

In Shortly About Thyroid Gland

Franjić S*

Independent Researcher

***Corresponding author:**

Dr. Siniša Franjić,
Independent Researcher,
E-mail: sinisa.franjic@gmail.com

Received: 06 Feb 2021
Accepted: 25 Feb 2021
Published: 01 Mar 2021

Copyright:

©2021 Franjić S, et al. This is an open access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and build upon your work non-commercially.

Keywords:

Thyroid Gland; Metabolic Change; Disorders; Health

Citation:

Franjić S, In Shortly About Thyroid Gland. Clin Surg. 2021; 4(9): 1-5

1. Abstract

The thyroid is a butterfly-like gland located in the lower half of the neck, below the larynx, and in front of the trachea. It consists of two lobes, each measuring between 2 and 4 centimeters, and interconnected by a central narrowed part. Thyroxine and triiodothyronine secrete their hormones into the blood. The work of the thyroid gland in physiological circumstances is controlled by the hypothalamus and pituitary gland. In the control of thyroid function, the most important is the pituitary gland, which uses thyrotropin to stimulate the thyroid gland to produce hormones, but also to grow, which can lead to an increase in the thyroid gland or goiter. The thyroid gland produces its hormones in normal amounts unless one of the thyroid disorders has developed.

2. Introduction

The thyroid gland forms as a diverticulum originating in the floor of the pharynx, and descends through the tongue, past the hyoid, to its position in the neck [1]. The diverticulum usually closes, leaving a pit at the base of the tongue (the foramen caecum, which lies in the midline at the junction of the anterior two thirds and the posterior third of the tongue). Failure of the thyroid to descend or incomplete descent of the track may result in ectopic thyroid tissue. Incomplete obliteration of the track may result in fistula or sinus formation. In all cases of unexplained midline nodules in the neck, thyroid tissue should be suspected. A radioiodine scan should be performed to ensure that there is normal thyroid tissue present in the correct place before the lump is removed.

The thyroid is derived from the foramen caecum at the junction of the anterior two-thirds and posterior third of the tongue [2]. The gland migrates downwards in front of the foregut to lie anterior to the trachea but occasionally may descend lower to the superior mediastinum. It leaves behind the thyroglossal duct which may persist and form a thyroglossal cyst.

The thyroid is fixed to the trachea by the pretracheal fascia so it moves up on swallowing. Aberrant thyroid tissues may be found anywhere along the embryological descent of the gland. Blood supply is from the superior and inferior thyroid arteries. Thyroid operations may damage important structures.

3. Anatomy

The main anlage of the thyroid gland develops as a median endodermal down growth from the first and second pharyngeal pouches [3]. During its migration caudally, it contacts the ultimobranchial bodies developing from the fourth pharyngeal pouches. When it reaches the position it occupies in the adult, with the isthmus situated just below the cricoid cartilage, the thyroid divides into two lobes. The site from which it originated persists as the foramen caecum at the base of the tongue. The path the gland follows may result in thyroglossal remnants (cysts) or ectopic thyroid tissue (lingual thyroid). A pyramidal lobe is frequently present. Agenesis of one thyroid lobe, almost always the left, may occur.

The normal thyroid weighs 15-25 g and is attached to the trachea by loose connective tissue. It is a highly vascularized organ that derives its blood supply principally from the superior and inferior

thyroid arteries. A thyroid ima artery may also be present.

The thyroid gland can cause two groups of symptoms and signs: those connected with the swelling in the neck, and those related to the endocrine activity of the gland [4]. Therefore, in order to appreciate fully the symptoms and signs that may be produced by diseases of the thyroid, a clear understanding of the physiology of the gland is essential. The history and examination should be directed towards detecting both local and general symptoms and signs that may be produced, either by any physical abnormality in the configuration of the thyroid or by any pathophysiological abnormality of its endocrine activity.

