

CALIFORNIA ASSOCIATION FOR MEDICAL LABORATORY TECHNOLOGY

DISTANCE LEARNING COURSE

**FRENETIC FLORA and FREEZING FRANK
(Thyroid Hormones and Thyroid Diseases)**

By

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**Course Number: DL-967
2.0 CE/Contact Hours
Level of Difficulty: Intermediate**

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DON'T PUT YOUR LICENSE IN JEOPARDY!**

This is a reminder that all the continuing education units required to renew your license must be earned no later than the expiration date printed on your license. If some of your units are made up of Distance Learning courses, please allow yourself enough time to retake the test in the event you do not pass on the first attempt. CAMLT urges you to earn your CE units early!

Frenetic Flora and Freezing Frank (Thyroid Hormones and Thyroid Diseases)

CASE #1: Frenetic Flora

Flora, a 46-year old female, visited her physician because of nervousness, tiredness, insomnia and recent weight loss. She told the doctor that she was eating Crisco out of the can with a tablespoon trying to keep her weight up. Her physician found her pulse to be 85 and noted a fine tremor of her hands. She also mentioned feeling irritable and having muscle aches. On examination the physician felt a mass in her lower neck and noted slightly protruding eyes. The physician ordered the usual CBC, chemistry panel and urinalysis. He also ordered serum TSH (thyroid stimulating hormone).

The results of the CBC, chemistry panel and urinalysis were in the normal range. The TSH was 0.06 mIU/L (normal = 0.4-4.0 mIU/L).

Questions, to be answered after reading the course:

1. What is Flora's possible diagnosis?
2. What is the incidence of this?
3. What tests should be done next?
4. What is the treatment and prognosis?

CASE #2: Freezing Frank

Frank came to medical attention after he was wearing an overcoat in a car with all the windows closed in 100 degree summer weather. He told the doctor that in addition to being cold, he was tired all the time and didn't feel like socializing. He also complained that he had been gaining weight even though he had a poor appetite. His physician noticed he had pale, dry skin and a puffy face. His voice was hoarse. The physician ordered a CBC, chemistry panel and TSH.

The results of the CBC were normal. The chemistry panel showed a high cholesterol level. The TSH was 15 mIU/L.

1. What is Frank's possible diagnosis?
2. What is the most common cause of this disease?
3. What test should be done next?
4. What is the treatment and prognosis?

INTRODUCTION

The thyroid gland produces two hormones, thyroxine and triiodothyronine. These hormones are essential for cell differentiation during development and for maintaining temperature and metabolic homeostasis in the adult.

Thyroid disease is common, affecting millions of Americans. Abnormalities usually involve thyroid hormone underproduction (hypothyroidism) or overproduction (hyperthyroidism). Laboratory tests are used to diagnose the abnormality and help the physician treat the patient.

This course will cover

- the thyroid gland, location, structure, and function
- thyroid hormone biosynthesis, control of production, and function
- laboratory tests
- thyroid diseases

OBJECTIVES

After reading the course, the participant will be able to:

1. describe the anatomy and location of the thyroid gland
2. outline the biosynthesis and control of thyroid hormone production
3. describe the transport and function of thyroid hormones
4. outline the laboratory tests used in evaluating thyroid status
5. describe hyperthyroid symptoms and the diseases which cause hyperthyroidism
6. describe hypothyroid symptoms and the diseases which cause hypothyroidism
7. construct a flow chart for identifying thyroid diseases by laboratory tests
8. discuss the treatment of various thyroid diseases
9. state the roles of antibodies in causing thyroid diseases

THE THYROID GLAND

The thyroid gland is located in the middle of the lower neck in front of the trachea just above the suprasternal notch. It is 12-20 g in size and has two butterfly shaped lobes connected by an isthmus. Four parathyroid glands are in the posterior region of each pole of the thyroid.

