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Thyroid Disorders. Part I: Hyperthyroidism

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The significant thyroid disorders that may be found in dental patients are presented in a series of 3 articles. This article (part I) deals with hyperthyroidism, part II with hypothyroidism and thyroiditis, and part III with neoplastic lesions of the thyroid. The signs and symptoms, laboratory tests used to diagnoses hyperthyroidism, and the medical management of patients with hyperthyroidism are presented in this paper. The dental management of patients with hyperthyroidism is discussed in detail. The dentist, by detecting the early signs and symptoms of hyperthyroidism, can refer the patient for medical diagnosis and treatment and avoid potential complications of treating patients with uncontrolled disease. These complications include the rare thyrotoxic crisis (thyroid storm) that may be precipitated by dental treatment, acute infection, or trauma in the patient with uncontrolled hyperthyroidism. Also, the use of epinephrine or other pressor ammines can cause a hypertensive crisis in the patient with uncontrolled hyperthyroidism. Patients will benefit from the early detection and referral by reducing the risks of the medical complications such as hypertension, cardiac arrhythmias, and congestive heart failure.
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The purpose of these articles is to discuss in 3 parts the more significant thyroid disorders that may be found in patients presenting for dental treatment. This article, part I, deals with the anatomy and physiology of the thyroid gland and hyperthyroidism (thyrotoxicosis), part II covers hypothyroidism (myxedema and cretinism) and thyroiditis, and part III describes neoplastic lesions of the thyroid gland. The dentist may detect early signs and symptoms of thyroid disease and refer the patient for medical evaluation and treatment. In some cases, this may be lifesaving, whereas in others the quality of life can be improved and complications of certain thyroid disorders avoided.[1]

THYROID GLAND – LOCATION AND FUNCTION

Location

The thyroid gland, located in the anterior portion of the neck just below and bilateral to the thyroid cartilage, develops from the thyroglossal duct and portions of the ultimobranchial body.[1-3] The thyroid consists of 2 lateral lobes connected by an isthmus. The right lobe is normally larger than the left,[4] and in some individuals a superior portion of glandular tissue, or pyramidal lobe, can be identified. Thyroid tissue may be found anywhere along the path of the thyroglossal duct, from its origin (midline posterior portion of the tongue) to its termination (thyroid gland, in the neck).[1,3] The thyroglossal duct passes through the region of the developing hyoid bone, and remnants of the duct can become enclosed or surrounded by the bone.[4] Ectopic thyroid tissue may secrete thyroid hormones or become cystic or neoplastic.[5] In a few individuals, the only functional thyroid tissue is in these ectopic locations.[2]

The parathyroid glands develop from the third and fourth pharyngeal pouches and become embedded in the thyroid gland.[3] Neural crest cells from the ultimobranchial branchial body give rise to thyroid medullary C cells that produce calcitonin, a calcium-lowering hormone.[2,3] The C cells are found throughout the thyroid gland.[2,3]

Function

The thyroid gland secretes 3 hormones: thyroxine (T_4), triiodothyronine (T_3), and calcitonin.[2] Calcitonin is involved, with parathyroid hormone and vitamin D, in regulating serum calcium and phosphorous levels

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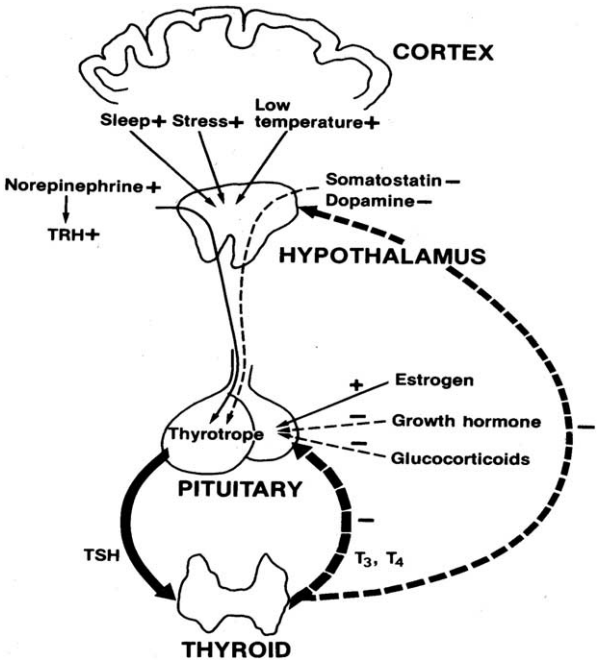

