

Sudan University of Science and Technology

College Of Graduate Studies

**Incidental Findings of Thyroid Ultrasound in Students
of the College of Medical Radiological Science –SUST**

**النتائج العرضية للغدة الدرقية بالمسح بالموجات فوق الصوتية لطلاب كلية
علوم الأشعة الطبية –جامعة السودان للعلوم والتكنولوجيا**

Thesis Submitted for Partial Fulfillment of the awards of
Master Degree in Diagnostic ultrasonography

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الآية الكريمة

قال تعالى:

قُلْ (لَوْ كَانَتِ حُرْمٌ لِكَلِمَاتِي لَنَنْزِلُهَا حُرًّا قَبْلَ أَنْ تُنكَهَدِمَ اتِّي وَيَوْمَ لَوْ جِئْتَنِي لَه مَدَدًا)

صدق الله العظيم

سورة الكهف الآية (109)

Dedication

To:

My family

My friends

My colleagues

My all teachers

Acknowledgment

First of all, I Thank Allah the almighty for helping me to complete this project. I Thank Dr. Dr. Karolin Edward my supervisor for her help and guidance, my great fullness for my all teachers in different educational levels.

Finally I would like to thank everybody who helped me in this project.

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List Abbreviation:

Abbreviation	Meaning
TSH	Thyroid Stimulating Hormone
cAMP	cyclic Adenosine Monophosphate
ATP	Adenosine Triphosphate
TRH	Thyrotropin Releasing Hormone
TSI	Thyroid Stimulating Immunoglobulin
AP	Anteroposterior

Abstract

This descriptive study was carried out to show the incidental findings of thyroid ultrasound scan in students of medical radiologic science college of Sudan university of science and technology. All the students were scanned for thyroid by measured thyroid diameters and findings on the affected subjects were evaluated by using (Alpinion-Ecube 7) device from July to September 2016. The study involved 60 subjects 47 (78.3%) of which were female whereas 13 (21.6%) were males aged (17_ 25years). the data analysed using excel Microsoft office programme and SPSS. The study was result that 71.6 % of sample were normal and 28.3% had thyroid findings 57.7% of these findings being cystic nodule ,15.4% solid nodule , 7.7% mixed , 7.7% calcification , 7.7% goiter,7.7%hypoechoic thyroid , The male had been affected more than female.

The study recommended the use of ultrasound in early detection of thyroid disease and further research in this direction by increasing the number of samples. The routine ultrasound examination to those who had family history of thyroid disease and suffer from symptoms of thyroid disorders is important.

ملخص البحث

اجريت هذه الدراسة الوصفية لتوضيح النتائج العرضية للمسح الذي تم بواسطة الموجات فوق الصوتية للغدة الدرقية على طلاب كلية علوم الاشعة الطبية بجامعة السودان للعلوم والتكنولوجيا. لقد تم مسح كل الطلاب لاكتشاف أمراض الغدة بواسطة قياس ب قياس ابعاد الغدة وتقييم النتائج للعينات واستخدم جهاز (Alpinion-Ecube 7). وقد شملت الدراسة 60 طالبا 47 منهم من النساء اي بنسبة 78.3% ، و 13 من الذكور اي بنسبة 21.6% ، وقد تراوحت أعمارهم بين 17 و 25 عاما، وذلك في الفترة من يوليو 2016 حتى سبتمبر 2016. تم تحليل البيانات باستخدام الحزمة الاحصائية للعلوم الاجتماعية (SPSS) وبرنامج مايكروسفت اكسل. وكانت نتيجة الدراسة ان 71.6% كانت طبيعية و 28.3% كانت مصابة وأن 57.7% من الحالات المصابة كانت اكياس في الغدة و(15.4%) كانت صلبه و(7.7%) كانت وصف الاصابة مختلطة و(7.7%) كان تضخم و(7.7%) تكلس و (7.7%) منها ناقص الصدى. ووضحت النتائج ان نسبة الاصابة في الذكور اكثر من النساء . وقد اوصت الدراسة باستخدام الموجات فوق الصوتية في الكشف المبكر لامراض الغدة الدرقية وزيادة البحوث في هذا الاتجاه بزيادة العينات واهمية الفحص الروتيني لمن لديهم تاريخ اسري لامراض الغدة الدرقية خصوصا إذا كانت لديهم اعراض اضطراب او اصابة بامراض الغده.

Chapter one

1.1 Introduction

Because of the superficial location of the thyroid gland, high-resolution real-time gray-scale and color Doppler sonography can demonstrate and provide noninvasive image of the highly sensitive thyroid gland, parathyroid, and lymph nodes anatomy and Congenital conditions (aplasia, hypoplasia, and ectopic), Pathology nodular thyroid disease include (cysts, Hyperplasia, Goiter, adenoma, Carcinomas (Papillary, medullary, follicular, and anaplastic) Lymphoma.

Diffuse thyroid disease include chronic autoimmune lymphocytic thyroiditis (Hashimoto's thyroiditis), colloid or adenomatous goiter, and Graves' disease.

There are various tests that can be carried out to find the cause of your thyroid problem. These include:

Blood tests – this is usually the first test performed and is done to ensure that the thyroid is not over- or under-active.

Fine needle aspiration cytology (FNAC) - removes cells from the thyroid for examination under a microscope. Usually use the ultrasound as guidance

X-rays - a CT scan (a special form of X-ray) may be performed to check the position of a retrosternal goiter in relation to the windpipe (trachea) and gullet (esophagus).

Nuclear medicine scan - this checks the size, shape, and position of the thyroid and detects areas that are over-active ('hot') or under-active ('cold').

1.2 Problem of study:

Usually thyroid diseases appear at middle age due hormonal changes but now a days we notice the appearance of this disease even in early ages so the study aims to evaluate the health of the students.

1.3 Objectives of the Study:

1.3.1 General Objective:

Incidental finding of ultrasound scan of Thyroid gland in student of collage of radiological science

1.3.2 Specific Objectives:

1. To measure the thyroid gland lobes (length –width- antro posterior-volume)
2. To evaluate the finding in both lobes
3. To correlate the finding with gender

1.4 Significant of study:

This study will be document and references for similar studies in the certain year and population

Chapter two

Literature review

2-1 Anatomy of the Thyroid Gland (Glandula Thyreioidea; Thyroid Body)

2-1-1 The **thyroid gland** is a highly vascular organ, situated at the front and sides of the neck; it consists of right and left lobes connected across the middle line by a narrow portion, the **isthmus**. Its weight is somewhat variable, but is usually about 30 grams. It is slightly heavier in the female, in whom it becomes enlarged during menstruation and pregnancy. (Gray,H,2001)

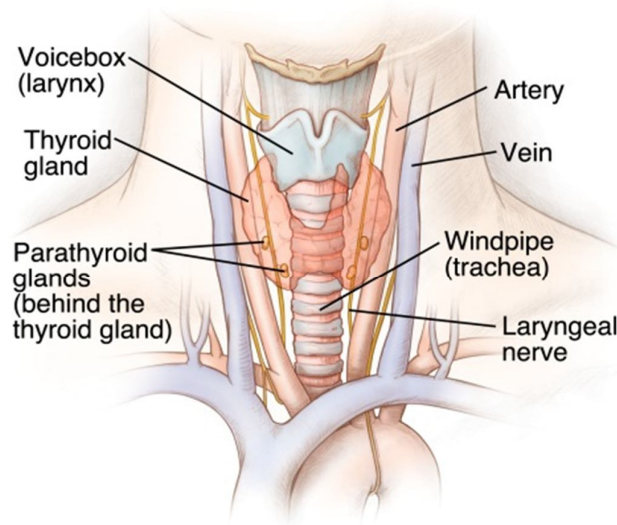


fig 2-1 Thyroid gland

<https://anatomy-medicine.com>

The **lobes** (*lobuli gl. thyreioideae*) are conical in shape, the apex of each being directed upward and lateral ward as far as the junction of the middle with the lower third of the thyroid cartilage; the base looks downward, and is on a level with the fifth or sixth tracheal ring. Each lobe is about 5 cm. long; its greatest width is about 3 cm., and its thickness about 2 cm.

The **lateral** or **superficial surface** is convex, and covered by the skin, the superficial and deep fasciæ, the Sternocleidomastoideus, the superior belly of the Omohyoideus, the Sternohyoideus and Sternothyreoides, and beneath the last muscle by the pretracheal layer of the deep fascia, which forms a capsule for the gland.

The **deep** or **medial surface** is moulded over the underlying structures, viz., the thyroid and cricoid cartilages, the trachea, the Constrictor pharyngis inferior and posterior part of the Cricothyreoides, the esophagus (particularly on the left side of the neck), the superior and inferior thyroid arteries, and the recurrent nerves

. The **anterior border** is thin, and inclines obliquely from above downward toward the middle line of the neck, while the **posterior border** is thick and overlaps the common carotid artery, and, as a rule, the parathyroids.

The **isthmus** (*isthmus gl. thyreoidea*) connects together the lower thirds of the lobes; it measures about 1.25 cm. in breadth, and the same in depth, and usually covers the second and third rings of the trachea. Its situation and size present, however, many variations. In the middle line of the neck it is covered by the skin and fascia, and close to the middle line, on either side, by the Sternothyreoides. Across its upper border runs an anastomotic branch uniting the two superior thyroid arteries; at its lower border are the inferior thyroid veins. Sometimes the isthmus is altogether wanting.

A third lobe, of conical shape, called the **pyramidal lobe**, frequently *arises* from the upper part of the isthmus, or from the adjacent portion of either lobe, but most commonly the left, and ascends as far as the hyoid bone. It is occasionally quite detached, or may be divided into two or more parts.

A fibrous or muscular band is sometimes found attached, above, to the body of the hyoid bone, and below to the isthmus of the gland, or its pyramidal lobe. When muscular, it is termed the **Levator glandulæ thyreoideæ**.

Small detached portions of thyroid tissue are sometimes found in the vicinity of the lateral lobes or above the isthmus; they are called **accessory thyroid glands** (*glandulae thyroideae accessoriae*). (Gray,H,2001)

2-1-2 Structure:-

The thyroid gland is invested by a thin capsule of connective tissue, which projects into its substance and imperfectly divides it into masses of irregular form and size. When the organ is cut into, it is of a brownish-red color, and is seen to be made up of a number of closed vesicles, containing a yellow glairy fluid, and separated from each other by intermediate connective tissue. The vesicles of the thyroid of the adult animal are generally closed spherical sacs (100 to 300 micrometers in diameter) filled with a secretory substance called *colloid* and lined with *cuboidal epithelial cells* that secrete into the interior of the follicles). This appearance is supposed to be due to the mode of growth of the gland, and merely indicates that an increase in the number of vesicles is taking place. Each vesicle is lined by a single layer of cubical epithelium. There does not appear to be a basement membrane, so that the epithelial cells are in direct contact with the connective-tissue reticulum which supports the acini. The vesicles are of various sizes and shapes, and contain as a normal product a viscid, homogeneous, semifluid, slightly yellowish, colloid material; red corpuscles are found in it in various stages of disintegration and decolorization, the yellow tinge being probably due to the hemoglobin, which is thus set free from the colored corpuscles. The colloid material contains an iodine compound, *iodothylin*, and is readily stained by eosin. The thyroid gland prepares and secretes into the vascular channels a substance, formed under normal conditions in the outer pole of the cell and excreted from it directly without passing by the indirect route through the follicular cavity. In addition to this direct mode of secretion there is an indirect mode which consists in the condensation of the secretion into the form of droplets, having high content of solids, and the extension of these

droplets into the follicular cavity. These droplets are formed in the same zone of the cell as that in which the primary or direct secretion is formed.

This internal secretion of the thyroid is supposed to contain a specific hormone which acts as a chemical stimulus to other tissues, increasing their metabolism (Gray,H,2001)

2-1-3 Blood Supply:-

As with other endocrine organs, the thyroid gland has a rich blood supply with abundant anastomoses. The arterial supply is bilateral from both the external ca-rotid system, through the superior thyroid artery, and the subclavian system, through the inferior thyroid branch of the thyrocervical trunk. There may be a single thyroid IMA artery that arises from thebrachiocephalic artery. The superior thyroid artery is normally the first branch of the external carotid artery, though frequently it may arise more inferiorly from the common carotid artery. This vessel descends to the superior pole of the thyroid along with the external laryngeal nerve. As it reaches the thyroid, the artery divides into anterior and posterior branches. The anterior branch parallels the medial border of the lobe and anastomoses in the midline with the anterior branch of the other side. The posterior branch anastomoses with branches of the inferior thyroid artery. The inferior thyroid artery takes a looping course. It ascends along the anterior scalene muscle. It turns medially to pass posteriorly to the carotid sheath and usually posteriorly to the sympathetic trunk as well. It descends along the longus colli to reach the inferior pole of the thyroid. There it passes to the thyroid either anteriorly or posteriorly to the recurrent laryngeal artery. At the thyroid, the artery branches into superior and inferior branches. The superior branch ascends on the posterior part of the gland to anastomose with the posterior branch of the superior thyroid artery. The inferior branch supplies the inferior part of the gland as well as the inferior parathyroid glands. The

inferior thyroid artery may be absent on either side. There is evidence that there are anthropologic differences in the incidence of thyroid ima arteries, as well as in the symmetric origin of the superior thyroid arteries.

