Although neoplastic diseases of the thyroid are the most common problem requiring surgical intervention in the neck, a thorough understanding of nonneoplastic diseases of the thyroid is required of the otolaryngologist. The most common worldwide nonneoplastic process of the thyroid is iodine deficiency, which is estimated to affect approximately three-quarters of a billion people. Iodine deficiency can lead to thyroid goiter, and severe deficiency to cretinism.

In the United States, the addition of iodine to food products such as salt leads to iodine excess, a process which some believe responsible for Hashimoto’s disease. Other problems iodine excess induce include thyrotoxicosis and hypothyroidism with Grave’s disease.

Thyroid function tests are the most common method of identifying nonneoplastic diseases of the thyroid, although some may be identified on physical exam alone. A thorough knowledge of the anatomy, embryology and especially physiology of the thyroid is a prerequisite to suspecting, understanding and treating these diseases.

**Anatomy**

The word thyroid is greek, and loosely translated means “shield”. The thyroid gland weighs approximately twenty grams, but is slightly larger in pregnant and menstruating females. It is located on the anterior superior aspect of the trachea, usually around the third tracheal ring, and spreads laterally both superiorly and inferiorly to “shield” the medial neck. The gland is encircled by the visceral layer of the deep cervical fascia. The overlaying strap muscles are encircled by the superficial layer of the deep cervical fascia. The lateral lobes are divided into superior and inferior poles, with the medial portion termed the isthmus. There is often a pyramidal lobe, which originates in the midline and rises superiorly towards the hyoid bone. The gland is firmly attached to the laryngotracea by septae interconnecting the visceral layers of the deep cervical fascia, as well as by firm fibrous tissue termed Berry’s ligament.

The **blood supply** is from the paired superior and inferior thyroid arteries, with an infrequent contribution from the thyroid ima artery, itself a branch from either the brachiocephalic artery or the aorta. The superior thyroid artery is usually the first anterior branch of the external carotid artery, although it rarely is a direct branch from the common carotid artery just before the bifurcation into the external and internal carotid arteries (a rare exception to the rule that there are no branches of the common carotid
artery). It descends in the neck under cover from the sternothyroid muscle before supplying the thyroid gland on its anterior aspect. Accompanying the superior thyroid artery above the level of the superior pole of the gland is the external branch of the superior laryngeal nerve, the motor nerve to the cricothyroid muscle. At the level of the superior pole, the nerve courses medial to the gland. High ligation of the artery therefore places the nerve at risk. Ligation of this nerve leads to a tremulous voice with difficulty obtaining a high pitch, and rotation of the posterior larynx towards the damaged nerve. High ligation of the nerve may also produce deficits of the internal branch of the superior laryngeal nerve, which provides sensation to the ipsilateral larynx and is responsible for the ipsilateral cough reflex.

The inferior thyroid artery arises from the thyrocervical trunk, itself a branch of the subclavian artery. The artery ascends the neck in the tracheoesophageal groove before supplying the thyroid gland from its medial aspect. During its ascent, it usually is in intimate contact with the recurrent laryngeal nerve. Its anatomical location with respect to the nerve is not constant, however, as it may be located anteriorly, posteriorly or even divide to encompass the nerve.

The venous drainage of the thyroid gland is via three paired veins, the superior, middle and inferior thyroid veins. The superior and middle veins drain directly into the internal jugular vein, whereas the inferior veins drain into the brachiocephalic and subclavian veins. The superior thyroid vein runs with the superior thyroid artery, but the inferior thyroid vein usually runs anteriorly on the gland, whereas the inferior thyroid artery is located posteriorly.

**Histology**

The functional units of the thyroid gland are the thyroid follicles, which are spheroidal structures made of cuboidal epithelial cells surrounded by a basement membrane. Each follicle contains variable amount of colloid, the stored form of tri- and tetraiodothyronine. Active follicles tend to be smaller. Within the basement membrane are the parafollicular “C” cells. The normal thyroid follicles are highly eosinophilic, whereas the parafollicular “C” cells cytoplasm tends not to stain.