Fig. 1. Diagram showing the hypothalamic-pituitary-thyroid axis involved in the control of thyroid secretion. The secretion of thyroid-stimulating hormone (TSH) is regulated by the interaction of thyroid-releasing hormone (TRH) and an inhibitory factor (somatostatin). Thyroid hormones (T3 and T4) act directly on the pituitary to inhibit TSH secretion. Thyroid hormones also act at the hypothalamic level to stimulate somatostatin release. T4 is converted to T3 in the liver, kidney, and heart and in the pituitary and hypothalamus. T3 is more potent than T4 at all sites. (With permission from: Little JW, Dental management of the medically compromised patient. 6th ed. St Louis: Mosby; 2002. p. 286.)

and skeletal remodeling. T_4 and T_3 are hormones that affect metabolic processes throughout the body and are involved with oxygen use.[2,6]

Blood levels of T_4 and T_3 are controlled through a servofeedback mechanism mediated by the hypothalamic-pituitary-thyroid axis (Fig. 1). Increased or decreased metabolic demand appears to be the main modifier of the system. Drugs, illness, thyroid disease, and pituitary disorders can affect the control of this balance.[2,6-8] Studies also show that age has some effect on the system.

Under normal conditions, thyrotropin-releasing hormone (TRH) is released by the hypothalamus in response to external stimuli (stress, illness, metabolic demand, low levels of T_3, and to a lesser degree, T_4). TRH stimulates the pituitary to release thyroid-stimulating hormone (TSH), which causes the thyroid gland to secrete T_4 and T_3.[2,6] T_4 and T_3 also have a direct influence on the pituitary. High levels turn off the release of TSH, and low levels turn it on.

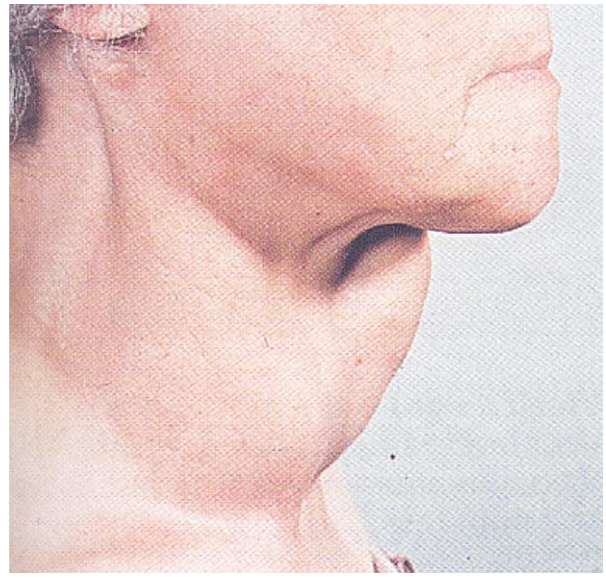

Fig. 2. Multinodular goiter that required surgical treatment because of retrosternal extension with tracheal compression. This patient was euthyroid. (With permission from Forbes CD, Jackson WF. Color atlas and text of clinical medicine. 3rd ed. St Louis: Mosby; 2003. p. 313.)

In the blood, T_4 and T_3 are almost entirely bound to plasma proteins. The binding plasma proteins are thyroxine-binding globulin (TBG), transthyretin, and thyroid-binding albumin (TBA). A small amount of T_3 and T_4 are bound to high-density lipoproteins.[2] The most important thyroid hormone-binding serum protein is TBG. TBG binds about 70% of T_4 and 75% to 80% of T_3.[2] Only 0.02% to 0.03% of free thyroxine (FT_4) and about 0.3% of free tri-iodothyronine (FT_3) are found in plasma.[2,6]

Antibodies to various structures within the thyroid are associated with autoimmune diseases of the thyroid. Graves' disease and Hashimoto's thyroiditis have such an association. Three autoantibodies are most often involved with autoimmune thyroid disease. These are TSH receptor antibodies (TSHRAb), thyroid preoxidase antibodies (TPOAb), and thyroglobulin antibodies (TgAb).[1,2,6,9]

THYROTOXICOSIS (HYPERTHYROIDISM)

