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Clinical Thyroidology® for the Public

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Wiersinga WM et al 2018 Predictive score for the development or progression of Graves' orbitopathy in patients with newly diagnosed Graves' hyperthyroidism. Eur J Endocrinol 178:635–643. Epub 2018 Apr 12. PMID: 29650691.

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Asban A et al 2018 Hyperthyroidism is underdiagnosed and undertreated in 3336 patients: an opportunity for improvement and intervention. Ann Surg 268:506–512. PMID: 30004926.

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Treatment-induced hypothyroidism reduces long-term posttreatment cardiovascular morbidity and mortality in Graves' Disease and Toxic Multinodular Goiter

Thyroid hormones have major effects on the heart and palpitations and irregular heart rhythms are frequent symptoms caused by hyperthyroidism. Because of this, hyperthyroidism is associated with increased cardiac problems. This study examines the effect of radioactive iodine therapy and surgery on long-term heart disease outcomes in patients with Graves' disease and toxic multinodular goiter and the relationship of these outcomes to post-treatment thyroid status

Essi R et al 2018 Cardiovascular morbidity and mortality after treatment of hyperthyroidism with either radioactive iodine or thyroidectomy. Thyroid. Epub 2018 Jul 23. PMID: 29882483.

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Quantitative uptake of sestamibi differentiates Type I from Type 2 amiodarone-induced thyrotoxicosis

In some patients taking amiodarone, thyrotoxicosis develops, releasing high levels of thyroid hormone into the blood. There are two types of amiodarone-induced thyrotoxicosis that are treated differently, but diagnosing the different types can be difficult. The aim of this study was to analyze the usefulness of sestamibi in differentiating between the types of amiodarone-induced thyrotoxicosis.

Censi S et al 2018 Amiodarone-induced thyrotoxicosis: differential diagnosis using 99mTc-sestamibi and target-to-background ratio (TBR). Clin Nucl Med 43:655–662. PMID: 30036255.

HYPOTHYROIDISM 11

A novel, futuristic method of thyroid hormone treatment

Since some hypothyroid patients do not feel well on T_4 alone, there has been interest in combination therapy with both T_4 and T_3. Currently, T_3 and T_4 treatments are available but do not provide the T_4:T_3 ratio seen in individuals with an intact thyroid gland. This study reports the potential use of 2D-printed gel films that can deliver very precise amounts of T_4 and/or T_3 as a novel, futuristic form of personalized thyroid replacement therapy.

Alomari M et al. Printing T_3 and T_4 oral drug combinations as a novel strategy for hypothyroidism. Int J Pharm 2018 Jul 29;549(1-2):363-369.

THYROID CANCER. 13

Thyroglobulin levels do not predict recurrence after lobectomy for low-risk papillary thyroid cancer

Patients with small cancers confined to the thyroid gland are more frequently being treated with a thyroid lobectomy. Patients are traditionally monitored for cancer recurrence serum thyroglobulin levels, which is accurate after a total thyroidectomy, but there are few large studies looking at how thyroglobulin predicts recurrence after a lobectomy. This study examines the role of thyroglobulin in predicting cancer recurrence after thyroid lobectomy.

Park S et al 2018 Changes in serum thyroglobulin levels after lobectomy in patients with low-risk papillary thyroid cancer. Thyroid. Epub 2018 May 30. PMID: 29845894.

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EDITOR'S COMMENTS

Welcome to another issue of *Clinical Thyroidology for the Public*. In this journal, we will bring to you the most up-to-date, cutting edge thyroid research. We also provide even faster updates of late-breaking thyroid news through Twitter at @thyroidfriends and on Facebook. Our goal is to provide patients with the tools to be the most informed thyroid patient in the waiting room. Also check out our friends in the **Alliance for Thyroid Patient Education**. The **Alliance** member groups consist of: the *American Thyroid Association, Bite Me Cancer, the Graves' Disease and Thyroid Foundation, the Light of Life Foundation, ThyCa: Thyroid Cancer Survivors' Association, Thyroid Cancer Canada, Thyroid Cancer Alliance* and *Thyroid Federation International.*

We invite all of you to join our **Friends of the ATA** community. It is for you that the American Thyroid Association (ATA) is dedicated to carrying out our mission of providing reliable thyroid information and resources, clinical practice guidelines for thyroid detection and treatments, resources for connecting you with other patients affected by thyroid conditions, and cutting edge thyroid research as we search for better diagnoses and treatment outcomes for thyroid disease and thyroid cancer. We thank all of the Friends of the ATA who support our mission and work throughout the year to support us. We invite you to help keep the ATA mission strong by choosing to make a donation that suits you – it takes just one moment to give online at: www.thyroid.org/donate and all donations are put to good work. The ATA is a 501(c)3 nonprofit organization and your gift is tax deductible.