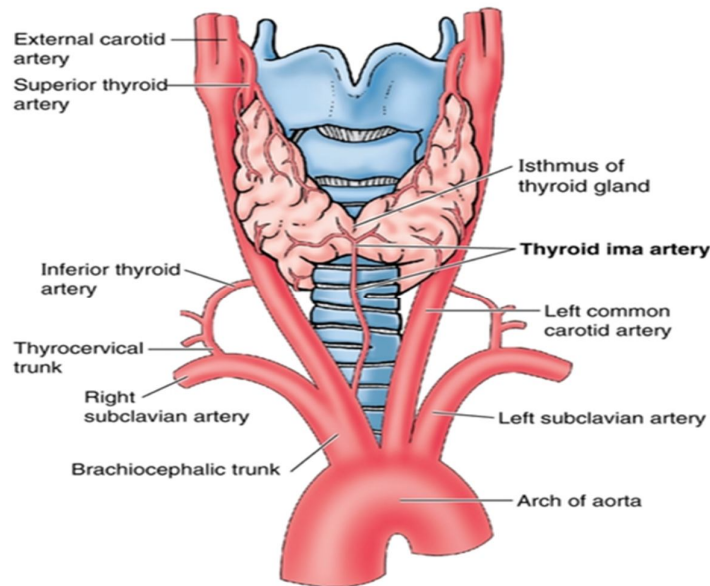


Fig2-2 Thyroid arteries

<https://quizlet.com>

There are three main venous pathways from the thyroid: the superior, middle, and inferior thyroid veins. The superior thyroid vein accompanies the superior thyroid artery and drains into the internal jugular vein. The middle thyroid vein is unaccompanied and drains directly into the internal jugular vein. Because of its posterior course, it is at risk when forward traction is applied to the gland, as in a thyroidectomy there are often a number of inferior thyroid veins that drain into the internal jugular or the brachiocephalic vein (www.endocrinesurgeon.co.uk/index.php/anatomy-of-the-thyroid-gland).

2-1-4 Lymphatic drainage:-

Lymphatic drainage of the thyroid gland is extensive and flows multidirectionally. Immediate lymphatic drainage courses to the periglandular nodes; to the prelaryngeal (Delphian), pretracheal, and paratracheal nodes along the recurrent laryngeal nerve; and then to mediastinal lymph nodes. Regional metastases of thyroid carcinoma can also be found laterally, higher in the neck along the internal jugular vein. This can be explained by tumor invasion of the pretracheal and paratracheal nodes causing an obstruction of normal lymph flow. (<http://fistweb.unhc.edu/student/selectives/luzietti/thyroid-anatomu.htm>)

2-1-5 Thyroid gland nerve supply:

The thyroid gland is innervated by nerves of the autonomic nervous system, arising from the sympathetic trunk in the neck (specifically, nerves from the superior, middle and inferior cervical sympathetic ganglia). It is important to note that the nerves do not influence the secretion of hormones from the gland; this is purely under the control of the pituitary gland, which releases thyroid-stimulating hormone into the bloodstream to signal to the thyroid gland to release thyroid hormones into the bloodstream. The nerves to the thyroid gland travel there via different plexuses, including the cardiac plexus, superior and inferior thyroid periarterial plexuses. In general, the nerves are often found accompanying the inferior and superior thyroid arteries. (<http://www.thyroid.com.au/thyroid-gland-anatomy>)

2-1-6 Initial Thyroid Embryology:

The thyroid gland is the first of the body's endocrine glands to develop, on approximately the 24th day of gestation. The thyroid originates from two main structures: the primitive pharynx and the neural crest. The rudimentary lateral thyroid develops from neural crest cells, while the median thyroid,

which forms the bulk of the gland, arises from the primitive pharynx. The foramen cecum begins rostral to the copula, also known as the hypobranchial eminence. This median embryologic swelling consists of mesoderm that arises from the second pharyngeal pouch (although the third and fourth pouches are also involved). The thyroid gland, therefore, originates from between the first and second pouches. <http://emedicine.medscape.com>

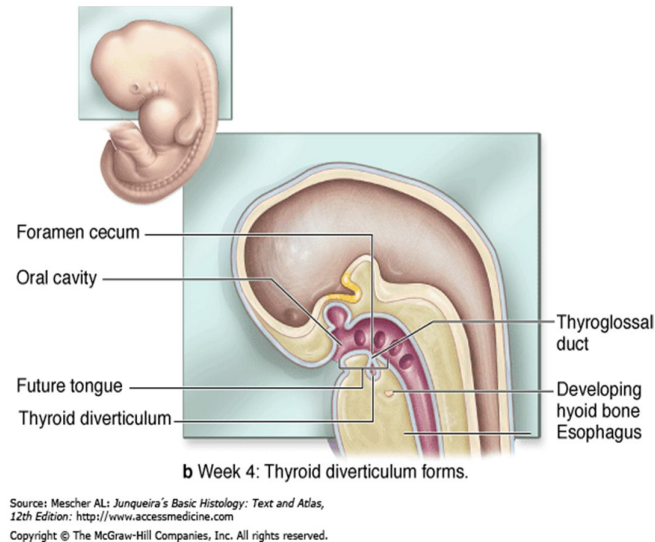


Fig 2-3 Thyroid in week 4

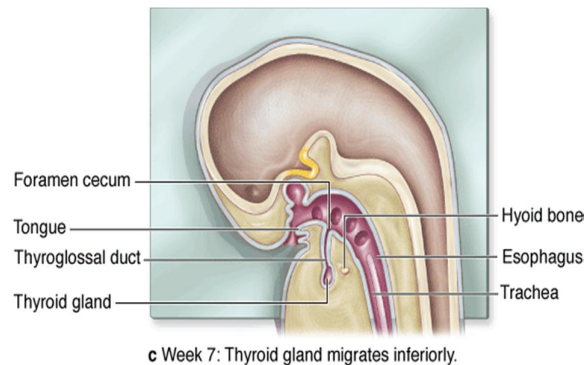
<http://www.accessmedicin.com>

The initial thyroid precursor, the thyroid primordium, starts as a simple midline thickening and develops to form the thyroid diverticulum. This structure is initially hollow, although it later solidifies and becomes bilobed. The stem usually has a lumen, the thyroglossal duct that does not descend into the lateral lobes. The 2 lobes are located on either side of the midline and are connected via an isthmus. . <http://emedicine.medscape.com>)

2-1-7 Descent of the Thyroid Gland:

The initial descent of the thyroid gland follows the primitive heart and occurs anterior to the pharyngeal gut. At this point, the thyroid is still

connected to the tongue via the thyroglossal duct. The tubular duct later solidifies into a cord of cells that will form the follicular elements. The proximal segment retracts and subsequently obliterates entirely, leaving only the foramen cecum at the posterior aspect of the tongue. Nonetheless, in some individuals, remnants of this duct may still persist



Source: Mescher AL: *Junqueira's Basic Histology: Text and Atlas, 12th Edition*: <http://www.accessmedicine.com>
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fig 2-4 Thyroid in week7

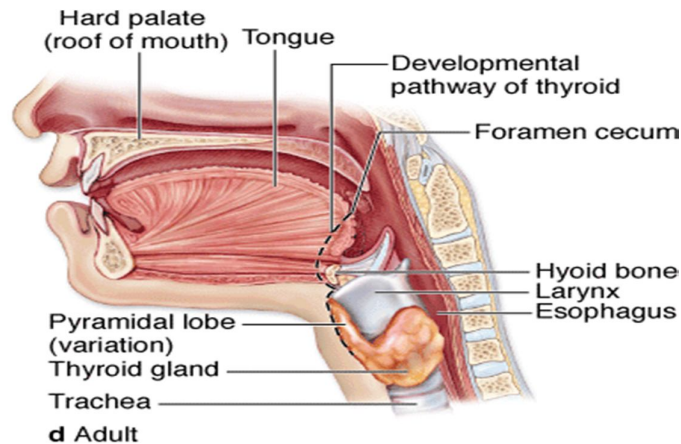
(<https://www.accessmedicin.com>)

The foramen cecum represents the opening of the thyroglossal duct into the tongue; its remains may be observed as a small blind pit in the midline between the anterior two thirds and the posterior third of the tongue.

A pyramidal lobe of the thyroid may be observed in as many as 50% of patients. This lobe represents a persistence of the inferior end of the thyroglossal duct that has failed to obliterate. [1] As such, the pyramidal lobe itself may be attached to the hyoid bone, similar to a thyroglossal duct cyst, or may be incorporated into a thyroglossal duct cyst. (<http://emedicine.medscape.com>).

The caudal segment of the thyroglossal duct develops as a bilobed, encapsulated gland while reaching its final, orthotopic position.

Descent of the thyroid gland carries it anterior (or ventral) to the hyoid bone and, subsequently, anterior (or ventral) to the laryngeal cartilages. As the thyroid gland descends, it forms its mature shape, with a median isthmus connecting the two lateral lobes. The thyroid completes its descent in the seventh gestational week, coming to rest in its final location immediately anterior to the trachea. (Defective embryogenesis of the thyroid gland as it descends to its target location can result in ectopic thyroid tissue, found in approximately 1 in 100,000-300,000 persons and in 1 in 4000-8000 patients with thyroid disease. (<http://emedicine.medscape.com>).



Source: Mescher AL: *Junqueira's Basic Histology: Text and Atlas, 12th Edition*: <http://www.accessmedicine.com>
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Fig 2-5 Thyroid in adult

<https://www.accessmedicin.com>)

The caudal segment of the thyroglossal duct undergoes histologic differentiation into follicular elements at gestational weeks 10 and 11.

The division of these primary follicles results in follicle formation during weeks 11 and 12. Intracellular canaliculi form connections with adjacent cells and these spaces subsequently fuse, forming a confluent lumen. Desmosomes, molecular complexes of adhesion proteins, connect the cells, keeping follicular contents confined within the lumen. Colloid then

accumulates within the follicles during gestational week 13. (<http://emedicine.medscape.com>)

2-2 physiology of the thyroid gland:

2-2-1 The thyroid secretes two major hormones, *thyroxine* and *triiodothyronine*, commonly called T4 and T3, respectively. Both of these hormones profoundly increase the metabolic rate of the body. Complete lack of thyroid secretion usually causes the basal metabolic rate to fall 40 to 50 per cent below normal, and extreme excesses of thyroid secretion can increase the basal metabolic rate to 60 to 100 per cent above normal. Thyroid secretion is controlled primarily by *thyroid-stimulating hormone (TSH)* secreted by the anterior pituitary gland. The thyroid gland also secretes calcitonin, an important hormone for calcium Metabolism.

About 93 per cent of the metabolically active hormones secreted by the thyroid gland is *thyroxine*, and 7 per cent *triiodothyronine*. However, almost all the thyroxine is eventually converted to triiodothyronine in the tissues, so that both are functionally important. The functions of these two hormones are qualitatively the same, but they differ in rapidity and intensity of action. Triiodothyronine is about four times as potent as thyroxine, but it is present in the blood in much smaller quantities and persists for a much shorter time than does thyroxine. (Guyton, A et al 2006)

oxine and triiodothyronine, these iodinated tyrosines also are freed from the thyroglobulin. Iodine Is Required for Formation of Thyroxine:

To form normal quantities of thyroxine, about 50 milligrams of ingested iodine in the form of iodides are required *each year*, or about *1 mg/week*. To prevent iodine deficiency, common table salt is iodized with about 1 part sodium iodide to every 100,000 parts sodium chloride. (Guyton, A et al 2006)

It takes all these steps and processes to make the thyroid hormones and so they become active in the body!

