Assessment and treatment of patients with hypothyroidism

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Assessment and treatment of patients with underactive thyroid.

Maggie Carson BN/RGN, MSc, Certificate in Adult Endocrine Nursing

Aim

The aim of this article is to assist nurses in their understanding of the management of patients with an underactive thyroid gland or hypothyroidism. Hypothyroidism is one of the most common endocrine conditions. It occurs when the thyroid gland does not produce enough of the hormone thyroxine (T4) causing many of the body's functions to slow down.

Intended learning outcomes:

After reading this article you should be able to:

1. Describe the structure and function of the thyroid gland and be able to give an example of a negative feedback mechanism.
2. Identify the causes of hypothyroidism.
3. Describe the main signs and symptoms associated with hypothyroidism.
4. Describe how hypothyroidism is diagnosed and treated.
5. Identify the need to provide patients with ongoing support and encouragement to adhere to treatment and to attend for annual check-ups.

Introduction

The thyroid gland secretes two hormones, triiodothyronine (T3) and thyroxine (T4). These hormones are iodinated molecules. They have various effects on metabolism and protein synthesis and, together with human growth hormone and insulin, are necessary

for growth and development in children. Consequently, any under activity of the gland can result in widespread clinical manifestations. Hypothyroidism is relatively common, affecting approximately 1 in 50 females and 1 in 300 males. Patients on replacement therapy require long term management and follow-up to monitor their condition by an endocrinologist, their GP or both.

Anatomy and physiology

The thyroid is a small butterfly shaped gland located in the neck that sits in front of the larynx and is attached to the second and third tracheal cartilages. It consists of two lobes connected by an isthmus. Structures that lie in close proximity include the oesophagus, trachea, the parathyroid glands, the recurrent laryngeal nerves and the carotid arteries. Enlargement of the thyroid (goitre) may compress the trachea and oesophagus. Surgery of the thyroid may damage the parathyroid glands and the recurrent laryngeal nerves.

The thyroid gland is highly vascular, receiving a rich blood supply (80-120mls of blood per minute) from the superior thyroid arteries, which branch from the external carotid arteries, and from the inferior thyroid arteries, which branch from the subclavian arteries. Venous return is to the internal jugular veins via the thyroid veins. (See Figure 1a – Tortora and Derrickson, 2006)

The thyroid is one of the largest endocrine glands in the body weighing about 30g (1 oz) and is the only endocrine gland that stores its own hormones in large quantities with enough thyroid hormones being stored for about 100 days supply (Tortora and Derrickson, 2006). Each lobe is composed of cuboidal epithelium which forms a number of microscopic sac-like spheres called follicles. Each follicle is surrounded by a basement membrane. These follicles produce and store a thick sticky protein called

colloid. Between the follicles are parafollicular cells ("C" cells), which secrete the hormone calcitonin; this together with parathormone from the parathyroid glands is involved in the metabolism of calcium (see Figure 1b – Tortora and Derrickson, 2006). The thyroid hormones, thyroxine or tetraiodothyronine (T4) so called because it contains four atoms of iodine and 15% of circulating tri-iodothyronine (T3) which contain three atoms of iodine are synthesized and secreted by the follicular cells as large precursor modules called thyroglobulin which is the major constituent of colloid. This synthesis of thyroid hormones is dependent on the availability of iodine in the diet, e.g. from table salt, seafood, milk and vegetables grown in iodine-rich soils. Dietary iodine is absorbed from the small intestine, changed to iodide and transported in the blood to the thyroid gland, where it is taken up by the thyroid cells. This process is called iodine trapping. More T4 than T3 is produced, but T4 is converted in some tissues, mainly the liver and skeletal muscle, to the more biologically active T3 (85% of T3 is produced in this way). Over 99% of all thyroid hormones are bound to plasma proteins during transport in the blood including thyroid-binding globulin (TBG). Only the free unbound hormone is available for use by the tissues entering the cells and regulating the pituitary feedback mechanism. If the levels of T4 and T3 in the blood fall, the hypothalamus releases thyrotrophin-releasing hormone (TRH), a tripeptide. The secretion of TRH is also stimulated by exercise, stress, malnutrition, low plasma glucose and sleep. As the levels of TRH rise, the anterior pituitary gland is stimulated and secretes thyroid stimulating hormone (TSH), a polypeptide, which in turn stimulates the thyroid gland to produce and release more T4 and T3. This is an example of negative feedback (see Figure 2 – Ross and Wilson).