**Embryology**

The thyroid gland develops as an invagination of the endoderm between the first and second pharyngeal pouches immediately caudal to the tongue bud. It grows inferiorly as the median diverticulum, the tip of which bifurcates into the lateral and medial lobes of the gland. The connection of the median diverticulum to the pharynx is termed the thyroglossal duct, and is marked in the oropharynx by the foramen cecum of the tongue. In the full term embryo, the duct runs from the foramen cecum just anterior to the hyoid bone, where a recurrent loop wraps under the the hyoid before progressing caudally once again to the thyroid gland. The distal part of the duct often differentiates into the
pyramidal lobe of the thyroid, or into the levator muscle of the thyroid. Occasionally, remnants of the thyroglossal duct persists, portions of which may become prominent midline neck masses known as thyroglossal duct cysts. Aberrant descent of the gland may result in ectopic thyroid glands. The most common location is the base of tongue, which is a result of the complete failure of the gland to migrate. Other locations are anywhere along the descent of the thyroglossal duct, including inferiorly to the normal location of the gland.

The parafollicular “C” cells of the thyroid gland are believed to derive from the ultimobranchial apparatus, the most caudal aspect of the pharyngeal pouches. Most lower vertebrates have a separate organ called the ultimobranchial organ, and this is known to arrive from the ultimobranchial apparatus. The organ secretes calcitonin. Parafollicular cells of the thyroid secrete calcitonin and are histologically similar to the ultimobranchial organ of lower vertebrates.

**Physiology**

The primary function of the thyroid gland is the synthesis and release of thyroid hormones, tetraiodothyronine (T4) and triiodothyronine (T3). The primary hormone released is T4, although smaller amounts of T3 are released. In the peripheral bloodstream, T4 is converted to T3. T3 has a more powerful action than T4, as well as a longer half-life. Many believe T4 serves only as a precursor to T3, and has little effect on function.

The production and release of thyroid hormones are stimulated by thyroid stimulation hormone (TSH), a hormone secreted by the anterior pituitary in response to thyroid releasing hormone (TRH), itself released by the hypothalamus. T4 and T3 also have a negative feedback on the release of TSH by the anterior pituitary. It is generally felt the negative feedback of T4 is the most important component in the level of TSH. For example, a two-fold decrease in free T4 leads to an approximate 100-fold increase in TSH.

Most cells within the body have T3 receptors, as coded by the c-erb A gene found on chromosomes 17 and 3. Once in the cell, T3 binds to specific nuclear sites and regulates gene function. Absence of the c-erb A genes leads to a relative peripheral resistance to thyroid hormone.

The production of thyroxine by the thyroid begins with the joining of TSH and the TSH receptor of the thyroid follicle. This causes a chain of events to occur within the thyroid gland, all of which are mediated by cAMP. The process begins with the Na/K ATPase mediated active transport of iodide against a concentration gradient into the cell. The synthesis of thyroglobulin by the endoplasmic reticulum is upregulated. Once in the cell, iodide is oxidized to a higher valence state similar to iodine via thyroperoxidase, and
the iodine then joins with tyrosine and thyroglobulin to form the stored form of thyroid hormone, called thyroid colloid. Once again, thyroperoxidase is the catalyst. Via pinocytosis, colloid is removed from the lumen where lysosomes degrade the endocytic vesicles and the thyroglobulin to T4, T3 and mono-and –diiodotyrosines. The T4 and T3 are secreted into the bloodstream.

**Thyroid Function Tests**

There is no adequate test to measure the response of tissue to thyroid hormones, therefore thyroid function is determined by measuring thyroid hormone levels, including T4, T3, TSH and thyroid iodine uptake.

The TSH level is the most commonly used test. The usual method involves the use of two monoclonal antibodies, each with an affinity for a portion of the TSH molecule. The use of the two monoclonal antibodies affords a high specificity and broad range of accurate test levels. This test is allows an accurate assessment of primary or peripheral causes of hypothyroidism.

