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" The Effect Of The Thyroid Gland Deficiency On Patients "

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قال تعالى

" بسم الله الرحمن الرحيم "

﴿ ٱقۡرَأۡ بِٱسۡمِ رَبِّكَ ٱلَّذِي خَلَقَ * خَلَقَ ٱلۡإِنسَٰنَ مِنۡ عَلَقٍ * ٱقۡرَأۡ وَرَبُّكَ ٱلۡأَكۡرَمُ*
ٱلَّذِي عَلَّمَ بِٱلۡقَلَمِ * عَلَّمَ ٱلۡإِنسَٰنَ مَا لَمۡ يَعۡلَمۡ ﴾

* صدق الله العلي العظيم *

العلق : 1 – 5

" شكر وإمتنان "

لابد لنا ونحن نخط وخطواتنا الْخيرة في الحياة الجامعية من وقفة نعود إلى أعوام قضيناها في رحاب الجامعة مع أساتذتنا الكرام الذين قدموا لنا الكثير باذلين بذلك جهودا كبيرة في بناء جيل الغد لتبعث الامة من جديد ....

وقبل أن نمض ي نقدم أسمى آيات الشكر والامتنان والتقدير والمحبة إلى الذين حملوا أقدس رسالة في الحياة ....

إلى الذين مهدوا لنا طريق العلم والمعرفة ....

إلى جميع أساتذتنا الأفاضل ....

" كن عالماً، فإن لم تستطع فكن متعلما، فإن لم تستطع فأحب العلماء، فإن لم تستطع فلا تبغضهم "

وأخص بالشكر والتقدير

الاستاذة: م.م نورس بهاء

الذي نقول لها بشراك قول رسول الله صلى الله عليه وآله وسلم:

" إن الحوت في البحر، والطير في السماء، ليصلون على معلم الناس الخير "

كما أنني أتوجه بشكر خاص وامتنان الى عمادة كلية الحلة الجامعة.

إلى من علمنا التفاؤل والمضي إلى الامام، إلى من رعانا وحافظ علينا، إلى من وقف إلى جانبنا عندما ضللنا الطريق .....

الاهداء

إلى من جرع الكأس فارغاً ليسقيني قطرة حب

إلى من كلت أنامله ليقدم لنا لحظة سعادة

إلى من حصد الاشواك عن دربي ليمهد لي طريق العلم

إلى القلب الكبير (والدي العزيـز)

إلى من أرضعتني الحب والحنان

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إلى القلوب الطاهرة الرقيقة والنفوس البريئة إلى رياحين حياتي (إخوتي).

الآن تفتح ا لاشرعة وترفع المرساة لتنطلق السفينة في عرض بحر واسع مظلم هو بحر الحياة وفي هذه الظلمة لا يضيء إلا قنديل الذكريات ذكريات الخوة البعيدة إلى الذين أحببتهم وأحبوني (أصدقائي).

"أهدي هذا البحث المتواضع راجياً من المولى عز وجل أن يجد القبول والنجاح"

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Abstract:

The thyroid gland, A vital endocrine organ located in the neck, Consists of two lobes connected by an isthmus. Responsible for synthesizing, Storing, And releasing thyroid hormones, Primarily T3 and T4, It plays crucial roles in metabolism, Growth, And development. Surrounding structures, Such as the thyroid cartilage housing the larynx and the cricoid cartilage providing tracheal support, Contribute to its function. The medium cricothyroid ligament, Situated between the thyroid and cricoid cartilages, Is a landmark for emergency airway procedures like cricothyroidotomy. Additionally, The parathyroid glands, located posteriorly to the thyroid, regulate calcium levels by secreting parathyroid hormone. Close proximity during thyroid surgery poses a risk of inadvertent injury, potentially leading to transient hypocalcemia and associated clinical manifestations. Understanding these anatomical and functional aspects is crucial for medical professionals involved in thyroid-related procedures. In summary, The symptoms of thyroid gland deficiency are diverse and can impact multiple systems of the body. These symptoms, Ranging from fatigue and weight gain to depression and cognitive impairment, Can significantly affect an individual's quality of life. Recognizing these symptoms is the first step toward diagnosis and treatment. It is important for individuals experiencing such symptoms to seek medical advice for proper evaluation and management. Understanding these symptoms not only aids in early detection but also emphasizes the importance of thyroid health in overall well-being.

In conclusion, the general treatment of thyroid gland deficiency is multifaceted and requires a personalized approach. Treatment primarily involves hormone replacement therapy, Which must be carefully monitored and adjusted to suit individual needs. Lifestyle modifications and diet also play a crucial role in managing symptoms and enhancing the effectiveness of medical treatment. Regular monitoring and consultation with healthcare providers are essential for optimal management of this condition. By understanding the complexity of treatment options and the importance of a tailored approach, individuals with thyroid gland deficiency can lead a healthy and fulfilling life.

The study aimed to prove that iodine plays a major role in the causes of hypothyroidism, apart from the causes of congenital and hereditary deficiency. Iodine deficiency is one of the most common causes. The study sample included several matters as well, The most important and most common of these primary causes was the autoimmune condition called (disease). Hashimoto's disease, Which is defined as chronic inflammation of the thyroid gland, Has an autoimmune origin. Hashimoto's thyroiditis results when antibodies in the body attack thyroid cells(autoimmunity). Some medications may be the cause of this deficiency, Such as lithium, Which is a medication used to treat a mental health condition sometimes referred to as manic depression, As lithium has many biological effects on the thyroid gland, Including: reducing the thyroid gland’s ability to produce thyroid hormone (T4) and triiodothyronine (T4). T3 Preventing the secretion of thyroid hormones from the thyroid gland.

1.1: Introduction:

The thyroid gland is a vital butterfly-shaped endocrine gland situated in the lower part of the neck. It is present in the front and sides of the trachea, inferior to the larynx. It plays an essential role in regulating the basal metabolic rate (BMR) and stimulates somatic and psychic growth, besides having a vital role in calcium metabolism. It is a gland consisting of two lobes, the right, and the left lobes, joined together by an intermediate structure, the isthmus. Sometimes a third lobe called the pyramidal lobe projects from the isthmus. It has a fibrous/fibromuscular band, i.e., levator glandulae thyroideae running from the body of the hyoid to the isthmus.. The thyroid gland is a richly vascular organ supplied by the superior and inferior thyroid arteries and sometimes by an additional artery known as the thyroid ima artery. The venous drainage is by superior, middle, and inferior thyroid veins. Sometimes a fourth thyroid vein might be present, called the vein of Kocher. The nerve supply is mainly from the middle cervical ganglion but also partly from the superior and inferior cervical ganglions. Two capsules completely cover the thyroid gland. The true capsule is made up of fibro-elastic connective tissue. The false capsule comprises the pre-tracheal layer of the deep cervical fascia. It consists of a deep capillary plexus deep into the true capsule. Hence, it is crucial to remove the plexus with the capsule during thyroidectomy**[1].**

The thyroid gland is a vital hormone gland: It plays a major role in the metabolism, growth, and development of the human body. It helps to regulate many body functions by constantly releasing a steady amount of thyroid hormones into the bloodstream. If the body needs more energy in certain situations – for instance, if it is growing or cold, or during pregnancy – the thyroid gland produces more hormones. The thyroid weighs between 20 and 60 grams on average. It is surrounded by two fibrous capsules. The outer capsule is connected to the voice box muscles and many important vessels and nerves. There is loose connective tissue between the inner and the outer capsule, so the thyroid can move and change its position when we swallow. The thyroid tissue itself consists of a lot of small individual lobules that are enclosed in thin layers of connective tissue. These lobules contain a great number of small vesicles (sacs) – called follicles – which store thyroid hormones in the form of little droplets **[2].**

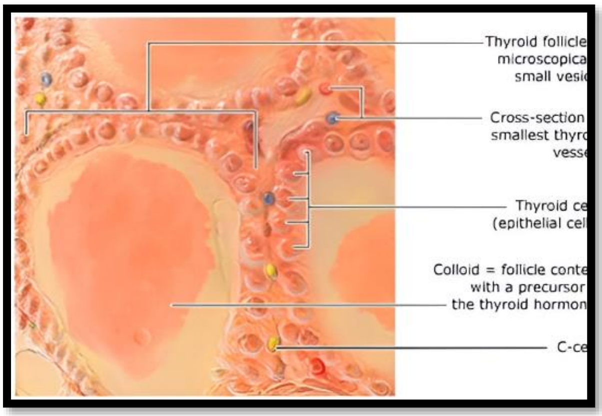

Fig. (1) Thyroid gland cells [2].