T4 and T3 secretion begins at the end of the twelfth week of life in utero. It remains fairly constant throughout life increasing only during puberty and in women during their reproductive years especially when pregnant.

The principal effect of thyroid hormones is to influence the metabolism of cells and, therefore, what is known as the basal metabolic rate (BMR). Low levels of hormone are associated with a low body temperature (patients will complain of feeling cold), slow heart rate and weight gain whereas patients with high concentrations of thyroid hormones experience a rapid heart rate, heat intolerance and weight loss despite an increased appetite with high calorie intake.

Hyposecretion of the thyroid hormones

The cause of underactivity of the thyroid gland may be primary, resulting from disease of the thyroid, or secondary ("central") due to pituitary or hypothalamic failure (see Table 1).

Table 1: Causes of hypothyroidism

Primary	**Secondary**
Spontaneous atrophic (autoimmune destruction of the gland)	Pituitary failure
Goitrous (Hashimoto's thyroiditis, drug induced, iodine deficiency, dyshormonogenesis)	Hypothalamic failure
Post ablative (following radioactive iodine)	Post pituitary surgery
Post thyroid surgery	

Transient	
Sub-clinical	
Congenital	

Aetiology

Primary hypothyroidism accounts for 95% of all cases of hypothyroidism in adults (Walsh 1997). It is six times more common in women than in men (Haslett et al 2002). It affects approx 1% of the adult population and is most common in elderly females.

Of these, primary hypothyroidism as a result of autoimmune disease is the commonest cause of thyroid underactivity. It may be associated with other autoimmune diseases such as pernicious anaemia, vitiligo, Addison's disease, hypoparathyroidism and insulin dependent diabetes mellitus (Lindsey and Toft 1997). Hypothyroidism may also be iatrogenic, i.e. caused by previous treatment for thyrotoxicosis by means of surgery or radioactive iodine.

Iodine deficiency is another cause of hypothyroidism ("endemic hypothyroidism") and is due to insufficient dietary intake of iodine which in turn is usually due to low levels of iodine in the soil in the locality. This leads to reduced thyroid hormone production. Goitre is a frequent feature of this condition. This is due to the excess secretion of TSH, a proliferation of thyroid gland cells and enlargement of the gland. Endemic hypothyroidism is rare in developed countries and where iodide supplementation is practiced but still occurs in areas where iodine levels in the water supply are low (usually inland areas which are far from the sea or in mountainous regions). It is therefore still seen in the following areas; parts of central Africa, central Asia and central and Eastern Europe as well as in the Andes, the Himalayas and the Alpine areas of central Europe (Delange and Dunn 2005). Interestingly, it is now also reappearing in developed countries such as New Zealand where the population has heeded public

health advice about the dangers of too much salt in the diet and reduced their intake (Mann and Aitken, 2003).

Some medication may also induce hypothyroidism. For example, Lithium Carbonate (used to manage bipolar disorders) can result in goitrous hypothyroidism (Haslett et al 2002), and Amioderone, an anti-arrythmic agent, may cause either transient or permanent hypothyroidism (Laurberg et al 2005).

Congenital hypothyroidism occurs in approximately 1 in 3,500 live births and usually results from congenital absence of the thyroid gland; it can also be caused by certain genetic enzyme defects (dyshormonogenesis). If congenital hypothyroidism is not detected and treated early, the child will not develop fully, either mentally or physically. Neonatal screening for congenital hypothyroidism is performed in most developed countries at 5-7 days.