Total T4 levels are misleading, as it is the free form of T4 that is active. Total T4 levels are altered by serum protein levels, including thyroid binding globulin (TBG), albumin and prealbumin. Free T4 can be directly determined via membrane dialysis, but the test is cumbersome, not readily available and lengthy. Instead, free T4 is estimated by multiplying the total T4 level by the percent of unoccupied protein binding sites on the thyroglobulin molecule. The percent of unoccupied binding sites is determined by the T3 Resin Uptake test (T3RU), which is performed by adding I131 labeled T3 to the patient’s serum and then adding the serum/I131 mixture to a resin with affinity for unoccupied sites. The amount of I131 labeled T3 is then measured. Of note, this test may be inaccurate when TBG concentration is markedly altered.

Although infrequently used anymore, an estimate of thyroid function can be made by the thyroid radioiodine uptake test (RAIU). This test administers I123 intravenously and scans the thyroid for radioactive uptake at set time intervals. Euthyroid patients generally have peak thyroid iodide trapping times between 24 and 48 hours of between 8 and 30% of total administered dose. This broad “normal” range of uptake makes assessment difficult. Further complicating matters in the United States is the high iodide diet, making selective uptake of I131 more difficult.

Thyroid autoantibodies to microsomes or thyroglobulin are sometimes used to assess autoimmune thyroiditis. These tests should be interpreted with caution, however, as antibody levels correlate with a patient’s age and are frequently found in both thyroid and nonthyroid diseases. A diagnosis of an autoimmune disease should therefore not be made on antibody presence alone, but also requires an abnormal TSH level.
HYPOTHYROIDISM

Acquired Hypothyroidism

Hypothyroidism is associated with numerous symptoms, including lethargy, hoarseness, deafness, thick dry skin, severe constipation, cold intolerance and a stiff gait. These symptoms are often attributed to the elderly, leading to a delay in or failure to diagnose. This is particularly troublesome as the incidence of hypothyroidism increases with age. More than 15% of those above the age of 65 have subclinical disease as determined by elevated TSH levels. Females are affected approximately five times more often. Risk factors other than age and gender include autoimmune diseases, goiter, prior radiation therapy or the presence of laryngectomy.

Myxedema coma is a thyroid emergency that is a late manifestation of severe hypothyroidism. Symptoms include coma, hypothermia, bradycardia, pleural effusions, electrolyte imbalances and hypoventilation. Seizures usually occur. Underlying illness usually exacerbates the underlying symptoms. Treatment is with large doses of corticosteroids and intravenous thyroxine. Mortality is 50%.

Hypothyroidism falls into one of four categories, primary, secondary, tertiary or peripheral resistance. Each type may be further subdivided into goitrous or nongoitrous. Primary hypothyroidism is caused by the thyroid gland, secondary by the pituitary, tertiary by the hypothalamus and peripheral resistance is often due to a genetic abnormality in the c–erb A gene from chromosomes 17 or 3. Primary hypothyroidism accounts for the majority of cases.

The most common cause of goitrous hypothyroidism are autoimmune diseases, primarily Hashimoto’s disease, although less common causes include medications such as lithium, amiodarone and iodide containing compounds. Of note, Hashimoto’s thyroiditis is transient in about 10% of patients, and a return of normal thyroid function can often be seen. This is usually found in those with large goiters and initially highly elevated TSH levels. Nongoitrous hypothyroidism is most often caused by concomitant thyroid disease such as glandular atrophy during the management of Grave’s disease with I131, but is more rarely caused by hypothalamic or pituitary abnormalities.

Excessive iodine intake can cause an autoimmune thyroiditis and subsequent hypothyroidism in certain patients, with a high correlation seen in those with spina bifida. Those in renal failure excrete iodide poorly and this can also lead to a thyroiditis and hypothyroidism.