Enlargements of the thyroid gland, termed a *goiter,* can be diffuse, nodular (Fig. 2), singular, functional, or nonfunctional.[2,3] Simple goiter accounts for about 75% of all thyroid swellings.[6] Most of these goiters are nonfunctional and thus do not cause hyperthyroidism. The goiter of Graves' disease is associated with hyperthyroidism.[10,11] Worldwide, the most common thyroid disorder is iodine deficiency (diet-related) goiter. This

Table I. Classification of hyperthyroid disorders[2,20,23]

Disorder	*Causes*
Hyperthyroidism (thyrotoxicosis)	Primary thyroid hyperfunction
	Graves' disease
	Toxic multinodular goiter
	Toxic adenoma
	Secondary thyroid hyperfunction
	Pituitary adenoma – TSH secretion
	Inappropriate TSH secretion (pituitary)
	Trophoblastic hCG-secretion
	Without thyroid hyperfunction
	Hormonal leakage
	Thyroid hormone use (factitia)
	Bovine thyroid in ground beef
	Metastatic thyroid cancer
	Iatrogenic (overdosage of thyroid hormone)

TSH, Thyroid-stimulating hormone; *hCG,* human chorionic gonadotropin

type of goiter is called *endemic* if more than 10% of a local population is affected.[12-15]

Subclinical hyperthyroidism is a common, well-defined condition that often progress to overt disease.[16-18] In addition, concerns are evident that the subclinical state may contribute to hyperlipidemia, cardiac dysfunction, and osteoporosis.[2,19,20]

Incidence, Prevalence, and Demographics

Several studies demonstrate the prevalence of thyroid hyperfunction.[9,21] Among patients with hyperthyroidism, 60% to 80% have Graves' disease. Studies in Great Britain have shown about 25 to 30 cases of hyperthyroidism per 10 000 women.[9] The mean age at the time of diagnosis was 48 years. Some 2% of the women had established cases. The incidence of new cases was 3 per 1000 women per year. These studies showed that hyperthyroidism was 10 times more common in women than in men.[9] The incidence of the disease in the United States has been reported to be 1 case per 1000 women.[21,22] It is much lower in men.

Etiology and Pathogenesis

The term *thyrotoxicosis* refers to an excess of T_4 and T_3 in the bloodstream.[22] This excess may be caused by ectopic thyroid tissue, Graves' disease, multinodular goiter, thyroid adenoma, subacute thyroiditis, ingestion of thyroid hormone (thyrotoxicosis factitia), food-containing thyroid hormone, or pituitary disease involving the anterior portion of the gland or may be iatrogenic due to overdosage (Table I).[23]

The signs and symptoms, laboratory tests, treatment, and dental considerations for the patient with Graves' disease are presented to serve as the model for other conditions that can cause hyperthyroidism. Multinodular goiter, ectopic thyroid tissue, and neoplastic causes of hyperthyroidism are rare compared with toxic goiter (Graves' disease).[10,20,23]

The basic cause of Graves' disease is not understood, but an immunoglobulin or family of immunoglobulins directed against the TSH receptor mediates the thyroid stimulation. These include TSHRAb and TSHR-blocking Ab, which inhibit the binding of TSH to its receptors. Graves' disease is considered to be an autoimmune disease.[9,10,23] Patients with Graves' disease often have in their serum high titers of TSHRAb and low titers of TSHR-blocking Ab. The level of TSHRAb in serum does not correlate with the severity of symptoms of the disease. In addition, infants whose mothers have Graves' disease will show a transient period of goiter, ophthalmopathy, and clinical manifestations of Graves' disease. As the disease disappears, the level of serum TSHRAb diminishes.[9,10,23]

A familial tendency has been noted for the transmission of Graves' disease, with an increased incidence of about 20% reported in monozygotic twins compared with a much lower rate in dizygotic twins.[9,23,24] No single gene is known to cause Graves' disease or be necessary for its development.[9] Graves' disease occurs in 3% to 5% of patients with myasthenia gravis and myasthenia gravis occurs in 1% of patients with Graves' disease.[23]

The chief risk factor for Graves' disease is female gender. This may be in part due to the modulation of the autoimmune response by estrogen.[23] This disorder is much more common in women (7:1) and may manifest itself at puberty, pregnancy, or menopause.[23] Graves' disease is the most common cause of hyperthyroidism in children.[25] Emotional stress such as severe fright or separation from loved ones has been reported to be associated with its onset.[23] The disease may occur in a cyclic pattern and then "burn itself out" or continue in an active state.[9]