1.1.1: The thyroid gland produces three hormones:

Triiodothyronine, also known as T3, Tetraiodothyronine, also called thyroxine or T4, Calcitonin, strictly speaking, only T3 and T4 are proper thyroid hormones. They are made in what are known as the follicular epithelial cells of the thyroid. Iodine is one of the main building blocks of both hormones. Our bodies can't produce this trace element, so we need to get enough of it in our diet. Iodine is absorbed into our bloodstream from food in our bowel. It is then carried to the thyroid gland, where it is eventually used to make thyroid hormones. Sometimes our bodies need more thyroid hormones, and sometimes they need less. To make the exact right amount of hormones, the thyroid gland needs the help of another gland: the pituitary gland. The pituitary gland "tells" the thyroid gland whether to release more or less hormones into the bloodstream. Also, a certain amount of thyroid hormones are attached to transport proteins in the blood. If the body needs more hormones, T3 and T4 can be released from the proteins in the blood and do their job. The third hormone produced by the thyroid gland is called calcitonin. Calcitonin is made by C-cells. It is involved in calcium and bone metabolism. T3 and T4 increase the basal metabolic rate. They make all of the cells in the body work harder, so the cells need more energy too. This has the following effects, for example: Body temperature rises, Faster pulse and stronger heartbeat, Food is used up more quickly because the energy stored in the liver and muscles is broken down, The brain matures (in children), Growth is promoted (in children), Activation of the nervous system leads to improved concentration and faster reflexes **[3].**

1.1.2: thyroid gland works to produce and release hormones:

The signal comes from a small gland located at the bottom of our brain called the pituitary gland. The pituitary gland produces and sends out a hormone called thyroid-stimulating hormone (TSH). TSH then tells the thyroid gland how much hormone to produce and secrete. TSH levels in your blood are rising and falling depending on your body's needs, to produce more or less thyroid hormones. The pituitary gland responds either directly to the thyroid hormones in the blood, but it also responds to signals from the hypothalamus, which sits above the pituitary gland as part of your brain. The hypothalamus releases its own hormone thyrotropin-releasing hormone (TRH). TRH in turn stimulates the release of TSH in the pituitary, which then signals to the thyroid gland. This whole network is also referred to as the hypothalamic-pituitary-thyroid axis (HPT) and it adapts to metabolic changes and your body's needs **[4].**

1.1.3: Thyroid hormone biosynthesis:

There are two biologically active thyroid hormones: thyroxine (T4) and 3,5,3'-triiodothyronine (T3) (figure 1). They are composed of a phenyl ring attached via an ether linkage to a tyrosine molecule. Both have two iodine atoms on their tyrosine (inner) ring. They differ in that T4 has two iodine atoms on its phenyl (outer) ring, whereas T3 has only one. The compound formed if an iodine atom is removed from the inner ring of T4 is 3,3',5'-triiodothyronine (reverse T3 [rT3]), which has no biological activity. T4 is solely a product of the thyroid gland, whereas T3 is a product of the thyroid and of many other tissues, in which it is produced by the deiodination of T4. The thyroid gland contains large quantities of T4 and T3 incorporated in thyroglobulin, the protein within which the hormones are both synthesized and stored. Being stored in this way, T4 and T3 can be secreted more quickly than if they had to be synthesized **[5].**

Iodine economy: Iodine is essential for normal thyroid function, and it can be obtained only by consumption of foods that contain it or to which it is added. Iodized salt The UZI is a safe and cost-effective mass fortification strategy that, if properly implemented, can meet the needs of all population groups. At a salt intake of about 10 g/day, WHO recommends salt fortification at 20−40 mg/kg, and iodization of ≥15 mg/kg at consumption is adequate to prevent population iodine deficiency. Food and food products Though most foods are poor in native iodine, foods from the sea, particularly certain seaweeds, are rich in iodine. Milk products are a major contributor of iodine to many diets, and depending on dairy practice can be iodine rich. Drinking water typically has an iodine content of approximately 1−10 μg/L **[6].**

1.1.4: Thyroid hormones are synthesized in the thyroid gland via the following steps:

1. Thyroid iodide transport:

Iodide is transported into thyroid follicular cells against a chemical and electrical gradient. Iodide transport is linked to the transport of sodium, is energy-dependent and saturable, and requires oxidative metabolism. The sodium iodide transporter is an intrinsic transmembrane protein located on the basolateral membrane of the thyroid follicular cells. Other ions such as perchlorate and pertechnetate also are transported into the thyroid by the same mechanism and, therefore, are competitive inhibitors of iodide transport. Activation of the mitogen-activated protein kinase (MAPK) pathway by BRAF mutations in differentiated thyroid cancer results in reduced synthesis of the transporter, causing radioiodine refractory tumors; BRAF inhibitors allow upregulation of the transporter and restore iodine avidity **[5].**

2. Tyrosyl iodination:

In thyroid follicular cells, iodide rapidly diffuses to the apical surface of the cells, where it is transported by pendrin, a membrane iodide-chloride transporter, to exocytotic vesicles fused with the apical cell membrane. In these vesicles, the iodide is rapidly oxidized and covalently bound (organified) to a few of the tyrosyl residues of thyroglobulin. The oxidation of iodide is catalyzed by thyroid peroxidase in a reaction that requires hydrogen peroxide. This enzyme catalyzes the iodination of approximately 10 percent of the tyrosine residues of thyroglobulin **[7].**

3. Coupling of iodotyrosyl residues of thyroglobulin:

T4 is formed by coupling of two diiodotyrosine residues and T3 by coupling of one monoiodotyrosine and one diiodotyrosine within a thyroglobulin molecule. These reactions also are catalyzed by thyroid peroxidase **[8].**

4. Thyroglobulin synthesis:

Thyroglobulin is a 660-kilodalton (kD) glycoprotein composed of two identical, noncovalently linked subunits. It is found mostly in the lumen of thyroid follicles. It is synthesized and glycosylated in the rough endoplasmic reticulum and then incorporated into exocytotic vesicles that fuse with the apical cell membrane. Only then are tyrosine residues iodinated and coupled to form T4 and T3. The coupling process is not random. T4 and T3 are formed in regions of the thyroglobulin molecule with unique amino acid sequences. Normal thyroglobulin contains approximately six molecules of monoiodotyrosine, four of diiodotyrosine, two of T4, and 0.2 of T3 per molecule **[5].**

1.1.5: Development of the thyroid gland:

The thyroid gland and its hormones play multifaceted roles in organ development and the homeostatic control of fundamental physiological mechanisms such as body growth and energy expenditure in all vertebrates. The thyroid is formed from a midline anlage in the pharyngeal floor consisting of foregut endoderm cells that are committed to a thyroid fate. These thyroid progenitors then give rise specifically to the follicular cell lineage that eventually will form hormone-producing units – the thyroid follicles – that make up the thyroid gland. Differentiated cells within these follicles, known as thyrocytes, are strictly epithelial: they possess an apical surface that delimits the follicle lumen and a basal (or basolateral) surface that faces the extrafollicular space. It is these cells that produce the thyroid hormones triiodothyronine and thyroxine (T3 and T4), which are iodinated dipeptides that are synthesized, stored, and secreted in a complex series of reactions involving bidirectional transport to and from the lumen. Thyroid hormone production thus requires that thyrocytes are both fully polarized and able to maintain a tight barrier between inside and outside; from this viewpoint, thyroid follicular cells share many properties with exocrine cells that distinguish the thyroid from other major endocrine glands. Thyroid-stimulating hormone (TSH or thyrotropin) produced by the pituitary is the main regulator of thyroid growth and function from late fetal life to adulthood. However, thyroid organogenesis and de novo follicle formation occur independently of pituitary control, indicating that the embryonic thyroid relies entirely on locally derived inductive signals and morphogens for its proper development **[9].**

1.2: Location of The Thyroid Gland: -

The thyroid gland is located in the front of your neck, straddling your windpipe (trachea). It's shaped like a butterfly, smaller in the middle with two wide wings that extend around the side of your throat. A healthy thyroid gland is not usually visible from the outside (there's no appearance of a lump on your neck), and you can't feel it when you press your finger to the front of your neck. There are two main parts of the thyroid: the two halves (lobes) and the middle of the thyroid that connects the two lobes (thyroid isthmus). thyroid is made of thyroid follicle cells (thyrocytes), which create and store thyroid hormone (mainly T3 and T4), and C-cells, which secrete the hormone calcitonin **[10].**

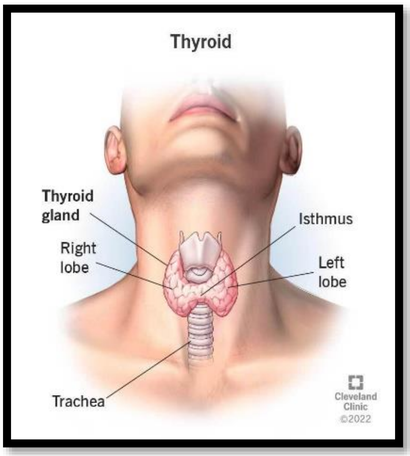

Fig. (2) Location of The Thyroid Gland [10].