Amiodarone given to pregnant women is known to cause transient hypothyroidism in the neonate, and every newborn whose mother is taking amiodarone should be checked. The hypothyroidism is exacerbated by many prenatal vitamins which may contain iodide.
Tertiary hypothyroidism is uncommon and is usually caused by large pituitary or hypophyseal tumors. Other causes include hypothalamic infarction infection.

**Iatrogenic Hypothyroidism**

Iatrogenic hypothyroidism is usually due to thyroid surgery, radioactive iodine therapy or laryngectomy with radiation therapy. All three procedures require periodic TSH levels and adjustment of thyroid hormone replacement therapy.

Hypothyroidism after thyroidectomy usually occurs within the first postoperative year, and is the most common cause of iatrogenic hypothyroidism. It is more common with autoimmune thyroid diseases, particularly those with thyroid blocking antibodies. Of note, a small percentage of these cases remit, and therefore TSH levels should be checked periodically and adjusted.

After I131 therapy for Graves’ disease or multinodular goiter, 30% of all patients develop hypothyroidism within a year, with an additional incidence of 6% per year thereafter.

After total laryngectomy with radiation therapy, hypothyroidism may develop. It usually is seen within 4 months, but hormone production is notoriously variable in these patients and a repeat TSH is required every 3-6 months to readjust therapy. Risk factors include high dose radiation and neck dissection.

**Congenital Hypothyroidism (Cretinism)**

The incidence of cretinism in the United States is 1/4000. The most common causes are maternal antithyroid drugs or excessive iodine intake, particularly in those with autoimmune thyroid disease. Maternal IgG crosses the placenta, and IgG thyroid blocking antibodies from the mother may lead to a transient or even permanent neonatal hypothyroidism. Genetic mutations of thyroid production, such thyroperoxidase deficiencies, or of the c-erb A gene lead to deficient or ineffective thyroid hormone.

Cretin children usually present with a protuberant abdomen and face, a flat nose and yellow skin. They usually have severe constipation, lethargy and feeding difficulties. The cry is usually hoarse, and glossoptosis is usually present. Many children are mentally retarded.

Differentiation between endemic cretinism and sporadic cretinism is usually made by the presence of a goiter. Endemic cretinism always has a goiter, whereas with most cases of sporadic cretinism there is thyroid agenesis. Most children with endemic cretinism have sensorineural hearing loss, whereas sporadic cretinism usually presents
with a mixed hearing loss. Pendred’s syndrome, an autosomal recessive disease, is characterized by a goiter due to a defect in thyroid hormone synthesis and a severe sensorineural hearing loss. The site of lesion is the cochlea.

Detection and management of neonatal hypothyroidism may reverse or inhibit many of the symptoms. Many countries, including the United States, have routine screening for hypothyroidism. In addition, maternal thyroid blocking antibodies are assessed, to determine which children are likely to have a transient hypothyroidism. In those mothers known to have blocking antibodies, a cordocentesis may be performed, and a single amniotic injection of T4 effectively controls fetal hypothyroidism. Replacement is with oral synthetic thyroxine.

**Juvenile Hypothyroidism**

Juvenile hypothyroidism occurs after infancy but before adulthood. Many of these children have a hormonal synthesis deficit or a c-erb A aberration. The remainder usually have an autoimmune etiology. Symptoms are thyroid goiter, delayed maturation, testicular enlargement or precocious menarche along with poor performance in school. These children are not usually mentally retarded. If diagnosed early enough and oral thyroxine instituted, recovery is the general rule.

**HYPERTHYROIDISM (THYROTOXICOSIS)**

Thyrotoxicosis is defined as the physiologic state where tissue is exposed to and responds to an excess of thyroid hormones. There are several causes of thyrotoxicosis, including Graves’ disease, thyroiditis, uni – and multinodular goiters, carcinoma and pituitary abnormalities. The most common cause is Graves’ disease, followed by multinodular goiter.