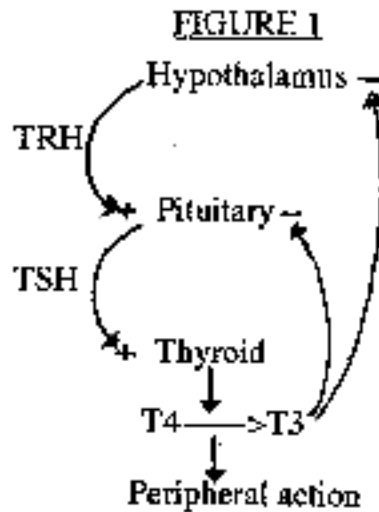
The gland contains numerous follicles that surround a colloid filled space containing thyroglobulin (the precursor to thyroid hormones). Thyroid stimulating hormone binds to receptors (TSH-R) on the follicular cells and controls the production and secretion of thyroid hormones into the blood stream.

THYROID HORMONES

Production: the two thyroid hormones, thyroxine (tetraiodothyronine (T4) and triiodothyronine (T3)) are produced in the thyroid follicles from dietary iodine and the amino acid, tyrosine. Thyroxine contains 4 iodine atoms. De-iodination of one iodine by the enzyme, deiodinase, leads to the production of the potent hormone, T3. Thyroid peroxidase (TPO) is involved in 2 steps of the biosynthetic pathway.

Dietary iodine is required for hormone production. Iodine is naturally available in areas of the world close to coastlines since products of the sea contain it. In inland areas of the world there may not be sufficient iodine available in the diet. In these areas hypothyroidism may result from lack of iodine unless the diet is supplemented. In the United States adequate iodine is generally available through iodized salt. However, in some inland areas of the world supplementation is not available or is not accepted. In these areas cretinism and goiter (enlarged thyroid gland) are still present.

Thyroid stimulating hormone (TSH), produced by the pituitary, stimulates thyroid hormone synthesis and secretion. TSH in turn is under control of thyroid regulating hormone (TRH) produced by the hypothalamus. This is an example of a biofeedback control system (Figure 1). Thyroid hormones feed back to control TRH and TSH production. Increase in thyroid hormones causes decrease in TRH and TSH. Decrease in thyroid hormones causes increase in TRH and TSH. TSH establishes the level or "set point" of this equation. The TSH level is a sensitive and specific marker of thyroid function.



Transport of thyroid hormones: The amount of T4 secreted from the thyroid gland is at least 20 times greater than T3. After secretion both are bound to plasma proteins, thyroxine-binding globulin (TBG), transthyretin (TTR) and albumin. About 99.98% of T4 is bound and 99.7% of T3 is bound. Protein bound thyroid hormones do not enter cells, thus are biologically inactive and serve as storage reservoirs for circulating thyroid hormones. Minute amounts of free hormones readily enter cells by specific membrane transport mechanisms. These thyroid hormones act by binding to nuclear receptors called thyroid hormone receptors (TR) α and β . Both are expressed in most tissues, but their relative levels of expression vary among organs. T3 has 10 to 15 times more affinity for the receptors than T4. The combination of decreased affinity of T3 for transport proteins (i.e., more free T3) combined with its increased affinity for tissue receptors helps explain its greater hormonal potency than T4.

Function: Thyroid hormones play a critical role in cell differentiation during development. Lack of thyroid hormones *in utero* and infancy can result in cretinism, a condition characterized by severe mental and growth retardation. Treatment is iodine or hormone replacement during pregnancy as well as in the neonate and in childhood.

In the adult the hormones maintain temperature and metabolic homeostasis. They maintain the rate at which the body uses fats and carbohydrates, influence the heart rate and help regulate the production of protein. Thyroid hormones increase tissue oxygen consumption and heat production. They increase the absorption of glucose from the intestine and by tissues, thus regulating the rate of metabolism.

Increase in thyroid hormones causes an increase in the body's metabolism called hyperthyroidism. Decrease in the hormones causes decrease in the body's metabolism, called hypothyroidism.

LABORATORY TESTING

The development of sensitive TSH tests and accurate free T4 (FT4) and free T3 (FT3) measurements have significantly changed laboratory testing for thyroid abnormalities. Several of the older thyroid tests used to estimate FT4 and FT3 levels are now considered obsolete. At present the recommended laboratory tests include TSH, FT4, FT3 and thyroid antibodies.

TSH

Serum TSH measurement is the single most reliable test to diagnose all common forms of hypothyroidism and hyperthyroidism.

