period.

Graves’ disease accounts for 80 per cent of thyrotoxicosis and is usually diagnosed clinically by the presence of a diffuse goitre and typical eye signs (see Figure 1), or a personal or family history of associated autoimmune disorders such as pernicious anaemia, vitiligo, type 1 diabetes mellitus, Addison’s disease or coeliac disease. TPO antibodies are present in 80 per cent of Graves’ patients. Recent improvements in assay techniques have now made measurement of TSH receptor antibodies the best specific test for the diagnosis of Graves’ disease, although this test is unnecessary if the patient has clinical signs of eye disease or if it will not alter management, eg if the patient is going to be treated with radioiodine.

The next most common causes are toxic multinodular goitre (see Figure 2) or a solitary toxic adenoma (see Figure 3). The main causes of thyrotoxicosis are listed in Table 2.

This review will focus mainly on the management of autoimmune and iatrogenic hypothyroidism and Graves’ disease. Extensive guidelines for the diagnosis and treatment of both
conditions have recently been published. The management of ophthalmopathy is beyond the scope of this article.

In most cases of primary hypothyroidism, management should be undertaken in primary care. Specialist referral is recommended for all patients with thyrotoxicosis at initial diagnosis and is necessary for the definitive management of those patients needing radiiodine, which is the treatment of choice in most patients with toxic adenoma or multinodular goitre.

Hypothyroidism

In a patient with no remaining thyroid tissue, very high TSH and undetectable free thyroxine (T₄), the normal replacement dose of levothyroxine is 1.6µg per kg per day, which is about 100–150µg per day. This dose can be given from the outset in patients below the age of 60 with no evidence of heart disease. Patients with lesser degrees of thyroid failure are usually commenced on 50–100µg per day. Currently, patients in the UK are entitled to an exemption certificate to allow them to obtain their prescription at no cost.

A frequent mistake is to assess the patient too soon after starting treatment. Dosage changes of 25–50µg levothyroxine per day should be made based on TSH levels measured two to three months after starting treatment. Levothyroxine should be taken at the same time each day, and ideally an hour before breakfast, if the TSH is to be maintained in a narrow, specific range.³

Patients should be warned not to expect a rapid, full improvement in symptoms, and also told that weight loss, a typical concern, will normally only occur when euthyroidism is restored and coupled with attention to exercise and diet.

Mild thyroid failure

In patients with mild thyroid failure, progression of thyroid destruction may occur over months or years, and therefore follow-up at three- to six-month intervals is worthwhile until the tempo of the hypothyroidism is established. Once on a full replacement dose, TSH levels should be checked every one to three years depending on the pattern of previous TSH estimations.

Older patients and heart disease

For older patients or those with heart disease, the usual starting dose of levothyroxine is 25µg daily or on alternate days, with gradual dosage increments of 12.5–25µg per day according to angina symptoms and TSH levels. Occasional patients with severe angina, which cannot be further improved by treatment, may be unable to tolerate the full replacement dose as the hypothyroid state acts as an effective antianginal in its own right. Daily levothyroxine requirements may decrease by 25–50µg in older patients.

TSH suppression

Some patients do not feel completely well despite restoration of normal TSH levels. There is indirect evidence that TSH levels in untreated individuals in the upper half of the reference range are associated with a slightly increased risk of future hypothyroidism, indicating that such TSH levels might already represent a degree of thyroid impairment, and therefore it seems sensible to titrate levothyroxine dosage to bring TSH levels into the lower half of the reference range (below 2mIU per litre) in patients requiring treatment who feel unwell.

TSH suppression, ie TSH levels below the lower limit of the reference range, is associated with an increased risk of atrial fibrillation and lower bone mineral density. The latter occurs particularly in postmenopausal women who have already had an episode of thyrotoxicosis and now have iatrogenic hypothyroidism. Therefore, dosages that suppress the TSH below the lowest part of the reference range should be avoided except when deliberate TSH suppression is desired, for instance in the treatment of thyroid cancer.