The diagnosis is often difficult, particularly in mild cases. Symptoms include nervousness, tremors, sweating, heat intolerance, palpitations, weight loss in the presence of an active appetite, amenorrhea and muscle weakness. Often, these symptoms are masked, particularly in the elderly, where many of the symptoms are overlooked as a natural result of aging.

Although often used interchangeably, thyrotoxicosis and hyperthyroidism are not synonymous. Thyrotoxicosis can occur with or without hyperthyroidism. Hyperthyroidism should be reserved for those diseases where hyperfunction of the thyroid gland itself leads to elevated levels of serum thyroid hormones. This has implication for treatment, as surgery, antithyroid agents and radioiodine are inappropriate with a normally functioning thyroid.
Graves Disease

Graves’ disease is an autoimmune disease of unknown etiology where IgG antibodies are made against the TSH receptors of the thyroid gland. Most of the antibodies stimulate thyroid hormone synthesis, but some are known to be blocking antibodies. This may make the distinction between Graves’ disease and Hashimoto’s thyroiditis somewhat difficult in cases of hypothyroidism.

Clinically, Graves’ disease is characterized by the triad of thyroid goiter, ophthalmopathy and dermopathy. The thyroid gland is enlarged, soft and vascular. Pathologically there is a lymphocytic infiltrate and hyperplasia and hypertrophy of the parenchyma that correlate with the level of antibodies in the bloodstream. The histologic hallmark is “too many follicular cells and too little colloid.” If given iodide, the thyroid gland becomes firm, usually due to an increase in colloid storage. An ophthalmopathy is often present, which is characterized by unilateral or bilateral exophthalmos. The ophthalmopathy is characterized by an inflammatory lymphocytic infiltrate of the orbital contents, particularly the extraocular muscles. A dermopathy is often present as pretibial edema characterized pathologically by a thickening of the dermis with mucopolysaccharides and lymphocytes.

There are three methods to treat Graves’ disease, including antithyroid medications, radioactive ablation of the gland, and surgical removal of the gland. Antithyroid medications include iodine, thionamides and beta blockers. Medications used in the past include monovalent anions and cations such as perchlorate and lithium, but these are no longer used due to toxic side effect.

When used in high concentration, iodine inhibits the organification and proteolysis of hormones. The effect is transient, however, and escape usually occurs within two weeks. Currently, iodine is now used preoperatively to make the gland less vascular and therefore reduce bleeding. Of note, iodide causes thyrotoxicosis in euthyroid Graves’ disease.

Thionamides such as propothiouracil (PTU) and methimazole work by competitively inhibiting thyroperoxidase and thus the production of hormones. In addition, PTU also blocks the peripheral conversion of T4 to T3 and is therefore appropriate when a quick onset is desired, such as in thyroid storm. These medications do not prevent the release of stored hormones, therefore their onset of action is 4 to 8 weeks. Most clinicians do no use thionamides except in pregnant women or in the thyrotoxic patient preoperatively. A rare but fatal complication of thionamides is massive hepatitis.

Beta blockers are valuable in the management of hyperthyroidism. In addition to ameliorating the adrenergic side effects of thyrotoxicosis, they block the peripheral action of thyroid hormones. Propanolol has the additional effect of inhibiting the conversion of T4 to T3. These are among the most common preoperative antithyroid medications.
In the United States radioiodine thyroid ablation is the procedure of choice in patients who fail or refuse antithyroid medications. Several studies have shown thionamides used before iodide ablation increases the efficacy of therapy and also requires lower radioactive doses. Typically, the thionamide is stopped three days prior to the procedure. The results of ablation usually require 2-4 months, and usually causes a transient exacerbation of thyrotoxicosis, but rarely thyroid storm. This usually occurs within the first two weeks, and is believed secondary to a transient thyroiditis. Radioiodine therapy is absolutely contraindicated in pregnancy and breast feeding.