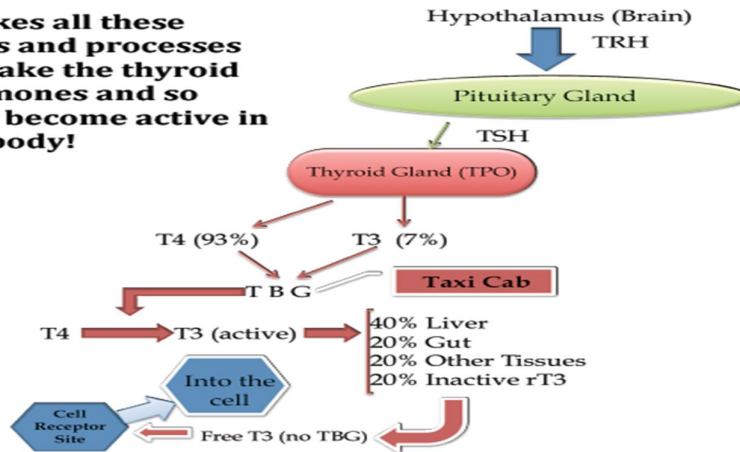


Fig 2-6 Processes to make thyroid hormones

<https://helpmychronicpain.com>

2-2-2 Fate of Ingested Iodides:

Iodides ingested orally are absorbed from the gastrointestinal tract into the blood in about the same manner as chlorides. Normally most of the iodides are rapidly excreted by the kidneys, but only after about one fifth are selectively removed from the circulating blood by the cells of the thyroid gland and used for synthesis of the thyroid hormones. (Guyton, A et al 2006)

2-2-3 Iodide Pump (Iodide Trapping)

The first stage in the formation of thyroid hormones, shown in is transport of iodides from the blood into the thyroid glandular cells and follicles. The basal membrane of the thyroid cell has the specific ability to pump the iodide actively to the interior of the cell. This is called *iodide trapping*. In a normal gland, the iodide pump concentrates the iodide to about 30 times its concentration in the blood. When the thyroid gland becomes maximally active, this concentration ratio can rise to as high as 250 times. The rate of iodide trapping by the thyroid is influenced by several factors, the most important being the concentration of TSH; TSH stimulates and

hypophysectomy greatly diminishes the activity of the iodide pump in thyroid cells. (Guyton, A et al 2006)

2-2-4 Thyroglobulin, and Chemistry of Thyroxine and Triiodothyronine Formation:

2-2-4-1 Formation and Secretion of Thyroglobulin by the Thyroid Cells:

The thyroid cells are typical protein-secreting glandular cells, as shown in. The endoplasmic reticulum and Golgi apparatus synthesize and secrete into the follicles a large glycoprotein molecule called *thyroglobulin*, with a molecular weight of about 335,000. Each molecule of thyroglobulin contains about 70 tyrosine amino acids, and they are the major substrates that combine with iodine to form the thyroid hormones. Thus, the thyroid hormones form *within* the thyroglobulin molecule. That is, the thyroxine and triiodothyronine hormones formed from the tyrosine amino acids remain part of the thyroglobulin molecule during synthesis of the thyroid hormones and even afterward as stored hormones in the follicular colloid. (Guyton, A et al 2006)

2-2-4-2 Oxidation of the Iodide Ion:

The first essential step in the formation of the thyroid hormones is conversion of the iodide ions to an *oxidized form of iodine*, either nascent iodine (I⁰) or I³ that is then capable of combining directly with the amino acid tyrosine. This oxidation of iodine is promoted by the enzyme *peroxidase* and its accompanying *hydrogen peroxide*, which provide a potent system capable of oxidizing iodides. The peroxidase is either located in the apical membrane of the cell or attached to it, thus providing the oxidized iodine at exactly the point in the cell where the thyroglobulin molecule issues forth from the Golgi apparatus and through the cell membrane into the stored thyroid gland colloid. When the peroxidase system is blocked or when it is hereditarily absent from the cells, the rate of formation of thyroid hormones falls to zero. (Guyton, A et al 2006)

2-2-5 Iodination of Tyrosine and Formation of the Thyroid Hormones— “Organification” of Thyroglobulin:

The binding of iodine with the thyroglobulin molecule is called *organification* of the thyroglobulin. Oxidized iodine even in the molecular form will bind directly but very slowly with the amino acid tyrosine. In the thyroid cells, however, the oxidized iodine is associated with an *iodinase* enzyme that causes the process to occur within seconds or minutes. Therefore, almost as rapidly as the thyroglobulin molecule is released from the Golgi apparatus or as it is secreted through the apical cell membrane into the follicle, iodine binds with about one sixth of the tyrosine amino acids within the thyroglobulin molecule. Tyrosine is first iodized to *monoiodotyrosine* and then to *diiodotyrosine*. Then, during the next few minutes, hours, and even days, more and more of the iodotyrosine residues become *coupled* with one another. The major hormonal product of the coupling reaction is the molecule *thyroxine* that remains part of the thyroglobulin molecule. Or one molecule of monoiodotyrosine couples with one molecule of diiodotyrosine to form *triiodothyronine*, which represents about one fifteenth of the final hormones. (Guyton, A et al 2006)

2-2-6 Storage of Thyroglobulin:

The thyroid gland is unusual among the endocrine glands in its ability to store large amounts of hormone. After synthesis of the thyroid has run its course, each thyroglobulin molecule contains up to 30 thyroxine molecules and a few triiodothyronine molecules. In this form, the thyroid hormones are stored in the follicles in an amount sufficient to supply the body with its normal requirements of thyroid hormones for 2 to 3 months. Therefore, when synthesis of thyroid hormone ceases, the physiologic effects of deficiency are not observed for several months.

2-2-7 Release of Thyroxine and Triiodothyronine from the Thyroid Gland:-

Thyroglobulin itself is not released into the circulating blood in measurable amounts; instead, thyroxine and triiodothyronine must first be cleaved from the thyroglobulin molecule, and then these free hormones are released. This process occurs as follows: The apical surface of the thyroid cells sends out pseudopod extensions that close around small portions of the colloid to form *pinocytic vesicles* that enter the apex of the thyroid cell. Then *lysosomes* in the cell cytoplasm immediately fuse with these vesicles to form digestive vesicles containing digestive enzymes from the lysosomes mixed with the colloid. Multiple *proteases* among the enzymes digest the thyroglobulin molecules and release thyroxine and triiodothyronine in free form. These then diffuse through the base of the thyroid cell into the surrounding capillaries. Thus, the thyroid hormones are released into the blood. About three quarters of the iodinated tyrosine in the thyroglobulin never becomes thyroid hormones but remains monoiodotyrosine and diiodotyrosine. During the digestion of the thyroglobulin molecule to cause release of thyrbulin molecules. However, they are not secreted into the blood. Instead, their iodine is cleaved from them by a *deiodinase enzyme* that makes virtually all this iodine available again for recycling within the gland for forming additional thyroid hormones. In the congenital absence of this deiodinase enzyme, many persons become iodine-deficient because of failure of this recycling process. (Guyton, A et al 2006)

2-2-8 Daily Rate of Secretion of Thyroxine and Triiodothyronine:

About 93 per cent of the thyroid hormone released from the thyroid gland is normally thyroxine and only 7 per cent is triiodothyronine. However, during the ensuing few days, about one half of the thyroxine is slowly deiodinated to form additional triiodothyronine. Therefore, the hormone finally delivered

to and used by the tissues is mainly triiodothyronine, a total of about 35 micrograms of triiodothyronine per day.

2-2-9 Transport of Thyroxine and Triiodothyronine to Tissues:

2-2-9-1 Thyroxine and Triiodothyronine Are Bound to Plasma Proteins:

On entering the blood, over 99 per cent of the thyroxine and triiodothyronine combines immediately with several of the plasma proteins, all of which are synthesized by the liver. They combine mainly with *thyroxine-binding globulin* and much less so with *thyroxine-binding prealbumin* and *albumin*. (Guyton, A et al 2006)

2-2-9-2 Thyroxine and Triiodothyronine Are Released Slowly to Tissue Cells:

Because of high affinity of the plasma-binding proteins for the thyroid hormones, these substances in particular, thyroxine are released to the tissue cells slowly. Half the thyroxine in the blood is released to the tissue cells about every 6 days, whereas half the triiodothyronine because of its lower affinity is released to the cells in about 1 day. On entering the tissue cells, both thyroxine and triiodothyronine again bind with intracellular proteins, the thyroxine binding more strongly than the triiodothyronine. Therefore, they are again stored, but this time in the target cells themselves, and they are used slowly over a period of days or weeks. (Guyton, A et al 2006)

2-2-9-3 Thyroid Hormones Have Slow Onset and Long Duration of Action:

After injection of a large quantity of thyroxine into a human being, essentially no effect on the metabolic rate can be discerned for 2 to 3 days, thereby demonstrating that there is a *long latent* period before thyroxine activity begins. Once activity does begin, it increases progressively and reaches a maximum in 10 to 12 days. Thereafter, it decreases with a half-life of about 15 days. Some of the activity persists for as long as 6 weeks to 2 months.

The actions of triiodothyronine occur about four times as rapidly as those of thyroxine, with a latent period as short as 6 to 12 hours and maximal cellular activity occurring within 2 to 3 days. Most of the latency and prolonged period of action of these hormones are probably caused by their binding with proteins both in the plasma and in the tissue cells, followed by their slow release. However, we shall see in subsequent discussions that part of the latent period also results from the manner in which these hormones perform their functions in the cells themselves.

2-2-10 Functions of the Thyroid Hormones:-

Thyroid Hormones Increase the Transcription of Large Numbers of Genes:

The general effect of thyroid hormone is to activate nuclear transcription of large numbers of genes. Therefore, in virtually all cells of the body, great numbers of protein enzymes, structural proteins, transport proteins, and other substances are synthesized. The net result is generalized increase in functional activity throughout the body. Thyroid Hormones Activate Nuclear Receptors. The thyroid hormone receptors are either attached to the DNA genetic strands or located in proximity to them.

The thyroid hormone receptor usually forms a heterodimer with *retinoid X receptor (RXR)* at specific *thyroid hormone response* elements on the DNA. On binding with thyroid hormone, the receptors become activated and initiate the transcription process. Then large numbers of different types of messenger RNA are formed, followed within another few minutes or hours by RNA translation on the cytoplasmic ribosomes to form hundreds of new intracellular proteins. However, not all the proteins are increased by similar percentages some only slightly, and others at least as much as six fold. It is believed that most, if not all, of the actions of thyroid hormone result from the subsequent enzymatic and other functions of these new protein.

2-2-10-1 Thyroid Hormones Increase Cellular Metabolic Activity:

The thyroid hormones increase the metabolic activities of almost all the tissues of the body. The basal metabolic rate can increase to 60 to 100 per cent above normal when large quantities of the hormones are secreted.

The rate of utilization of foods for energy is greatly accelerated. Although the rate of protein synthesis is increased, at the same time the rate of protein catabolism is also increased. The growth rate of young people is greatly accelerated. The mental processes are excited, and the activities of most of the other endocrine glands are increased. (Guyton, A et al 2006)

2-2-10-2 Thyroid Hormones Increase the Number and Activity of Mitochondria:

When thyroxine or triiodothyronine is given to an animal, the mitochondria in most cells of the animal's body increase in size as well as number. Furthermore, the total membrane surface area of the mitochondria increases almost directly in proportion to the increased metabolic rate of the whole animal. Therefore, one of the principal functions of thyroxine might be simply to increase the number and activity of mitochondria, which in turn increases the rate of formation of adenosine triphosphate (ATP) to energize cellular function. However, the increase in the number and activity of mitochondria could be the *result* of increased activity of the cells as well as the cause of the increase. (Guyton, A et al 2006)

2-2-10-3 Thyroid Hormones Increase Active Transport of Ions Through Cell Membranes:

One of the enzymes that increases its activity in response to thyroid hormone is *Na⁺-K⁺ ATPase*. This in turn increases the rate of transport of both sodium and potassium ions through the cell membranes of some tissues. Because this process uses energy and increases the amount of heat produced in the body, it has been suggested that this might be one of the mechanisms by which thyroid hormone increases the body's metabolic rate. In fact,

thyroid hormone also causes the cell membranes of most cells to become leaky to sodium ions, which further activates the sodium pump and further increases heat production. (Guyton, A et al 2006)

2-2-11 Effect of Thyroid Hormone on Growth:

Thyroid hormone has both general and specific effects on growth. For instance, it has long been known that thyroid hormone is essential for the metamorphic change of the tadpole into the frog. In humans, the effect of thyroid hormone on growth is manifest mainly in growing children. In those who are hypothyroid, the rate of growth is greatly retarded. In those who are hyperthyroid, excessive skeletal growth often occurs, causing the child to become considerably taller at an earlier age. However, the bones also mature more rapidly and the epiphyses close at an early age, so that the duration of growth and the eventual height of the adult may actually be shortened. An important effect of thyroid hormone is to promote growth and development of the brain during fetal life and for the first few years of postnatal life. If the fetus does not secrete sufficient quantities of thyroid hormone, growth and maturation of the brain both before birth and afterward are greatly retarded, and the brain remains smaller than normal. Without specific thyroid therapy within days or weeks after birth, the child without a thyroid gland will remain mentally deficient throughout life. (Guyton, A et al 2006)

2-2-12 Effects of Thyroid Hormone on Specific Bodily Mechanisms:

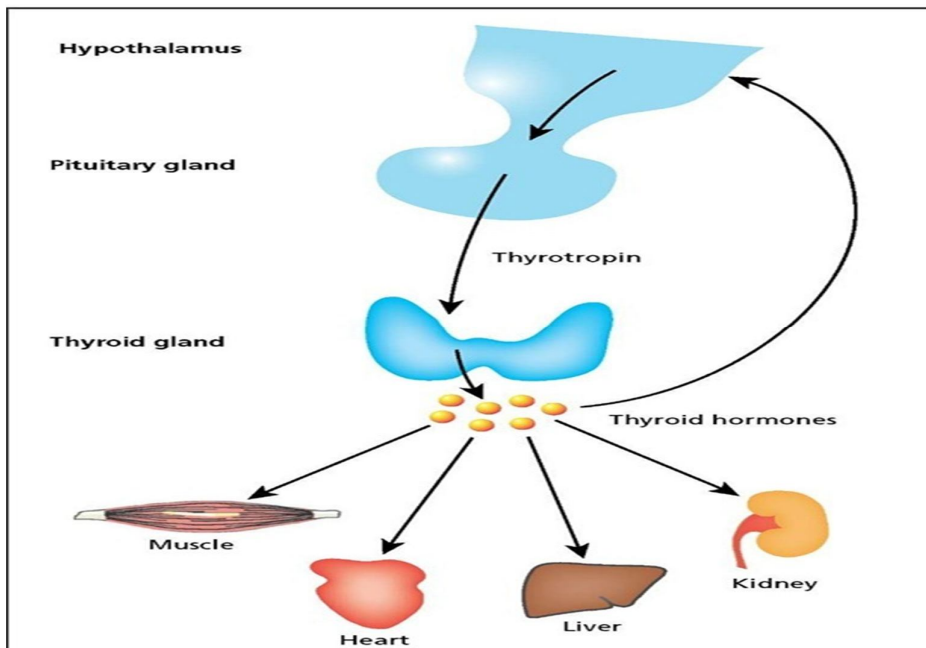
Stimulation of Carbohydrate Metabolism: Thyroid hormone stimulates almost all aspects of carbohydrate metabolism, including rapid uptake of glucose by the cells, enhanced glycolysis, enhanced gluconeogenesis, increased rate of absorption from the gastrointestinal tract, and even increased insulin secretion with its resultant secondary effects on carbohydrate metabolism. All these effects probably result from the overall increase in cellular metabolic enzymes caused by thyroid hormone. (Guyton, A et al 2006)

