

**Graduation Document**

**Thyroid gland problems and their relation to oral and dental health**

**This graduation project is submitted to the dental Department**

**in partial fulfillment of the requirements for the degree**

**of Bachelor of dentistry**

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**SUPERVISOR CERTIFICATION**

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## **Abstract**

Thyroid disease in a patient who presents for dental treatment is a cause for concern on several fronts. Undiagnosed or poorly controlled thyroid disorders can be expected to compromise outcomes with otherwise perfectly appropriate dental management plans. Detection of early signs and symptoms of such disorders during the dentist’s head and neck evaluation can lead to referral of the patient for medical evaluation and treatment. In some instances, such intervention may be lifesaving, and in others, quality of life can be improved and complications of certain thyroid disorders avoided, particularly in the context of delivery of dental care. The patient with a thyroid dysfunction, as well as the patient taking medications for it, requires proper risk management before considering dental treatment by the dentist. Thus, communication of dentist with endocrinologist must be bidirectional, to maintain patient's oral and thyroid health.

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**Introduction**

The thyroid gland is the major regulator of metabolism and affects all of the bodily functions. Thyroid dysfunction is the second most common glandular disorder of the endocrine system which may rear its head in any system in the body including the mouth. The thyroid gland and its hormones play an important role in the regulation of growth, development and metabolic functions of the body. Thyroid diseases include a group of conditions that can affect the delivery of dental care.

**Thyroid gland relation with oral cavity**

The oral cavity is the mirror of the body health, so the dentist should be familiar with the oral manifestations of the thyroid diseases. An estimated 15 percent of the general population has abnormalities of thyroid anatomy on physical examination, this means Patients with undiagnosed hypothyroidism or hyperthyroidism are seen in the dental chair, where routine treatment has the potential to result in adverse outcomes. In this article, we explore the function and assessment of the thyroid gland and the impact of its dysfunction on the provision of dental care.

**Our problem as a dentists**

Unfortunately, we sometimes forget to or do not pay a lot of attention to the thyroid gland thinking of it as a part out of our zone as a dentist, ignoring its problems on our treatment plan and even misdiagnosing it.

**Thyroid gland normal structure**

**1- Thyroid gland anatomy**

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.[16,5]

The thyroid gland is formed from the pharyngeal epithelium during the third week of fetal development; it then migrates caudally to its final position, which is posterior to the cricoid and arytenoid cartilages in the neck midline. The adult gland comprises a bilobular structure, which weighs between 15 and 20 grams, and is connected by a 2-centimeter–wide isthmus in the middle. The gland is palpable in most healthy adults. [13]

**2-Thyroid gland physiology**

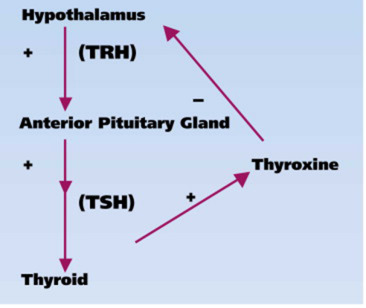
The internal anatomy of the thyroid gland consists of follicles that contain a mucinous colloid where the protein thyroglobulin is found. Thyroglobulin is the basic building block for the two main hormones produced by the thyroid: triiodothyronine, or T3, and thyroxine, or T4. In addition to thyroglobulin, iodine is needed for T3 and T4 synthesis.[13] Iodine is transported into the thyroid follicular cells and is combined with thyroglobulin to form the thyroid hormone precursors monoiodotyrosine and diiodotyrosine. These precursors are transformed into T3 and T4 and later released into the bloodstream. T4 is produced only in the thyroid, while T3 also can be produced in extraglandular tissues. Once in the plasma, T4 is bound primarily to T4-binding globulin, or TBG, and less efficiently to T4-binding prealbumin (transthyretin) and albumin. [13-36].