November is Hyperthyroidism Awareness Month.

In this issue, the studies ask the following questions:

- Can we identify patients at risk for Graves' eye disease?
- Are patients with a suppressed TSH being appropriately evaluated for hyperthyroidism?
- Should the goal of definitive therapy for hyperthyroidism be hypothyroidism?
- Is there a better way to differentiate between the 2 types of amiodarone-induces thyrotoxicosis?
- Is there a new formulation for thyroid hormone therapy in the future?
- Is it worth it to follow thyroglobulin levels in thyroid cancer patients that only have undergone a lobectomy?

We welcome your feedback and suggestions. Let us know what you want to see in this publication. I hope you find these summaries interesting and informative.

— Alan P. Farwell, MD, FACE

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THYROID EYE DISEASE

A validated tool can predict risk of developing thyroid eye disease during antithyroid drug treatment of Graves' Disease

BACKGROUND

Over-activity of the thyroid gland, called hyperthyroidism, can sometimes be caused by a flaw in a person's immune system (the body's natural defense system again infection). This problem is a type of autoimmune disorder called Graves' disease. In addition to thyroid gland over-activity, Graves' disease causes eye problems in some people, including eye bulging, loss of color vision, double vision, eye pain and dry eyes.

Not everyone diagnosed with Graves' disease will develop eye problems. Identifying people diagnosed with Graves' disease who do develop eye disease is important, however, because early treatment may make these eye problems less severe. The goal of this study is to identify which people diagnosed with Graves' disease will eventually have eye problems. Such people could then be treated for these eye problems before these problems become severe, and possibly irreversible.

THE FULL ARTICLE TITLE

Wiersinga WM et al 2018 Predictive score for the development or progression of Graves' orbitopathy in patients with newly diagnosed Graves' hyperthyroidism. Eur J Endocrinol 178:635–643. Epub 2018 Apr 12. PMID: 29650691.

SUMMARY OF THE STUDY

This study started with 348 men and women living in Europe who had just been diagnosed with Graves' disease. Treatment for hyperthyroidism was started in these 348 people and they were then tested for development of new eye problems over the next 18 months. To try and understand why eye problems happen in Graves' disease, the people in this group who did develop eye problems during this18 month time period were compared to the people in the group who did not develop new eye problems.

The study identified 53 people who developed eye problems from the starting group of 348 people with Graves' disease. Comparing these 53 people with new eye problems to those in the group who did not develop new eye problems showed that eye problems were associated with Graves' disease if:

1. A person already has eye problems at the time their Graves' disease is discovered.
2. A person diagnosed with Graves' disease is a cigarette smoker.
3. The immune system flaw that causes Graves' disease is more active in a person diagnosed with Grave's disease.
4. If Graves' disease is not discovered early (a person with Graves' disease does not receive prompt treatment).

WHAT ARE THE IMPLICATIONS OF THIS STUDY?

This study identified 4 findings that could predict which people with Graves' disease will or will not develop eye problems. This testing showed that these 4 findings are most useful for identifying Graves' patients who will not develop eye problems – if absent, a person with Graves' disease is very unlikely to develop eye problems. On the other hand, these 4 findings were found to be less good at predicting which people diagnosed with Graves' disease will develop eye problems. This study is important because it helps identify those people with Graves' disease who are unlikely to develop eye problems avoid associated unneeded testing, and possibly unneeded treatment.