Pathophysiology

In primary hypothyroidism insufficient thyroxine is being produced by the thyroid gland. Serum T4 is low and levels of TSH are high. The onset is slow and insidious and, because the affected individual is often an older person, it may be accepted as a normal part of ageing and it may therefore be some time before a medical opinion is sought. Symptoms may include lethargy, depression, which can be severe, and an inability to 'think quickly'. The patient may report sensitivity to cold, weight gain and a general slowing down of body functions. Peristalsis becomes markedly slower resulting in constipation which may be chronic and sometimes leads to faecal impaction or ileus (Greenspan, 2004).

As a result of infiltration of the dermis, with mucopolysaccharide, non pitting oedema or myxoedema occurs which is most marked in the skin of the hands, feet and eyelids

(Haslett et al, 2002). The face may be puffy in appearance and is often either pale, due to a combination of vasoconstriction and anaemia, or may be yellowish in colour due to carotinaemia resulting from the reduced conversion of carotene to vitamin A and the consequent higher levels of carotene in the blood (Greenspan, 2004). The hair may be sparse, coarse and brittle and often the outer portion of the eyebrows disappears (See Figure 3).

Renal function is also affected; there is a decrease in the glomerular filtration rate and an impaired ability to excrete a water load.

In women, hypothyroidism impairs the conversion of oestrogen precursors to oestrogens affecting Luteinising Hormone (LH) and Follicle Stimulating Hormone (FSH) production which in turn can lead to anovulatory cycles (menstrual cycles which vary in length and which have no ovulatory or leuteal phase). The absence of ovulation results in infertility. Hypothyroidism can also cause severe menorrhagia (abnormally heavy and prolonged regular menstrual bleeding) which may explain why many patients are also anaemic though this may also be due to impaired haemoglobin synthesis or impaired iron absorption in the gut (Greenspan, 2004).

Medical Management

Physical examination

An assessment should first be made of the patient's general appearance. Changes that may indicate hypothyroidism include paleness, facial puffiness and a lack of facial expression. Their skin and hair may appear dry and brittle and they may report they have noticed a change in texture. Pulse and respiratory rate should be measured as in hypothyroidism these tend to slow down. Blood pressure, however may be raised. In

patients with severe and prolonged hypothyroidism an ECG should be performed. This may reveal sinus bradycardia with low voltage complexes and ST-T wave abnormalities (Haslett et al 2002). A lipid profile should also be done as cholesterol levels may be raised.

Diagnosis

Thyroid function tests (TFTs) are used to diagnose both an underactive and overactive thyroid gland before treatment is initiated. Diagnosis of hypothyroidism is confirmed by low plasma levels of T4 (usually < 5 pmol/L) and raised TSH levels (usually >20 mU/L).

Approximately 10 million TFTs are carried out in the UK each year at an estimated cost of £30 million (BTF News 2006). This simple blood test measures levels of circulating TSH and free T4 (see Table 2). T3 is not usually measured as it is not reliable enough to distinguish between euthyroid (normal thyroid function) and hypothyroid patients (Haslett et al, 2002).

Table 2: Thyroid Function Tests

Hormone	**Reference range**	**Primary hypothyroidism**	**Secondary hypothyroidism**	**Sub-clinical hypothyroidism**
T4		< 5 pmol/L i.e. low	Low	Normal (usually lower end of normal reference range)
Free T4 (measures unbound portion of T4 that is free	10-27 pmol/L			

to enter cells)				
T3		Low*	Low or normal*	Normal *
TSH		>20 mU/L i.e. high	Undetectable, normal or mildly elevated (5-20mU/L)	Mildly elevated (5-20 mU/L)

* T3 is not a sensitive indicator of hypothyroidism and should therefore not be measured.

Nursing Assessment

Assessment begins by taking a comprehensive history from the patient. The commonest symptoms of hypothyroidism are tiredness and lethargy although there may be no symptoms or signs present. Symptoms usually develop gradually and may be vague and general in nature slowly worsening over months or years as the levels of thyroxine in the body fall.