2-2-12-1 Stimulation of Fat Metabolism:

Essentially all aspects of fat metabolism are also enhanced under the influence of thyroid hormone. In particular, lipids are mobilized rapidly from the fat tissue, which decreases the fat stores of the body to a greater extent than almost any other tissue element. This also increases the free fatty acid concentration in the plasma and greatly accelerates the oxidation of free fatty acids by the cells

Effect on Plasma and Liver Fats:

Increased thyroid hormone *decreases* the concentrations of cholesterol, phospholipids, and triglycerides in the plasma, even though it *increases* the free fatty acids. Conversely, *decreased* thyroid secretion greatly *increases* the plasma concentrations of cholesterol, phospholipids, and triglycerides and almost always causes excessive deposition of fat in the liver as well. The large increase in circulating plasma cholesterol in prolonged hypothyroidism is often associated with severe atherosclerosis. (Guyton, A et al 2006)



Effects of Thyroid Hormone (2-7)

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One of the mechanisms by which thyroid hormone decreases the plasma cholesterol concentration is to increase significantly the rate of cholesterol secretion in the bile and consequent loss in the feces. A possible mechanism for the increased cholesterol secretion is that thyroid hormone induces increased numbers of low-density lipoprotein receptors on the liver cells, leading to rapid removal of low-density lipoproteins from the plasma by the liver and subsequent secretion of cholesterol in these lipoproteins by the liver cells. (Guyton, A et al 2006)

2-2-12-2 Increased Requirement for Vitamins:

Because thyroid hormone increases the quantities of many bodily enzymes and because vitamins are essential parts of some of the enzymes or coenzymes, thyroid hormone causes increased need for vitamins. Therefore, a relative vitamin deficiency can occur when excess thyroid hormone is secreted, unless at the same time increased quantities of vitamins are made available.

2-2-12-3 Increased Basal Metabolic Rate:

Because thyroid hormone increases metabolism in almost all cells of the body, excessive quantities of the hormone can occasionally increase the basal metabolic rate 60 to 100 per cent above normal. Conversely, when no thyroid hormone is produced, the basal metabolic rate falls almost to one-half normal. . Extreme amounts of the hormones are required to cause very high basal metabolic rates. (Guyton, A et al 2006)

2-2-12-4 Decreased Body Weight:

Greatly increased thyroid hormone almost always decreases the body weight and greatly decreased hormone almost always

2-2-12-5 increases the body weight:

These effects do not always occur, because thyroid hormone also increases the appetite, and this may counterbalance the change in the metabolic rate. (Guyton, A et al 2006)

2-2-13 Effect of Thyroid Hormones on the Cardiovascular System:

2-2-13-1 Increased Blood Flow and Cardiac Output:

Increase metabolism in the tissues causes more rapid utilization of oxygen than normal and release of greater than normal quantities of metabolic end products from the tissues. These effects cause vasodilation in most body tissues, thus increasing blood flow. The rate of blood flow in the skin especially increases because of the increased need for heat elimination from the body. As a consequence of the increased blood flow, cardiac output also increases, sometimes rising to 60 per cent or more above normal when excessive thyroid hormone is present and falling to only 50 per cent of normal in very severe hypothyroidism. (Guyton, A et al 2006)

2-2-13-2 Increased Heart Rate:

The heart rate increases considerably more under the influence of thyroid hormone than would be expected from the increase in cardiac output. Therefore, thyroid hormone seems to have a direct effect on the excitability of the heart, which in turn increases the heart rate. This effect is of particular importance because the heart rate is one of the sensitive physical signs that the clinician uses in determining whether a patient has excessive or diminished thyroid hormone production. (Guyton, A et al 2006)

2-2-13-3 Increased Heart Strength:

The increased enzymatic activity caused by increased thyroid hormone production apparently increases the strength of the heart when only a slight excess of thyroid hormone is secreted. This is analogous to the increase in heart strength that occurs in mild fevers and during exercise. However, when thyroid hormone is increased markedly, the heart muscle strength becomes depressed because of long-term excessive protein catabolism. Indeed, some severely thyrotoxic patients die of cardiac decompensation secondary to myocardial failure and to increased cardiac load imposed by the increase in cardiac output. (Guyton, A et al 2006)

2-2-13-4 Normal Arterial Pressure:

The *mean* arterial pressure usually remains about normal after administration of thyroid hormone. Because of increased blood flow through the tissues between heartbeats, the pulse pressure is often increased, with the systolic pressure elevated in hyperthyroidism 10 to 15 mm Hg and the diastolic pressure reduced a corresponding amount. (Guyton, A et al 2006)

2-2-14 Increased Respiration:

The increased rate of metabolism increases the utilization of oxygen and formation of carbon dioxide; these effects activate all the mechanisms that increase the rate and depth of respiration.

2-2-15 Increased Gastrointestinal Motility:

. In addition to increased appetite and food intake thyroid hormone increases both the rates of secretion of the digestive juices and the motility of the gastrointestinal tract. Hyperthyroidism often results in diarrhea. Lack of thyroid hormone can cause constipation

2-2-15 Excitatory Effects on the Central Nervous System:

In general, thyroid hormone increases the rapidity of cerebation but also often dissociates this; conversely, lack of thyroid hormone decreases this function. The hyperthyroid individual is likely to have extreme nervousness and many psychoneurotic tendencies, such as anxiety complexes, extreme worry, and paranoia.

Effect on the Function of the Muscles.

Slight increase in thyroid hormone usually makes the muscles react with vigor, but when the quantity of hormone becomes excessive, the muscles become weakened because of excess protein catabolism. Conversely, lack of thyroid hormone causes the muscles to become sluggish, and they relax slowly after a contraction.

2-2-15-1 Muscle Tremor:

One of the most characteristic signs of hyperthyroidism is a fine muscle tremor. This is not the coarse tremor that occurs in Parkinson's disease or in shivering, because it occurs at the rapid frequency of 10 to 15 times per second. The tremor can be observed easily by placing a sheet of paper on the extended fingers and noting the degree of vibration of the paper.

This tremor is believed to be caused by increased reactivity of the neuronal synapses in the areas of the spinal cord that control muscle tone. The tremor is an important means for assessing the degree of thyroid hormone effect on the central nervous system. (Guyton, A et al 2006)

2-2-15-2 Effect on Sleep:

Because of the exhausting effect of thyroid hormone on the musculature and on the central nervous system, the hyperthyroid subject often has a feeling of constant tiredness, but because of the excitable effects of thyroid hormone on the synapses, it is difficult to sleep. Conversely, extreme somnolence is characteristic of hypothyroidism, with sleep sometimes lasting 12 to 14 hours a day.

2-2-16 Effect on Other Endocrine Glands:

Increased thyroid hormone increases the rates of secretion of most other endocrine glands, but it also increases the need of the tissues for the hormones. For instance, increased thyroxine secretion increases the rate of glucose metabolism everywhere in the body and therefore causes a corresponding need for increased insulin secretion by the pancreas. Also, thyroid hormone increases many metabolic activities related to bone formation and, as a consequence, increases the need for parathyroid hormone.

Thyroid hormone also increases the rate at which adrenal glucocorticoids are inactivated by the liver. This leads to feedback increase in adrenocorticotrophic hormone production by the anterior pituitary and,

therefore, increased rate of glucocorticoid secretion by the adrenal glands.
.(Guyton, A et al 2006)

2-2-17 Effect of Thyroid Hormone on Sexual Function:

For normal sexual function, thyroid secretion needs to be approximately normal. In men, lack of thyroid hormone is likely to cause loss of libido; great excesses of the hormone, however, sometimes cause impotence. In women, lack of thyroid hormone often causes *menorrhagia* and *polymenorrhea*— that is, respectively, excessive and frequent menstrual bleeding. .(Guyton, A et al 2006)

Yet, strangely enough, in other women thyroid lack may cause irregular periods and occasionally even *amenorrhea*. A hypothyroid woman, like a man, is likely to have greatly decreased libido. To make the picture still more confusing, in the hyperthyroid woman, *oligomenorrhea*, which means greatly reduced bleeding, is common, and occasionally amenorrhea results. The action of thyroid hormone on the gonads cannot be pinpointed to a specific function but probably results from a combination of direct metabolic effects on the gonads as well as excitatory and inhibitory feedback effects operating through the anterior pituitary hormones that control the sexual functions

2-2-18. Regulation of Thyroid Hormone Secretion:

To maintain normal levels of metabolic activity in the body, precisely the right amount of thyroid hormone must be secreted at all times; to achieve this, specific feedback mechanisms operate through the hypothalamus and anterior pituitary gland to control the rate of thyroid secretion. These mechanisms are as follows:-

TSH (from the Anterior Pituitary Gland) Increases Thyroid Secretion. TSH, also known as *thyrotropin*, is an anterior pituitary hormone, a glycoprotein with a molecular weight of about 28,000. This hormone, increases the

secretion of thyroxine and triiodothyronine by the thyroid gland. Its specific effects on the thyroid gland are as follows:

1. *Increased proteolysis of the thyroglobulin* that has already been stored in the follicles, with resultant release of the thyroid hormones into the circulating blood and diminishment of the follicular substance itself
2. Increased activity of the iodide pump, which increases the rate of “iodide trapping” in the glandular cells, sometimes increasing the ratio of intracellular to extracellular iodide concentration in the glandular substance to as much as eight times normal.
3. Increased iodination of tyrosine to form the thyroid hormones
4. Increased size and increased secretory activity of the thyroid cells
5. Increased number of thyroid cells plus a change from cuboidal to columnar cells and much infolding of the thyroid epithelium into the follicles

In summary, TSH increases all the known secretory activities of the thyroid glandular cells. The most important early effect after administration of TSH is to initiate proteolysis of the thyroglobulin, which causes release of thyroxine and triiodothyronine into the blood within 30 minutes. The other effects require hours or even days and weeks to develop fully.

2-2-19 Cyclic Adenosine Monophosphate Mediates the Stimulatory Effect of TSH:

In the past, it was difficult to explain the many and varied effects of TSH on the thyroid cell. It is now clear that most, if not all, of these effects result from activation of the “second messenger” cyclic adenosine monophosphate (cAMP) system of the cell.

The first event in this activation is binding of TSH with specific TSH receptors on the basal membrane surfaces of the thyroid cell. This then activates adenylyl cyclase in the membrane, which increases the formation of cAMP inside the cell. Finally, the cAMP acts as a second messenger to activate protein kinase, which causes multiple phosphorylations throughout

the cell. The result is both an immediate increase in secretion of thyroid hormones and prolonged growth of the thyroid glandular tissue itself. This method for control of thyroid cell activity is similar to the function of cAMP as a “second messenger” in many other target tissues of the body.

Anterior Pituitary Secretion of TSH Is Regulated by Thyrotropin-Releasing Hormone from the Hypothalamus:

Anterior pituitary secretion of TSH is controlled by a hypothalamic hormone, thyrotropin-releasing hormone (TRH), which is secreted by nerve endings in the median eminence of the hypothalamus. From the median eminence, the TRH is then transported to the anterior pituitary by way of the hypothalamichypophysial portal blood, as explained in. TRH has been obtained in pure form. It is a simple substance, a tripeptide amide—pyroglutamyl-histidylproline- amide. TRH directly affects the anterior pituitary gland cells to increase their output of TSH. When the blood portal system from the hypothalamus to the anterior pituitary gland becomes blocked, the rate of secretion of TSH by the anterior pituitary decreases greatly but is not reduced to zero. The molecular mechanism by which TRH causes the TSH-secreting cells of the anterior pituitary to produce TSH is first to bind with TRH receptors in the pituitary cell membrane. This in turn *activates the phospholipase second messenger system* inside the pituitary cells to produce large amounts of phospholipase C, followed by a cascade of other second messengers, including calcium ions and diacyl glycerol, which eventually leads to TSH release. .(Guyton, A et al 2006)

2-2-20 Effects of Cold and Other Neurogenic Stimuli on TRH and TSH Secretion:

One of the best-known stimuli for increasing the rate of TRH secretion by the hypothalamus and therefore TSH secretion by the anterior pituitary gland, is exposure of an animal to cold. This effect almost certainly results from excitation of the hypothalamic centers for body temperature control.