The majority of thyroid swellings grow slowly and painlessly. Quite often the patient will come across a swelling coincidentally when washing, or a member of their family or a close friend will point it out to them. Other swellings may have been there for some years before the patient suddenly decides to seek advice concerning their nature and management. In a few patients, a lump will appear suddenly and may be painful, or a long-standing lump may enlarge quickly.

4. Examination

Inspection should be carried out initially from the front [5]. Confirm that there is a mass in the neck in the area of the thyroid gland and that it moves up on swallowing. Place a finger in the suprasternal notch and check that the trachea is central. Examine the thyroid from behind. Place the thumbs on the vertebra prominens and the fingers on the anterior part of the neck on either side. Allow the head to tilt forwards slightly to relax the neck muscles. Feel up and down in the area of the thyroid. Applying gentle pressure to one side of the neck over the thyroid facilitates examination of the contralateral lobe. Decide whether there is a single nodule, many nodules or whether there is diffuse enlargement of the thyroid gland.

5. Symptoms

- Smooth non-toxic enlargement of the gland [5]. This is characteristic of a physiological goitre, which may occur at puberty or in pregnancy
- A smooth toxic enlargement of the gland associated with Graves' disease
- A smooth firm enlargement of the gland (occasionally asymmetrical), usually in middle-aged females and often associated with hypothyroidism, e.g. Hashimoto's disease
- A nodular non-toxic enlargement of the gland. This is characteristic of multinodular goitre
- A solitary nodule in a lobe of the thyroid gland. This may be due to a palpable dominant nodule in a multinodular goitre, a cyst, an adenoma or a carcinoma
- A rapid increase in the size of nodule associated with haemorrhage into a cyst, a rapidly growing carcinoma or

thyroiditis, which may be painful

- A hard irregular goitre with infiltration of muscles and lymphadenopathy in the older patient, e.g. anaplastic carcinoma

In addition to the examination of the thyroid itself, the physical examination should include a search for signs of abnormal thyroid function and the extra thyroidal features of ophthalmopathy and dermopathy [6]. Examination of the neck begins by inspecting the seated patient from the front and side, and noting any surgical scars, obvious masses, or distended veins. The thyroid can be palpated with both hands from behind or while facing the patient, using the thumbs to palpate each lobe. It is best to use a combination of these methods, especially when the nodules are small. The patient's neck should be slightly flexed to relax the neck muscles. After locating the cricoid cartilage, the isthmus can be identified and followed laterally to locate either lobe (normally the right lobe is slightly larger than the left). By asking the patient to swallow sips of water, thyroid consistency can be better appreciated as the gland moves beneath the examiner's fingers.

Features to be noted include thyroid size, consistency, nodularity, and any tenderness or fixation. An estimate of thyroid size (normally 12 to 20 g) should be made, and a drawing is often the best way to record findings. However, ultrasound is the method of choice when it is important to determine thyroid size accurately. The size, location, and consistency of any nodules should also be defined. A bruit over the gland indicates increased vascularity, as occurs in hyperthyroidism. If the lower borders of the thyroid lobes are not clearly felt, a goiter may be retrosternal. Large retrosternal goiters can cause venous distention over the neck and difficulty breathing, especially when the arms are raised (Pemberton's sign). With any central mass above the thyroid, the tongue should be extended, as thyroglossal cysts then move upward. The thyroid examination is not complete without assessment for lymphadenopathy in the supraclavicular and cervical regions of the neck.

6. Metabolic Change

Metabolic complications include glucose intolerance, ketoacidosis, metabolic acidosis, hypermetabolic states, thyroid insufficiency, and adrenal impairment [7]. In fact, almost any medical metabolic problem can arise after surgery, although it may not be related to the anesthesia or surgery per se, but is frequently included as a "complication of surgery". These instances are often indistinguishable from "true" complications following surgery. After surgery on the thyroid or parathyroid, hypocalcemia may develop, either related to injury or removal of the parathyroid glands. The hypocalcemia may be transient, if the stress or injury recovers, and may need oral or rarely intravenous calcium replacement in the shorter term. If the hypocalcemia is slight, no treatment is typically required. However, if hypocalcemia is permanent, continued

calcium and vitamin D replacement may be required. Nutritional and trace mineral deficiencies are common, especially in chronically unwell patients, and require careful balancing and supplementation to correct. Magnesium and manganese, in addition to the classical electrolytes sodium and potassium, are essential to monitor. Nutritional complications are dealt with in a separate section later. The endocrine and metabolic changes can sometimes be surprisingly subtle and go undetected for long periods, unless actively sought. Recent reliance on gastrointestinal hyperalimentation, where possible, has helped correct some of the absorptive and metabolic problems encountered previously with long-term parenteral nutrition.