**3-Thyroid gland function**

T3 is the main metabolic effector, with a 10-fold greater affinity over T4 or nuclear thyroid receptor proteins. The action of this hormone at a molecular level includes the activation of genetic material (mainly transcription and formation of messenger ribonucleic acid) and translation to proteins coding for multiple hormonal and constituent tissues such as growth hormone; thyrotropin-releasing hormone, or TRH; malic enzyme; myosin; and the calcium pump complex of the sarcoplasmic reticulum[17].Thyroid hormone increases oxygen consumption, thermogenesis, and expression of the low-density lipoprotein (LDL) receptor, resulting in accelerated LDL cholesterol degradation. Other physiologic effects of thyroid hormone include increased mental alertness, ventilatory drive, gastrointestinal motility, and bone turnover. During fetal development, thyroid hormone plays a critical role in brain development and skeletal maturation. [20,41]

**4-Regulation of thyroid hormones**

Thyroid function, like many hormonal somatic regulators, is controlled by feedback mechanisms, in which the thyroid hormones act as direct inhibitors of TRH, thus regulating their own production. figure 1. [29-15]



**Figure1.** Regulation of thyroid hormones

**Assessment of thyroid gland**

**1- Evaluation of thyroid disease**

The American Thyroid Association’s Guidelines for Detection of Thyroid Dysfunction [18-33] suggest a screening model for all patients. It is recommended that patients have a serum thyroid-stimulating hormone–, or TSH–, level screen starting at age 35 years and every five years after that, regardless of sex. People from families with history of and risk factors for thyroid disease may be followed more closely. Risk factors include pernicious anemia; diabetes mellitus, or DM; previous surgery or radiation to the head and neck region; vitiligo; family history of thyroid disorders; autoimmune disease; and intake of iodine containing medications (for example, contrast media for imaging purposes). [18]

**2- Laboratory studies:**

Several tests are available that measure thyroid function. Highly specific and sensitive radioimmunoassays are used most often to measure serum T4 and T**3** concentrations and rarely to measure reverse T**3** (rT**3**) concentration. Normal



**Figure2**. Thyroid hormones level

**Thyroid gland diseases**

**1- Hyperthyroidism**

he term *thyrotoxicosis* refers to an excess of T4 and T3 in the bloodstream. This

excess may be the result of production by ectopic thyroid tissue, multinodular

goiter, or thyroid adenoma or may be associated with subacute thyroiditis

(painful and painless), pituitary disease involv- ing the anterior portion of the

gland, or ingestion of thyroid hormone (thyrotoxicosis factitia) or foodstuffs

containing thyroid hormone. When thyrotoxicosis occurs, it is most commonly

associated with Graves disease, toxic nodular goiter, or acute

thyroiditis. **[**19,35,42,27] Graves disease is an autoimmune disease in which

thyroid-stimulating immunoglobulins bind to and activate thyrotrophic

receptors, causing the gland to grow and stimulating the thyroid follicles to

increase T4 and T3 synthesis. [19,35,27] This disorder is much more common in

women (with a male-to-female ratio of 10:1) and may manifest during puberty

or pregnancy or at menopause.

**Thyroid storm**

is the main complication of persistent hyperthyroidism. It is defined as the

body’s response to maintained thyrotoxicosis. Thyroid storm commonly is

expressed as extreme irritability and delirium, a temperature of higher than 41

tachycardia, hypotension, vomiting and diarrhea. Thyroid storm is the body’s

response to maintained thyrotoxicosis. This is common in postoperative states

in patients who have uncontrolled or undiagnosed hyperthyroidism. It also can

be triggered by a surgical emergency, sepsis and trauma. Some case reports

describe acute renal failure, lactic acidosis and absence of fever.[14]

**Clinical manifestations of hyperthyroidism**

**Signs and Symptoms.**

The most common symptoms and signs are nervousness, fatigue, rapid

heartbeat or palpitations, heat intolerance, and weight loss.

These manifestations are reported in more than 50% of all diagnosed patients.

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 of age but occurs in approximately 20% of older

patients. The patient’s skin is warm and moist and the complexion rosy; the

patient may blush readily. Palmar erythema may be present, profuse sweating is

common, and excessive melanin pigmentation of the skin is evident 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. [11,19,35,27] Graves ophthalmopathy, which is identified in approxi-

mately 50% of patients, is characterized by edema and inflammation of the

extraocular muscles, as well as an increase in orbital connective tissue and fat.

ophthalmopathy often produces the greatest long-term disability for patients

with this disease. [37,3]

**Oral manifestations of hyperthyroidism**

In children, the teeth and jaws develop rapidly, and premature loss of deciduousteeth with early eruption of permanent teeth is common. Increase susceptibility to caries. Euthyroid infants of hyper- thyroid mothers have been reported to have erupted teeth at birth. A few patients with thyrotoxicosis have been found to have a lingual “thyroid” consisting of thyroid tissue posterior to the foramen cecum.Burning mouth syndrome also could be found.[28,6]