The thyroid is located in the midline of the neck, anterior to the trachea and inferior to the larynx. It is found within the pre-tracheal fascia, one of the fascial compartments of the neck. It is a thin fascia in the anterior part of the neck and is composed of two parts: The muscular part: contains the infrahyoid muscles. Visceral part: contains the thyroid, trachea, and oesophagus. The thyroid gland is comprised of two lateral lobes connected by a central isthmus. It is surrounded by a fibrous capsule and located at the level of vertebrae C5-T1. The pyramidal lobe, an embryological remnant from the descent of the thyroid, typically projects upwards from the isthmus, however, there is a wide degree of variation between individuals. The primary function of the thyroid gland is to produce thyroid hormones (T3 and T4) following stimulation by thyroid-stimulating hormone (TSH) **[11].**

The thyroid gland is comprised of left and right lobes which are connected to each other by an isthmus, a horizontal band of thyroid tissue. Each thyroid lobe extends from midthyroid cartilage superiorly to the 4th or 5th tracheal ring inferiorly and common carotid arteries laterally. Though thyroid lobes are normally flat or globular yet partially cover the trachea posteriorly by a three-dimensional shape. Variations are observed in the occurrence of lobes, isthmus, arterial supply, venous drainage, and innervation patterns of the thyroid gland. The thyroid gland is enveloped by two capsules namely true and false. The true capsule is formed by condensation of connective tissue of the gland and the false capsule is derived from pre tracheal layer of deep cervical fascia. The dense capillary plexus is located deep in the true capsule. The main vessels lie between true and false capsules. It is difficult to ligate vessels in the capillary plexus while large vessels laying between true and false capsules can be easily ligated. Hence during thyroidectomy, the thyroid gland is removed along with the true capsule. The thyroidea ima artery may arise from the aortic arch or innominate artery and courses to the inferior portion of the isthmus or inferior thyroid poles. The thyroidea ima artery can be quite enlarged in patients with thyroid disease such as goiter or hyperthyroidism. It has also been reported to be a compensatory artery when one or both of the inferior thyroid arteries are absent and, in a few cases, the only source of blood to the thyroid gland. The thyroid ima artery is very important because of its relatively small size and infrequent presence but it can cause complications such as severe bleeding in surgery of the thorax, trachea, thyroid, or parathyroid glands if variations in the origin and course of this artery are not known to surgeons. Identification of this artery is very essential during tracheostomy and thyroidectomy as it is smaller than the other thyroid vessels, and originates from one of the large vessels, injury to it while performing the surgery may cause complications such as severe hemorrhage and significant blood loss **[12].**

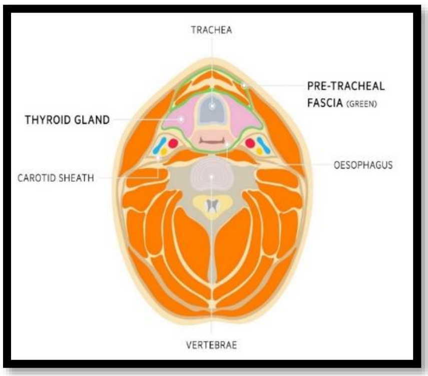

Fig. (3) Anatomical Location of The Thyroid Gland [11].

1.2.1: The Thyroid gland is surrounded by several important structures:

1. Thyroid Cartilage:

The thyroid cartilage is one of the three unpaired laryngeal cartilages (the others being the cricoid cartilage and epiglottis). It is formed by hyaline cartilage and is found above the thyroid gland, sitting at the level of vertebrae C4-5. It forms a median projection, more prominent in males, termed the Adam's apple. Superior to this projection is the superior thyroid notch. The cartilage has both superior and inferior horns **[13].**

2. Cricoid Cartilage:

The cricoid cartilage sits at the level of vertebrae C6. It is the only complete ring of cartilage in the trachea. It is composed of a posterior component termed the lamina and an anterior component termed the arch. It is attached to the first tracheal ring by the cricotracheal ligament **[14].**

3. Medium Cricothyroid Ligament:

This ligament connects the inferior border of the thyroid cartilage with the superior border of the cricoid cartilage. An incision is made through the medium cricothyroid ligament to establish an emergency airway - a cricothyroidotomy **[15].**

4. Parathyroid Glands:

The parathyroid glands (typically four) are located posterior to the thyroid gland. It is important to note there is a great deal of variety in both the location and number of glands. They are responsible for the release of parathyroid hormone, a key part of the calcium homeostasis pathways. Due to their location, they are frequently removed, injured, or de-vascularised during total thyroidectomy, typically resulting in transient hypocalcemia **[11].**

1.3: Effect of thyroid gland deficiency on body:

1. Impact on Metabolism:

The thyroid gland exerts a profound influence over the body's metabolic processes through the secretion of key hormones, primarily thyroxine (T4) and triiodothyronine (T3), These hormones play a pivotal role in orchestrating the metabolic rate, a critical determinant of energy utilization and overall physiological equilibrium **[15]**. Hypothyroidism, characterized by an underproduction of thyroid hormones, disrupts this delicate balance **[15]**. With diminished levels of T3 and T4, the metabolic rate decelerates, resulting in a cascade of metabolic imbalances. Individuals with hypothyroidism often experience weight gain, lethargy, and intolerance to cold temperatures due to the reduced efficiency in energy utilization. Moreover, hypothyroidism can lead to alterations in lipid metabolism, contributing to increased cholesterol levels and cardiovascular risks **[17]**.

The impact of hypothyroidism on metabolism is not limited to energy production alone. It extends to the regulation of glucose metabolism, potentially leading to insulin resistance and disturbances in blood sugar levels. These metabolic disruptions further underscore the intricate interplay between thyroid function and overall metabolic health **[16].**

2. Effects on Energy Levels:

The thyroid gland, as a central player in the endocrine system, exerts a profound impact on energy levels, influencing both the production and efficient utilization of energy throughout the body **[18]**. Thyroid hormones, particularly T3, play a pivotal role in cellular energy production **[19]**.

By enhancing the efficiency of mitochondrial oxidative phosphorylation, T3 ensures a constant supply of adenosine triphosphate (ATP), the primary energy currency of cells. This orchestrated energy production is vital for sustaining the myriad cellular processes that collectively contribute to overall vitality **[18].**

The role of thyroid hormones in energy regulation extends beyond ATP synthesis. T3 also influences the metabolism of carbohydrates, fats, and proteins, ensuring a balanced and efficient use of available energy substrates. This multifaceted regulation ensures that the body adapts its energy utilization based on the availability of different nutrients **[20]**.