Euthyroid (normal) levels have been 0.4-4.0 mIU/L. However, the American Association of Clinical Endocrinologists (1) recommends that the upper limit of the serum TSH euthyroid reference range be reduced to 3.0 mIU/L. The National Association of Clinical Biochemists (2) recommends that the normal upper limit be 2.5 mIU/L. These recommendations are because of the possible inclusion of mild hypothyroid individuals in the population used to establish the higher normals.

For over 25 years TSH methods have been able to detect the TSH elevations that are characteristic of primary hypothyroidism. Modern-day TSH methods have enhanced sensitivity and are also capable of detecting the low TSH values typical of hyperthyroidism. These new methods are often based on monoclonal antibody based immunometric (IMA) principles and are available on a variety of automated immunoassay analyzer platforms. Most of the current methods are capable of achieving a functional sensitivity of 0.02mIU/L or less, which is a necessary detection limit for the full range of TSH values observed between hypo- and hyperthyroidism. With this level of sensitivity, it is possible to distinguish the profound TSH suppression typical of severe Graves' thyrotoxicosis (TSH <0.01mIU/L) from the TSH suppression (0.01-0.1 mIU/L) observed with mild hyperthyroidism and in some patients with a non-thyroidal illness.

However, other conditions may also cause decrease or suppression of TSH. These include endogenous depression, central hypothyroidism (pituitary or hypothalamic disease), resistance to thyroid hormone and corticosteroid therapy. TSH can also be low or suppressed when TSH receptor antibodies are present, even in patients with normal or low thyroid function. TSH receptor antibodies may be blocking or stimulating antibodies. Stimulating TSH receptor antibodies, which are also known as thyroid stimulating immunoglobulins or TSI, are the cause of Graves' disease, and for this reason the TSH test may be misleading in the diagnosis of Graves' disease.

In the last decade the diagnostic strategy for using TSH measurements has changed as a result of the sensitivity improvements in these assays. It is now recognized that the TSH measurement is a more sensitive test than FT4 for detecting both hypo- and hyperthyroidism. As a result, some countries now promote a TSH-first strategy for diagnosing thyroid dysfunction in ambulatory patients. Other countries still favor the TSH + FT4 panel approach, because the TSH-first strategy can miss patients with central hypothyroidism or TSH-secreting pituitary tumors.

Free T4 (FT4) and Free T3 (FT3):

Free T4 or Free T3 tests are frequently sent to reference laboratories to be performed. The most accurate procedure, equilibrium dialysis, is demanding and not cost effective for doing in a clinical laboratory.

The older thyroid tests for estimating free T4 and T3 are now seldom performed in modern clinical laboratories. These outmoded tests include total T4 (TT4) and total T3 (TT3), T3 uptake, and FT4 index (FTI) or estimate. The reasons for eliminating these tests include the following:

TT4 and TT3 levels measure the thyroid hormone bound to the proteins that carry the hormone through the blood (biologically inactive form) plus the free hormone

(biologically active form). When binding protein levels are elevated, levels of T4 and T3 are elevated. Thus the measure of total T4 when binding proteins are increased does not reflect actual thyroid function and is considered a false elevation. It was found that increased total T4 or T3 was more often due to increase in binding proteins than to abnormal thyroid function. Binding proteins are elevated in pregnancy, in patients on estrogens including contraceptives, and in patients taking many other medications.

T3 uptake test and the FT4 index: The T3 uptake does not measure T3. It is used to measure the binding proteins that carry thyroid hormone through the bloodstream. From this result and a total T4 result, the approximate amount of free T4 (FT4) can be calculated. This result is the FT4 index, (FTI). Values of FTI do not correlate well with direct measurements of FT4, and the FT4 assay is considered more reliable.

Thyroid Antibody Testing:

Tests for three thyroid antibodies are available. These tests are helpful in evaluating thyroid cases that are difficult to diagnose.