Clinical presentation

Direct and indirect effects of the excessive thyroid hormones cause the clinical picture in Graves' disease. The most common symptoms are nervousness, fatigue, a rapid heartbeat or palpitations, heat intolerance, and weight loss.[9,10,20,23] These symptoms are present in more than 50% of all patients who have the disease. With increasing age, weight loss and decreased appetite become more common, and irritability and heat intolerance are less common. Atrial fibrillation is rare in patients younger than 50 years old but is found in approximately 20% of older patients.[23]

The patient's skin is warm and moist, the complexion rosy, and the patient may blush readily. Palmar erythema may be present, profuse sweating is common,

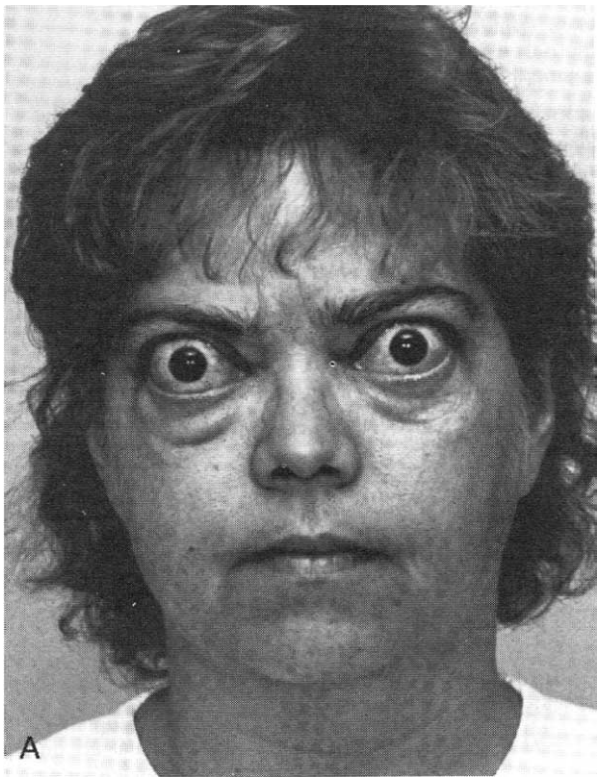

Fig. 3. Classic signs of Graves' orbitopathy showing the thyroid stare, the asymmetry, the proptosis, and the periorbital edema. (With permission from Wilson JD, Foster DW, Kronenberg HM, Larsen PR. Williams textbook of endocrinology. 9th ed. Philadelphia: W. B. Saunders Company; 1998. p. 439.)

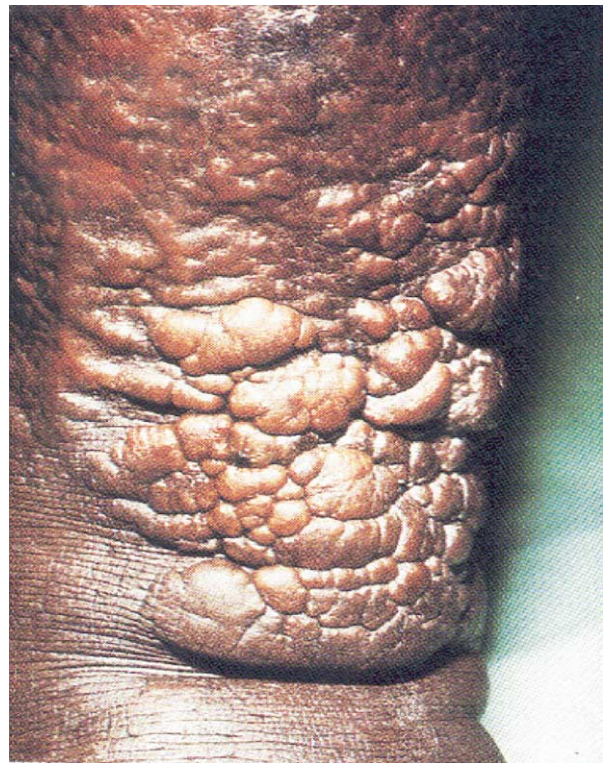

Fig. 4. Chronic pretibial myxedema (dermopathy) in a patient with Graves' disease and orbitopathy. The lesions are firm and nonpitting with a clear edge to feel. (With permission from Wilson JD, Foster DW, Kronenberg HM, Larsen PR. Williams textbook of endocrinology. 9th ed. Philadelphia: W. B. Saunders Company; 1998. p. 437.)