— Jason D. Prescott, MD PhD

ATA THYROID BROCHURE LINKS

Hyperthyroidism (Overactive): https://www.thyroid.org/hyperthyroidism/

Graves' Disease: https://www.thyroid.org/graves-disease/

THYROID EYE DISEASE, continued

ABBREVIATIONS & DEFINITIONS

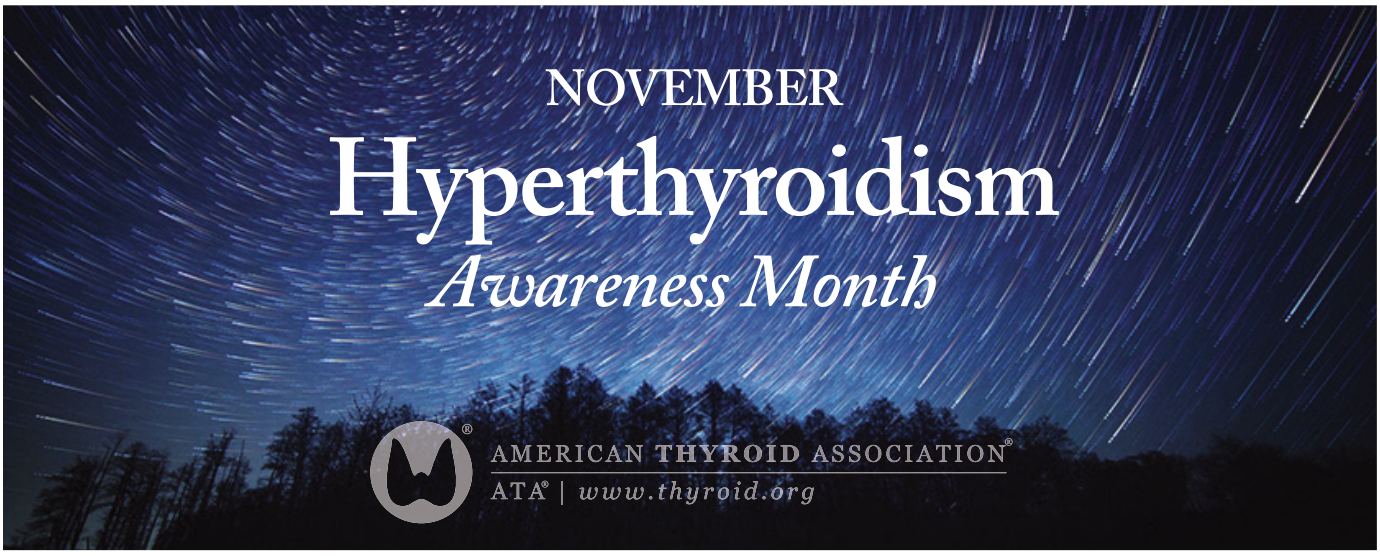
Autoimmune disorders: A diverse group of disorders that are caused by antibodies that get confused and attack the body's own tissues. The disorder depends on what tissue the antibodies attack. Graves' disease and Hashimoto's thyroiditis are examples of autoimmune thyroid disease. Other Autoimmune disorders include: type 1 diabetes mellitus, Addison's disease (adrenal insufficiency), vitiligo (loss of pigment of some areas of the skin), systemic lupus erythematosus, pernicious anemia (B12 deficiency), celiac disease, inflammatory bowel disease, myasthenia gravis, multiple sclerosis, and rheumatoid arthritis.

Autoimmune thyroid disease: a group of disorders that are caused by antibodies that get confused and attack the thyroid. These antibodies can either turn on the thyroid (Graves' disease, hyperthyroidism) or turn it off (Hashimoto's thyroiditis, hypothyroidism).

Hyperthyroidism: a condition where the thyroid gland is overactive and produces too much thyroid hormone. Hyperthyroidism may be treated with antithyroid meds (Methimazole, Propylthiouracil), radioactive iodine or surgery.

Graves' disease: the most common cause of hyperthyroidism in the United States. It is caused by antibodies that attack the thyroid and turn it on.

Thyroid eye disease (TED): also known as Graves ophthalmopathy. TED is most often seen in patients with Graves' disease but also can be seen with Hashimoto's thyroiditis. TED includes inflammation of the eyes, eye muscles and the surrounding tissues. Symptoms include dry eyes, red eyes, bulging of the eyes and double vision.



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HYPERTHYROIDISM

Some patients do not undergo subsequent evaluation following an abnormally suppressed TSH suggestive of hyperthyroidism

BACKGROUND

The best screening test when a thyroid problem is suspected is measurement of thyroid stimulating hormone (TSH) levels. However, when an abnormal TSH is discovered, additional testing is usually required to complete the evaluation. This additional testing usually includes measurement of the thyroid hormones (T_4 and T_3), thyroid antibodies and possibly imaging studies to determine if the abnormal TSH identifies a thyroid problem that needs to be treated.

Overt hyperthyroidism, which occurs in about 1-2% of people, is a serious disorder that can be harmful to the heart by causing a rapid heart rate and possibly an abnormal heart rhythm that can lead to heart and brain damage. TSH levels are low/suppressed and the thyroid hormone levels (T_4 and T_3) are increased in patients with overt hyperthyroidism and these patients need to be treated. Subclinical hyperthyroidism occurs when TSH levels are low/suppressed but the thyroid hormone levels are normal. This is seen much more frequently and may not require treatment initially.