It has been suggested that a combination of levothyroxine and liothyronine may improve well-being when compared to levothyroxine alone. However, liothyronine as presently formulated is unsatisfactory for replacement as the half-life is short, leading to widely fluctuating thyroid hormone levels. The evidence to date suggests there is no overall benefit from the addition of current preparations of liothyronine and combined treatment is not recommended for hypothyroid patients.

Table 1. Principal causes of hypothyroidism

<table>
<thead>
<tr>
<th>Primary hypothyroidism</th>
</tr>
</thead>
<tbody>
<tr>
<td>• autoimmune hypothyroidism</td>
</tr>
<tr>
<td>• iatrogenic: treatment of hyperthyroidism or external radiation of the thyroid (e.g. for lymphoma)</td>
</tr>
<tr>
<td>• drugs: excess iodine, amiodarone, lithium, antithyroid drugs, alpha-interferon</td>
</tr>
<tr>
<td>• congenital</td>
</tr>
<tr>
<td>• destructive thyroiditis: silent and postpartum thyroiditis, subacute thyroiditis</td>
</tr>
<tr>
<td>• infiltrative disorders</td>
</tr>
<tr>
<td>• iodine deficiency: common worldwide but absent in UK</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Secondary hypothyroidism</th>
</tr>
</thead>
<tbody>
<tr>
<td>• hypopituitarism</td>
</tr>
<tr>
<td>• hypothalamic disease</td>
</tr>
</tbody>
</table>

Figure 1. Exophthalmos (left eye) due to thyrotoxicosis
to an endocrinologist should be considered for patients who wish to discuss combination treatment further.

Another fairly frequent management problem is the patient who has erratic TSH levels despite a constant dose of levothyroxine, or who appears to require ever-increasing doses. The usual cause is poor adherence, and levothyroxine requirements in excess of 200µg per day suggest that tablets are being missed, although it is important first to rule out malabsorption (especially coeliac disease and conditions in which there is impaired acid secretion, including H. pylori infection and autoimmune gastritis), out-of-date tablets and the effect of certain drugs on levothyroxine absorption or kinetics (see Table 3).

A tactful discussion usually reveals a problem with adherence. This can be improved by telling patients that the long half-life of levothyroxine (seven days) makes it safe to take any tablets that have been missed during a week.

Pregnancy
Hypothyroidism should be diagnosed and treated prior to pregnancy as an elevated maternal TSH in the first trimester of pregnancy is associated with a minor degree of intellectual impairment in the baby. Fertility is also impaired and the risk of miscarriage increased.

A preconception TSH-level check is therefore ideal in women already taking levothyroxine to ensure that replacement is adequate. TSH and free T4 levels should be measured once pregnancy is confirmed and at the beginning of the second and third trimesters.

Levothyroxine requirements increase by about 50 per cent in pregnancy and ideally this increase should be anticipated, the patient being encouraged to increase thyroxine by 25–50µg per day as soon as they are pregnant, with a thyroid function test being done four to six weeks later. A simple way to achieve this is by asking patients to take two extra daily doses of levothyroxine per week once pregnancy is confirmed. The aim should be to maintain free T4 levels in the upper part of the reference range. The normal T4 dose is resumed after delivery, and there are no implications for breast-feeding.

Subclinical hypothyroidism
Because of increased awareness and improved thyroid-function tests, patients are often identified at an early stage of thyroid failure, when the TSH is elevated but free T4 levels are normal.

Although called subclinical hypothyroidism, implying that this state is asymptomatic, up to 25 per cent of such patients may have symptoms of hypothyroidism, including cognitive impairment. Thyroid-function tests should be repeated after three months, and if the TSH is still elevated a trial of levothyroxine may be warranted to determine whether symptoms improve, although guidelines suggest that there may be little or no benefit from this approach (see Resources). On the other hand there is recent evidence that ischaemic heart disease is increased in those who develop subclinical hypothyroidism at a younger age.