Subtotal and total thyroidectomy for thyrotoxic Graves’ disease is the most rapid and effective treatment. Some prefer surgery as a first line defense, particularly in pregnant women where iodine therapy is contraindicated. Thyrotoxicosis should be controlled preoperatively with thionamides or beta blockers. Some add iodine, believing vascularity of the gland is reduced. Complications or sequelae of surgery include hypothyroidism and recurrent laryngeal nerve damage. Thyroid storm can occur, particularly if preoperative control of thyrotoxicosis is inadequate.

**Toxic Adenoma (“Hot Nodule”)**

Thyrotoxicosis may be caused by a hot nodule, but not all hot nodules cause thyrotoxicosis. In general, nodules larger than 3 cm are required. These nodules are 4 times more likely in females.

The thyrotoxicosis is generally mild and caused by T3. The TSH level is generally extremely low or absent, and the I123 scan shows only the functioning nodule, with the remainder of the gland silent. Treatment is therefore by either I131 or surgery, although rarely a ultrasound guided ethanol injection is used. Radioiodine therapy does not usually affect the remainder of the gland, as it is usually in an extremely low state of thyroid hormone synthesis and therefore iodide uptake. After therapy, there is often a transient hypothyroidism.

**Toxic Multinodular Goiter**

Toxic multinodular goiter is relatively rare in the United States. It is more common in areas of endemic iodine deficiency. Its course is indolent, and usually begins as a euthyroid goiter which subsequently develops multiple nodules which secrete hormones independent of TSH levels. Attempted therapy with thyroxine therefore often leads to a thyrotoxic patient. Unlike Graves’ disease, spontaneous remission does not occur.

Pathologically the nodules give the impression of being encapsulated, but are merely surrounded by compressed stromal tissue. Histologically the goiter demonstrates a large amount of scarring with a variation in the size of follicles. It is therefore difficult to distinguish the multinodular goiter from an adenoma without examining the entire specimen.
Radioiodine and surgery are the treatment of toxic multinodular goiter. Thionamides are less effective, but are usually given prior to attempted surgery or radioablation.

**Thyrotropin Induced Thyrotoxicosis**

Thyrotoxicosis with a high TSH is usually caused by an abnormality of the pituitary gland. Excess TSH hormone production by the anterior pituitary is an adenoma until proven otherwise. Carcinomas and hyperplasia alone are rare. A pituitary adenoma is confirmed by MRI in combination with an inappropriately elevated TSH. The treatment of choice is surgical excision.

**Trophoblastic Tumors**

Thyrotoxicosis may be caused by bHCG secreting trophoblastic tumors such as hydatidiform moles in women and germ cell tumors in men. The bHCG secreted by these moles is much more thyrotropic than the hCG produced in normal pregnancy. Treatment is excision.

**THYROID STORM**

Thyroid storm is a rare emergency encountered during thyrotoxicosis. It presents in times of high stress, such as infection or surgery. Manic behavior is followed by obtundation and even coma. Heart failure or atrial fibrillation often follow. A high fever ensues. Treatment is with high dose PTU to block thyroid hormone formation along with high dose steroids and propanolol to curb peripheral T4 conversion. Ice baths are used to control hyperthermia. In resistant cases, plasmapheresis is used. Mortality is 20%.

**THYROIDITIS**

Thyroid disorders marked by prominent infiltration of the gland by leukocytes, fibrosis or both are termed thyroiditis. There are several categories, including: acute suppurative; fibrous (Riedel’s); subacute, Hashimoto’s and painless. In general, these diseases mimic other thyroid diseases, and cause both hyper and hypothyroidism. The thyroid gland ranges from goitrous to nonpalpable.

**Acute Suppurative Thyroiditis**

Acute suppurative thyroiditis is an infection of the thyroid gland, usually by *S. pneumoniae* or *S. aureus*. The infection is usually caused by trauma, but bacterial
seeding and locoregional spread are also seen. It is more common in children. The prodrome begins with fever, malaise and pain and progresses to an immobile head with a fixed neck. Treatment is with intravenous antibiotics and incision and drainage as necessary.