Knowledge of the clinical signs and symptoms of hypothyroidism (see Table 3) enable the nurse to formulate specific questions to identify specific health problems the patient may not have considered relevant and may fail to mention but which taken together help to confirm the diagnosis. The patient should be asked about changes in their physical appearance, their activity and energy levels, sleep patterns, sensitivity to cold, muscle tone, body weight, loss of appetite, hair, skin tone, menstrual cycle and thought processes. Questions specific to their usual daily routine and how organized they are may provide the nurse with clues as to subtle changes and how the patient is compensating. With questioning the patient may, for example, recognize that they have

become more forgetful but identify that they have compensated for this by writing themselves lists of things to do.

Condition specific questionnaires have been designed and validated for use with patients with hypothyroidism: the Underactive Thyroid-Dependent Quality of Life Questionnaire (ThyDQoL) and the Underactive Thyroid Treatment Satisfaction Questionnaire (ThyTSQ). The ThyDQoL has eighteen domains covering quality of life

Table 3: Signs and symptoms of hypothyroidism (adapted from Kumer and Clark 1998 and Haslett et al 2002)

Cardiac/respiratory	Bradycardia
	Hypotension
	Angina
	Heart failure
	Pericardial and pleural effusions
	Coma
Dematological	Dry, course, flaky skin
	Dry, brittle, unmanageable hair
	Loss of eyebrows (especially outer third)
	Myxoedema
	Puffy eyes
Gastrointestinal	Constipation
	Anorexia
General	**Tiredness**
	Weight gain
	Fluid retention
	Cold intolerance/cold peripheries
	Change in appearance
	Goitre
	Hoarse, deep voice
Haematological	Anaemia
Neuromuscular	Arthralgia
	Myalgia
	Carpal tunnel syndrome
	Deafness
Psychological	**Lethargy**
	Depression
	Poor memory
	Confusion
	Psychosis/dementia
Reproductive	Poor libido
	Impotence
	Menorrhagia or oligomenorrhoea in

	women
	Galactorrhoea
	Infertility

measures such as energy levels, physical capabilities, motivation, physical appearance and weight together with other aspects of life affected by hypothyroidism (McMillan et al 2004). It is an individualized measure of the perceived impact of hypothyroidism on the patients' quality of life and is a useful tool to use when assessing a new patient.

It is important to remember that many symptoms are relatively non-specific and can be seen in patients without thyroid disease. With the advent of widely available and accurate thyroid function testing, relatively minor disturbances of thyroid function are frequently detected and are not always the cause of the patient's symptoms.

Management

Current thyroid hormone replacement therapy as a treatment for hypothyroidism was established by beginning of the 1970's (O'Reilly 2000). Clinical guidelines recommend replacement therapy to relieve symptoms, restore euthyroid state and normalize serum TSH levels (Vanderpump et al 1996). Uncomplicated cases of hypothyroidism are treated with replacement doses of Thyroxine (T4) which is available as 25, 50 and 100 mcg tablets. Treatment should commence with a low dose of 25-50 mcg orally and increase by increments of 25-50mcg every 3-4 weeks until serum TSH is normal. A typical dose would be 100-200 mcg/daily. The half life of thyroxine is 6-7 days allowing for small dose adjustments to be made by adding or withdrawing a tablet once or twice weekly so the patient will not notice immediate relief of their symptoms. Thyroxine is only partly absorbed after ingestion and food (especially those rich in calcium or iron), minerals, other drugs and tablet composition can all influence its absorption. Tablets should be taken in the morning on an empty

stomach and as a single daily dose. Symptomatic relief should become apparent 2-4 weeks after starting replacement therapy with improvements to puffy eyes and a reduction in weight often noticed in this time, but it will take 6-8 weeks for a steady state of thyroid hormones to be achieved (Elliot 2000). The patient and their family should therefore be advised that there will only be a gradual improvement in symptoms until this state is reached. Until then, it is important to remind the patient's family that symptoms of lethargy and mental slowness are part of the disease process. Improvement to hair and skin texture can take 3-6 months. Patients should be encouraged to moisturize their skin if dry and flakey.

Patients may need to wear extra clothing and bedding to help with cold intolerance and in some instances, prevent hypothermia. In an attempt to reduce constipation patient should be advised to eat a high fibre diet.