and excessive melanin pigmentation of the skin occurs in many patients, however pigmentation of the oral mucosa has not been reported. In addition, the patient's hair becomes fine and friable, and the nails soften.[9, 10, 23]

Graves' ophthalmopathy, found in approximately 50% of the patients, is characterized by edema and inflammation of the extraocular muscles, and an increase in orbital connective tissue and fat.[9,23] The ophthalmopathy is an organ-specific autoimmune process strongly linked to Graves' hyperthyroidism. Although the hyperthyroidism can be successfully treated, the ophthalmopathy often produces the greatest long-term disability for patients with this disease.[9,23] Fig. 3 demonstrates the signs associated with ophthalmopathy (eyelid retraction, proptosis, periorbital edema, chemosis, and bilateral exophthalamos). The disease may progress to visual loss by exposure keratopathy or compressive optic neuropathy.[9,23]

Most thyrotoxic patients show eye signs not related to the ophthalmopathy of Graves' disease. These signs (stare with widened palpebral fissures, infrequent blinking, lid lag, jerky movements of the lids, and failure to wrinkle the brow on upward glaze) result from sympathetic overstimulation and usually clear when the thyrotoxicosis is corrected.[9,23]

Another complication, found in about 1% to 2% of the patients with Graves' disease, is dermopathy (Fig 4). In focal areas of the skin, the hyaluronic acid and chondroitin sulfate concentrations in the dermis are increased. This may occur from lymphokine activation of fibroblasts. The accumulation causes compression of the dermal lymphatics and nonpitting edema. Early lesions contain a lymphocytic infiltrate. Nodular and plaque formation may occur in chronic lesions. The lesions are most common over the anterolateral aspects of the shin. Patients with dermopathy almost always will have severe ophthalmopathy.[9,10,23]

The increased metabolic activity caused by excessive hormone secretion increases circulatory demands, and an increased stroke volume and heart rate often develop in addition to widened pulse pressure, resulting in the patient complaining of palpitations.[23] Supraventricular

cardiac dysrythmias develop in many patients. Congestive heart failure may occur and often is somewhat resistant to the effects of digitalis.[23] Patients with untreated or incompletely treated thyrotoxicosis are highly sensitive to the actions of epinephrine or other pressor amines, and these agents must not be administered to them; however, once the patient is well managed from a medical standpoint, these agents can be resumed.[23]

Dyspnea not related to the effects of congestive heart failure may occur in some patients. The respiratory effect is caused by reduction in the vital capacity secondary to weakness of the respiratory muscles.[23]

Weight loss even with an increased appetite is a common finding in younger patients. Stools are poorly formed, and the frequency of bowel movements is increased. Anorexia, nausea, and vomiting are rare but, when they occur, may be the forerunners of thyroid storm. Gastric ulcers are rare in patients with thyrotoxicosis. Many of these patients have achlorhydria, and about 3% develop pernicious anemia.[23]

Thyrotoxic patients tend to be nervous and often show a great deal of emotional lability, losing their tempers easily and crying often; severe psychic reactions may occur.[10,22] These patients cannot sit still and are always moving. A tremor of the hands and tongue, along with lightly closed eyelids, is often present; in addition, a generalized muscle weakness may lead to the patient complaining of easy fatigability.[23]

Excessive thyroid hormone results in accelerated bone turnover caused by direct stimulation of bone cells by the hormone.[26] The serum osteocalcin (bone formation marker) and phosphorus levels are increased as is the urinary calcium/creatinine ratio.[26] Thyrotoxic patients have an increased excretion of calcium and phosphorus in their urine and stools, and radiographs demonstrate increased bone loss.[23] Hypercalcemia occurs sometimes, but the serum levels of alkaline phosphatase usually are normal.[23]

The individual red blood cells (RBCs) in patients with thyrotoxicosis are usually normal; however, the RBC mass is enlarged to carry the additional oxygen needed for the increased metabolic activities.[23] In addition to the increase in total numbers of circulating RBCs, the bone marrow reveals an erythroid hyperplasia, and requirements for vitamin B_{12} and folic acid are increased.[23] The white blood cell (WBC) count may be decreased because of a reduction in the number of neutrophils, whereas the absolute number of eosinophils may be increased.[23] Enlargement of the spleen (10% of patients) and lymph nodes occurs in some patients.[23] The platelets and clotting mechanism usually are normal, but thrombocytopenia has been reported.[23]