Exposure of rats for several weeks to severe cold increases the output of thyroid hormones sometimes to more than 100 per cent of normal and can increase the basal metabolic rate as much as 50 per cent. Indeed, persons moving to arctic regions have been known to develop basal metabolic rates 15 to 20 percent above normal. Various emotional reactions can also affect the output of TRH and TSH and therefore indirectly affect the secretion of thyroid hormones. Excitement and anxiety conditions that greatly stimulate the sympathetic nervous system cause an acute decrease in secretion of TSH, perhaps because these states increase the metabolic rate and body heat and therefore exert an inverse effect on the heat control center. Neither these emotional effects nor the effect of cold is observed after the hypophysial stalk has been cut, demonstrating that both of these effects are mediated by way of the hypothalamus. (Guyton, A et al 2006)

2-2-21 Feedback Effect of Thyroid Hormone to Decrease Anterior Pituitary Secretion of TSH:

Increased thyroid hormone in the body fluids decreases secretion of TSH by the anterior pituitary. When the rate of thyroid hormone secretion rises to about 1.75 times normal, the rate of TSH secretion falls essentially to zero. Almost all this feedback depressant effect occurs even when the anterior pituitary has been separated from the hypothalamus. Therefore increased thyroid hormone inhibits anterior pituitary secretion of TSH mainly by a direct effect on the anterior pituitary gland itself. Regardless of the mechanism of the feedback, its effect is to maintain an almost constant concentration of free thyroid hormones in the circulating body fluids

2-2-22 . Antithyroid Substances:

Drugs that suppress thyroid secretion are called antithyroid substances. The best known of these substances are *thiocyanate*, *propylthiouracil*, and high concentrations of *inorganic iodides*. The mechanism by which each of these

blocks thyroid secretion is different from the others, and they can be explained as follows.

2-2-22-1 Thiocyanate Ions Decrease Iodide Trapping:

The same active pump that transports iodide ions into the thyroid cells can also pump thiocyanate ions, perchlorate ions, and nitrate ions. Therefore, the administration of thiocyanate (or one of the other ions as well) in high enough concentration can cause competitive inhibition of iodide transport into the cell— that is, inhibition of the iodide-trapping mechanism. The decreased availability of iodide in the glandular cells does not stop the formation of thyroglobulin; it merely prevents the thyroglobulin that is formed from becoming iodinated and therefore from forming the thyroid hormones. This deficiency of the thyroid hormones in turn leads to increased secretion of TSH by the anterior pituitary gland, which causes overgrowth of the thyroid gland even though the gland still does not form adequate quantities of thyroid hormones. Therefore, the use of thiocyanates and some other ions to block thyroid secretion can lead to development of a greatly enlarged thyroid gland, which is called a *goiter*.

Propylthiouracil Decreases Thyroid Hormone Formation. Propylthiouracil (and other, similar compounds, such as methimazole and carbimazole) prevents formation of thyroid hormone from iodides and tyrosine. The mechanism of this is partly to block the peroxidase enzyme that is required for iodination of tyrosine and partly to block the coupling of two iodinated tyrosines to form thyroxine or triiodothyronine. Propylthiouracil, like thiocyanate, does not prevent formation of thyroglobulin. The absence of thyroxine and triiodothyronine in the thyroglobulin can lead to tremendous feedback enhancement of TSH secretion by the anterior pituitary gland, thus promoting growth of the glandular tissue and forming a goiter.

Iodides in High Concentrations Decrease Thyroid Activity and Thyroid Gland Size:

When iodides are present in the blood in high concentration (100 times the normal plasma level), most activities of the thyroid gland are decreased, but often they remain decreased for only a few weeks. The effect is to reduce the rate of iodide trapping, so that the rate of iodination of tyrosine to form thyroid hormones is also decreased. Even more important, the normal endocytosis of colloid from the follicles by the thyroid glandular cells is paralyzed by the high iodide concentrations. Because this is the first step in release of the thyroid hormones from the storage colloid, there is almost immediate shutdown of thyroid hormone secretion into the blood. Because iodides in high concentrations decrease all phases of thyroid activity, they slightly decrease the size of the thyroid gland and especially decrease its blood supply, in contradistinction to the opposite effects caused by most of the other antithyroid agents. For this reason, iodides are frequently administered to patients for 2 to 3 weeks before surgical removal of the thyroid gland to decrease the necessary amount of surgery, especially to decrease the amount of bleeding. (Guyton, A et al 2006)

2-3 pathology of the thyroid gland:

Patients with thyroid disease may present with general enlargement of the gland (goitre), with a single nodule, or with evidence of thyroid hormone excess or lack. The latter presentations cause well-recognized syndrome

2-3-1 Hyperthyroidism:

Most effects of hyperthyroidism are obvious from the preceding discussion of the various physiologic effects of thyroid hormone. Often known as thyrotoxicosis, the symptoms and signs are related to a general increase in metabolic activity. It is usually due to autoimmune thyroid disease (Graves' disease) Patients are hyperkinetic, show emotional liability and complain of

heat intolerance, excessive sweating and weight loss despite a good appetite. There is tachycardia, increased cardiac output and palpitation. . Older patients may have atrial fibrillation and cardiac failure. Some of these effects are direct, while others – such as the characteristic eyelid retraction – are due to increased sensitivity to sympathetic stimulation; the levator palpebrae superioris muscle has sympathetic innervation. In some patients with Graves' disease there is proptosis; this is protuberance of the eyeball due to inflammatory infiltration of the extraocular tissues of the orbit (Levison, A. David et al 2008)

2-3-1-1. Causes of hyperthyroidism:

- Graves' disease – 80%
- Toxic nodular goitre – 10%
- Thyroid adenoma – 5–10%
- Early Hashimoto's thyroiditis
- TSH-secreting pituitary adenoma
- Ingestion of thyroid hormones

2-3-2 Hypothyroidism:

In adults, this condition is called myxoedema, and the symptoms depend on the severity of hormone deficiency which causes a reduction in general metabolic activity. There is weight gain and general lethargy with cold intolerance. Skin and hair are dry and accumulation of mucopolysaccharides in connective tissue result in a thickening of the skin, hoarseness, and pain and paraesthesia when nerves are trapped. There is intellectual impairment. Change of mood may progress to psychosis. In severe deficiency, hypothermia and coma can develop. Raised blood cholesterol levels increase the risk of cardiovascular disease. Cretinism is due to severe hypothyroidism in infancy.

Thyroid hormones are critical for normal brain development, and these children show signs of mental retardation, neuromuscular abnormalities, deaf-mutism and retarded growth. There is a goiter when it is caused by severe iodine deficiency, or by inherited defects of the enzymes involved in thyroid hormone synthesis. Rarely, thyroid agenesis or hypoplasia occurs, and in these cases goiter is absent. It is extremely important to make an early diagnosis because hormone replacement permits normal development. (Levison, A. David et al 2008)

2-3-2-1 Causes of hypothyroidism:-

- Autoimmune thyroid disease
- Hashimoto's thyroiditis primary myxoedema
- Severe iodine deficiency
- Dyshormonogenesis
- Following thyroid surgery or radio-iodine therapy
- Ingestion of goitrogens
- Hypopituitarism

2-3-3 Functional Disorders: Non-toxic Nodular Goiter:

Non-toxic nodular goiter is the most common lesion in thyroid pathology. When there is absolute or relative iodine deficiency, reduced levels of thyroid hormones result in increased TSH secretion by the pituitary. This induces hyperplasia in an attempt to increase thyroid hormone output. The demands are usually intermittent, and the gland undergoes cycles of growth and involution, resulting in the well-recognized picture of multinodular goiter, with nodules consisting of follicles of varying size, fibrosis, haemorrhage and focal inflammation. Enlargement is usually asymmetrical, and the gland may weigh up to several hundred grams occasionally, simple goiter may produce signs or symptoms suggestive of tumor. When one nodule is larger than the others (dominant nodule), fine needle aspiration cytology or even partial thyroidectomy is required to distinguish the two.

Occasionally, there may be compression of trachea, oesophagus or recurrent laryngeal nerve. On an epidemiological basis, two forms are defined:-

- Endemic goiter affects more than 10% of the population, occurring in areas with absolute deficiency of iodine, usually far from the sea, reflecting seafood as the major source of iodine. These areas include the Andes, Himalayas and Alps. The introduction of iodized salt has reduced the incidence. Goiter usually develops in childhood, and the sexes are equally affected.

- Sporadic goiter is due to a relative lack of iodine in individuals. It reflects inadequate intake; inherited abnormalities in thyroid hormone production; and ingestion of goitrogens, i.e. substances that interfere with hormone

. These include vegetables of the *Brassica* family, excessive fluoride, or drugs such as *p*-aminosalicylic acid and sulphonylureas. Some people also suggest that autoimmune mechanisms may be involved (Levison, A. David et al 2008)

2-3-4. Autoimmune Thyroid Disease:

Autoimmune thyroid disease is associated with antibodies to thyroid antigens. The clinical picture varies according to the antibodies produced. Women are more commonly affected than men.

This group of diseases Graves' disease, Hashimoto's thyroiditis, and primary myxoedema are characterized by lymphoid infiltration of the gland and by the presence of circulating antibodies to various components of thyroid follicular cells some of which are thought to play an active role in pathogenesis. Thyroid-stimulating immunoglobulins (TSI) bind to and activate the TSH receptor, causing the increased secretion of thyroid hormones usually seen in Graves' disease. Other antibodies are thought to stimulate growth, and may be important in goitrogenesis in Hashimoto's thyroiditis. Receptor-blocking antibodies may contribute to hypothyroidism and to thyroid atrophy in primary myxoedema. These diseases may be

familial, and are associated with other organ-specific autoimmune diseases. (Levison, A. David et al 2008)

Familial associations of thyroid autoimmune disease:

- Addison's disease
- Pernicious anaemia
- Type 1 diabetes mellitus
- Graves' Disease

This is characterized by a diffuse goitre and hyperthyroidism. It mainly affects women aged 20 to 40 years. The gland is diffusely hyperplastic and hyperaemic, clinically resulting in a bruit on auscultation. In the untreated case, the thyroid epithelium is hyperplastic and there is little colloid storage. Lymphocytic infiltration is usually less marked than in Hashimoto's thyroiditis. It is unusual now to see the classical histological features of the disease because of effective drug therapy. A minority of patients relapse and come to surgery, but are usually euthyroid at the time of operation because of treatment with antithyroid drugs, with or without the addition of Iodine Their thyroids show complex histological features, antithyroid drugs inducing more marked follicular hyperplasia, while iodine reduces vascularity and increases colloid storage. This emphasizes the importance of a full clinical history in the interpretation of the histological appearances in endocrine disease. (Levison, A. David et al 2008)

2-3-5 Hashimoto's Thyroiditis:-

This is a disease of middle-aged women, in whom it occurs 20 times more commonly than in men. There is a diffuse, firm, painless goiter initially, the patient is usually euthyroid, but 80% become hypothyroid. The occasional patient is hyperthyroid at presentation, presumably due to the presence of TSI. High-titer antiperoxidase antibody is usually present. The gland is widely infiltrated and replaced by lymphocytes, plasma cells and

macrophages, often with germinal center formation. The thyroid follicular cells are enlarged with eosinophilic granular cytoplasm due to accumulation of mitochondria (Askanazy or Hürthle cell change). (Levison, A. David et al 2008)

2-3-6 Primary Myxoedema:

This disease affects mainly elderly women. The thyroid is atrophic, largely replaced by fibrous tissue with a lymphoid infiltrate. The patients are severely hypothyroid and are the most likely to present with hypothermia and coma. Focal chronic thyroiditis is seen in 15–20% of autopsies from patients with no clinical evidence of thyroid disease. This may represent subclinical autoimmune disease, as the incidence is similar to that of thyroid autoantibodies in the general population. Lymphocytic thyroiditis occurs in children and young adults who present with goitre and sometimes hyperthyroidism. It may be a precursor of Hashimoto's disease.

Other Forms of Thyroiditis:

- Acute thyroiditis may occasionally develop in bacteraemia or by local extension of inflammation.
- Giant cell (de Quervain's) thyroiditis, also known as *subacute thyroiditis*, presents as a painful goitre. It is probably viral in origin, and there are often preceding general or upper respiratory signs and symptoms. Women are affected three times as often as men. Hyperthyroidism and the presence of thyroid antibodies are usually transient. Although there is an initial acute inflammation, followed by a granulomatous response, the whole process may resolve. Even if some fibrosis persists, there are no long-term functional effects.
- Riedel's thyroiditis. In this very rare disease the thyroid is replaced by dense fibrous tissue, which often extends into perithyroidal tissues, mimicking invasive carcinoma. It may present as a goitre or with symptoms

related to involvement of trachea or recurrent laryngeal nerve. The aetiology is unknown, but some patients also have retroperitoneal or mediastinal fibrosis (Levison, A. David et al 2008)

2-3-7 Thyroid Tumors:

2-3-7-1 Thyroid tumors include:-

- follicular adenoma
- follicular carcinoma
- papillary carcinoma
- medullary carcinoma
- anaplastic carcinoma
- lymphoma.