**Painful Thyroiditis (de Quervain’s)**

Painful thyroiditis is common, but the frequency is difficult to discern. It is more common in females in their 2 through fifth decades. It is likely due to a virus, although the virus is yet to be identified.

The presentation is one of a large, painful thyroid following flu-like symptoms. The disease lasts two to five months and begins with a transient thyrotoxicosis lasting less than one month. A subsequent hypothyroidism lasts for several weeks. The euthyroid state ensues, followed by the disappearance of the goiter. Pathologically the gland exhibits cellular destruction and enlarged and disrupted thyroid follicles. Histiocytes are common, leading to pseudogiant cells.

Therapy consists of symptomatic treatment, including beta blockers during the thyrotoxicosis stage. In severe cases, steroids are required. Complete recovery is the rule.

**Postpartum Thyroiditis**

Postpartum thyroiditis is a silent thyroiditis of pregnancy and the immediate postpartum period. It is usually not identified for several weeks after delivery. It presents in a multitude of ways, including hypothyroidism alone, thyrotoxicosis alone, or a combination of the two. It is associated with the onset of Graves’ disease and antimicrosomal antibodies.

The diagnosis is usually based on the finding of mild thyrotoxicosis with a small goiter and low RAIU. Less commonly, hypothyroidism is identified. Treatment is symptomatic with beta blockers or thyroxine, as necessary. Of note, high titer antimicrosomal antibodies are predictors of long term disease.

**Hashimoto’s Thyroiditis**

Hashimoto’s thyroiditis is the most common thyroiditis. It is associated with numerous other autoimmune diseases, including Sjogren’s syndrome, rheumatoid arthritis and systemic lupus erythematosus. Most patients present with an asymptomatic goiter. Although the exact autoimmune mechanism is unknown, more than 90% of patients have antithyroglobulin and anti thyroperoxidase antibodies. A smaller percentage of patients have TSH receptor antibodies.
Clinically most patients are euthyroid, although many develop either thyrotoxicosis or hypothyroidism. When thyrotoxicosis is present along with TSH receptor antibodies, the disease is indistinguishable from Graves’ disease. Lymphoma should be expected in elderly patients with a growing goiter or hoarseness. On physical exam, the gland is large, firm and avascular. Histologically, the gland shows follicular cell degeneration (“Askanazy changes”) with a predominant lymphocytic infiltrate with germinal centers.

Management depends upon thyroid state. Thyrotoxic patients are managed with antithyroid medications. Euthyroid and hypothyroid patients are given thyroxine. Surgery is reserved for failure of suppression or suspicion of lymphoma.

**Fibrous Thyroiditis (Reidel’s Thyroiditis)**

Reidel’s thyroiditis is a rare idiopathic fibrosis of the thyroid gland and surrounding structures. It is often associated with fibrosing cholangitis, orbital pseudotumor and retroperitoneal fibrosis. Thyroid function tests are usually normal. There is a female preponderance, and the gland is found to rapidly enlarge, causing dyspnea and dysphagia.

Surgery is often required to establish the diagnosis or to relieve symptoms. The surgery is often difficult due to fibrotic planes.

**EUTHYROID GOITER**

Virtually any disease that affects the thyroid may cause a goiter. Those that do not alter thyroid function are termed euthyroid goiter. Endemic goiter is uncommon in the United States, but there are an estimated 250 million people affected worldwide. The usual cause is iodide deficiency.

Before the diagnosis of euthyroid goiter can be made, TSH levels, estimated free T4 and antithyroid antibodies must all be normal. Many also require a thyroid iodine scan, to attempt to determine hypo or hyperfunctioning nodules.

Management of euthyroid goiter consists of exogenous thyroid hormone replacement. Surgery is used for symptomatic goiters, failure of suppression or when an underlying malignancy is suspected. Radioiodine is used in patients who are not surgical candidates.
References