A. Impact on daily life and productivity:

The ramifications of thyroid deficiency extend beyond physiological symptoms, permeating into daily life and productivity. The chronic fatigue experienced by individuals with hypothyroidism can impede cognitive function, concentration, and overall mental acuity. This, in turn, can hinder professional and personal pursuits, affecting work performance, academic achievements, and the ability to engage in social activities. Beyond cognitive effects, the impact on physical stamina can limit the ability to engage in regular physical activities, leading to a sedentary lifestyle that further exacerbates fatigue and contributes to a cycle of reduced energy levels. Additionally, the association between thyroid dysfunction and mood disorders, such as depression and anxiety, adds another layer of complexity to the overall well-being of individuals with thyroid disorders **[21].**

B. Connection between hypothyroidism and memory/concentration issues:

Hypothyroidism, characterized by a deficiency in thyroid hormones, is frequently associated with cognitive impairments, notably in memory and concentration, The reduced availability of T3 compromises the efficient functioning of neurotransmitter systems, leading to difficulties in concentration, memory retrieval, and overall cognitive performance. Studies have shown that individuals with hypothyroidism often exhibit alterations in brain structure and function, contributing to cognitive deficits. Magnetic resonance imaging (MRI) studies have revealed changes in the size and activation of certain brain regions, further highlighting the impact of thyroid hormones on cognitive processing**[23].**

C. Emotional implications, such as mood swings and depression:

Thyroid deficiency extends its impact beyond cognitive realms to emotional well-being. Individuals with hypothyroidism often experience mood swings and an increased susceptibility to depression. The intricate relationship between thyroid hormones and neurotransmitters, such as serotonin and dopamine, underscores the physiological basis for these emotional fluctuations **[24].**

The influence of thyroid hormones on neurotransmitter synthesis and receptor sensitivity has been extensively studied **[22].**

Changes in thyroid function can lead to alterations in the levels of these neurotransmitters, affecting mood regulation. Additionally, the role of thyroid hormones in the hypothalamic-pituitary-adrenal (HPA) axis, a key player in the body's stress response, further contributes to the emotional impact of thyroid dysfunction **[25].**

3. Cardiovascular Consequences:

The thyroid's regulatory influence extends to the cardiovascular system, where thyroid hormones orchestrate vital processes that maintain heart function and overall cardiovascular health **[26].**

A. Association between hypothyroidism and cardiovascular diseases:

Hypothyroidism can significantly impact cardiovascular health, leading to an increased risk of cardiovascular diseases **[27].**

The reduced levels of thyroid hormones in hypothyroid individuals can result in elevated cholesterol levels, impaired blood vessel function, and atherosclerosis. These factors collectively contribute to an increased susceptibility to conditions such as hypertension, coronary artery disease, and heart failure **[29].**

The relationship between hypothyroidism and cardiovascular risk is complex. Studies have shown that hypothyroidism is associated with changes in lipid metabolism, including elevated levels of LDL cholesterol and triglycerides, contributing to a pro-atherogenic environment. Additionally, hypothyroidism can lead to endothelial dysfunction, further exacerbating the risk of atherosclerosis and cardiovascular events **[28].**

B. Importance of thyroid function in maintaining a healthy cardiovascular system:

Thyroid function is integral to maintaining a healthy cardiovascular system **[29].** The balance of thyroid hormones ensures optimal cardiac output, blood vessel function, and overall circulatory efficiency. Disruptions in thyroid function, whether excess or deficiency, can lead to imbalances in these crucial cardiovascular parameters, potentially culminating in serious cardiovascular complications **[30].**

4. Reproductive and Hormonal Effects:

The thyroid's influence extends to the realms of reproductive health and hormonal balance, playing a crucial role in ensuring the proper functioning of the reproductive systems in both men and women **[31].**

A. Thyroid hormones and their role in reproductive health:

Thyroid hormones contribute significantly to reproductive health by influencing the production and function of sex hormones. T3 and T4 regulate the synthesis of sex hormones, such as estrogen and progesterone in women, and testosterone in men. Beyond their role in sex hormone synthesis, thyroid hormones also influence the responsiveness of reproductive tissues to these hormones. For instance, They modulate the sensitivity of the uterus and ovaries to estrogen and progesterone, Ensuring proper menstrual cycle regulation and fertility in women **[33]**.

In men, thyroid hormones influence the responsiveness of the testes to gonadotropins, impacting testosterone production and sperm quality **[32].**

B. Impact on menstrual cycles and fertility in women:

In women, Thyroid dysfunction, Especially hypothyroidism, Can disrupt the menstrual cycle and fertility. Insufficient levels of thyroid hormones can lead to irregular menstrual periods, anovulation, and difficulties in conceiving **[34].**

Studies have shown that untreated hypothyroidism during pregnancy is associated with an increased risk of complications, including preterm birth, Low birth weight, And neurodevelopmental issues in the offspring. Proper management of thyroid function in reproductive-aged women is crucial for optimizing fertility and ensuring healthy pregnancy outcomes **[32].**

C. Effects on testosterone levels and fertility in men:

Thyroid hormones also play a role in male reproductive health by influencing testosterone production and overall fertility. Imbalances in thyroid function, Particularly hypothyroidism, Can lead to a decrease in testosterone levels, Affecting sperm production and quality. This can result in fertility issues and impact reproductive outcomes in men **[31].** The relationship between thyroid function and male reproductive health is complex. Thyroid hormones influence the function of Leydig cells in the testes, which are responsible for testosterone production. Disruptions in this process can lead to not only decreased testosterone levels but also alterations in sperm morphology and motility **[34].**

1.4: Common Symptoms of Thyroid Gland Deficiency:

1.4.1: Fatigue and Weakness:

a. Impact on Daily Functioning:

Individuals grappling with thyroid gland deficiency face an ongoing battle with persistent fatigue and weakness that significantly hampers their ability to carry out daily activities **[35].**

This isn't merely a transient feeling of tiredness; it's a pervasive state of exhaustion that can make even the simplest tasks seem overwhelming. The thyroid gland, a small but mighty organ situated in the neck, Plays a pivotal role in the regulation of metabolism. Through the production of hormones like thyroxine (T4) and triiodothyronine (T3), The thyroid gland essentially serves as the body's metabolic control center **[36].**

The intricate dance of these hormones orchestrates the efficient utilization of energy within the body. However, when there's a deficiency in thyroid hormones, This balance is disrupted, Leading to a compromised state of energy production. The consequences are felt on a daily basis as individuals find themselves grappling with fatigue that seems unrelenting**[35].**

b. Connection to Decreased Energy Production:

Delving deeper into the molecular underpinnings, Thyroid hormones, Especially T4 and T3, Emerge as key players in the conversion of nutrients into energy within cells. Imagine these hormones as conductors orchestrating a symphony of metabolic processes. In the absence of this orchestration due to thyroid deficiency, The body's ability to convert food into usable energy is impeded. This disruption forms the core of the fatigue experienced by those with thyroid gland deficiency **[37].**

A crucial aspect of energy production takes place within the cellular powerhouses known as mitochondria. These organelles are responsible for generating the energy currency of the cell, ATP (adenosine triphosphate). In the context of thyroid deficiency, The scarcity of thyroid hormones further disrupts the harmonious workings of mitochondria, Throwing a wrench into the intricate machinery of energy production. This disturbance not only amplifies the fatigue but also contributes to the overall weakness experienced by individuals with thyroid gland deficiency **[38].**

1.4.2: Weight Gain and Difficulty Losing Weight:

a. Relationship Between Thyroid Hormones and Metabolism:

The intricate dance between thyroid hormones and metabolism plays a crucial role in the body's energy balance. According to various studies, including research conducted by the American Thyroid Association **[39].**

Thyroid hormones are indispensable regulators of metabolism, Directly influencing the rate at which the body burns calories to generate energy. T3, Identified as the active form of thyroid hormone, Emerges as a key player in this process. This hormone stimulates the breakdown of nutrients and facilitates their conversion into energy, Thereby ensuring a healthy and efficient metabolic rate **[40].**

The delicate equilibrium maintained by thyroid hormones is vital for weight management. When thyroid hormone levels are insufficient, The consequence is a deceleration in metabolism. This slowdown reduces the body's calorie-burning capacity, subsequently increasing the likelihood of weight gain **[41]**.