- Thyroperoxidase antibodies (TPOAb)
- Thyroglobulin antibodies (TgAb)
- Thyrotropin receptor antibodies (TRAb)

THYROID DISEASES

Diseases of the thyroid are common in adults, with higher incidence in women than in men. They are divided into two main categories, hyperthyroidism and hypothyroidism. The American Thyroid Association (ATA) (3) states, "Thyroid dysfunction is common in adults and frequently has significant clinical consequences. Hypothyroidism and hyperthyroidism can be accurately diagnosed with laboratory tests and are readily treatable. Clinical manifestations of thyroid dysfunction vary considerable among patients in their character and severity. Associated symptoms and signs are often nonspecific and progress slowly. Consequently, the accuracy of clinical diagnosis is limited. If only patients presenting with clearly suggestive symptoms and signs are evaluated, many affected individuals will remain undiagnosed. For these persons, appropriate treatment or monitoring can only be implemented when routine laboratory screening identifies them."

ATA's recommendations include

- screening of all newborn children for hypothyroidism (already a widely accepted and legislatively mandated practice).
- performing serum TSH measurement every 5 years starting at 35 years of age.

Disorders of the thyroid are most frequently due to autoimmune processes that either cause glandular destruction and underproduction of thyroid hormones (hypothyroidism) or stimulate the overproduction of thyroid hormones (thyrotoxicosis—hyperthyroidism). Autoimmune thyroid disease (AITD) causes cellular damage and alters thyroid gland function by humoral and cell-mediated mechanisms. Cellular damage occurs when sensitized T-lymphocytes and/or autoantibodies bind to thyroid cell membranes causing cell lysis and inflammatory reactions. Alterations in thyroid gland function result from the action of stimulating or blocking autoantibodies on cell membrane receptors.

Antibodies to three principal thyroid antigens are involved in AITD. These antigens are thyroperoxidase (TPO), the TSH receptor and thyroglobulin (Tg). TPO antibodies (TPOAb) have been involved in the tissue destructive processes associated with the hypothyroidism observed in Hashimoto's and atrophic thyroiditis. The appearance of TPOAb usually precedes the development of thyroid dysfunction. TSH receptor autoantibodies (TRAb) are heterogeneous and may either mimic the action of TSH and cause hyperthyroidism as observed in Graves' disease or alternatively, antagonize the action of TSH and cause hypothyroidism. The latter occurs most notably in the neonate as a result of a mother with antibodies due to AITD. The pathologic role of TgAb remains unclear.

Other disorders include thyroiditis and neoplastic processes which can result in benign nodules or thyroid cancer.

HYPERTHYROIDISM

Increase in thyroid hormones causes an increase in the body's metabolism called hyperthyroidism. Thyrotoxicosis is the clinical term applied to the state of thyroid hormone excess, but the term hyperthyroidism is commonly used.

The signs and symptoms of hyperthyroidism are listed in Table I.

Table I Hyperthyroidism

Fatigue	Weight loss
Heat intolerance	Warm, smooth skin
Nervousness/emotional lability/insomnia	Muscle cramps/myalgias
Tachycardia/palpitations	Dyspnea
Hyperdefecation	Menstrual irregularity-menorrhagia
Eyestare, lid lag, proptosis (Graves' disease)	

The causes of hyperthyroidism include

- Graves' disease
- Hyperfunctioning thyroid nodules (toxic multinodular goiter, toxic adenoma)
- Thyroiditis

Graves' Disease: The most common cause of hyperthyroidism is Graves' disease, named after Dr. Robert Graves, an Irish physician, who first described the disease over 150 years ago. Graves' disease occurs in up to 2% of women, most frequently between 20-50 years of age. It is 10 times more common in women than men.

The disease is triggered by an autoimmune response in which antibodies (TRAb) bind to the surface of thyroid cells and stimulate the overproduction of thyroid hormone. In a number of cases the triggering mechanism seems to be emotional stress such as the death of a loved one. However, not all cases can be traced to such a stressful incident; viral infection and unknown etiology have also been noted. Genetic predisposition has been shown; monozygotic twins have 20-30% concordance rate and the risk is increased among siblings. HLA-DR3 in Caucasians is associated with increased incidence of Graves' disease as well as autoimmune hypothyroidism.

The disease is diagnosed by a combination of signs and symptoms found in hyperthyroidism (Table 1) along with a TSH below 0.1 mIU/L. In some patients, signs specific to Graves' disease, ophthalmopathy and dermopathy, occur.