Levothyroxine should be given long term to those with a TSH >10mIU per litre. Those with a lower but still elevated TSH level and positive TPO antibodies (see Figure 4) are at high risk of developing overt hypothyroidism (roughly 5 per cent per year) and levothyroxine treatment should be considered, or if there are any symptoms suggestive of hypothyroidism. Annual thyroid-function testing is required in patients with subclinical hypothyroidism who do not start levothyroxine.

Many patients complain of symptoms suggestive of hypothyroidism but have normal TSH and free T4 levels. There has been an unconventional view, particularly held by some private practitioners, that such patients warrant a trial of levothyroxine or desiccated thyroid extract (Armour; not licensed in the UK). However, there is no scientific basis for the suggestion that this group of patients has a state of subtle thyroid dysfunction, and trials of levothyroxine, when suitably controlled, show no benefit. A normal TSH rules out primary hypothyroidism, and if there are suggestive features of hypothyroidism a normal free T4 level will rule out the much rarer type of secondary (pituitary-dependent) hypothyroidism. If both tests are normal, the patient is euthyroid and an alternative cause for the symptoms should be sought.

Thyrotoxicosis
Antithyroid drugs reduce thyroid hormone synthesis and can also reduce the level of autoantibodies that cause Graves’ disease. However, antithyroid drugs only achieve a cure in
40–50 per cent of patients with Graves’ disease. These drugs will control the symptoms of thyrotoxicosis in other forms of hyperthyroidism (see Table 2) but recurrence will ensue as soon as the drug is discontinued. Hospital referral is needed in cases of relapse for definitive treatment with radiiodine or surgery.

Destructive thyroiditis causing transient thyrotoxicosis generally settles spontaneously within four weeks. Antithyroid drugs are ineffective as the thyrotoxicosis is caused by stored hormone release rather than increased synthesis. A beta-blocker, such as propranolol 40–80mg three times daily, may be useful in controlling symptoms; atenolol 50mg a day is an alternative but is less effective as it is more cardioselective. If the patient has severe subacute (or viral) thyroiditis, prednisolone – starting at a dose of 40mg a day – may be indicated, which should be tapered off over two to four weeks depending on the symptomatic response. Many patients have only mild thyroid tenderness that responds well to analgesics such as paracetamol.

Antithyroid drugs
The antithyroid drugs in use are shown in Table 4. Carbimazole is the favoured drug in the UK, propylthiouracil being used when a patient develops side-effects with carbimazole. The inhibitory effect of propylthiouracil on T₄ to T₃ conversion is only of practical benefit in severe thyrotoxicosis (thyrotoxic crisis or ‘storm’) and is offset by the need for more frequent dosing and the small tablet size (50mg) necessitating the use of sometimes 9–12 tablets a day.

Propylthiouracil is more protein bound than methimazole, the active metabolite of carbimazole, and therefore less likely to cross the placenta or to enter milk. However, these benefits are modest and meticulous control of thyroid function is still needed with any thyroid drug used in pregnancy or during lactation.

Administration Antithyroid drugs can be given in two ways: the titration and block-replace regimens. In the first, the patient is given carbimazole 20mg two or three times daily, and the dose is lowered every three to six weeks, based on free T₆ measurements, to achieve a maintenance daily dosage of 5–10mg; 5mg carbimazole is equivalent to 50mg propylthiouracil.

The same starting dosage of carbimazole or propylthiouracil is given in the block-replace regimen, but thereafter levothyroxine is added to maintain euthyroidism. Levothyroxine 100µg is usually needed about four weeks after starting the antithyroid drug when free T₆ levels are near normal range. The dose of levothyroxine is adjusted based on free T₄ levels, but the dose of antithyroid drug remains constant, usually 40mg daily for carbimazole.

Both regimens have their advocates. I prefer the block-replace regimen because the titration regimen must be continued for 18–24 months to achieve a remission rate of 40–50 per cent, whereas the same rate is achieved using the block-replace regimen for six months. Unless there is frequent monitoring of thyroid-hormone levels with the titration regimen, fluctuation of thyroid hormone levels is likely, but this is much less of a problem with the block-replace regimen.