The patient and their family should also be advised that they will need to take thyroxine replacement for life. This entitles the patient to free prescriptions including all other prescribed medication in addition to the thyroxine. The majority of patients in the United Kingdom on thyroid replacement are taking a synthetic preparation containing Levothyroxine Sodium BP, Lactose, Maize Starch, Magnesium Sterate and sometimes Sucrose and/or powdered Acacia. It is possible to get lactose free thyroxine for patients who are lactose intolerant. Some UK clinicians are now prescribing T3 as well as T4 for their patients. These patients may be on synthetic T3 or T3/T4 combinations. It is also possible to get natural thyroxine made from the thyroid glands of pigs but no advantages have been scientifically proven. Side effects are rare and almost entirely related to either under-replacement (in which case the symptoms of hypothyroidism will not have been corrected) or over replacement producing symptoms of hyperthyroidism. Adverse effects of over replacement include anxiety, restlessness,

palpitations, angina, diarrhoea and vomiting, muscle wasting, osteoporosis (especially in post menopausal women) and atrial fibrillation (Clarke and Kabadi 2004). The patient should also be informed about the signs and symptoms of over-replacement although this is uncommon.

Once the level of serum TSH is within the normal reference range, the correct maintenance dose can be individualized on the basis of the patient's symptoms and levels of TSH and T4. The elderly usually require lower doses than young adults and may need their dose of thyroxine reduced.

As thyroxine potentiates the effects of some other commonly prescribed medications (anticoagulants, amitriptyline, insulin, oral hypoglycaemics and tricyclic antidepressants) it is important to establish if the patient is on any of these and ensure the doses are adjusted accordingly. In addition, the metabolism of thyroid hormones is accelerated by some drugs (barbiturates, carbamazipine, phenytoin, rifampicin) and the absorption of thyroxine is reduced by others (colestipol, colestyramine, calcium salts, cimetidine, iron) and so doses of T4 may need to be increased or decreased depending on the patients concomitant medication (BNF 2007).

Nursing priorities and management:

It is usual to treat patients with hypothyroidism on an ongoing basis in the community. The community nursing team should follow up medical treatment and explanations and ensure that patients understand the reasons for the thyroxine replacement therapy and the importance of attending for regular checks to monitor TFTs to ensure that a euthyroid state is both achieved and maintained. Patients are seen in hospital for annual review in out-patient clinics which nowadays are often nurse-led. Questionnaires such as the ThyTSQ mentioned earlier, a seven item measure of satisfaction with current

treatment and a four item measure of satisfaction with past treatment (around the time of diagnosis) on a scale of 0 (very dissatisfied) to 6 (very satisfied) can be used by the nurse to assess patient satisfaction with their care. Studies show that patients are least satisfied with their present understanding of their condition and the information provided at the time of their diagnosis (McMillan et al 2006).

It is important that the patient is encouraged to comply with their treatment and to learn self management in regard to this and symptom control. Despite this, studies suggest that 75% of patients remain overweight and 80% of patients report still feeling depressed even though their thyroid function tests are normal (O'Malley et al 2000).

Specific problem groups and issues:

Hypothyroidism and pre-existing Ischaemic Heart Disease

Patients with a history of ischaemic heart disease should be started on a low dose of Thyroxine, 25mcg, which is gradually titrated every three to four weeks by 25mcg increments to the full replacement dose. Care should be taken to ensure that these patients are not over replaced with T4 because of the risk of exacerbating further cardiac events. Myocardial infarction and sudden death are recognized complications of increasing the metabolic rate in patients with a compromised coronary artery circulation. Symptoms of angina may be exacerbated when thyroxine is first started. Approximately 40% of patients with angina cannot tolerate full replacement therapy despite the concomitant use of B-blockers and vasodilators. These patients may need to undergo successful coronary angioplasty or coronary artery bypass grafting before they are able to tolerate the full replacement dose (Walker and Toft, 2002).