These findings are corroborated by a comprehensive review published in the Journal of Clinical Endocrinology & Metabolism **[42].**

b. Effects on Body Weight Regulation:

Beyond influencing metabolism, thyroid gland deficiency wreaks havoc on the intricate hormonal balance responsible for appetite and satiety regulation. Notably, hormones like leptin and ghrelin, Known for their roles in signaling hunger and fullness, Become imbalanced **[43].**

Extensive research from institutions such as the National Institute of Diabetes and Digestive and Kidney Diseases **[44],** Underscores that individuals with an underactive thyroid often grapple with an increase in appetite. This heightened appetite can lead to overeating, Contributing significantly to weight gain. The interconnectedness of these factors forms a complex web that exacerbates the challenges faced by those with thyroid-related weight issues **[45].**

Furthermore, the accumulation of excess weight can create a detrimental feedback loop. Adipose tissue, Or body fat, has been identified as more than just a passive storage depot. It actively produces hormones and molecules that can interfere with the proper functioning of the thyroid gland **[46].**

This interference sets the stage for a vicious cycle: as weight increases, thyroid function may further decline, And as thyroid function falters, Weight gain becomes more challenging to manage **[47].**

1.4.3: Changes in Mood and Mental Health:

a. Depression and Anxiety:

The impact of thyroid hormones on mental health extends beyond their role in metabolism. A growing body of evidence, Including studies cited by the World Journal of Psychiatry **[48],** Emphasizes the critical role of thyroid hormones in maintaining neurotransmitter balance in the brain, Particularly influencing mood regulation **[49].**

Thyroid gland deficiency, Especially in the form of hypothyroidism, Has been identified as a potential contributor to an increased risk of depression and anxiety disorders. The relationship between thyroid hormones and mental health is complex, Involving the modulation of neurotransmitters such as serotonin and norepinephrine, Which are vital for mood stability [**50**]. As outlined in research published in the Journal of Clinical Psychiatry, reduced thyroid hormone levels may disrupt the delicate balance of these neurotransmitters, potentially leading to the development or exacerbation of depressive and anxious symptoms**[51].**

b. Cognitive Impairment and Memory Issues:

Cognitive function, including memory and concentration, relies heavily on optimal thyroid hormone levels. Numerous studies, Including those featured in the Journal of Neurology, Neurosurgery & Psychiatry, Highlight the essential role of thyroid hormones in maintaining cognitive well-being **[52].**

In cases of thyroid gland deficiency, cognitive impairment can manifest as difficulties with concentration, Attention, And memory recall. The hippocampus, A brain region crucial for memory formation, Emerges as particularly sensitive to changes in thyroid hormone levels. Insufficient thyroid hormones may hinder neurogenesis and synaptic plasticity in this area, Contributing to memory-related issues **[53].**

Moreover, Hypothyroidism has been associated with a phenomenon known as "brain fog," characterized by a sense of mental confusion and difficulty organizing thoughts. The Journal of Neuropsychiatry and Clinical Neurosciences **[54],** Has explored the intricate connections between thyroid dysfunction and cognitive issues.

Beyond cognitive function, thyroid dysfunction's impact on mental health extends to motivation, energy levels, And overall emotional well-being. A study published in the Journal of Affective Disorders **[55].**

1.4.4: Hair and Skin Changes:

a. Dry Skin and Brittle Nails:

The influence of thyroid hormones extends beyond internal processes, significantly impacting the health and appearance of the skin. Research, such as studies conducted by the American Academy of Dermatology, Emphasizes the crucial role of thyroid hormones in maintaining healthy skin through the regulation of skin cell turnover and moisture levels**[56].**

In cases of thyroid gland deficiency, particularly hypothyroidism, A prevalent symptom is dry, rough, And scaly skin. This dermatological manifestation arises from the decreased metabolic activity associated with an underactive thyroid, leading to reduced oil production by skin glands and contributing to skin dryness **[57].**

Furthermore, Brittle nails emerge as another consequence of compromised thyroid function. Thyroid hormones play an integral role in the growth and maintenance of nails**[58].**

b. Hair Loss and Thinning:

Hair follicles, the dynamic structures responsible for hair growth, Are highly sensitive to thyroid hormone levels, Hypothyroidism, Characterized by an underactive thyroid, Has been associated with hair loss, Including the thinning of hair on the scalp and other body areas **[59].**

The disruption of the normal hair growth cycle is a key factor, as thyroid dysfunction shifts a higher percentage of hair follicles into a resting phase, resulting in increased shedding **[60].**

Additionally, Thyroid dysfunction may not only impact hair quantity but also alter its texture, Making it coarse and brittle. Are often reversible with appropriate thyroid hormone replacement therapy **[61].**

1.4.5: Temperature Sensitivity:

a. Feeling Excessively Cold or Hot:

The intricate dance between thyroid hormones and the body's core temperature regulation is a dynamic process, as highlighted by research from the Journal of Clinical Endocrinology & Metabolism. Thyroid hormones are crucial players in maintaining the delicate balance of the body's core temperature within a narrow range **[62].**

Individuals with thyroid gland deficiency, especially those experiencing hypothyroidism, may find themselves intolerant to cold temperatures. This intolerance stems from a slowed metabolic rate and reduced heat production, both of which are hallmarks of an underactive thyroid. that the decreased metabolic activity associated with hypothyroidism can limit the body's ability to generate sufficient heat, leading to a heightened sensitivity to cold **[63].**

Conversely, The spectrum of thyroid-related temperature sensitivity extends to individuals with hyperthyroidism. In this condition, An excess of thyroid hormones accelerates metabolic processes, leading to increased heat production. As a result, individuals with hyperthyroidism may exhibit heightened sensitivity to heat, Experiencing discomfort and warmth even in moderate temperatures **[64].**

b. Role of Thyroid Hormones in Temperature Regulation:

The thyroid gland's role in temperature regulation is multi-faceted, Involving its influence on metabolic rate and energy production. In-depth studies, Such as those published in the Journal of Physiology, Emphasize the thyroid's impact on how quickly the body produces and expends energy, Directly affecting temperature regulation **[65].**

Thyroid hormones exert their influence on the efficiency of heat production within cells, Influencing the body's response to external temperature changes. This intricate regulation is crucial for maintaining thermal homeostasis, Ensuring the body adapts appropriately to various environmental conditions **[66].**

An imbalance in thyroid hormones can disrupt this delicate thermoregulation, Leading to aberrations in temperature sensitivity. For instance, An underactive thyroid may compromise the body's ability to generate sufficient heat, Resulting in a predisposition to feeling excessively cold. Conversely, hyperthyroidism may lead to an overactive metabolic state, causing heightened sensitivity to heat **[67].**

This interplay between thyroid hormones and temperature regulation underscores the importance of thyroid function not only in metabolic processes but also in the body's ability to adapt to the surrounding environment **[68].**

1.5: Common Symptoms of Thyroid Gland Deficiency:

1.5.1: Fatigue and Weakness:

a. Impact on Daily Functioning:

Individuals grappling with thyroid gland deficiency face an ongoing battle with persistent fatigue and weakness that significantly hampers their ability to carry out daily activities **[69].**

This isn't merely a transient feeling of tiredness; it's a pervasive state of exhaustion that can make even the simplest tasks seem overwhelming. The thyroid gland, A small but mighty organ situated in the neck, Plays a pivotal role in the regulation of metabolism. Through the production of hormones like thyroxine (T4) and triiodothyronine (T3), The thyroid gland essentially serves as the body's metabolic control center **[70].**

The intricate dance of these hormones orchestrates the efficient utilization of energy within the body. However, when there's a deficiency in thyroid hormones, This balance is disrupted, Leading to a compromised state of energy production. The consequences are felt on a daily basis as individuals find themselves grappling with fatigue that seems unrelenting**[69].**

b. Connection to Decreased Energy Production:

Delving deeper into the molecular underpinnings, Thyroid hormones, especially T4 and T3, Emerge as key players in the conversion of nutrients into energy within cells. Imagine these hormones as conductors orchestrating a symphony of metabolic processes. In the absence of this orchestration due to thyroid deficiency, The body's ability to convert food into usable energy is impeded. This disruption forms the core of the fatigue experienced by those with thyroid gland deficiency **[71].**

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The delicate equilibrium maintained by thyroid hormones is vital for weight management. When thyroid hormone levels are insufficient, the consequence is a deceleration in metabolism. This slowdown reduces the body's calorie- burning capacity, Subsequently increasing the likelihood of weight gain **[75].** These findings are corroborated by a comprehensive review published in the Journal of Clinical Endocrinology & Metabolism **[76].**

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Beyond influencing metabolism, thyroid gland deficiency wreaks havoc on the intricate hormonal balance responsible for appetite and satiety regulation. Notably, hormones like leptin and ghrelin, Known for their roles in signaling hunger and fullness, Become imbalanced **[77].**

Extensive research from institutions such as the National Institute of Diabetes and Digestive and Kidney Diseases **[78],** Underscores that individuals with an underactive thyroid often grapple with an increase in appetite. This heightened appetite can lead to overeating, Contributing significantly to weight gain. The interconnectedness of these factors forms a complex web that exacerbates the challenges faced by those with thyroid-related weight issues **[79].**

Furthermore, The accumulation of excess weight can create a detrimental feedback loop. Adipose tissue, or body fat, Has been identified as more than just a passive storage depot. It actively produces hormones and molecules that can interfere with the proper functioning of the thyroid gland **[80].**

This interference sets the stage for a vicious cycle: as weight increases, Thyroid function may further decline, And as thyroid function falters, Weight gain becomes more challenging to manage **[81].**

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The impact of thyroid hormones on mental health extends beyond their role in metabolism. A growing body of evidence, Including studies cited by the World Journal of

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Moreover, Hypothyroidism has been associated with a phenomenon known as "brain fog" characterized by a sense of mental confusion and difficulty organizing thoughts. The Journal of Neuropsychiatry and Clinical Neurosciences **[88],** Has explored the intricate connections between thyroid dysfunction and cognitive issues.