Thyroid tumors usually present as solitary nodules, and most are ‘cold’ on scanning, as they concentrate radioactive iodine less actively than the surrounding gland. Some 70% of clinically apparent solitary nodules are, however, dominant nodules in a multinodular goiter; the rest are tumors (mostly benign). Thyroid cancer is rare, and accounts for less than 1% of all cancers and less than 0.5% of deaths from cancer. Cold nodules in men and in younger women should be regarded as more suspicious than those in middle-aged women. Most tumors arise from follicular cells, the majority being follicular adenomas. There are two types of carcinomas—follicular and papillary – which develop and behave differently. The C cells give rise to medullary carcinoma. (Levison, A. David et al 2008)

2-3-7-2 Follicular Adenoma:

These are the most common thyroid neoplasms, presenting most frequently in women aged over 30 years. They are usually non-functional, but may occasionally secrete excess thyroid hormones. They are generally encapsulated and compress the surrounding gland. Haemorrhage, degeneration and fibrosis may occur. The lesions show a variety of

histological appearances, but these have no clinical importance. It can sometimes be difficult for a pathologist to distinguish between an adenoma and a hyperplastic (adenomatoid) nodule in a non-toxic goitre, but again this has no clinical significance, (Levison A. David et al 2008)

2-3-7-3 Follicular Carcinoma:-

These comprise 15–20% of all thyroid cancers, but they are more common in areas of iodine deficiency. Their peak incidence is in the fifth decade of life, with one-third of cases occurring over the age of 50 years. These lesions are also more common in women. They metastasize by the vascular route, particularly to bone and lung. The overall survival is reported at about 50%. Two variants are recognized: encapsulated, so-called ‘minimally invasive’ carcinoma, where vascular and capsular invasion is seen only on microscopic examination and ‘widely invasive’ tumors, where obvious spread is seen throughout the gland or beyond. The latter have a poorer prognosis than the former. (Levison, A. David et al 2008)

2-3-7-4 Papillary Carcinoma:

All papillary tumours of the thyroid are regarded as malignant. They account for 60–70% of all thyroid cancers, occur in young adults (aged 30–40 years), and are three times more common in women than in men. They have good prognosis overall, with a 5-year survival approaching 90%. They are not usually encapsulated, and may be multifocal. For this reason, some surgeons perform total thyroidectomy. Some 40% of patients have metastases in local lymph nodes at presentation, but this does not seem to alter the prognosis. The patient may first present with an enlarged lymph node if the thyroid primary is very small. In the small minority where the tumour has spread through the thyroid capsule or there are distant metastases, the prognosis is worse. Some have fibrosis, and psammoma bodies are present in about half. There are characteristic cytological features, with clear or grooved nuclei.

Some tumours with these nuclear features have mixed papillary and follicular, or only follicular, architecture (the follicular variant of papillary carcinoma). These behave in the same way as the classical papillary tumours. Some less common variants (e.g. tall cell variant) exhibit more aggressive behavior. (Levison, A. David et al 2008)

2-3-7-5 Medullary Carcinoma:

These account for 5–10% of thyroid cancers. Between 10% and 20% are familial, forming part of the MEN2 syndrome

2-3-7-6 Other Tumours:

Occasionally in older women, a rapidly growing anaplastic carcinoma occurs. These are highly malignant and usually present as a rapidly growing mass. They probably represent progression of a pre-existing follicular or papillary tumour. Benign and malignant tumours may rarely arise from the connective tissue or vascular components (e.g. haemangiomas). Occasional metastases are seen, usually from the lung, breast or gastrointestinal tract. In 1–2% of patients with Hashimoto's thyroiditis, B-cell lymphoma develops. This may also present as a rapidly expanding mass. Differentiation from anaplastic carcinoma can be made using fine needle aspiration cytology.

2-3-8 Aetiology:

The molecular genetic pathways involved differ in follicular and papillary tumours. Mutations in *ras* oncogenes play a role in some follicular tumours, and are said to be associated with metastases. Iodine deficiency seems to favour *ras* mutation.

- Congenital conditions of the thyroid gland include **aplasia** of one lobe or the whole gland, varying degrees of **hypoplasia**, and **ectopia**

(Levison, A. David et al 2008)

2-4 Sonographic of thyroid gland:

Normal thyroid parenchyma has a homogeneous, medium-level to high-level echogenicity that makes detection of focal cystic or hypoechoic thyroid lesions relatively easy in most cases. The thin, hyperechoic line around the thyroid lobes is the capsule, which is often identifiable on ultrasound.

2-4-1 Nodular Thyroid Disease

2-4-1-1 Hyperplasia and Goiter:

. Pathologically, they are often referred to as hyperplastic, adenomatous, or colloid nodules. Many (if not all) cystic thyroid lesions are hyperplastic nodules that have undergone extensive liquefactive degeneration. Sonographically, most hyperplastic or adenomatous nodules are isoechoic compared to normal thyroid tissue but may become hyperechoic because of the numerous interfaces between cells and colloid substance. Less frequently, a hypoechoic spongelike or honeycomb pattern is seen. When the nodule is isoechoic or hyperechoic, a thin peripheral hypoechoic halo is typically seen, most likely caused by perinodular blood vessels and mild edema or compression of the adjacent normal parenchyma. The degenerative changes of goitrous nodules correspond to their sonographic appearances: purely anechoic areas are caused by serous or colloid fluid. Echogenic fluid or moving fluid-fluid levels correspond to hemorrhage. Bright echogenic foci with comet-tail artifacts are likely caused by microcrystals or aggregates of colloid substance, which may also move slowly, like snowflakes, within the fluid collection. Thin, intracystic septations probably correspond to attenuated strands of thyroid tissue and appear completely avascular on color Doppler ultrasound. These degenerative processes may also lead to the formation of calcifications, which may be either thin, peripheral shells (“eggshell”) or coarse, highly reflective foci with associated acoustic shadows, scattered throughout the gland.

2-4-2 Adenoma Sonographically:

Adenomas are usually solid masses that may be hyperechoic, isoechoic, or hypoechoic. They often have a thick, smooth peripheral hypoechoic halo resulting from the fibrous capsule and blood vessels, which can be readily seen by color Doppler imaging. Often, vessels pass from the periphery to the central regions of the nodule, sometimes creating a “spoke and wheel” appearance.

2-4-3 Carcinoma:-

- Hypoechoogenicity (90% of cases), resulting from closely packed cell content, with minimal colloid substance.
- Microcalcifications, appearing as tiny, punctuate hyperechoic foci, either with or without acoustic shadows in rare, but usually aggressive cases of papillary carcinomas of childhood, microcalcifications may be the only sonographic sign of the neoplasm, even without evidence of a nodular lesion
- Hypervascularity (90% of cases), with disorganized vascularity, mostly in well-encapsulated forms
- Cervical lymph node metastases, which may contain tiny, punctate echogenic foci caused by microcalcifications

The sonographic appearance of follicular carcinoma: Irregular tumor margins thick, irregular halo Tortuous or chaotic arrangement of internal blood Vessels.

The sonographic appearance of medullary carcinoma: is usually similar to that of papillary carcinoma and is seen most often as a hypoechoic solid mass. Calcifications are often seen (histologically caused by calcified nests of amyloid substance) and tend to be more coarse than the calcifications of typical papillary carcinoma Calcifications can be seen not only in the primary tumor but also in lymph node metastases and even in hepatic metastases.

The sonographic appearance of anaplastic carcinoma: Large, hypoechoic mass .Encase or invade blood vessels .Invade neck muscles. Sonographically, lymphoma of the thyroid appears as an extremely hypoechoic and lobulated mass. Large areas of cystic necrosis may occur, as well as encasement of adjacent neck vessels.

Metastases usually are from melanoma (39%), breast (21%), and renal cell (10%) carcinoma. Metastases may appear as solitary, well-circumscribed nodules or as diffuse involvement of the gland. On sonography, thyroid tumors are solid, homogeneously hypoechoic masses, without calcifications. (Rumack. Carol et al.2011)

2-4-4 Diffuse Thyroid Disease:

Several thyroid diseases are characterized by diffuse rather than focal involvement. This usually results in generalized enlargement of the gland (goiter) and no palpable nodules. Specific conditions that produce such diffuse enlargement include chronic autoimmune lymphocytic thyroiditis (Hashimoto's thyroiditis), colloid or adenomatous goiter, and Graves' disease. Acute suppurative thyroiditis, on ultrasound images, an abscess is seen as a poorly defined, hypoechoic heterogeneous mass with internal debris, with or without septa and gas. Adjacent inflammatory nodes are often present.

Subacute granulomatous thyroiditis or de Quervain's disease: Sonographically, the gland may appear enlarged and hypoechoic, with normal or decreased vascularity caused by diffuse edema of the gland, or the process may appear as focal hypoechoic regions.

Hashimoto's thyroiditis is diffuse, coarsened, parenchymal echotexture, generally more hypoechoic than a normal thyroid in most cases the gland is enlarged. Multiple, discrete hypoechoic micronodules from 1 to 6 mm in diameter are strongly suggestive of chronic thyroiditis; this appearance has been called micronodulation Painless (silent) thyroiditis has the typical

histologic and sonographic pattern of chronic autoimmune thyroiditis (hypoechoogenicity, micronodulation, and fibrosis), Graves' disease is a common diffuse abnormality of the thyroid gland and is usually biochemically characterized by hyperfunction (thyrotoxicosis). The echotexture may be more inhomogeneous than in diffuse goiter, mainly because of numerous large, intraparenchymal vessels. Further, especially in young patients, the parenchyma may be diffusely hypoechoic because of the extensive lymphocytic infiltration or the predominantly cellular content of the parenchyma, which becomes almost devoid of colloid substance. Color Doppler sonography often demonstrates a hypervascular pattern referred to as the thyroid inferno (Rumack. Carol et al.2011)

2-5 previous studies:

study of Early detection of thyroid gland disorder for students in the faculty of applied medical sciences done by Ibrahim _et al (2015) university department diagnostic radiology in Jeddah Saudi Arabia among total number of 70 student 26% was found with abnormal of ultrasound findings 17% of them with cystic nodules while solid and mixed nodules 4% for each (Ibrahim _et al 2015)

study of Role of ultrasound in thyroid pathologies done by Dr Awad Mohamed.E 2013 To thyroid patients for ultrasound (2007-2009) at different hospitals and dispensaries in the kingdom of Saudi Arabia (eastern area) This study was carried out on 303 patients of various types of thyroid disease in Saudi Arabia 232 (77%) were female patients and 71 (23%) males .along this series there were 25% of enlarged thyroid which act as most common ultrasound findings of thyroid disorders followed by 23% solitary thyroid nodules. 10% multinodular goiter 8% simple cyst. 7% nonpalpable thyroid nodule .4% goiter and hashimoto thyroiditis. the most common ultrasound findings the enlargement of gland .the least common is nodal enlargement .adenoma thyroiditis microcalcification small size thyroid multiple thyroid nodules .

study of early detection of thyroid disorders using ultrasonography

Done by Salma .M (2016) study was conducted cases were 65% female and 34.5% male aged (16-75 years) has been selected random samples do not suffer from any disease of thyroid gland 67.3% were normal 18.2% had cystic nodules 14.5% solid nodules and also the study showed that female were affected more than male .

Study of role of high frequency ultrasound in diagnosis of thyroid pathologies done by Abdel Haleem M (2005) that using ultrasound the imaging of foci of disease within thyroid gland are easily identified

especially using high frequency probes which enables solid nodules up to 3mm to be revealed with 10MHZ probes. Abdel Haleem M.Abdalla -2005). Study of measurement of thyroid volume in normal adult Sudanese women by using ultrasound done by Samah .A (2013) the study revealed that the right lobe of the thyroid had significantly higher volume than the left lobe .

Chapter three

Materials & Methods

3-1 Materials:

1. All studies were obtained with Ultrasound machine with High-frequency linear transducer (7.5-15.0 MHz).
2. Pad.
3. Gel.
4. Volunteer.
5. ultrasound machine: Alpinion-Ecube 7 with CD, PD, PW and CW feature.

Sampling:

3.1.1 Inclusion Criteria:

The students ages between 17 to 25years old both gender

3.1.2 Exclusion Criteria:

The students age below 17 and above 25 years old

3.2Methodology :

3.2.1Method of thyroid scanning measurement and technique :

The thyroid measured in two planes (for each lobe) transvers plane to measure the AP and the width of thyroid gland, and in longitudinal to measure the length .the volume was measured outomatically using the machine.

Technique:

Using High-frequency transducers (7.5-15.0 MHz), the patient is typically examined in the supine position, with the neck extended. A small pad was placed under the shoulders to provide better exposure of the neck, particularly in patients with a short, stocky habitus. The thyroid gland was examined thoroughly in both transverse and longitudinal planes. Imaging of the lower poles can be enhanced by asking the patient to swallow, which

momentarily raises the thyroid gland in the neck. The entire gland, including the isthmus, was examined the examination must also be extended laterally to include the region of the carotid artery and jugular vein in order to identify enlarged jugular chain lymph nodes, superiorly to visualize submandibular adenopathy, and inferiorly to define any pathologic supraclavicular lymph nodes

3.2.2 Method of pathology evaluation:

Any finding in the thyroid lobes were evaluated according to its sonograph so if we found solid masses that may be hyperechoic, isoechoic, or hypoechoic with thick, smooth peripheral hypoechoic halo that features of solid nodule (adenoma). and because of the numerous interfaces between cells and colloid substance Less frequently, a hypoechoic spongelike or honeycomb pattern is seen and that is feature of complex nodule (adenoma) If there is tiny, punctuate hyperechoic foci, either with or without acoustic shadows in rare that the feature of calcifications.

If there is purely anechoic areas which caused by serous or colloid fluid and some of the nodules have tiny echogenic foci that are thought to be microcrystals a few of these foci are associated with comet-tail artifacts posteriorly. These nodules are mostly cystic (Colloid cysts).

If there is generalized enlargement of the gland this wil be (goiter).

If the echotexture of whole thyroid gland is hypoechoic this feature either outoimmune disease or in the patient of morbid obesity.

The diagnosis was done by expert sonographer and the researcher.

3.3 Data collection:

The data collected by using data collecting sheet it is designed to cover the gender ,age family history of thyroid disease,(AP, width ,length, volume) for each lobe and the findings if found .