7. Thyroiditis

Thyroiditis (inflammation of the thyroid) can be acute, subacute, or chronic [8]. Each type is characterized by inflammation, fibrosis, or lymphocytic infiltration of the thyroid gland. Acute thyroiditis is a rare disorder caused by infection of the thyroid gland. The causes are bacteria (*Staphylococcus aureus* most common), fungi, mycobacteria, or parasites. Subacute cases may be granulomatous thyroiditis (de Quervain's thyroiditis) or painless thyroiditis (silent thyroiditis or subacute lymphocytic thyroiditis). This form often occurs in the postpartum period and is thought to be an autoimmune reaction.

Chronic thyroiditis occurs most frequently in women aged 30 to 50 years and is termed Hashimoto's disease, or chronic lymphocytic thyroiditis. Diagnosis is based on the histologic appearance of the inflamed gland. The chronic forms are usually not accompanied by pain, pressure symptoms, or fever, and thyroid activity is usually normal or low. Cell-mediated immunity may play a significant role in the pathogenesis of chronic thyroiditis. A genetic predisposition also appears to be significant in its etiology. If untreated, the disease slowly progresses to hypothyroidism.

8. Thyroid Storm

Thyroid storm (thyrotoxic crisis) is a form of severe hyperthyroidism, usually of abrupt onset and characterized by high fever (hyperpyrexia), extreme tachycardia, and altered mental state, which frequently appears as delirium [8]. Thyroid storm is a life-threatening condition that is usually precipitated by stress, such as injury, infection, surgery, tooth extraction, insulin reaction, diabetic ketoacidosis, pregnancy, digitalis intoxication, abrupt withdrawal of antithyroid drugs, extreme emotional stress, or vigorous palpation of the thyroid. These factors precipitate thyroid storm in the partially controlled or completely untreated patient with hyperthyroidism. Untreated thyroid storm is almost always fatal, but with proper treatment the mortality rate can be reduced substantially.

Thyroid storm is an entity characterized by severe hyperthyroidism [9]. Multiple etiologies exist, and care of the surgical patient can precipitate this disease. Thyroid storm can have a high mor-

tality and knowledge of this entity can provide the physician with valuable tools to assist the care for the patient.

The initial diagnosis of hyperthyroidism is usually made on the basis of thyroid function tests in addition to a detailed history and physical exam. In thyroid storm, serum total and free triiodothyronine (T) levels are elevated. In addition, thyroid stimulating hormone (TSH) is generally depressed. However, the differentiation between thyroid storm and simple thyrotoxicosis is not easily made.

9. Thyroid Cancer

Thyroid cancer is not very common, since it accounts for 1-2% of all cancers, with an incidence of 4 cases per 100,000 populations with a higher incidence among women than men (4:1) [10].

There are various types of thyroid cancer: the benign variant is called adenoma and the most common form is follicular, which is capsulated and presents a morphology similar to follicular epithelium. The adenoma differs from carcinoma because it does not invade the capsule or the supplying vessels and generally does not exceed 2 cm in diameter.

There are several types of malignant thyroid tumors: follicular, papillary, medullary and anaplastic. Follicular carcinomas have cytologic features similar to adenomas and are differentiated only by infiltration of the capsule and angioinvasion.

Pre-surgery diagnosis is based on fine needle aspiration cytology (FNAC), but in 20-30% of cases the cell alteration does not allow a clear identification between follicular adenoma and follicular carcinoma. In these situations, as a precaution, the thyroid is removed and the surgical findings are subjected to histologic examination.