Hypothyroidism and pregnancy

During pregnancy there is an increase in the concentration of TBG resulting in a decrease in serum free T4 in patients with partial or complete thyroid failure. This cannot be compensated for by increased secretion of T3/T4 (Walker and Toft, 2002). If left untreated, pregnant women with primary hypothyroidism are at greater risk of developing pre-eclampsia, anaemia, going into premature labour and of having a stillbirth or a low birth weight baby (Lazarus and Othman 1991). Pregnant women with known hypothyroidism should therefore have their dose of thyroxine increased by 25-50 mcg daily when conception is confirmed and their levels of serum TSH closely monitored each trimester to maintain a normal TSH (Kaplan 1992).

Hypothyroidism and Myxodematous Coma

In severe hypothyroidism, the patient may be admitted in a coma and perhaps be thought to be suffering from hypothermia. This represents a medical emergency in which intensive treatment and care are essential. It is characterised by a depressed level of consciousness, a severe fall in body temperature which may be as low as 25C and sometimes convulsions. The pressure of cerebrospinal fluid (CSF) and its protein content are raised. There is a 50% mortality rate (Strachan and Walker 2006). Treatment must begin immediately the diagnosis is suspected which is often before it can be confirmed biochemically. The patient should be slowly rewarmed using a space blanket. Intravenous T3 is given as a bolus every 8 hours until symptoms improve. The patient can then be switched to oral T4 at a starting dose of 50mcg/day.

Hypothyroidism and ageing

Some degree of hypothyroidism is common in older people with 5-20% of women and 3-8% of men affected (Laurberg et al 2005). Patients in their 70s and 80s who are

already on replacement therapy may need a reduced dose of thyroxine from that which they were taking previously usually as a result of lower body weight and/or concomitant heart disease.

Sub clinical hypothyroidism

This is a term used to describe patients without the symptoms of hypothyroidism who are clinically euthryoid. The diagnosis is entirely based on laboratory findings of an elevated serum TSH levels (>10mU/L) but normal free T4 levels (usually at the lower end of the reference range). It is frequently observed in patients following surgery or Iodine 131 therapy for hyperthyroidism but also occurs spontaneously in over 5% of the female population due to sub-clinical auto-immune thyroid disease. These patients are seen as mildly hypothyroid and there is currently some debate as to whether or not they should be treated. Patients with sub clinical hypothyroidism who also have the anti-thyroid peroxidase (TPO) antibodies have a greater risk of becoming overtly hypothyroid and treatment for these patients is recommended (Lock et al 2004). Only some patients will feel better on replacement therapy (Cooper 2001): prevention may be better than cure but not all these patients will progress to have thyroid failure.

Inappropriate thyroxine replacement

Symptoms of hypothyroidism can be non-specific and include fatigue, low mood and weight gain. A minority of individuals, looking for an explanation for and resolution from these symptoms may press their doctor for a trial of thyroxine replacement either as T4 alone or in combination with T3. This may be initiated without biochemical confirmation of the diagnosis or despite their thyroid function tests being normal.

Pollock et al (2001) have demonstrated that individuals with non-specific symptoms of hypothyroidism and normal T4 and TSH levels do not benefit from replacement therapy.

In some patients , thyroxine replacement may be continued for years for what was in fact a short lived episode of thyroid failure. In these individuals, thyroxine replacement should be stopped and TFTs checked 4-6 weeks later (Walker and Toft, 2002)

Adherence to management regimens in hypothyroidism

Patients are often poor at remembering to take long term medications routinely and poor adherence to thyroid replacement is not uncommon (McMillan et al 2006, Walsh 2002). While the daily tablet regimen is not in itself complicated patients may not appreciate the need to titrate their dose to achieve a euthyroid state and prevent side effects and/or complications associated with hypo- or hyperthyroidism. Other patients may feel despondent if the treatment does not make them feel as they did before the onset of their disease and consequently not adhere to their treatment (Walsh 2002). Flynn et al (2005) demonstrate an association between being placed on a thyroid register and increased compliance with thyroid monitoring and improvement in biochemical control. In the UK the new General Medical Services contract financially rewards primary care practices for regularly measuring thyroid function tests in patients with hypothyroidism (Flynn et al 2005). In some parts of the country disease specific automated registers prompt hypothyroid patients on replacement therapy to attend for blood tests (TSH levels) at 18-monthly intervals (Jung et al 1991).