3.4 Area of the study:

This study was conducted at Khartoum state at Sudan University of Science and Technology collage of radiological science's department.

3.5 Duration of the study:

This study was conducted during the period from July to September 2016.

3.6 Method of data analysis:

This data will analyze using Statistical Package for Social Science (SPSS) and an excel Microsoft office program.

3.7 Ethical issues:

There were official written permission to collage to take the data.

No volunteer data will publish.

Chapter Four: Results

The following figures and tables presented the result of the study which done in 60 subjects 47 were female 13male the mean age was 21 years old .the affected sample was 17subjects 13 female and 4male

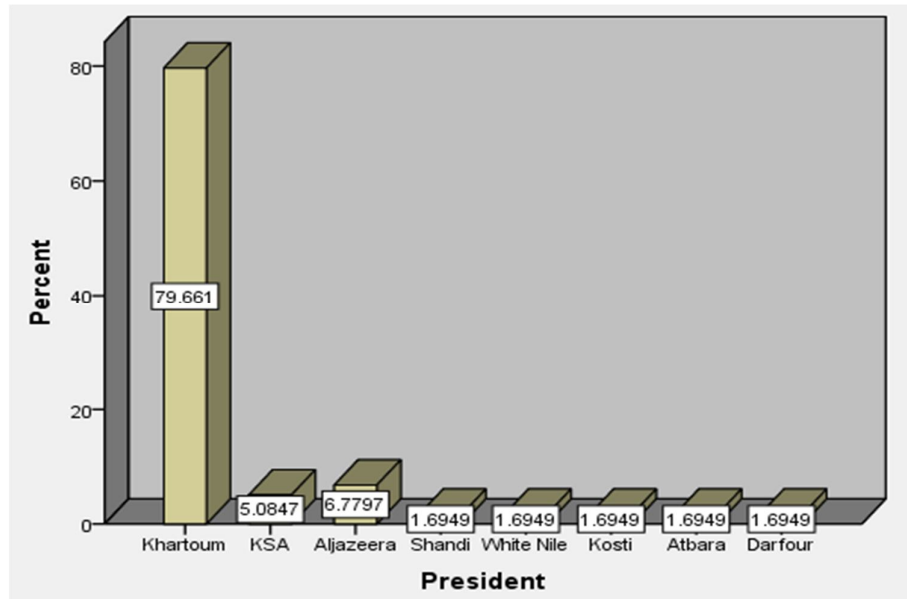


Figure (4-1)Resident distribution of the students

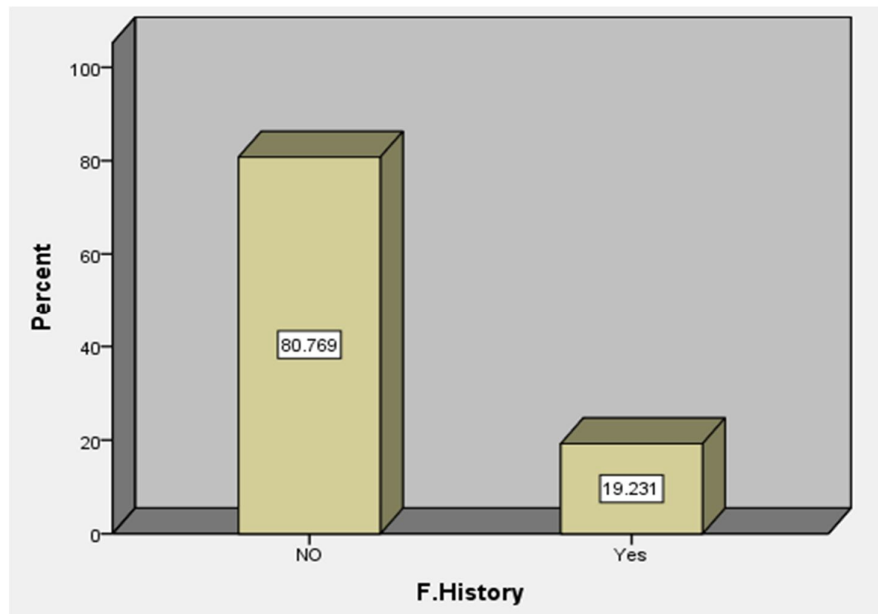


Figure (4-2) Family history of thyroid disease

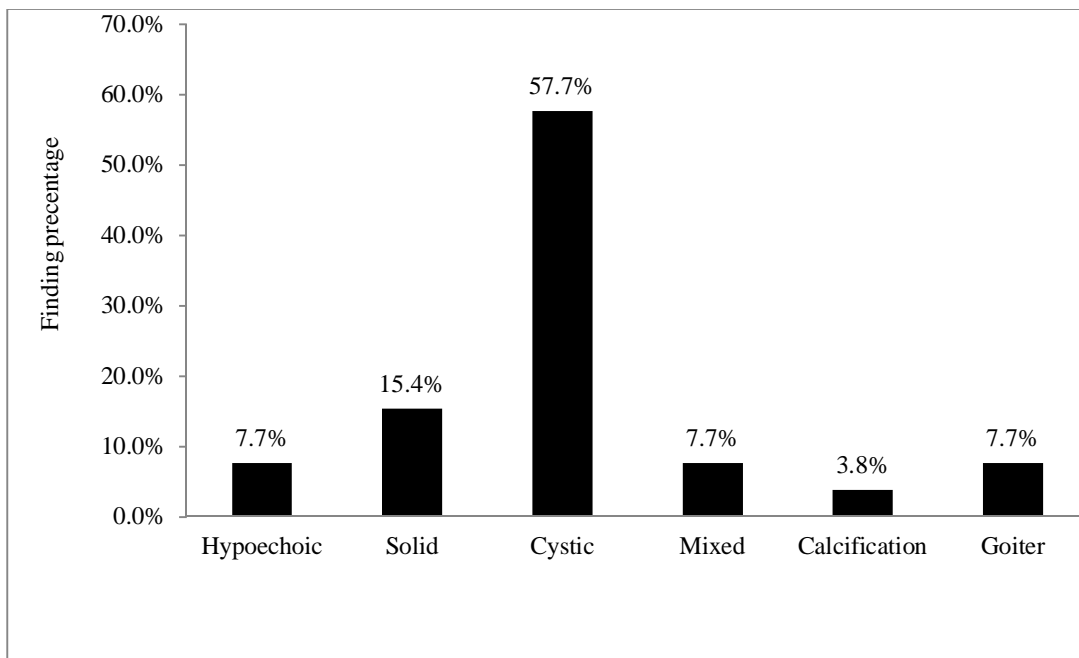


Figure (4-3) Percentage of Thyroid findings

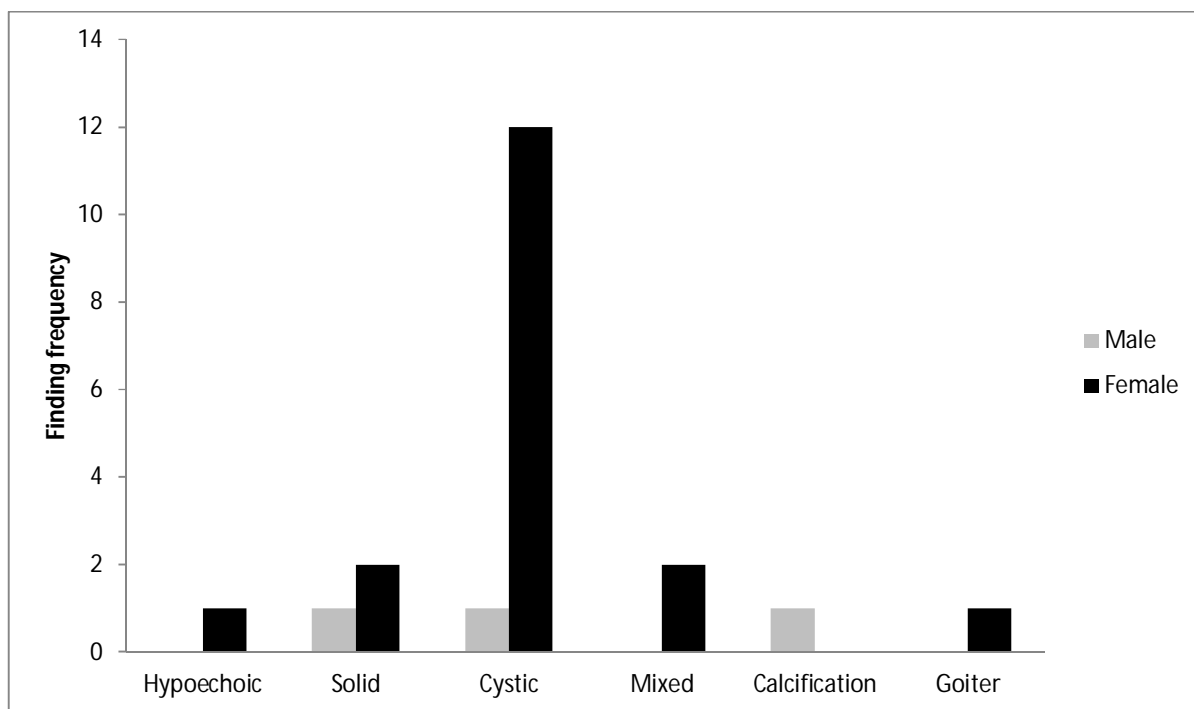


Figure (4-4) Male and female with thyroid finding

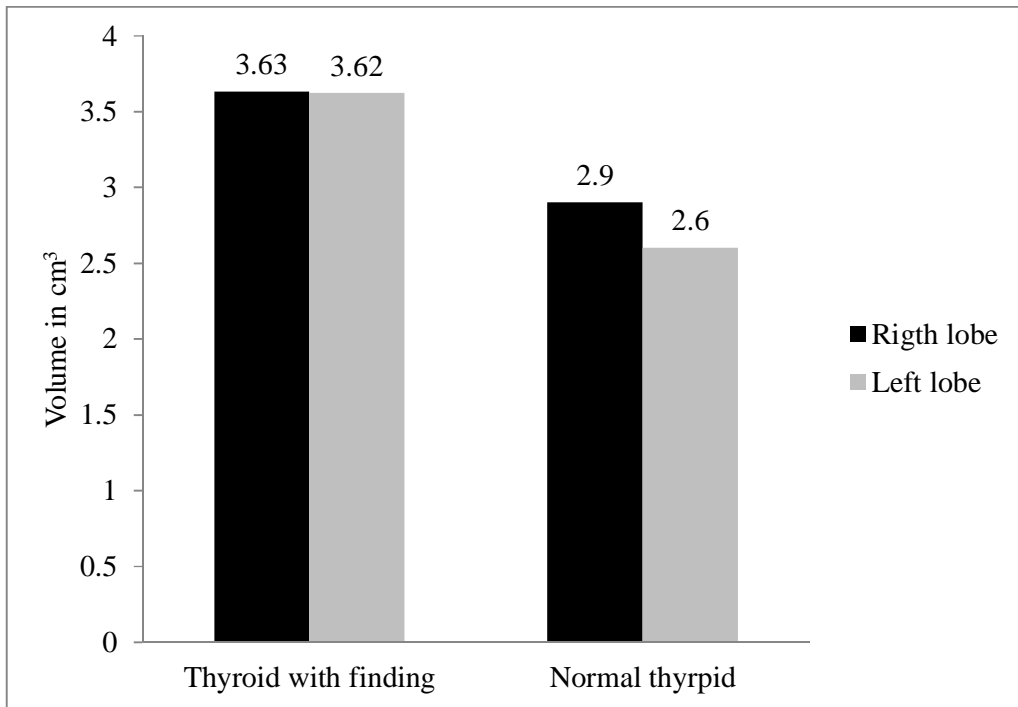


Figure (4-5) Mean of the volume in normal and abnormal thyroid

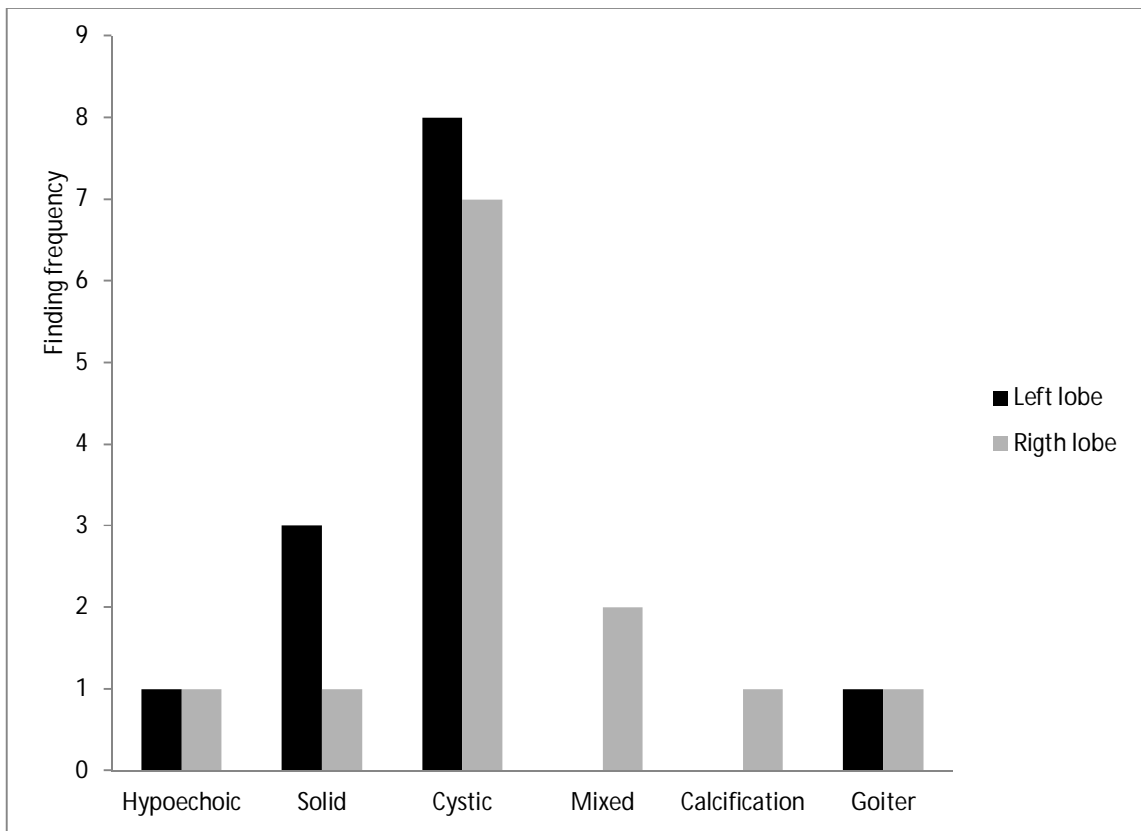


Figure (4-6) the findings and its frequency in lobes

Table 4-1 age range and frequency of thyroid findings

Age range	CYST	CALCIFICATIO N	SOLI D	MIXE D	GOITE R	HYPO
17-19	6	0	0	0	1	0
20-22	6	1	2	2	0	1
23-25	1	0	1	0	0	0

Table 4-2 Thyroid Volume (min-max) (mean±SD)