Only 25% of these cases prove to be malignant, while the remaining 75% of patients have undergone an unnecessary thyroidectomy, thus with an associated waste of resources and money, in addition to the fact that the patient will need to take synthetic thyroid hormones for the rest of their life.

10. Oral Surgery

The thyroid gland problem of primary significance in oral surgery is thyrotoxicosis because thyrotoxicosis is the only thyroid gland disease in which an acute crisis can occur [11]. Thyrotoxicosis is the result of an excess of circulating triiodothyronine and thyroxine, which is caused most frequently by Graves' disease, a multinodular goiter, or a thyroid adenoma. The early manifestations of excessive thyroid hormone production include fine, brittle hair, hyperpigmentation of skin, excessive sweating, tachycardia, palpitations, weight loss, and emotional lability. Patients frequently, although not invariably, have exophthalmos (a bulging forward of the globes caused by increases of fat in the orbit). If hyperthyroidism is not recognized early, the patient may have heart failure. The diagnosis is made by the demonstration of elevated circulating

thyroid hormones, using direct or indirect laboratory techniques.

Thyrotoxic patients are usually treated with agents that block thyroid hormone synthesis and release, with a thyroidectomy, or with both. However, patients left untreated or incompletely treated can have a thyrotoxic crisis caused by the sudden release of large quantities of preformed thyroid hormones. Early symptoms of a thyrotoxic crisis include restlessness, nausea, and abdominal cramps. Later symptoms are a high fever, diaphoresis, tachycardia, and, eventually, cardiac decompensation. The patient becomes stuporous and hypotensive, with death resulting if no intervention occurs.

The dentist may be able to diagnose previously unrecognized hyperthyroidism by taking a complete medical history and performing a careful examination of the patient, including thyroid gland inspection and palpation. If severe hyperthyroidism is suspected from the history and inspection, the gland should not be palpated because that manipulation alone can trigger a crisis. Patients suspected of being hyperthyroid should be referred for medical evaluation before oral surgery.

11. Thyroid in Pregnancy

In pregnancy, normal physiologic changes result in alterations to thyroid function [12]. Biochemical hyperthyroidism (gestational transient thyrotoxicosis) is found in about 3% of normal pregnancies in the first and early second trimester, usually associated with hyperemesis gravidarum. The transient changes spontaneously normalize to a euthyroid state by late second trimester. This is attributed to the thyrotrophic effect of human chorionic gonadotropin (HCG); thus conditions with higher levels of HCG, such as molar pregnancies and multiple pregnancies, may be particularly associated with transient thyrotoxicosis.

Patients with previously undiagnosed thyroid disorders often have non-specific symptoms or are asymptomatic and clinical detection of new cases can be promoted by having a high index of suspicion.

Hyperthyroidism is the excessive production of thyroid hormone (TH), most commonly due to an autoimmune disorder known as Graves' disease or to a multinodular goiter. Thyrotoxicosis describes the presence of excessive circulatory TH and is diagnosed by a suppressed thyroid-stimulating hormone (TSH) associated with elevated free thyroxine (FT4) and free triiodothyronine (FT3) serum concentrations. It is characterized by sympathetic overactivity and increased metabolic rate despite normal levels of catecholamine's.

Clinical features of thyrotoxicosis include tremor, palpitations, weight loss, sweating, anxiety, and heat intolerance. Cardiac arrhythmias are common, typically sinus tachycardia but can also take the form of atrial fibrillation.

Hypothyroidism is diagnosed by an elevated serum TSH concentration accompanied by a low serum FT4 concentration. While iodine deficiency is the most common cause worldwide, autoimmune

Hashimoto thyroiditis is the most common cause in iodine-replete areas. The clinical picture can range from being asymptomatic to a life-threatening "myxedema coma.