Graves' ophthalmopathy is caused by swelling of tissues and muscles behind the eye. This condition can vary from lid retraction to proptosis, where the eyeballs are pushed forward and protrude.

Thyroid dermopathy occurs in less than 5% of patients with Graves' disease. It usually manifests as swelling and pink to purple discoloration of the skin over the lateral and anterior shin.

Subclinical hyperthyroidism (TSH low, but FT4 and FT3 normal) has been shown to have deleterious effects on the patient's health. It contributes to increased rate of bone loss, cardiac hypertrophy and atrial fibrillation. In addition, there is increased risk of development of overt hyperthyroidism. Identifying and treating these patients may prevent these sequelae.

Laboratory findings in Graves' disease:

For the diagnosis of Graves' disease, the TSH, FT4, and FT3 are the primary laboratory tests needed to establish the presence of hyperthyroidism. TSH level is decreased below 0.1 mIU/L along with increase in the T4 level. In 2-5% of the patients the T4 is normal but the T3 is increased. When the FT4 is normal, it is important that tests for FT3 be run because many patients with both Graves' disease and toxic multinodular goiter will have elevated FT3 levels and normal FT4 levels.

Antibodies to TSH receptor (TRAb) are usually present.

Treatment of Graves' disease:

The treatment of choice at present in the United States is radioactive iodine. The radioactive iodine causes ablation of thyroid tissue, the amount of tissue destroyed depends on the radioactive dose given to the patient. Most patients become hypothyroid eventually after this treatment and need to be followed to determine when thyroid replacement therapy is needed. This treatment regimen usually resolves the hyperthyroidism with a minimum of hypothyroid problems.

Other treatments include surgical removal, now uncommonly done in the U.S., or the administration of antithyroid drugs, methimazole or propylthiouracil; however remission rates are variable and relapses are frequent with these drugs. This latter therapy is most commonly used in pregnant women with hyperthyroidism since radioactive iodine crosses the placenta and damages the fetus.

Hyperfunctioning thyroid nodules:

Nodular thyroid disease is characterized by disordered growth of thyroid follicles. Nodular disease is common, occurring in 3-7% of adults when assessed by physical examination. In multinodular disease palpation of the thyroid reveals an enlarged gland (goiter) with irregular nodules of varying size. The TSH is low. T4 may be normal or slightly increased. T3 is often elevated more than T4. Thyroid scan shows heterogeneous uptake with multiple regions of increased and decreased uptake. Treatment is difficult and may consist of antithyroid drugs, radioactive iodine or surgery.

A "hot" solitary nodule is called toxic adenoma. The TSH level is low and free T4 or T3 may or may not be increased. A thyroid scan reveals the hyperfunctioning nodule.

Thyroiditis:

Acute thyroiditis is rare and is due to a suppurative infection of the thyroid. The patient has pain, a tender goiter and is febrile. A fine needle aspirate of the thyroid is cultured and antibiotic treatment appropriate to the organism involved is instituted.

Subacute thyroiditis usually has a viral etiology. Various viruses have been implicated. The patient presents with a painful and enlarged thyroid and may be febrile. The hyperthyroidism is due to release of stored thyroid hormone from the inflamed gland. The disease is self limited.

Silent thyroiditis (painless) is thought to have an autoimmune etiology and has a course similar to subacute thyroiditis. It is common in postpartum women.

HYPOTHYROIDISM

Decrease in thyroid hormones causes a slow-down in metabolism, called hypothyroidism. Women, especially those over 40, are more likely to have decreased thyroid activity. By age 60, up to 17% of women may have hypothyroidism.

The symptoms of hypothyroidism are related to the duration and severity of the disease, the rapidity with which it occurs, and the psychological characteristics of the patient. At first the symptoms may be subtle, but as the disease progresses, more obvious symptoms develop.

The signs and symptoms of hypothyroidism are listed in Table II.