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The disruption of the normal hair growth cycle is a key factor, as thyroid dysfunction shifts a higher percentage of hair follicles into a resting phase, resulting in increased shedding **[94].**

Additionally, thyroid dysfunction may not only impact hair quantity but also alter its texture, making it coarse and brittle. are often reversible with appropriate thyroid hormone replacement therapy **[95].**

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Individuals with thyroid gland deficiency, Especially those experiencing hypothyroidism, May find themselves intolerant to cold temperatures. This intolerance stems from a slowed metabolic rate and reduced heat production, Both of which are hallmarks of an underactive thyroid. That the decreased metabolic activity associated with hypothyroidism can limit the body's ability to generate sufficient heat, leading to a heightened sensitivity to cold **[97].**

Conversely, The spectrum of thyroid-related temperature sensitivity extends to individuals with hyperthyroidism. In this condition, An excess of thyroid hormones accelerates metabolic processes, Leading to increased heat production. As a result, Individuals with hyperthyroidism may exhibit heightened sensitivity to heat, Experiencing discomfort and warmth even in moderate temperatures **[98].**

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Thyroid hormones exert their influence on the efficiency of heat production within cells, Influencing the body's response to external temperature changes. This intricate regulation is crucial for maintaining thermal homeostasis, Ensuring the body adapts appropriately to various environmental conditions **[100].**

An imbalance in thyroid hormones can disrupt this delicate thermoregulation, Leading to aberrations in temperature sensitivity. For instance, An underactive thyroid may compromise the body's ability to generate sufficient heat, Resulting in a predisposition to feeling excessively cold. Conversely, Hyperthyroidism may lead to an overactive metabolic state, Causing heightened sensitivity to heat **[101].**

This interplay between thyroid hormones and temperature regulation underscores the importance of thyroid function not only in metabolic processes but also in the body's ability to adapt to the surrounding environment **[102].**

1.6: Causes of failure and symptoms of hypothyroidism:

Hypothyroidism is a deficiency or absence of the secretion of the hormone thyroxine and results from many causes, including congenital ones that the newborn is born with. What is new, Including what is the acquisition that begins later, Although congenital hypothyroidism may begin months after birth, And what concerns us here is congenital hypothyroidism due to its close connection to mental abilities and delayed development in the affected child. As a result, The birth screening program aims to detect the disease and start... By treating it before its symptoms appear and serious complications occur, Especially on the brain, Because thyroid hormone is very necessary for the development of the central nervous system in the first three years of life **[103].**

The thyroid gland is essential for life, Because hypothyroidism causes mental and functional impairment **[104].**

Thyroid hormones are found in all chordate animals, And they are the only biochemical molecules known to combine with iodine. Iodine is necessary for the synthesis of TH, And iodine deficiency is the main cause of hypothyroidism in underdeveloped countries **[105].**

1.6.1: Causes of hypothyroidism:

1. Thyroid autoimmunity

2. Not consuming a sufficient amount of iodine in food

3. Removing the thyroid gland while not taking a sufficient amount of thyroid hormone tablets or taking them incorrectly.

4. Taking therapeutic radioactive iodine.

5. Insufficiency in the function of the pituitary gland **[106]**.

Hormones (3T) and triiodothyronine (4T) Hypothyroidism is a deficiency of hormones secreted by the thyroid gland - the thyroid gland. Present from birth, the main problem is probably either A gland is essential for normal growth and cell function. It may be hypothyroidism itself or in the hypothalamus or pituitary gland, Which is responsible for stimulating the thyroid gland. A routine blood test for hypothyroidism is performed on every newborn to ensure early diagnosis and treatment of the problem, And it is a good test to detect thyroid problems **[107].**

Studies indicate that high levels of the hormone leptin in the event of obesity have serious effects, The most important of which are an increase in the incidence of heart clots and an increase in the incidence of hypothyroidism **[108].**

Stop taking thyroid hormone replacement pills for a period ranging between 3 to 6 weeks before using the iodine assigned to you. It is worth noting that stopping the use of pills will lead to an increase in the level of the TSH hormone to 30 or higher, And this percentage is much higher than the normal maximum Clear, And you may feel symptoms of hypothyroidism **[109].**

1.6.2: Causes of hypothyroidism:

The majority of the causes of congenital hypothyroidism result from maldevelopment of the thyroid gland during fetal life, And its most common form is an ectopic thyroid migration defect, Where the thyroid gland does not complete its migration from its origin to its place of stability, Is small, And is unable to secrete normal amounts of thyroid hormone, And there is aplasia. Or hypoplasia, these causes are often not hereditary and constitute more than 80% of the causes. Some of the remainder is hereditary and results from a deficiency of one of the yeasts necessary for the production of the hormone yshormonogenesd%. It constitutes 10 of the causes and is associated with an enlarged thyroid gland, And less of it is caused by iodine deficiency or the transmission of immunomodulators. From the infected mother to the fetus or because of Taking medications that harm the thyroid gland and are transmitted to the fetus **[110].**

Thyroxine (T4) is one of the two main hormones secreted by the thyroid gland. If the thyroid gland does not secrete a sufficient amount of the hormone thyroxine, It is possible to suffer from thyroid symptoms **[111].**

Posterior hypothyroidism often occurs when the thyroid gland does not form properly, Either because it does not exist at all, Is too small, Or They end in the wrong part of the neck Sometimes the gland is formed correctly but does not produce the hormone the right way Sometimes it loses the signal from the (controlling) pituitary gland to the thyroid gland to produce thyroid hormone in a small number of cases. Taking some medications during pregnancy, especially to treat hyperthyroidism, can lead to congenital hypothyroidism Temporarily, in most cases, posterior hypothyroidism is usually not caused by genetic causes. This means that if one child is affected, It is not possible to the same condition is likely to happen to other births in the family **[112]**.

Hypothyroidism occurs when the thyroid gland does not secrete enough hormones. Medical conditions or problems that can lead to hypothyroidism include: Autoimmune disease. The most common cause of hypothyroidism is an autoimmune disease known as Hashimoto's disease. Autoimmune diseases occur when the immune system produces antibodies that attack healthy tissue. This process sometimes extends to the thyroid gland and affects its ability to secrete hormones **[113].**

Thyroid surgery Surgery to remove all or part of the thyroid can reduce or stop its ability to produce thyroid hormones altogether. Radiotherapy. Radiation used to treat head and neck cancers can affect the thyroid gland and may lead to hypothyroidism. Thyroiditis. Thyroiditis occurs when the thyroid gland becomes inflamed, possibly due to an infection, autoimmune disease, or other condition that affects the thyroid gland. Inflammation stimulates the thyroid gland to secrete all its stores of thyroid hormone at once, causing a sudden increase in thyroid activity, a condition known as hyperthyroidism. After that, the thyroid gland becomes inactive. pharmaceutical. A number of medications may lead to hypothyroidism, including lithium, which is used to treat some psychological disorders. Therefore, if you are taking medication, ask your doctor about its effects on the thyroid gland. Less commonly, hypothyroidism may be caused by: Problems present from birth. Some children are born with a thyroid gland that does not function properly, and others are born without athyroid gland. In most cases, the reason why the thyroid gland does not develop properly is not clearly known. But some children have a hereditary type of thyroid disorder. Often, Symptoms of hypothyroidism in children born with this condition are not noticeable at first. This is one reason why most states require newborn thyroid screening. Pituitary gland disorder. A relatively rare cause of hypothyroidism failure of the pituitary gland to produce enough thyroid-stimulating hormone (TSH), Usually due to a benign pituitary tumor. Pregnancy. Some women develop hypothyroidism during or after pregnancy. If hypothyroidism occurs during pregnancy and is not treated, it increases the risk of miscarriage, premature birth, and preeclampsia. Preeclampsia causes a significant rise in blood pressure during the last three months of pregnancy. Hypothyroidism can also affect fetal development **[114].**