The side-effects of antithyroid drugs are, to some extent, dose related but there is little practical difference between the

Table 2. Main causes of thyrotoxicosis

<table>
<thead>
<tr>
<th>Category</th>
<th>Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Primary hyperthyroidism</td>
<td>Graves’ disease, toxic multinodular goitre, toxic adenoma, ectopic thyroid tissue: struma ovarii, functioning thyroid cancer metastases, congenital: TSH-receptor mutation, iodine excess (Jod-Basedow phenomenon)</td>
</tr>
<tr>
<td>Thyrotoxicosis without hyperthyroidism</td>
<td>silent and postpartum thyroiditis, subacute thyroiditis, excess thyroid hormone ingestion, amiodarone-induced thyrotoxicosis type 2 (secondary to a toxic effect of the drug on thyroid cells)</td>
</tr>
<tr>
<td>Secondary hyperthyroidism</td>
<td>pituitary tumour secreting TSH, excess human chorionic gonadotrophin (hCG): hyperemesis gravidarum, hCG-secreting tumours</td>
</tr>
</tbody>
</table>

Table 3. Possible drug interactions with levothyroxine

| Reduced absorption (give levothyroxine several hours after drug) | colestyramine, colestipol, sucralfate, ferrous sulphate, aluminium hydroxide, charcoal, proton-pump inhibitors |
| Increased metabolism | rifampicin, phenytoin, carbamazepine |
| Interactions | enhances anticoagulant effect of warfarin, acenocoumarol and phenindione, avoid lofepramine, enhances metabolism of propranolol |
needed if agranulocytosis develops and no antithyroid drug should be given thereafter. Lesser degrees of leucopenia are more common, and indeed may be a feature of Graves’ disease itself. It is recommended that the drug is stopped in the granulocyte count is less than \(1.0 \times 10^9\) per litre, whereas continuation with close monitoring is possible if the count is between 1.0 and \(1.5 \times 10^9\) per litre.\(^{11}\)

Propylthiouracil should be avoided in children as they are particularly susceptible to severe hepatotoxicity and death as a complication from taking this drug.\(^{12}\)

**Outcome after antithyroid drugs**

About two-thirds of relapses occur in the year after treatment is stopped, and relapse 10 or more years after antithyroid drug treatment is unusual. Relapse occurs most frequently in those with large goitres and severe hyperthyroidism. Contrary to conventional opinion, younger patients are also less likely to enter remission than patients older than 50.\(^{13}\) A high dietary iodine intake (from seaweed or kelp tablets, for instance) also reduces the chances of remission.

Other tests to predict outcome, including measurement of TSH receptor antibodies, are insufficiently specific and sensitive to be of value in the management of individual cases.

Patients should be assessed about 6 and 12 weeks after treatment is stopped and then every three months to the end of a year. Thereafter, annual follow-up is sufficient, usually via a computerised recall scheme. About 15 per cent of patients successfully treated with antithyroid drugs develop spontaneous hypothyroidism 10–15 years later due to the associated autoimmune thyroiditis.

Symptoms of Graves’ disease usually resolve slowly once treatment begins and patients with severe symptoms secondary to sympathetic overactivity, eg palpitations, sweating or tremor, may benefit from a beta-blocker for the first few weeks of treatment.

For patients who are intolerant of thionamide drugs, there is little medical alternative. Potassium perchlorate or lithium can reduce thyroid hormone synthesis but have serious side-effects and should only be used on advice from an endocrinologist. Potassium perchlorate, however, is sometimes very useful for hyperthyroidism caused by excessive iodine intake (the Jod-Basedow phenomenon), including one type of amiodarone-induced thyrotoxicosis,\(^{14}\) as antithyroid drugs are relatively ineffective in this setting.

Atrial fibrillation secondary to hyperthyroidism reverts to sinus rhythm in about half of patients when rendered euthyroid, and warfarin should always be considered in this case. Control of the heart rate in a thyrotoxic patient with atrial fibrillation can be achieved with digoxin, but often at higher doses than normal.