Diagnosis

Direct tests of thyroid function involve the administration of radioactive iodine.[10,22] Measurement of the thyroid radioactive iodine uptake (RAIU) is the most common of these tests. ^{131}I has been used for this test, but ^{123}I is preferred because it exposes the patient to a lower radiation dose. The RAIU is measured 24 hours after the administration of the isotope**.** The RAIU varies inversely with the plasma iodide concentration and directly with the functional state of the thyroid. In the United States the normal 24-hour RAIU is 10% to 30%. The RAIU discriminates poorly between normal and hypothyroid states. Values above the normal range usually indicate thyroid hyperfunction.[6]

The measurement of the basal serum TSH concentration is useful in the diagnosis of hyperthyroidism and hypothyroidism.[10,22] Very sensitive methods are now available, such as immunoradiometric or chemiluminescent, to measure serum TSH. In cases of hyperthyroidism the TSH level is almost always low or nondetectable**.** Higher levels indicate hypothyroidism, and lower levels signify hyperthyroidism.[6]

Three types of thyroid autoantibodies can be measured for diagnostic uses. The autoantibodies that can be tested for are thyroglobulin (Tg Ab), peroxidase (TPO Ab), and TSH receptor (TSHR Ab).[2]

A thyroid scan is a common test used to localize thyroid nodules and to locate functional ectopic thyroid tissue. ^{123}I or ^{99}Tc is injected, and a scanner localizes areas of radioactive concentration. This technique allows for the identification of nodules 1 cm or larger.[2,6]

Several tests are available that measure the thyroid hormone concentration and binding in blood. Highly specific and sensitive radioimmunoassays are used to measure serum T_4 and T_3 concentrations and rarely to measure reverse tri-iodothyronine (rT_3) concentration.[10,22] Elevated levels usually indicate hyperthyroidism, and lower levels usually indicate hypothyroidism. The free hormone levels usually correlate better with the metabolic state than do total hormone levels. Indirect assays are used to estimate the free T_4 level.[6]

Current practice is to screen patients suspected of being hyperthyroid with the TSH serum level and measure or estimate the free T_4 concentration.[10,22] A low TSH level and a high free T_4 concentration are classic for hyperthyroidism.[10,22] Some patients are hyperthyroid with a low TSH level and normal free T_4 concentration, but they have an elevated free T_3 level. A few patients have a normal or elevated TSH and a high free T_4. These patients either have a TSH-secreting pituitary adenoma or have thyroid hormone–resistance syndrome.[9]

No general agreement exists on whether serum TSH receptor antibodies should be measured in the differen-

tial diagnosis of Graves' disease.[10,22] The newer assays have a high degree of sensitivity—up to 99%. A positive assay may indicate the presence of either TSHRAb or TSHR-blocking Ab. A positive test in a clinically hyperthyroid patient would indicate the presence of TSHRAb.[9]

Ultrasonography is used to detect thyroid lesions. Nodules 1 to 2 mm in size can be identified.[10,22] The technique also is used to distinguish solid from cystic lesions, measure the size of the gland, and guide needles for aspiration of cysts or biopsy of thyroid masses. Computed tomography (CT) and magnetic resonance imaging (MRI) are expensive procedures helpful mainly in the postoperative management of patients with thyroid cancer.[2,10] They are used for the preoperative evaluation of larger lesions of the thyroid, greater than 3 cm, that extend beyond the gland into adjacent tissues.[2,6]

Medical Management and Treatment

Treatment of patients with thyrotoxicosis may involve antithyroid agents that block hormone synthesis, iodides, radioactive iodine, or subtotal thyroidectomy.[20,22,23,27,28] The most common antithyroid agents used are propylthiouracil, carbimazole, and methimazole, all of which inhibit thyroid preoxidase and thus the synthesis of thyroid hormone. Antithyroid agents may cause a mild leukopenia, but drug therapy is not stopped unless the WBC count is more severely depressed.[9,23] In rare cases, agranulocytosis may occur. If sore throat, fever, and/or mouth ulcers develop, most physicians advise the patient to stop the antithyroid medication and have a WBC count performed.[9,20,22,23]

