Disorders of thyroid function

There are two main hormones produced by the thyroid gland:*thyroxin* (T4) and *triiodothyronine* (T3). Both hormones are derived from the amino acid *tyrosine* and contain *iodine* that is extracted from the blood.

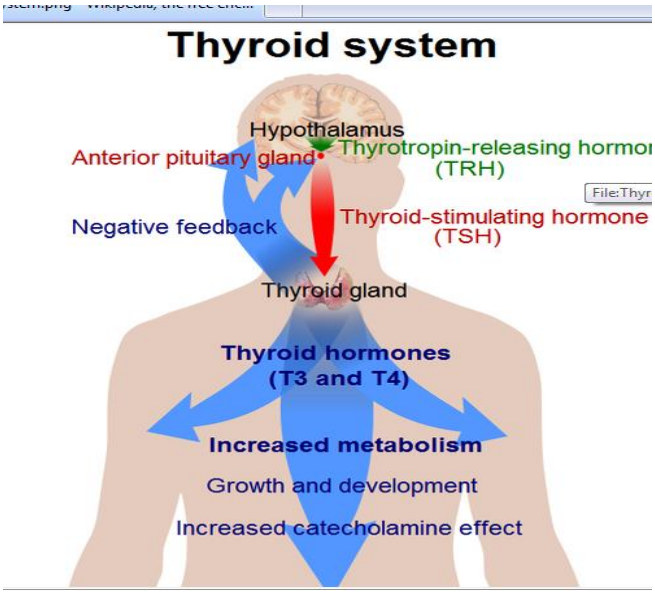
The release of thyroid hormones from the thyroid is regulated by TRH (thyrotropin releasing hormone) secreted from the hypothalamus which inturn induce the anterior pituitary gland to produce TSH(thyroid stimulating hormone). The main actions of T3 and T4 are outlined in Table.

Hypothyroidism

• May be a primary condition resulting from a defect within the thyroid itself or can be secondary to a lack of stimulation by TSH.

• Dietary deficiency of *iodine* may lead to hypertrophy of the thyroid gland that presents as a *goiter* (Table).

• The most common cause of hypothyroidism is an autoimmune condition Called *Hashimoto's thyroiditis* in which antibodies are produced against the tissue of the thyroid. Although the exact etiology of this autoimmune disorder is unknown, it can lead to progressive destruction of the thyroid gland and loss of thyroid function.



Manifestations

Cretinism

• Hypothyroidism that occurs during fetal development

• May occur as a result of a congenital defect in thyroid development
• Severe mental retardation due to poor development of the brain
• Poor overall development and growth retardation

Table :
-Physiologic Effects of Thyroid Hormones
Increased basal metabolic rate
Maintenance of normal metabolic function
Development of the nervous system in the fetus
Tabl:
Toxic Goiter and Nontoxic Goiter
-Nontoxic goiter — Hypertrophy of the thyroid gland that is not accompanied by excess secretion of thyroid hormones. May occur as a result of dietary iodine deficiency. Symptoms are those of hypothyroidism.
-Toxic goiter — Hypertrophy of the thyroid that is accompanied by excess thyroid production. May be associated with *Grave's disease* . Symptoms are those of hyperthyroidism.
-*Thyrotoxicosis*
is a term that is used to describe the "toxic" effects of excess thyroid hormones on the body.

Myxedema
• Hypothyroidism in the adult
• May result from autoimmune destruction of the thyroid or thyroid injury or removal
• Presents with signs of hypometabolism including:
Cold intolerance
Weight gain
Fatigue
Bradycardia
Cool, dry skin
Anorexia
Constipation
Edema of the face (swelling around the eyes), hands and ankles;drooping eyelids.
• Possible long-term complications of untreated hypothyroidism, including cardiac hypertrophy, heart failure, and *myxedema coma,*which presents with hypothermia, seizures and respiratory depression
Treatment
Thyroid hormone replacement therapy. A variety of synthetic and natural T3/T4 preparations are available for use orally.

Hyperthyroidism

Increased synthesis and release of T3and T4. Hyperthyroidism may be a primary condition that results from an overactive thyroid gland or it may occur as a result of excessive stimulation of the thyroid by TSH from the pituitary. Hyperthyroidism is also referred to as *thyrotoxicosis* . One of the most common causes of hyperthyroidism is *Grave's disease.*

Hyperthyroidism can also be caused by a toxic goiter not associated with Grave's disease (*Plummer's disease*) or by a tumor of the thyroid. In rare cases carcinomas arising outside of the thyroid may produce thyroid hormone or TSH.

Grave's disease

Patients with Grave's disease produce antibodies that bind TSH receptors on the thyroid and mimic the actions of TSH leading to excess production of thyroid hormones.

Manifestations

The manifestations of hyperthyroidism are essentially the same regardless of the cause of the hyperthyroidism; they include the following:

• Increased basal metabolic rate
• Increased heat production, patient always feels "hot"
• Tachycardia
• Increased catecholamine sensitivity; patients are at risk for cardiac arrhythmias
• Increased appetite
• Weight loss
• Enhanced bowel activity
• Behavioral changes including possible nervousness and hyperactivity

Treatment

•Blocking drugs to blunt the effects of excess adrenergic stimulation.
• Antithyroid drugs (*propylthiouracil, carbimazole, methimazole*) that block production of thyroid hormone.
• *Radioiodine*
— Given orally and taken up by hormone-producing cells of the thyroid as if it were normal iodine. The cytotoxic actions of the iodine and radiation destroy the hormone-producing cells of the thyroid. After treatment the patient usually becomes hypothyroid and must be managed with thyroid hormone replacement therapy.

Radioactive iodine should not be used in patients of childbearing age due to the possible effects on offspring.

• Surgical ablation of a portion of the thyroid may also be used. Following surgery, patients may likewise become hypothyroid and require thyroid hormone replacement therapy.