The findings of a raised serum TSH in a patient known to have been previously well controlled can indicate poor compliance but may also be due to the patient being on another medication which interferes with the absorption of T4 e.g. Ferrous Sulphate

or with its metabolism e.g. anti-malarials. The combination of a raised free T4 and raised serum TSH implies that a patient who is known to be poorly compliant with their treatment has taken excessive quantities of T4 for a few days prior to their appointment (Toft 1999).

If compliance issues are suspected or there is doubt as to whether thyroxine is being absorbed patients can be asked to attend clinic and levels of T4 and TSH checked at hourly intervals for 8 hours following the administration of oral thyroxine given by the nurse who observes the patient taking their tablet (s).

Some patients notice a difference in symptoms if the brand of thyroxine they have been using is changed. This can happen when their dose is altered as not all brands manufacture 25, 50 and 100 mcg tablets. It can also happen because their local pharmacy switches to a different supplier.

Annual review

TFTs should be measured yearly in order to improve compliance and to ensure that the correct replacement dose is still being prescribed. For this reason the patient should be asked if there have been any changes to their concomitant medications. A full blood count and fasting serum lipids should also be measured.

Conclusion

There are currently over 500 000 people taking thyroid hormone therapy (Thyroxine) in the UK (Saravanan et al 2002) In addition, there are 180,000 people with undiagnosed hypothyroidism and a suspected 3.6 million people with undiagnosed sub-clinical hypothyroidism. Consequently it is vital that nurses working in both primary and

secondary care settings are aware of the signs and symptoms of hypothyroidism and know how to monitor and support patients with this disease.

Patient Support Groups

British Thyroid Foundation

www.btf-thyroid.org

British Thyroid Association

www.british-thyroid-association.org

Time Out Activities

Time Out 1

Revise the normal anatomy and physiology of the thyroid gland. Refamiliarise yourself with the principles of negative feedback mechanisms.

Time Out 2

What symptoms might a patient complain about that would prompt a GP to consider a diagnosis of hypothyroidism? Write a list of symptoms.

Time Out 3

Consider the consequences if a patient stopped taking their thyroid replacement therapy a) in the short term and b) long term?

Time Out 4

Using the internet find out what, if any, support is available to patients with hypothyroidism. Is there a national support group? Does it have local groups and is there one in your area? Could you go along to a meeting?

Time Out 5

Why is it important to monitor a patient's thyroid levels while on replacement? What signs and symptoms are associated with excessive doses of replacement Thyroxine?

References

British National Formulary 53rd ed (2007) British Medical Association and Royal Pharmaceutical Society of Great Britain, London

BTF News No 58 – Autumn 2006

Clarke N and Kabadi U M (2004) Optimizing treatment of hypothyroidism. *Treatments in Endocrinology* **3** (4): 217-221

Cooper D S (2001) Subclinical hypothyroidism. *New England Journal of Medicine* **345;** 260-265

Delange F M and Dunn J T (2005) Iodine deficiency. In: Braverman L E, Utiger (eds) Werner and Ingbar's the thyroid: a fundamental and clinical text, 9th edn. Lippincott, Williams and Wilkins, Baltimore.

Elliot B (2000) Diagnosing and treating hypothyroidism. *Nurse Practitioner* **25** (2); 92-105

Flynn R W V, Morris A D, Jung R T, MacDonald T M and Leese G P (2005) Does an automated thyroid register improve the clinical measurement of hypothyroid patients? An observational study. *Clinical Endocrinology* **63** (1); 116-118

Haslett C, Chilvers E R, Boon N A, Colledge N R eds (2002) Davidson's principles and practice of medicine, 19th ed. Churchill Livingstone, Edinburgh

Jung R T, Scott A, Chong P, Browning M and Waugh N (1991)
A new Pick-based computer thyroid register based on the national SAFUR requirements for local usage. *Health Bulletin* **49**; 244-249

Kaplan M M (1992) Monitoring thyroxine treatment during pregnancy. *Thyroid* **2**; 147-152