Radioactive iodine is the preferred initial treatment for Graves' disease in North America.[9,23] It is contraindicated in pregnant women and those who are breastfeeding. Radioactive iodine can induce or worsen ophthalmopathy, particularly in smokers. Weetman[9] recommends antithyroid drug treatment for patients younger than 50 years of age with their first episode of Graves' disease and radioactive iodine for those 50 years of age and older. The main side effect of radioactive iodine treatment is hypothyroidism. The incidence of cancer is unchanged or slightly reduced in patients treated with radioactive iodine, but the risk of death from thyroid cancer and possibly other cancers is slightly increased. Patients with severe hyperthyroidism should be treated with an antithyroid drug for 4 to 8 weeks before radioactive iodine therapy is initiated.[9,23] This approach reduces the slight risk of thyrotoxic crisis if radioactive iodine was given initially.[9]

Subtotal thyroidectomy is preferred by some patients with a large goiter and is indicated in patients with a coexistent thyroid nodule whose nature is unclear.[9,23] The patient is first treated with an antithyroid drug until euthyroidism is achieved. Then inorganic iodide is administered for 7 days before surgery.[9,23] In major centers, hyperthyroidism is cured in more than 98% of the cases, with low rates of operative complications. Postoperative hypothyroidism is a complication of the surgical treatment becoming more common as near-total thyroidectomy is approached.[9]

If exophthalmos is present, it follows a course independent of the therapeutic metabolic response to antithyroid treatment modalities and usually is irreversible.[23] Neither antithyroid drug treatment nor surgery affect the natural course of Graves' ophthalmopathy. In contrast, radioiodine therapy, in about 15% of cases, will cause progression of the eye disease.[29] Graves' ophthalmopathy progression after radioiodine therapy can be prevented by treatment with glucocorticoids.[29] The adrenergic component in thyrotoxicosis can be managed by using beta-adrenergic antagonists such as propranolol. Propranolol alleviates adrenergic manifestations such as sweating, tremor, and tachycardia.[9,10,23]

The clinical presentations of thyroid disorders often are subtle in older adults and may be confused with "normal" aging. To avoid delay in diagnosis, some authors recommend routine TSH screening of all patients age 60 and older in the primary care practice. When hyperthyroidism is caused by Graves' disease, symptomatic therapy with a beta-blocker or antithyroid drugs is initiated, followed by definitive thyroid ablation with radioiodine.[2,6,23]

Complications

Complications associated with hyperthyroidism include osteoporosis, atrial fibrillation, hypertension, and congestive heart failure.[22] Patients with thyrotoxicosis who are untreated or incompletely treated may develop thyrotoxic crisis, a serious but fortunately rare complication that may occur at any age and has an abrupt onset.[10,23] Thyrotoxic crisis occurs in less than 1% of the patients hospitalized for thyrotoxicosis.[8] Most patients who develop thyrotoxic crisis have a goiter, wide pulse pressure, eye signs, and long history of thyrotoxicosis.[10,23] Precipitating factors are infections, trauma, surgical emergencies, and operations.[9,10,23] Early symptoms are extreme restlessness, nausea, vomiting, and abdominal pain; fever, profuse sweating, marked tachycardia, cardiac arrhythmias, pulmonary edema, and congestive heart failure soon develop.[9,10,23] The patient appears to be in a stupor, and coma may follow. Severe hypotension develops, and death may occur. These reactions appear to be associated, at least in part, with adrenocortical insufficiency.[10,23]

Immediate treatment for the patient in a thyrotoxic crisis consists of large doses of antithyroid drugs (200

mg of propylthiouracil); potassium iodide; propranolol (to antagonize the adrenergic component); hydrocortisone (100 to 300 mg); dexamethasone (2 mg orally every 6 hours, to inhibit release of hormone form the gland and the peripheral conversion of T_4 to T_3); intravenous (IV) glucose solution; vitamin B complex; wet packs; fans; and ice packs. Cardiopulmonary resuscitation is sometimes needed.[10,23]

Prognosis

The prognosis of hyperthyroidism is good with proper therapy. There will be some failures with initial medical therapy. However, therapy can be repeated or a different treatment can be tried. Patients may develop serious allergic reactions to thioamides requiring an alternative therapy, usually radioiodine.[22] Patients who fail to respond to radioactive iodine within 6 to 9 months need to be retreated with a larger dose.[22]