Iodine deficiency. The thyroid gland needs iodine to produce thyroid hormones. Iodine is found mainly in seafood, seaweed, And plants that grow in soil rich in iodine and iodized salt. Severe iodine deficiency can lead to hypothyroidism, and too much iodine can also worsen hypothyroidism in People who already have this condition. In some areas of the world, iodine deficiency is common in the food people eat. Adding iodine to table salt has eliminated this problem in the United States. Hypothyroidism occurs from a malfunction of the thyroid gland itself (primary hypothyroidism) or from insufficient stimulation by thyroid stimulating hormone (central hypothyroidism). Primary hypothyroidism is about a thousand times more common than central hypothyroidism. Iodine deficiency is the most common cause of primary hypothyroidism and endemic goiter worldwide. In areas with sufficient amounts of iodine, hypothyroidism usually results from an autoimmune disease called Hashimoto's thyroiditis (chronic autoimmune thyroiditis) **[115].**

For hypothyroidism and the diseases associated with it, The thyroid isn’t producing enough thyroid hormone, Which also results in a lack of energy. Causes of hypothyroidism include an autoimmune disorder such as Hashimoto's disease. This is a condition in which the body attacks the thyroid and causes it to produce fewer thyroid hormones **[116].**

Hypothyroidism can also be a result of exposure to high amounts of radioactive iodine from hyperthyroidism treatment, Which then results in permanent hypothyroidism. Hypothyroidism could also be a side effect of radiation therapy used to treat cancers in the head and neck. Pregnancy can also cause hypothyroidism, As well as a congenital defect from birth in which the thyroid does not develop properly. Hypothyroidism is when there isn't enough thyroid hormone in your bloodstream and your metabolism slows down. Hypothyroidism happens when your thyroid doesn't create and release enough thyroid hormone into your body. This makes your metabolism slow down, affecting your entire body. Also known as underactive thyroid disease, hypothyroidism is fairly common. When your thyroid levels are extremely low; this is called myxedema. A very serious condition, myxedema can cause serious symptoms, including: A low body temperature, Anemia, Heart failure, Confusion, Coma, This severe type of hypothyroidism is life-threatening **[117].**

In general, Hypothyroidism is a very treatable condition. It can be managed with regular medications and follow-up appointments with your healthcare provider. Hypothyroidism can affect people of all ages, Genders and ethnicities. It's a common condition, Particularly among women over age 60. Women are generally more likely to develop hypothyroidism after menopause than earlier in life. Hypothyroidism can have a primary cause or a secondary cause. A primary cause is a condition that directly impacts the thyroid and causes it to create low levels of thyroid hormones. A secondary cause is something that causes the pituitary gland to fail, which means it can't send thyroid stimulating hormone (TSH) to the thyroid to balance out the thyroid hormones. Primary causes of hypothyroidism are much more common. The most common of these primary causes is an autoimmune condition called Hashimoto's disease. Also called Hashimoto's thyroiditis or chronic lymphocytic thyroiditis, this condition is hereditary (passed down through a family). In Hashimoto's disease, The body's immune system attacks and damages the thyroid. This prevents the thyroid from making and releasing enough thyroid hormone **[118].**

An underactive thyroid(hypothyroidism) is where the gland doesn't make enough hormones. Both of these imbalances can lead to a great number of symptoms **[119].**

1.7: General treatment of thyroid gland deficiency:

1.7.1: Treatment of Thyroid Gland Deficiency:

1. Levothyroxine (Synthroid, Levo-T):

A Comprehensive Overview Levothyroxine, A synthetic derivative of the thyroid hormone thyroxine (T4), Reigns supreme as the cornerstone of hypothyroidism treatment. This widely prescribed medication, often marketed under brand names like Synthroid and Levo-T, Effectively acts as a vital replacement for insufficient endogenous thyroid hormone production, Meticulously restoring normal physiological functions in the body. Levothyroxine's prominence as the most prevalent choice for thyroid hormone replacement therapy underscores its well- established efficacy and wide-ranging global application **[120].**

Levothyroxine functions as an exogenous replacement for the T4 hormone, Undergoing a critical conversion to the active T3 form within peripheral tissues. This orchestrated process mimics the effects of naturally produced thyroid hormones, contributing to the restoration of optimal thyroid function **[121].**

Determining the right levothyroxine dosage is a nuanced task, Considering factors such as age, Weight, And the severity of hypothyroidism. Initiated with a conservative dose, Treatment involves careful titration based on regular monitoring of thyroid function tests, Ensuring a personalized and effective approach **[122].**

Vigilant monitoring of thyroid function through blood tests (TSH, FT4, and FT3) is essential for managing hypothyroidism with levothyroxine. These assessments offer insights into therapy efficacy, Allowing adjustments to optimize outcomes and prevent complications. While generally well- tolerated, levothyroxine's potential side effects, Such as palpitations and insomnia, require understanding. Overmedication indicators, such as weight loss, Necessitate dosage adjustments, emphasizing the importance of regular clinical assessment and proactive management for a safe and effective therapeutic experience **[123].**

2. Antithyroid Medications: Orchestrating Equilibrium in Hyperthyroidism:

Antithyroid medications emerge as indispensable components in the nuanced management of hyperthyroidism-a dynamic condition marked by the overproduction of thyroid hormones. This class of medications takes center stage in the therapeutic landscape, Orchestrating a meticulous interplay to rein in excessive hormonal secretion and alleviate the myriad symptoms associated with hyperthyroidism. Among the pharmacological protagonists in this domain are thionamides, Exemplified by methimazole and propylthiouracil (PTU) **[124].**

Thionamides, exemplified by methimazole and propylthiouracil, Intricately interfere with thyroid peroxidase, A key enzyme in thyroid hormone synthesis. Methimazole suppresses thyroxine (T4) and triiodothyronine (T3) production by inhibiting iodine incorporation into thyroglobulin, While propylthiouracil hinders T4 to T3 conversion in peripheral tissues **[125],** Guiding the therapeutic journey involves personalized dosage determination based on factors like hyperthyroidism severity and patient responsiveness. Initiated with higher doses, The regimen is meticulously tapered to the lowest effective maintenance dose, Accompanied by harmonious monitoring through thyroid function tests (FT4, TSH) for precise adjustments **[126].**

Vigilant monitoring remains crucial, Ensuring therapeutic efficacy and preventing disharmony in the form of adverse effects. Blood tests offer a melodic guide for dosage adjustments, with special attention to potential severe reactions like agranulocytosis or hepatotoxicity, Requiring swift intervention to maintain therapeutic harmony **[127],** While generally well- received, Thionamides introduce the possibility of adverse effects, Such as agranulocytosis and hepatotoxicity. Regular blood monitoring becomes a vital movement in the symphony of patient care, allowing the conductor to balance treatment benefits against risks, Crafting a personalized melody for each patient **[128].**

3. Radioactive Iodine (RAI): Precision in the Therapeutic Landscape of Hyperthyroidism:

Radioactive Iodine (RAI) emerges as an intricately designed therapeutic modality, Standing as a beacon of precision in the comprehensive management of hyperthyroidism. This innovative approach presents a meticulous strategy to modulate thyroid function, offering targeted benefits through the focused destruction of thyroid tissue facilitated by beta radiation, primarily emitted by the radioactive isotope iodine-131 **[129].**