Volume (min-max) (mean±SD)		
	Right lobe	Left lobe
Thyroid with Finding	(1.98-6.9) (3.63±1.2)	(1.5-8.7) (3.62±1.6)
Normal thyroid	(1.5-5.3) (2.9±.83)	(1.4-4.5) (2.6±0.77)

Table (4-3) normal Thyroid lobes parameters

LT LOBE					RT LOBE			
	volume	length	width	AP	volume	length	width	AP
Min	1.42	2.20	1.06	0.8	1.50	2.53	1.08	0.83
Max	4.50	3.74	2.07	1.57	5.34	3.90	2.18	1.80
Mean	2.63	3.10	1.46	1.10	2.92	3.22	1.46	1.19
SD	0.77	0.32	0.24	0.19	0.83	0.31	0.25	0.19

Table(4-4) thyroid with findings parameters

	Left lobe				Right lobe			
	Volume (cm ³)	Length (cm)	Width (cm)	Ap (cm)	Volume (cm ³)	Length (cm)	Width (cm)	Ap (cm)
min	1.5	2.79	1.21	0.8	1.98	2.6	1.1	0.93
max	8.7	4	2.4	1.8	6.9	4	2.6	1.3
mean	3.59	3.18	1.71	1.19	3.53	3.28	1.61	1.27
SD	1.7	0.31	0.31	0.33	1.17	0.35	0.37	0.22

Chapter Five

5-1 Discussion:

Figure (4-2) show Family history of thyroid disease 19.2% had history which indicate that the history of thyroid disease in family may had a role in thyroid disease in this study.

Figure (4-3) show the thyroid findings in percentage the most common finding 57.7% was cystic nodule and that is similar to Salma .M (2016) result due to over growth of normal thyroid tissue also as Carol. M Rumak et al 2011 that (Many (if not all) cystic thyroid lesions are hyperplastic nodules that have undergone extensive liquefactive degeneration).

15.4% were solid isoechoic surrounded by a halo rim which indicated the benign feature as Carol. M. Rumak et al 2011, also it was easy to identified solid nodules by using high frequency ultrasound probe as Abdel haleem(2005) study revealed

7.7% was mixed nodule which indicate to degenerative changes in nodule as in Carol. M Rumak et al 2011. and 7.7% was hypoechoic thyroid that is may due to Graves ' disease or morbid obesity. 7.7% was calcification and goitre.

Figure (4-4) show the frequency of the findings with the gender the male in this study were affected more , and this result is not similar to (Carol. M. Rumak et al 2011) and Salma .M(2016) that, with women affected more frequently than men

Figure (4-5) show the changes in Mean of the volume in normal and abnormal thyroid in both lobes which indicate the increase in volume in the effected thyroid as Carol. M. Rumak et al 2011 that hyperplasia leads to an overall increase in size or volume of the gland ,and similar t Dr Awad ,M,E (2013) that most common findings were enlarge of thyroid.

Table (4-2) show the volume mean and min and max which indicate increase in affected thyroid.

Table (4-3) show mean of normal thyroid parameters that right lobe increase in volume than the left lobe and this result is similar to Samah Maglad et al 2013

5-2 Conclusion:

The study concluded that the mean normal thyroid dimension according to the findings of this study was found to be for the left lobe (volume 2.63 ± 0.77 , length 3.10 ± 0.32 , width 1.46 ± 0.24 , AP 1.10 ± 0.19) and for right lobe (volume 2.92 ± 0.83 , length 3.22 ± 0.3 , width 1.46 ± 0.25 , AP 1.19 ± 0.19). The most common finding was cystic nodule (57.7%) and the solid one come in to the second (15.4%) and the female were affected more than male by thyroid disease.

5-3 Recommendation:

Ultrasound examination is very important to identify thyroid nodule. So routine examination to those who had family history of thyroid disease and suffer from symptoms of thyroid disorders is important method to avoid the complication and the disturbance of hormone due to this disease

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Appendices



Image 1 Solid nodule in let lobe found in Female 20 years old

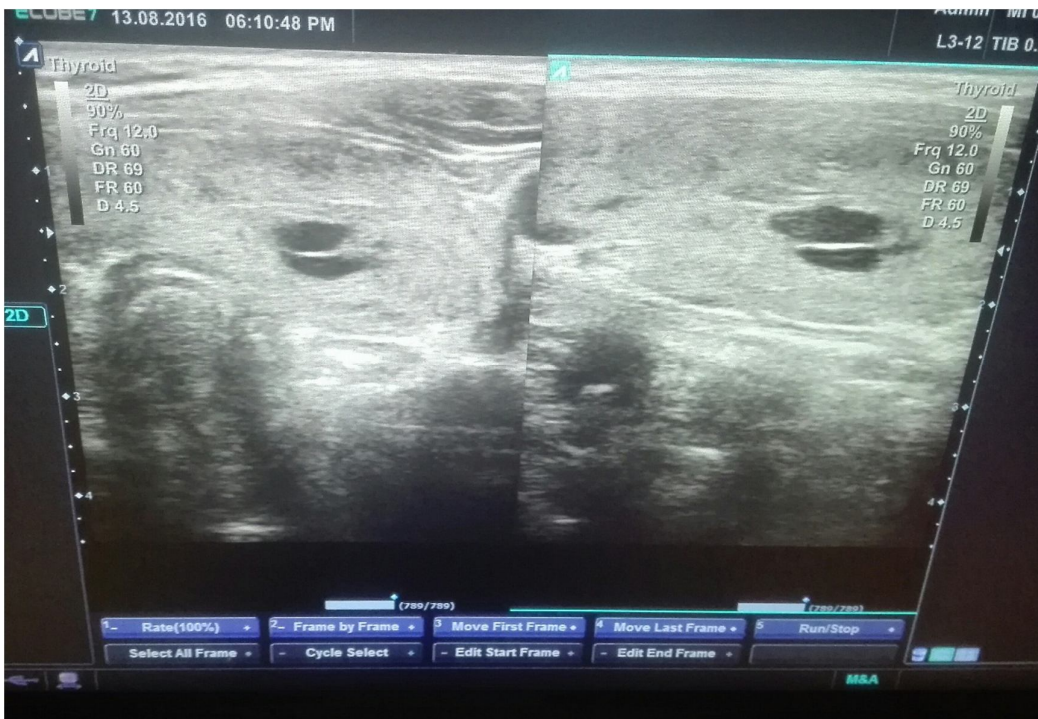


Image 2 septated cyst in left lobe with thyroid goiter in femal 18 years old



imag 3 cyst in right lobe with thyroid goiter in femal 18 years old



Image 4 cyst in left lobe in femal 22 years old

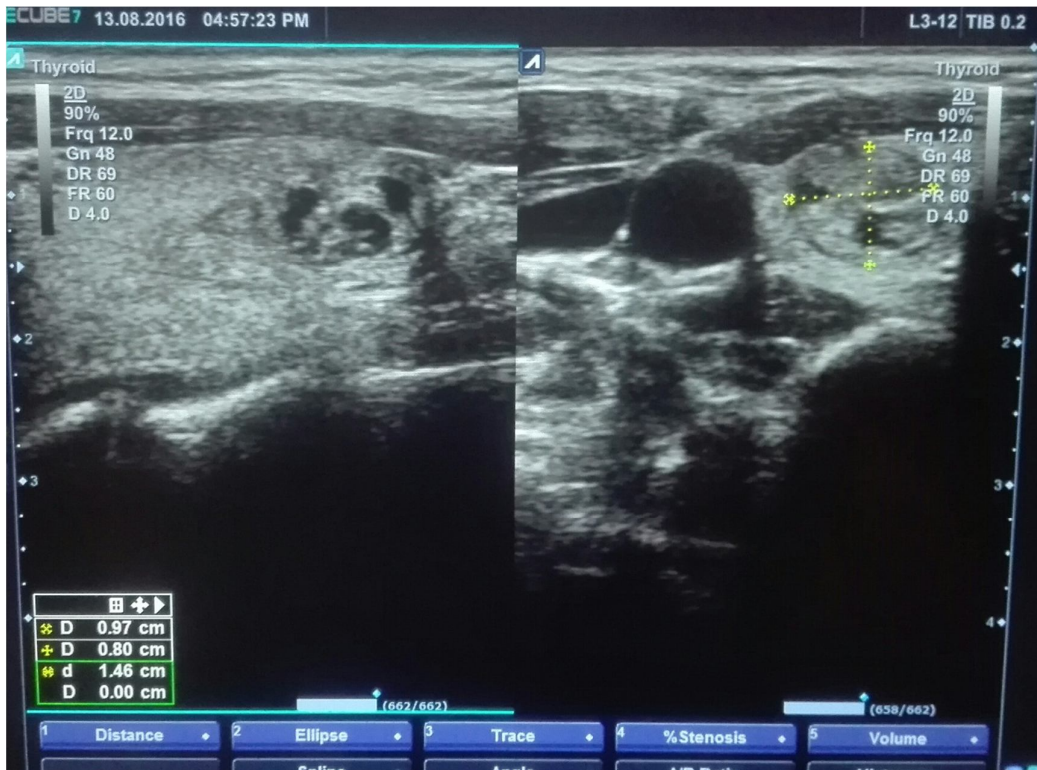


Image 5 Complexed solid nodule in right lobe female 20 years old



Image 6 Cyst in right lobe in male 19 years old



Image 7 Solid nodule in right lobe in female 22 years old

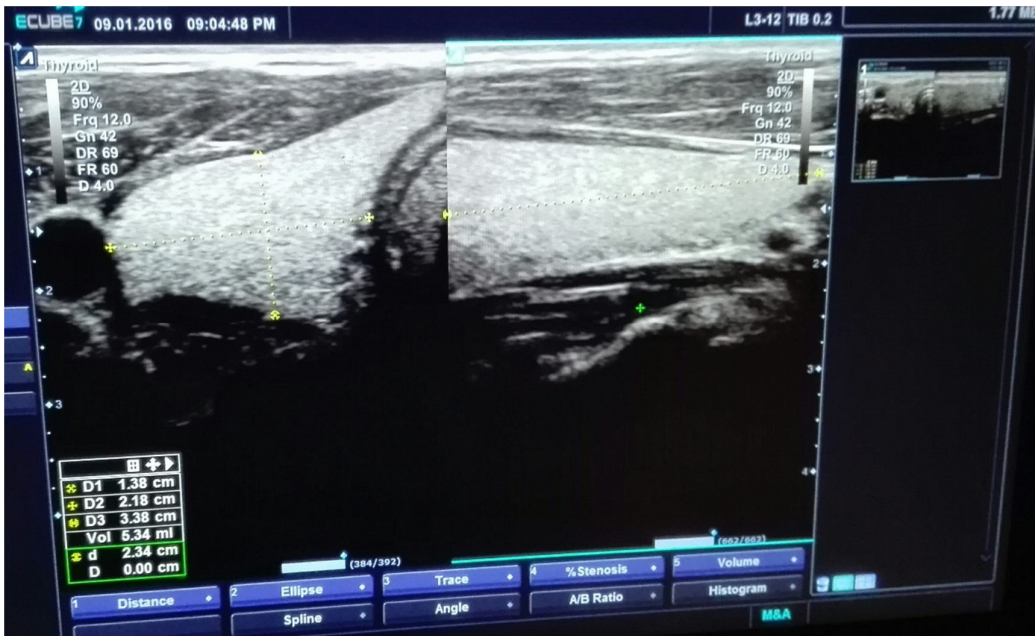


Image 8 Measurement of right lobe parameters in transvers and longitudinal plane