Dental management

Examination of the thyroid gland should be part of a head and neck examination performed by the dentist. The anterior neck region can be scanned for indications of old surgical scars; the posterior dorsal region of the tongue should be examined for a nodule that could represent lingual thyroid tissue; and the area just superior and lateral to the thyroid cartilage should be palpated for the presence of a pyramidal lobe. Although difficult to detect, the normal thyroid gland can be palpated in many patients.[30,31] It may feel rubbery and may be more easily identified by having the patient swallow during the examination.[1] As the patient swallows, the thyroid rises; lumps in the neck that may be associated with it also rise (move superiorly). Nodules in the midline area of the thyroglossal duct move upward with protrusion of the patient's tongue.[1]

An enlarged thyroid gland caused by hyperplasia (goiter) feels softer than the normal gland. Adenomas and carcinomas involving the gland are firmer on palpation and are usually seen as isolated swellings.[30,31] Patients with Hashimoto's disease or Riedel's thyroiditis have a much firmer gland on palpation than the normal gland.[32] The dentist should be aware of the clinical manifestations of thyrotoxicosis so that undiagnosed or poorly treated disease can be detected and the patient referred for medical evaluation and treatment (Table II). By doing this, dentists may be able to help reduce the morbidity and mortality rates associated with thyrotoxicosis.[1]

Patients with untreated or poorly treated thyrotoxicosis are susceptible to developing an acute medical emergency *thyrotoxic crisis,* which is another important reason for detection and referral. Symptoms include restlessness, fever, tachycardia, pulmonary edema, tremor, sweating, stupor, and finally coma and death if treatment is not provided. If a surgical procedure is performed on these patients, a crisis may then be precipitated. In addition, an acute oral infection could precipitate a crisis. If a crisis occurs, the dentist should be able to recognize what is happening, begin emergency treatment, and seek immediate medical assistance (Table II). The patient can be cooled with cold towels, given an injection of hydrocortisone (100 to 300 mg), and started on an IV infusion of hypertonic glucose (if equipment is available). Vital signs must be monitored, and cardiopulmonary resuscitation initiated if necessary.[1]

Although the role of chronic infection and thyrotoxicosis is unclear, these sources should be treated as in any other patient. Once the patient has been identified and referred for medical management, the treatment of oral foci of infection can be accomplished. Patients with extensive dental caries or periodontal disease, or both, can be treated after medical management of the thyroid problem has been effected.[1]

The use of epinephrine or other pressor amines (in local anesthetics, gingival retraction cords, or to control bleeding) must be avoided in the untreated or poorly treated thyrotoxic patient (Table II). However, the well-managed or euthyroid thyrotoxic patient presents no problem in this regard and may be given normal concentrations of these vasoconstrictors.[1] Once the thyro-

Table II. Dental management of the hyperthyroid patient

Disease status	*Clinical action*
Detection of undiagnosed disease	Symptoms Signs Refer for medical Dx and Rx
Diagnosed disease	Determine original diagnosis and Rx Past treatment Current treatment Lack of signs and symptoms Presence of any complications
Untreated or poorly controlled	Avoid surgical procedures Treat any acute infection Avoid use of epinephrine or pressor amines
Well controlled	Treat acute infection (avoid if possible) Treat chronic infection Implementation of normal procedures and management
Medical crisis (rare)	Recognition and initial management of thyrotoxic crisis Seek medical aid Wet packs, ice packs Hydrocortisone (100 to 300 mg) IV glucose solution Cardiopulmonary resuscitation

toxic patient is under good medical management, the dental treatment plan is unaffected. If acute oral infection occurs, however, consultation with the patient's physician is recommended as part of the management program.[1]

Oral findings

Osteoporosis may be found involving the alveolar bone. Dental caries and periodontal disease appear more rapidly in these patients. The teeth and jaws develop more rapidly, and premature loss of the deciduous teeth with early eruption of the permanent teeth is common. Euthyroid infants of hyperthyroid mothers have been reported with erupted teeth at birth. A few patients with thyrotoxicosis have been found to have a lingual thyroid**,** consisting of thyroid tissue below the area of the foramen cecum.[1] When a lingual tumor is found, the presence of a normal thyroid gland should be established before the mass is surgically removed. This usually is done by radioactive iodine scanning.[2,23]

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