The effectiveness of Radioactive Iodine (RAI) hinges on its targeted accumulation in the thyroid gland, Leveraging the organ's affinity for iodine. Administered orally or through a capsule, iodine-131 undergoes beta decay, Releasing radiation that precisely damages thyroid cells, aiming to recalibrate hormone production and alleviate hyperthyroidism symptoms **[130],** RAI deployment is tailored with a bespoke prescription, Considering factors like hyperthyroidism severity, Thyroid size, And uptake capability. The administration process is carefully orchestrated, typically involving a single dose to balance therapeutic efficacy and minimize potential adverse effects **[131].**

After RAI, Vigilant monitoring of thyroid function becomes paramount. Assessments of thyroid hormone levels (TSH, FT4, FT3) guide treatment adjustments, confirming the gradual normalization of thyroid function as a key indicator of therapeutic success. While generally well-tolerated, RAI is not without potential side effects **[132],** Transient exacerbation of hyperthyroid symptoms may occur post-treatment, Requiring short-term management. Long-term considerations include the risk of hypothyroidism, Necessitating a nuanced approach with ongoing hormone replacement therapy, Especially in specific populations like pregnant individuals, Where fetal thyroid function risks demand careful consideration **[133].**

4. Thyroid Surgery: A Surgical Symphony in Gland Management:

Thyroid surgery, an unequivocal intervention in the nuanced management of the thyroid gland, is a sophisticated procedure involving the meticulous removal of all or part of the thyroid gland. This decisive surgical modality, Referred to as thyroidectomy, Necessitates the expertise of a skilled surgical team and is typically contemplated when alternative treatments, Such as medication or radioactive iodine, Are either contraindicated or have proven ineffective **[134].**

1.7.2: Diverse Array of Thyroid Surgeries:

1. Total Thyroidectomy: A comprehensive removal of the entire thyroid gland.

2. Subtotal or Near-Total Thyroidectomy: A partial removal strategy, Leaving a carefully determined portion of the thyroid intact **[135].**

1.7.3: Indications Paving the Path to Thyroid Surgery:

1. Uncontrolled Hyperthyroidism: When hyperthyroidism persists despite rigorous attempts with medication or radioactive iodine therapy.

2. Large Goiters: Enlarged thyroid glands causing compression on adjacent structures, Warranting surgical intervention.

3. Thyroid Nodules or Tumors: Whether suspected or confirmed as malignancies, Surgical excision becomes a crucial diagnostic and therapeutic maneuver **[136].**

1.7.4: Surgical Artistry Unveiled:

1. Conventional Thyroidectomy: The time-honored approach involves traditional open surgery with a meticulous incision in the neck.

2. Minimally Invasive Thyroidectomy: Embracing technological advancements, endoscopic or robotic-assisted approaches offer reduced scarring and expedited recovery times, exemplifying a contemporary paradigm in thyroid surgical techniques **[137].**

1.7.5: Preoperative Symphony of Assessment:

A symphony of evaluations precedes the surgical act, Encompassing comprehensive thyroid function tests, advanced imaging modalities such as ultrasound and CT scans, and, In cases of suspected malignancy, The nuanced precision of fine-needle aspiration biopsy. The harmonious collaboration between surgeons, Endocrinologists, And pathologists ensures a thorough and tailored preoperative plan **[138].**

1.7.6: Intraoperative Precision Dance:

Surgical precision takes center stage during the performance to avoid inadvertent damage to adjacent structures, including the delicate parathyroid glands and recurrent laryngeal nerves. The surgical ballet is further enhanced by intraoperative monitoring of nerve function and parathyroid hormone levels, an intricate dance that elevates safety to an art form **[139].**

1.8: Primary Prevention of Thyroid Disease:

For many years, Thyroidologists have been researching factors other than iodine that may affect thyroid health; Of various factors that have been documented as having some effects on thyroid size and function, Tobacco abuse and alcohol are thought to be more prominent **[140].**

1. Tobacco Smoking:

Smoking is one of the leading preventable causes of various health derangements and death. Cigarette smoking introduces a large number of chemical substances to the human body that may affect the thyroid gland in different ways. The competitive inhibitory effect of thiocyanate on the sodium-iodine symporter (NIS) worsens iodine deficiency. Tobacco smoking causes a small decrease in serum TSH and an increase in serum-free T4. The effects of smoking on increased risk of Graves' hyperthyroidism, And Graves' orbitopathy, in particular **[140].**

2. Alcohol Consumption:

Alcohol Consumption Alcohol abuse leads to considerable morbidity and mortality. However, Scientists from Denmark report that a reduction in the risk of Graves' disease with hyperthyroidism may be seen with moderate alcohol consumption, whereas others report conflicting results; 3 studies found no effect of alcohol consumption in Graves' patients, while 1 showed a decreased risk in women but not in men **[140].**

3. Thyroid Autoimmunity (Positive Thyroid Antibodies):

It has been recommended that the presence of thyroid antibodies per se is not a reason to initiate thyroid hormone therapy. Despite the fact that the presence of antibodies alone can cause thyroid-related symptoms, Few endocrinologists treat those with positive antibodies and TSH within the normal range who feel unwell. The presence of thyroid antibodies alone can affect fertility or the ability to maintain a pregnancy; Therefore, Some thyroidologists treat euthyroid pregnant women with positive TPOAb. Thyroid autoimmunity is characterized by infiltration of sensitized T lymphocytes in the thyroid gland and increased thyroid autoantibodies in the serum; It is an inherited defect in immune surveillance, which leads to alteration of presenting antigens in the gland, or abnormal regulation of immune responsiveness **[140].**

1.8.1: Prevention of Hypothyroidism:

It is possible to avoid developing hypothyroidism, It can be prevented with a proper diet and lifestyle management. In addition, These can help in the prevention of hypothyroidism:

1. A diet rich in iodine, Selenium, And zinc.

2. Giving up smoking.

3. Keep stress and anxiety under control by doing regular exercise and yoga.

4. Avoid self-medication.

5. Avoid gluten, processed foods, and fast food.

6. Low exposure to environmental radiation **[141].**

1.8.2: Important Nutrients and Vitamins for the Prevention of hypothyroidism:

• Iodine: Iodine is an essential mineral that is needed to make thyroid hormones, And a deficiency can lead to hypothyroidism. In fact, Insufficient iodine intake is the most common cause Trusted Source of hypothyroidism worldwide.

• **Selenium**: Selenium is another mineral that's necessary for thyroid health and thyroid hormone production. It helps protect Trusted Source the thyroid from damage caused by oxidative stress.

• **Zinc:** Like selenium, zinc Trusted Source is needed for thyroid hormone production and thyroid function.

Not getting enough zinc can negatively affect your thyroid function and many other aspects of health, so it's essential to get enough of this nutrient in your diet.

• **Vitamin D:** Having low vitamin D levels can negatively affect thyroid function and worsen hypothyroid symptoms. Because vitamin D isn't concentrated in many foods, supplementation is often necessary.

• **Vitamin B12:** B12 deficiency is common among people with hypothyroidism.

• **Magnesium:** Low or deficient magnesium levels are associated Trusted Source with thyroid dysfunction and can increase the risk of developing hypothyroidism.

• **Iron:** Low iron levels or iron deficiency anemia can impair thyroid function. Supplementation is often necessary to reach and maintain healthy iron levels **[142].**

1.8.3: Preventing Thyroid Diseases with a Plant-Based Diet:

While the lack of iodine in the diet is the biggest risk factor for hypothyroidism worldwide, Obesity is a significant risk factor for hypothyroidism. The relation between obesity and hypothyroidism appears to have several explanations. Obesity may result in raised Thyroid Stimulating Hormone [TSH] levels, Partly due to a proinflammatory milieu and other endocrine derangements. Persons with obesity are prone to develop autoimmune hypothyroidism, And even mild thyroid failure contributes to the progressive increase in body weight, Which ultimately results in overt obesity. So elevated TSH levels may be both the consequence and the cause of obesity. Vegans have a lower BMI on average thus reducing their risk of hypothyroidism. However, A lower risk of hypothyroidism among vegans exists, Even after controlling for BMI and potential demographic confounders. One study showed that following a vegan diet tended to be associated with a 22% reduced risk of hypothyroidism. Data from the Adventist Health Study showed that a vegan diet reduced the risk of hyperthyroidism by 51% while a vegetarian diet reduced the risk by 28% showing a dose-response relationship. This is to be expected since epidemiological studies have shown that vegetarians have lower risks of autoimmune diseases in general **[143].**

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