**Choice of medical versus other treatments**

At diagnosis, a careful discussion with the patient is necessary to detail the likely outcome after antithyroid drugs and to offer the patient the choice of alternative treatment with radioiodine or surgery.

Most patients with Graves’ disease elect to try a course of antithyroid drugs initially as there is a low rate of subsequent hypothyroidism. Radioiodine and, less commonly, surgery are then used for relapses. UK guidelines for the use of radioiodine in benign thyroid disorders have been published.\(^{15}\)

Some UK centres, however, recommend radioiodine as initial treatment in all patients, in line with North American practice, in the belief that definitive treatment is a worthwhile goal from the outset. This approach is generally acceptable for the older patient with cardiovascular disease.

About 90 per cent of Graves’ patients are successfully treated with a single dose of radiiodine, and most of the rest are rendered euthyroid by a second dose usually given six months after the first. One drawback with radiiodine is hypothyroidism, which occurs in 10–20 per cent of patients within a year and in 3–5 per cent per year thereafter; another is the need for...
Some centres prefer to give a high ‘ablative’ dose of radioiodine. There is a much higher incidence of post-treatment hypothyroidism but this method has the advantages of a more predictable outcome and almost no need for retreatment.

Pregnancy and breast-feeding are absolute contraindications and ophthalmopathy may appear or worsen after radioiodine, especially in smokers. Caution is needed in the latter group and prophylactic prednisolone for three months after radioiodine should be considered.

Surgery remains a useful option, provided there is appropriate surgical expertise. Most surgeons aim to leave as small a remnant as possible (near-total thyroidectomy) to reduce the chance of recurrence. This is especially true for patients whose Graves’ disease has relapsed and who have an unallayable fear of radioiodine, who cannot avoid close contact with their children after radioiodine, or who want rapid removal of a goitre for cosmetic reasons. It is also indicated if there is a coincidental thyroid nodule whose nature is uncertain after fine-needle aspiration biopsy.

**Graves’ disease in pregnancy**

Meticulous control of Graves’ disease in pregnancy is necessary to maintain normal fetal thyroid-hormone levels and, ideally, women contemplating pregnancy should be euthyroid before conception. In this regard, there are no sustained teratogenic risks from radioiodine, provided more than six months have elapsed since treatment. When a woman does become pregnant and has active Graves’ disease, the block-replace regimen is contraindicated as the disproportionate transfer of antithyroid drug may cause fetal hypothyroidism.

Carbimazole treatment in pregnancy has been associated with fetal aplasia cutis and choanal atresia. As propylthiouracil has never been associated with these abnormalities, this drug is preferable during the first trimester in women who wish to become or who are pregnant.

Free T4 levels need monitoring at least every four weeks during pregnancy, and the lowest dose of antithyroid drug that maintains maternal free T4 levels in the upper part of the reference range should be given.

The autoimmune process tends to ameliorate in the later stages of pregnancy, necessitating a reduction in dose, and the antithyroid drug can usually be stopped at the beginning of the third trimester. Such spontaneous remission lasts into the postpartum period, but relapse is common three to six months after delivery and should be anticipated, with arrangements for regular thyroid-function tests at this time.

**Graves’ disease in neonates and children**

About 1 per cent of pregnancies in women with Graves’ disease are associated with the development of fetal and neonatal thyrotoxicosis due to the transplacental passage of TSH receptor-stimulating antibodies. This complication can be predicted by identifying a high maternal level of these antibodies during the last trimester, and by fetal tachycardia and failure to thrive. The fetus can be treated by giving the mother antithyroid drugs, but this obviously requires close monitoring. The neonate usually requires an antithyroid drug for one to three months until the maternal antibodies disappear.

Breast-feeding is safe with antithyroid drugs, but the lowest dose should be given and thyroid function should be checked in the baby if the mother is receiving 20mg or more of carbimazole daily.