Table II Hypothyroidism

Fatigue	Weight gain
Cold intolerance	Dry, coarse, thick skin (myxedema)
Hair dryness/loss	Depression/memory impairment
Dementia ("myxedematous madness")	Muscle weakness/tremor/decreased reflexes
Bradycardia	Edema, esp. pre-orbital and peripheral
Constipation	Menstrual irregularity-hypomenorrhea
Infertility	Thick tongue/hoarseness/slow speech

The main causes of hypothyroidism in the United States are

- chronic autoimmune thyroiditis
- iatrogenic
 - removal of the thyroid gland
 - treatment with radioactive iodine
 - external irradiation
 - drugs such as lithium or interferon
- replacement of the gland by tumor

Laboratory tests to diagnose hypothyroidism:

High TSH indicates that hypothyroidism exists. This is followed by FT4 which is decreased in symptomatic disease, normal in subclinical hypothyroidism. Thyroperoxidase antibodies are present in Hashimoto's disease. A thyroid scan or ultrasonography may be necessary to evaluate evidence of structural abnormalities.

Autoimmune thyroiditis (Hashimoto's disease):

The patient presents with a number of symptoms outlined in Table II. The diagnosis is established by a high TSH and low FT4. Thyroid nodules may be present and are associated with a 5% risk of development of thyroid cancer.

Autoantibodies (TPO) are present in 95% of the cases and high titers are useful to make the diagnosis. These antibodies cause destruction of thyroid tissue, resulting in a decrease in production of thyroid hormones. A virus or bacterium may trigger the response, or genetic abnormality may be involved.

The treatment is hormone replacement by levothyroxine. The dose is tailored to the individual patient and periodic follow-ups are done.

Subclinical hypothyroidism:

Subclinical hypothyroidism is important to diagnose and treat. The condition occurs in 1-10% of the adult population with the higher frequency in women and the elderly. It is usually found on a routine screening TSH. Although patients can be asymptomatic, some have subtle findings including alterations in lipid metabolism, cardiac, gastrointestinal, neuropsychiatric and reproductive abnormalities and an increased likelihood of developing overt hypothyroidism.

Laboratory findings are an increase in the TSH but with a normal FT4.

Treatment is controversial but the American Association of Clinical Endocrinologists (1) recommends that treatment with levothyroxine is indicated when the TSH is over 10 mIU/L or in patients with TSH levels between 5-10 mIU/L if goiter or positive TPO antibodies are present.

Hypothyroidism in pregnancy:

Untreated symptomatic hypothyroidism in pregnancy may increase the risk of hypertension, pre-eclampsia, anemia, cardiac dysfunction, abortion, fetal death, low birth weight and possible abnormal brain development. Even subclinical hypothyroidism may decrease the cognitive function of the fetus, which can be prevented by hormone replacement.

Levothyroxine therapy is safe during pregnancy and should be given if there is evidence of hypothyroidism.

The AACE recommends that TSH measurement be done before pregnancy or during the first trimester.

Thyroid cancer:

Tumor of the thyroid can replace normal thyroid tissue causing decrease in thyroid hormone production. The incidence of thyroid cancer (about 9/100,000) increases with age and plateaus after age 50. It is easy to diagnose because thyroid nodules are readily palpable allowing early detection and biopsy by fine needle aspiration. Radioactive iodine can be used to diagnose and treat differentiated cancer. The incidence is increased in those who had external radiation of the neck, face and head when young, or exposure to environmental radiation such as in the Chernobyl disaster. Treatment is surgery, TSH suppression therapy or radioiodine.

Other Causes of hypothyroidism:

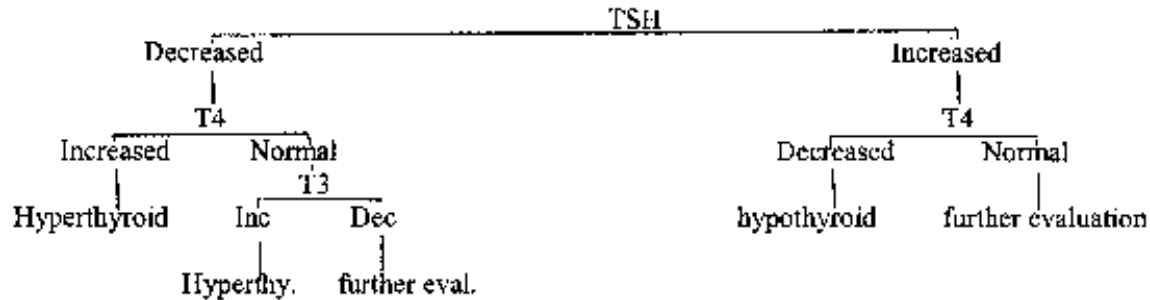
- Iatrogenic—surgical removal, gland ablation with radioiodine, external irradiation, or drugs such as lithium or interferon.
- Secondary causes—pituitary or hypothalamic disease.