**Medical management of hyperthyroidism**

Treatment of patients with thyrotoxicosis may involve antithyroid agents that

block hormone synthesis, iodides, radioactive iodine RAI, or subtotal

thyroidectomy. [11] Administration of RAI is the preferred initial treatment for

patients with Graves disease. RAI is contraindicated in pregnant women and

women who are breastfeeding.The main adverse effect associated with RAI is

hypothyroidism. [19,27,37]

**Dental management of hyperthyroidism**

**Antibiotics and Risk of Infection.** Chronic infection should be treated as in any

other patient; that is, patients with extensive dental caries or periodontal disease

should be treated after medical management of the thyroid problem has been

instituted. If acute oral infection occurs in a patient with uncontrolled

hyperthyroid disease, consulta- tion with the patient’s physician is

recommended before initiated dental therapy.[22,30]

**Bleeding*.*** There is little to no risk of bleeding abnor- malities in patients with

hyperthyroidism except in patients concurrently taking warfarin and

propylthiouracil.

**Capacity to Tolerate Care*.*** When a thyrotoxic patient is under good medical

management, dental treatment can proceed without alteration. However,

patients with untreated or poorly treated thyrotoxicosis are susceptible to

developing an acute medical emergency, called thyro- toxic crisis (thyroid

storm). [10]

**drug Considerations.**Use of epinephrine or other pressor amines (in local

anesthetics or gingival retraction cords or to control bleeding) must be avoided

in untreated or poorly treated thyrotoxic patients. However, well-managed

(euthyroid) thyrotoxic patients with thyroid disease require no special

consideration in this regard and may be given normal concentrations of these

vasoconstrictors.[24] Adverse reactions to propylthiouracil include agranu-

locytosis and leukopenia. If these occur, the patient is at risk for serious

infection. The physician should monitor the patient for these adverse

reactions.The dentist can consult with the patient’s physician or can order a

completeblood count to rule out the presence of these complications before

undertaking surgical pro-

cedures.

**Emergencies.** If a thyrotoxic crisis occurs, the dentist must recognize the

features, begin emergency treatment, and seek immediate medical assistance.

The patient given an injection of hydrocortisone (100–300 mg), and started on

an IV infusion of hypertonic glucose (if equipment is available). Vital signs

must be monitored. Immediate medical assistance should be sought, and when

available, other measures such asantithyroid drugs and potassium iodide may be

started.[16,10,23]

**2- Hypothyroidism**

Hypothyroidism is defined by a decrease in thyroid hormone production and thyroid gland function. It is caused by severe iron deficiency, chronic thyroiditis (Hashimoto’s disease), lack of stimulation, radioactive iodine that causes follicle destruction, surgery and pharmacological agents such as lithium and amiodarone, the latter of which is a commonly used antidysrhythmic. [2-4]

This condition can be classified into two categories: primary hypothyroidism, in which the defect is intrathyroid; or secondary hypothyroidism, in which other pathologies can cause an indirect decrease of circulating hormone (for example, surgical or pathological alteration of the hypothalamus). Congenital hypothyroidism refers to alteration in formation of the thyroid gland. It can be caused by dysgenesis, agenesia, inborn defect in hormone production or secretion. Defects in pituitary or hypothalamic metabolism account for some cases. Acquired hypothyroidism is commonly caused by irradiation of the thyroid gland (RAI), surgical removal, or excessive antithyroid drug therapy.

Iatrogenic hypothyroidism can be caused by surgery or radiation therapy to the gland. Hashimoto’s disease is an autoimmune thyroiditis, in which there is a lymphocytic infiltrate into the gland and the production of autoantibodies directed toward thyroglobulin and thyroid peroxidase. Consequently, both the building unit and the enzyme in charge of production of the thyroid hormones are blocked.

**Hashimoto thyroiditis**

Hashimoto thyroiditis is the most common cause of primary hypothyroidism in

. [9,32] It is an autoimmune disorder that manifests most often as an

asymptomatic diffuse goiter. High titers of circulating thyroid autoantibodies and

thyroid antigen–specific T cells are observed. It usually affects young and

middle-aged women and is three to four times more frequent in women than men.