Graves’ disease is rare in children and uncommon in adolescents. Theoretical concerns over the safety of radioiodine have in the past made many endocrinologists cautious about using radioiodine in these groups, and in general such patients are usually treated with long-term antithyroid drugs to allow undisturbed schooling. Surgery or radioiodine when older can then be planned to fit in with circumstances, although it has been argued that radioiodine should be considered more frequently as first-line treatment in children with Graves’ disease.

---

**Table 4. Properties of antithyroid drugs**

<table>
<thead>
<tr>
<th></th>
<th>Carbimazole*</th>
<th>Propylthiouracil</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Serum half-life</strong></td>
<td>6–8 hours</td>
<td>1–2 hours</td>
</tr>
<tr>
<td><strong>Intrathyroidal turnover</strong></td>
<td>slow</td>
<td>moderate</td>
</tr>
<tr>
<td><strong>Protein binding</strong></td>
<td>negligible</td>
<td>yes</td>
</tr>
<tr>
<td><strong>Influence of disease</strong></td>
<td>nil</td>
<td>nil</td>
</tr>
<tr>
<td><strong>Renal</strong></td>
<td>nil</td>
<td>nil</td>
</tr>
<tr>
<td><strong>Hepatic</strong></td>
<td>prolonged half-life</td>
<td>nil</td>
</tr>
<tr>
<td><strong>Excretion</strong></td>
<td>renal</td>
<td>renal</td>
</tr>
<tr>
<td><strong>Inhibitor of T4 to T3 deiodination</strong></td>
<td>no</td>
<td>yes</td>
</tr>
</tbody>
</table>

*rapidly metabolised to methimazole

---

**Table 5. Side-effects of antithyroid drugs**

**Minor (2–5% of patients)**
- pruritus
- urticarial or maculopapular rash
- arthralgia
- fever
- gastrointestinal upset
- altered taste

**Major (less than 0.2% of patients)**
- agranulocytosis
- aplastic anaemia
- thrombocytopenia
- hepatitis (propylthiouracil)
- cholestatic jaundice (carbimazole)
- SLE-like syndrome

---

**Drugs Review**

**Thyroid disorders**

---

Prescriber July/August 2013

prescriber.co.uk
*KEY POINTS*

- The normal replacement dose of levothyroxine in complete hypothyroidism is 1.6µg per kg per day.
- An elevated or fluctuating TSH level in patients taking 200µg or more of levothyroxine a day usually indicates poor adherence; interference by drugs and malabsorption must be ruled out.
- Up to a quarter of patients with subclinical hypothyroidism are asymptomatic and may benefit from levothyroxine; treatment is recommended in all patients whose TSH is >10mU per litre.
- Levothyroxine treatment is not indicated in those whose symptoms suggest hypothyroidism but whose thyroid function tests (TSH and free T₄) are normal.
- Patients with hyperthyroidism require referral to an endocrinologist to establish the cause of the disease and determine optimal treatment.
- Graves’ disease is usually treated initially with antithyroid drugs; relapse is usually treated with radiiodine or surgery depending largely on patient preference.
- Pregnancy in a patient with Graves’ disease or hypothyroidism requires meticulous control of thyroid function; ideally patients should ensure that they are euthyroid before becoming pregnant.

**Conclusion**

Disorders of thyroid function are common. Hypothyroidism is readily treated with levothyroxine in dosages that normalise the TSH level. Patients with thyrotoxicosis, however, require an accurate diagnosis to determine the optimum therapy, and for Graves’ disease the best treatment initially is usually a course of antithyroid drugs. Relapse occurs in about 40–50 per cent of patients and can be treated definitively with radiiodine or surgery.

This drug review is an update of the review published in November 2010.

**References**


**Declaration of interests**

Professor Weetman has received honoraria from the Novo Nordisk Foundation, Springer and Elsevier.