The patient should be assessed for the cause and given treatment appropriate to the condition.

Sick Euthyroid Syndrome:

Any severe acute or chronic illness may cause abnormalities of circulating TSH or thyroid hormone levels without thyroid disease. The major cause of these changes is the release of cytokines. These misleading measurements in acutely ill patients make evaluation of the patient's thyroid status difficult for the physician. Unless a thyroid disorder is strongly suspected, thyroid testing should be delayed until the patient has recovered.

FLOW CHART FOR LABORATORY DIAGNOSIS OF THYROID DISEASE



DISCUSSION OF CASE STUDIES

Case Study #1: Frenetic Flora

Question 1. What is Flora's probable diagnosis?

According to her symptoms and TSH below 0.1 mIU/L, Flora probably has hyperthyroidism—Graves' disease.

Question 2. What is the incidence of this?

It occurs in about 2% of women, most frequently between 20 and 50 years of age.

Question 3. What tests should be done next?

A FT4 should be done. If that is increased, no further tests are necessary. If the FT4 is normal, a FT3 should be done because some Graves' disease patients may show an increased FT3 but a normal FT4.

Question 4. What is the treatment and prognosis?

Treatment is thyroid tissue ablation using radioactive iodine. The amount of radioactive iodine is tailored by the physician to her condition. This treatment eventually results in development of a hypothyroid state. Flora should be evaluated regularly for her thyroid status. When hypothyroidism develops, she should be treated with levothyroxine.

Case Study #2: Freezing Frank

Question 1. What is Frank's probable diagnosis?

According to his symptoms and TSH above 10 mIU/L Frank has hypothyroidism.

Question 2. What is the most common cause of this disease?

The most common cause of this disease is thyroperoxidase antibodies which destroy thyroid tissue, a condition called Hashimoto's thyroiditis.

Question 3. What test should be done next?

The Free T4 test should be done. It should show reduced T4.

Question 4. What is the treatment and prognosis?

The treatment is to give levothyroxine. Frank should be evaluated regularly for his thyroid status.

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Review Questions

Course #056-967

Choose the **one** best answer

1. Thyroid hormone synthesis is under control of
 - a. thyroperoxidase
 - b. thyroglobulin
 - c. thyroid stimulating hormone
 - d. transthyretin
2. An enzyme involved in thyroglobulin synthesis is
 - a. TPO
 - b. THR
 - c. TSI
 - d. Tg
3. Thyroglobulin is synthesized from
 - a. iodine and alanine
 - b. bromine and methionine
 - c. lithium and tyrosine
 - d. iodine and tyrosine
4. Lack of iodine in the fetus/neonate may cause
 - a. Graves' disease
 - b. Cretinism
 - c. Hashimoto's thyroiditis
 - d. thyrotoxicosis
5. Increased binding proteins in the peripheral blood cause an increase in
 - a. Total T4
 - b. Free T4
 - c. thyroperoxidase
 - d. Free T3
6. A TSH of .03 mIU/L is indicative of
 - a. Hashimoto's thyroiditis
 - b. Graves' disease
 - c. thyroid cancer
 - d. euthyroid
7. Antibodies most often found in Graves' disease include
 - a. anti-de-iodinase
 - b. anti-peroxidase
 - c. anti-Tg
 - d. anti-thyrotropin receptor
8. A patient with increased irritability, heat intolerance and weight loss has a TSH of 0.01 mIU/L and a normal free T4. What test should be done next?
 - a. anti-Tg antibody
 - b. TRH hormone
 - c. Total T4
 - d. Free T3