Common features of hypothyroidism include fatigue, weight gain, cold intolerance, hair loss, dry skin, water retention, and depression. Cardiovascular effects include bradycardia, reduced cardiac contractility, and increased systemic vascular resistance resulting in hypertension. Chronic hypothyroidism can lead to congestive heart failure with impaired systolic and diastolic function.

12. Conclusion

Thyroid hormones affect the function of all organs. Without them, there is no normal development or functioning of the central nervous system. They are important for maintaining body temperature and maintaining the normal activity of the respiratory center, controlling energy expenditure and oxygen consumption. They act on the heart by increasing the contractility of the heart muscle and speeding up the work of the heart, and also increase intestinal motility, affect bone remodeling, sugar homeostasis and blood fat. The thyroid gland produces its hormones in normal amounts unless one of the thyroid disorders has developed. It can then have either excessive or deficient production called hyperthyroidism or hypothyroidism. The symptoms are opposite: on the one hand, overproduction causes weight loss, palpitations, sensitivity to heat and sweating, accelerated metabolism and shivering. On the other hand, insufficient production leads to weight gain, sensitivity to cold, constipation, fatigue and sleepiness.

References

1. Ellis H, Caine, Sir R, Watson C. General Surgery - Lecture Notes, 13th Edition, John Wiley & Sons, Ltd, Chichester, UK. 2016; 313.
2. Franklin IJ, Dawson PM, Rodway AD. Essentials of Clinical Surgery, Second Edition, Saunders, Elsevier, Edinburgh, UK. 2012; 181.
3. Doherty GM. Thyroid & Parathyroid in Doherty, G. M. (ed). Current Diagnosis and Treatment - Surgery, 14th Edition, McGraw-Hill Education, New York, USA. 2015; 277.
4. Browse NL, Black J, Burnand KG, Thomas WEG. Browse's Introduction to the Symptoms & Signs of Surgical Disease, Fourth Edition, CRC Press, Taylor & Francis Group, Boca Rato, USA. 2005; pp. 288.
5. Raftery AT, Delbridge MS, Wagstaff MJD. Surgery, Fourth Edition, Churchill Livingstone, Elsevier, Edinburgh, UK. 2011; 178-9.
6. Jameson JL, Weetman AP. Disorders of the Thyroid Gland in Kasper DL, Braunwald E, Fauci AS, Hauser SL, Longo DL, Jameson JL. Harrison's Principles of Internal Medicine, 16th Edition, The McGraw-Hill Companies, Inc., New York, USA. 2005; 2108.
7. Coventry BJ, Bruening M, Whitfield R, Yong J. General Perioperative Complications in Coventry, BJ. (ed). General Surgery Risk Reduction, Springer-Verlag, London, UK. 2014; 61.

8. Johnson JY. Brunner and Suddarth's Textbook of Medical-Surgical Nursing, Twelfth Edition, Wolters Kluwer, Lippincott Williams & Wilkins, Philadelphia, USA. 2010; 617-20.
9. Busken C, Coefield R, Kelly R, Brower S. Surgical Endocrine Emergencies in Cohn, SM. (ed). Acute Care Surgery and Trauma - Evidence-Based Practice, Informa Healthcare, London, UK. 2009; 451-3.
10. Cafiero G, Papale F, Barbarisi A. Advanced Diagnostic Applications in Bechi, P. New Approach to Diagnosis and Prognosis in Barbarisi A, Bechi P, Innocenti P, Redi CA, Rosso F. Biotechnology in Surgery, Springer-Verlag Italia, Mila, Italy. 2011; 31.
11. Hupp J.R. Preoperative Health Status Evaluation in Hupp JR, Ellis III E, Tucker MR. (eds). Contemporary Oral and Maxillofacial Surgery, Fifth Edition, Mosby, Elsevier, St. Louis, USA. 2008; 16-17.
12. Labib R, Chan S. Patient with Thyroid Disease in Coomarasamy A, Shafi MI, Davilla GW, Chan KK. (eds). „Gynecologic and Obstetric Surgery - Challenges and Management Options, John Wiley & Sons, Ltd, Chichester, UK. 2016; 30-31.