*Anthony Weetman is professor of medicine at the University of Sheffield, and honorary consultant endocrinologist at the Sheffield Teaching Hospitals Foundation Trust*

**Resources**

**Guidelines**


**Useful websites**

www.allthyroid.org. The Thyroid Foundation of America provides up-to-date information for patients.


www.thyroidmanager.org. Thyroid disease manager (USA). This is aimed at helping clinicians care for their patients and has extensive reviews on all aspects of thyroid disease.

www.endocrine.org. The Endocrine Society homepage (USA) provides research and education.

**Organisations**

The British Thyroid Association is an organisation of endocrinologists with a special interest in thyroid disease. Website: wwwbritish-thyroid-association.org.

The Society for Endocrinology is a specialist society for endocrinologists worldwide. Tel: 01454 642200, website: www.endocrinology.org.

**Patient information**

The British Thyroid Foundation. A patient self-help group that produces information leaflets and organises local meetings. Tel: 01423 709707, website: www.btf-thyroid.org.
CPD: Management of thyroid disorders

Answer these questions online at Prescriber.co.uk and receive a certificate of completion for your CPD portfolio. Utilise the Learning into Practice form to record how your learning has contributed to your professional development.

For each section, one of the statements is false – which is it?

1a. Graves’ disease accounts for 50 per cent of cases of thyrotoxicosis.
1b. Measurement of TSH receptor antibodies is unnecessary if a patient with suspected Graves’ disease has clinical signs of eye disease.
1c. In a patient with no remaining thyroid tissue, very high TSH and undetectable free thyroxine, the normal replacement dose of levothyroxine is about 100–150µg per day.
1d. In a patient with hypothyroidism, dose changes of 25–50µg levothyroxine per day should be made based on TSH levels measured two to three months after starting treatment.

2a. TSH levels should be checked every one to three years in patients taking a full replacement dose of levothyroxine for mild thyroid failure.
2b. Daily levothyroxine requirements may decrease by 25–50µg in older patients.
2c. A woman with hypothyroidism planning to become pregnant should be encouraged to increase the dose of thyroxine by 25–50µg per day as soon as she is pregnant.
2d. Women taking levothyroxine should not breastfeed.

3a. Up to 25 per cent of patients with ‘subclinical’ hypothyroidism may have symptoms of hypothyroidism.
3b. A levothyroxine requirement in excess of 200µg per day may be due to malabsorption or drug interactions.
3c. Combined treatment with levothyroxine and liothyronine is not recommended for hypothyroid patients.
3d. There is good evidence to support a trial of levothyroxine in patients who complain of symptoms suggestive of hypothyroidism but have normal TSH and free T₄ levels.

4a. Antithyroid drugs achieve a cure in 40–50 per cent of patients with Graves’ disease.
4b. Propranolol is more effective than atenolol in controlling symptoms due to destructive thyroiditis.
4c. Propylthiouracil is more likely than carbimazole to cross the placenta.
4d. Mild thyroid tenderness due to severe subacute or viral thyroiditis responds well to paracetamol.

5a. In the block-replace regimen, antithyroid treatment is initiated with carbimazole 20mg per day plus levothyroxine 100µg per day.
5b. Adverse effects are most common in the first three months with both the titration and block-replace regimens.
5c. Patients taking antithyroid drugs should receive a written warning about symptoms suggestive of agranulocytosis.
5d. About 15 per cent of patients successfully treated with antithyroid drugs develop spontaneous hypothyroidism 10–15 years later.

6a. About 90 per cent of Graves’ patients are successfully treated with a single dose of radioiodine.
6b. The block-replace regimen is the treatment of choice for a woman with active Graves’ disease who becomes pregnant.
6c. The autoimmune process tends to ameliorate in the later stages of pregnancy in a woman with Graves’ disease.
6d. A neonate born to a woman with active Graves’ disease usually requires an antithyroid drug for one to three months.

Keeping up to date with the latest thinking in prescribing and therapeutics can be a challenge. However, it is a requirement for all health professionals to maintain their competency through continued professional development.

Learning Central provides automatically marked questions linked to articles that have been published in the journal.

Visit http://bit.ly/prescriberCPD to see the latest online modules.