Kumar P and Clark M (1998) Endocrinology, Diabetes Mellitus and other Disorders of metabolism. In Clinical Medicine: a textbook for medical students and doctors. 4th ed. Balliere Tindall

Ladenson P W (2002) Pyschological wellbeing in patients. *Clin Endocrinol* **57**; 575-576

Laurberg P, Andersen S, Bulow Pedersen I and Carle A (2005) Hypothyroidism in the elderly: pathophysiology, diagnosis and treatment. *Drugs and Aging* **22**(1): 23-38

Lazarus J H and Othman S (1991) Thyroid disease in relation to pregnancy. *Clinical Endocrinology* **34**; 91-98

Lindsay R S and Toft A D (1997) Hypothyroidism. *Lancet* **349**; 413-417

Lock R J, Marden N A, Kemp H J, Thomas P H, Goldie D J and Gompels M M (2004) Subclinical hypothyroidism: a comparison of strategies to achieve adherence to treatment guidelines. *Annals of Clinical Biochemistry* **41** (3); 197-200

Mann J and Aitken E (2003) The re-emergence of iodine deficiency in New Zealand? *Journal of the New Zealand Medical Association* **116** (1170); 351-355

McMillan C V, Bradley C, Woodcock A, Razvi S and Weaver J U (2004) Design of new questionnaires to measure quality of life and treatment satisfaction in hypothyroidism. *Thyroid* **14** (11): 916-925

McMillan C, Bradley C, Razvi S and Weaver J (2006) Psychometric evaluation of a new questionnaire measuring treatment satisfaction in hypothyroidism: the ThyTSQ. *Value in Health* **9** (2): 132-139

O'Malley B, Hickey J and Nevens E (2000) Thyroid disfunction: weight problems and the psyche, the patient's perspective. *Journal of Human Nutrition and Dietetics* **13**(4): 243-248

O'Reilly D StJ (2000) Thyroid function tests – time for a reassessment. *British Medical Journal* **320**: 1332-1334

Pollock MA, Sturrock A, Marshall K, Davidson K M, Kelly C J G, McMahon A D et al (2001) Thyroxine treatment in patients with symptoms of hypothyroidism but thyroid function tests within the reference range; randomized double blind placebo controlled cross over trial. *BMJ* **323**: 891-895

Saravanan P, Chau W F, Roberts N, Vedhara K, Greenwood R and Dayan C M (2002) Psychological well-being in patients on 'adequate' doses of L-thyroxine: results of a large, controlled community-based questionnaire study. *Clinical Endocrinology* **57**; 577-585

Strachan M W J and Walker B R (2006) Endocrine Disease. In: Davidson's Principles and Practice of Medicine 20th ed. Boon N A, Colledge N R and Walker B R (eds). Churchill Livingstone

Toft A D (1999) Thyroid hormone replacement – one hormone or two? *New England Journal of Medicine* **340;** 469-470

Tortora G J and Derrickson B H (2006) The Endocrine System. In: Principles of Anatomy and Physiology 11th ed. Wiley and Sons

Vanderpump M P, Ahlquist J A, Franklin J A and Clayton R N (1996) Consensus statement for good practice and audit measures in the management of hypothyroidism and hyperthyroidism. *BMJ* **313**, 539-544

Walker B R and Toft AD (2002) Endocrine Disease Chapter 16. In: Davidson's Principles and Practice of Medicine 19th ed. Haslett C, Chilvers E R, Boon N A and Colledge N R (eds) Churchill Livingstone, Edinburgh

Walsh M ed (1997) Watson's Clinical Nursing and Related Science, 5th ed. Bailliere Tindall, London

Walsh J P (2002) Dissatisfaction with thyroxine therapy – could the patients be right? *Curr Opin Pharmacology* **2**; 717-722

Waugh A and Grant A (2006) Ross and Wilson Anatomy and Physiology in Health and Illness 10th ed. Churchill Livingstone

List of Tables and Figures

Figures

Tables

Table 3: Signs and Symptoms of Hypothyroidism