**Signs and Symptoms**

Goiter is the clinical hallmark of Hashimoto thyroiditis. The goiter usually is

moderate in size and rubbery firm in consistency, and it moves freely with

swallowing. In cases of sudden onset, the clinical picture suggests subacute

thyroiditis with pain. [19,9]

**Clinical manifestations of hypothyroidism**

**Signs and Symptoms :**Neonatal hypothyroidism is characterized by dwarfism; overweight; well-recognized facial features consisting of a broad flat nose, wide-set eyes, thick lips, and a large protruding tongue; poor muscle tone; pale skin; stubby hands; retarded bone age; delayed eruption of teeth; malocclusions; a hoarse cry; an umbilical hernia; and mental retardation. All of these abnormalities can be prevented by early detection and treatment.

The onset of hypothyroidism in older children and adults is manifested by characteristic changes in physical appearance: a dull expression; puffy eyelids; alopecia of the outer third of the eyebrows; palmar yel- lowing; dry and rough skin; and dry, brittle, and coarse hair, along with increased size of the tongue. Other features include slowing of physical and mental activity, slurred and hoarse speech, anemia, constipation, increased sensitiv- ity to cold, increased capillary fragility, weight gain, muscle weakness, and deafness. [16,29]Untreated patients with severe myxedema may develop hypothyroid coma, which usually is fatal.[7,39

**Oral manifestations of hypothyroidism**

Infants with hypothyroidism may present with thick lips, enlarged tongue, and delayed eruption of teeth with resulting malocclusion. Adults with acquired hypothyroid- ism can display an enlarged tongue and low salivary flow.[25,34]

**Medical management**

Thyroxine is administrated in all cases of hypothyroidism and is considered the mainstay of therapy. Following administration, thyroxine will peripherally convert into T3. The initial dose of thyroxine is determined based on several factors that include age, weight, comorbidities, and the presence of arrhythmias. The usual dose in adults is 1.8 µg/kg body weight. However, cautious measures should be taken when dealing with elderly, and the complete dose should not be started suddenly. Following administration of therapy, TSH levels should be measured every 6 weeks until it becomes normal. Then, it will be measured once a year for follow up [7,39]. Extremely severs hypothyroidism will cause myxedema coma, and usually develops following a long untreated case of hypothyroidism. A large dose of 400 g intravenous thyroxine should be initially given. After two days, normal dose is given. Hydrocortisone should also be given on the first day, along with other modalities to treat associated hypoglycemia, hypothermia, hypercalcemia, hypotension, and/or hyponatremia [25]. On the other hand, overestimation of thyroxine dose has been associated with several severe consequences like atrial fibrillation, coronary artery disease, and osteoporosis (in women older than 65 years). [34]

**Dental management**

**Antibiotics and Risk of Infection.** Acute oral infection in an uncontrolled

hypothyroid patient could trigger a myxedema coma; such a patient should

receive immediate consultation with the patient’s physician as part of the

management program.

**Bleeding.** There is little to no risk of bleeding abnormalities in patients with

hypothyroidism.

**Capacity to Tolerate Care.**In general, patients with mild symptoms of

untreated hypothyroidism are not in danger when receiving dental therapy.

Also, when hypothyroid patients are under good medical care, no special

problems in terms of dental management remain. However, patients with untreated severe symptoms of hypothyroidism may be in danger if dental treatment is rendered. This is particularly true of patients with poorly controlled disease who have infection and older adults with myxedema. A myxedematous coma can be precipi- tated by CNS depressants, surgical procedures, and infections; thus, the major concerns of dental management of patients with this condition are detection and referral for medical management before any dental treatment is rendered.

**Drug Considerations*.*** CNS depressants, sedatives, and narcotic analgesics may

cause an exaggerated response in patients with mild to severe hypothyroidism.

These drugs must be avoided in all patients with severe hypo- thyroidism and

must be used with care (reduced dosage) in patients with mild

hypothyroidism. [19,40,21]

**Emergencies.**If myxedema coma occurs, the dentist should call for medical

aid; while waiting for this assis- tance, the dentist can inject 100 to 300 mg of

hydrocor- tisone, cover the patient to conserve heat, and applycardiopulmonary

resuscitation if indicated. When medical aid becomes available, parental

levothyroxine is admin- istered, and IV hypertonic saline and glucose are given

as needed.[8]

**Conclusion**

Dental treatment modifications may be necessary for dental patients who are under medical management and follow-up for a thyroid condition even if there are no comorbid conditions. Stress reduction, awareness of drug side effects or interactions, and vigilance for appearance of signs or symptoms of hormone toxicity are among the responsibilities of the oral health care provider.

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