9. The hormone with the greatest amount of biologic activity is
 - a. Total T4
 - b. Free T4
 - c. thyroglobulin
 - d. Free T3
10. The most reliable test to diagnose thyroid disease is
 - a. TSH
 - b. Free T4
 - c. T3 uptake
 - d. Free T3
11. All but which of the following are characteristic of hypothyroidism
 - a. cold intolerance
 - b. Myxedema
 - c. proptosis
 - d. bradycardia
12. Thyroperoxidase antibodies are commonly found in
 - a. Graves' disease
 - b. toxic adenoma
 - c. patients on lithium therapy
 - d. Hashimoto' thyroiditis
13. The recommended treatment for hyperthyroidism in pregnancy is
 - a. propylthiouracil
 - b. radioactive iodine
 - c. interferon
 - d. surgery
14. Subacute thyroiditis
 - a. causes formation of thyroid nodules
 - b. is due to a suppurative bacterial infection
 - c. has a viral etiology
 - d. requires treatment with radioactive iodine
15. A patient without any specific symptoms has a TSH of 8 mIU/L and a normal T4. What is the most probable diagnosis?
 - a. euthyroid
 - b. subclinical hyperthyroidism
 - c. overt hypothyroidism
 - d. subclinical hypothyroidism
16. If the patient in question #15 has increased TPOAb, what is the recommended treatment?
 - a. nothing
 - b. levothyroxine
 - c. radioactive iodine
 - d. methimazole
17. Thyrotropin receptor antibodies (TRAb)
 - a. usually cause destruction of thyroid tissue
 - b. can be either stimulating or blocking
 - c. are found in Hashimoto's thyroiditis
 - d. are implicated in thyroid tumor formation

18. Thyroid hormone transport proteins include all the following except
- thyroglobulin
 - transthyretin
 - albumin
 - thyroid-binding globulin
19. Secondary hypothyroidism is due to
- cancer of the thyroid
 - resistance to thyroid hormones
 - pituitary disease
 - over-treatment with levothyroxine
20. The T3 uptake test
- is currently used in lieu of the free T4 assay
 - measures the amount of total T3 present
 - is a calculation of the free T3 plus the free T4
 - does not correlate well with direct measurements of FT4

Record your answers on the Distance Learning Course Registration Form & Answer Sheet below.

Thyroid Hormones and Thyroid Diseases

CAMLT Course # DL-967

2.0 CE Credits

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2.0 CE Fee = \$24.00 for members (\$30.00 for nonmembers)

Please circle the one best answer for each question.

- | | | | | | | | | | |
|-----|---|---|---|---|----|---|---|---|---|
| 1. | a | b | c | d | 11 | a | b | c | d |
| 2. | a | b | c | d | 12 | a | b | c | d |
| 3. | a | b | c | d | 13 | a | b | c | d |
| 4. | a | b | c | d | 14 | a | b | c | d |
| 5. | a | b | c | d | 15 | a | b | c | d |
| 6. | a | b | c | d | 16 | a | b | c | d |
| 7. | a | b | c | d | 17 | a | b | c | d |
| 8. | a | b | c | d | 18 | a | b | c | d |
| 9. | a | b | c | d | 19 | a | b | c | d |
| 10. | a | b | c | d | 20 | a | b | c | d |

Distance Learning Evaluation Form

According to state regulations, this evaluation must be completed and returned in order to receive CE hours. Your comments help us to provide you with better continuing education materials in the distance learning format. Please circle the number that agrees with your assessment.

- Overall, I was satisfied with the quality of this Distance Learning course.
(strongly agree) 5 4 3 2 1 (strongly disagree)
- The objectives of this Distance Learning course were met.
(strongly agree) 5 4 3 2 1 (strongly disagree)
- The difficulty of this Distance Learning course was consistent with the number of CE hours.
(strongly agree) 5 4 3 2 1 (strongly disagree)
- I will use what I learned from this Distance Learning course.
(strongly agree) 5 4 3 2 1 (strongly disagree)
- The time to complete this Distance Learning course was: _____ hours
- Please comment on this Distance Learning course on the back of this sheet.

What did you like or dislike?