This study was done to see what kind of testing and treatment occurs after a patient is discovered to have a low TSH test.

THE FULL ARTICLE TITLE

Asban A et al 2018 Hyperthyroidism is underdiagnosed and undertreated in 3336 patients: an opportunity for improvement and intervention. Ann Surg 268:506–512. PMID: 30004926.

SUMMARY OF THE STUDY

This was a study where the researchers reviewed the medical record of over 3000 patients at the University of Alabama-Birmingham who had at least one very low/suppressed level of TSH (<0.05 mU/L) at least one time. Patients with a known history of thyroid disease were not included in the study.

There were 3386 patients studied and the average age was 52 years old. Almost 80% were women and almost 60% were white. Most patients were healthy at the time of the abnormal TSH blood test. Over 21 months of review, only 1 out of 3 patients had a follow up test to determine how serious the overactive thyroid condition was and to understand why the thyroid was overactive.

The most common tests were blood tests to see if the immune system was the cause of the overactive thyroid (thyroid antibodies) and these were only checked half the time. The least common test was a radioactive iodine thyroid scan to determine why the thyroid is overactive. This was ordered in less than 1 out of 5 patients.

WHAT ARE THE IMPLICATIONS OF THIS STUDY?

This study shows that only 1 out of 3 patients with a low/suppressed TSH had the diagnosis hyperthyroidism written in the medical record and had any follow up testing performed. This study is important for patients in that it shows that even at a teaching hospital there is not necessarily best follow up of abnormal thyroid testing. This type of study does not allow us to understand all of the reasons why further testing may or may not have been performed, but it does indicate that patients should be aware of their own testing results and if there is an abnormality, make sure it is not something that needs to be further investigated.

— Josh Klopper, MD

HYPERTHYROIDISM, continued

ATA THYROID BROCHURE LINKS

Hyperthyroidism (Overactive): https://www.thyroid.org/hyperthyroidism/
Thyroid Function Tests: https://www.thyroid.org/thyroid-function-tests/

ABBREVIATIONS & DEFINITIONS

TSH: thyroid stimulating hormone – produced by the pituitary gland that regulates thyroid function; also the best screening test to determine if the thyroid is functioning normally.

Thyroxine (T_4): the major hormone produced by the thyroid gland. T_4 gets converted to the active hormone T_3 in various tissues in the body.

Triiodothyronine (T_3): the active thyroid hormone, usually produced from thyroxine, available in pill form as Cytomel™.

Hyperthyroidism: a condition where the thyroid gland is overactive and produces too much thyroid hormone. Hyperthyroidism may be treated with antithyroid meds (Methimazole, Propylthiouracil), radioactive iodine or surgery.

Subclinical Hyperthyroidism: a mild form of hyperthyroidism where the only abnormal hormone level is a decreased TSH.

Thyroid scan: this imaging test uses a small amount of a radioactive substance, usually radioactive iodine, to obtain a picture of the thyroid gland. A "cold" nodule means that the nodule is not functioning normally. A patient with a "cold" nodule should have a fine needle aspiration biopsy of the nodule. A "functioning", or "hot", nodule means that the nodule is taking up radioactive iodine to a degree that is either similar to or greater than the uptake of normal cells. The likelihood of cancer in these nodules is very low and a biopsy is often not needed.

Autoimmune thyroid disease: a group of disorders that are caused by antibodies that get confused and attack the thyroid. These antibodies can either turn on the thyroid (Graves' disease, hyperthyroidism) or turn it off (Hashimoto's thyroiditis, hypothyroidism).

TPO antibodies: these are antibodies that attack the thyroid instead of bacteria and viruses, they are a marker for autoimmune thyroid disease, which is the main underlying cause for hypothyroidism and hyperthyroidism in the United States.

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HYPERTHYROIDISM

Treatment-induced hypothyroidism reduces long-term posttreatment cardiovascular morbidity and mortality in Graves' Disease and Toxic Multinodular Goiter

BACKGROUND

Hyperthyroidism is caused when the thyroid is overactive and produces too high of levels of thyroid hormones. The 2 most common causes of hyperthyroidism are Graves' disease and toxic multinodular goiter. Antithyroid drugs, radioactive iodine therapy and surgery are all used to treat hyperthyroidism.

Thyroid hormones have major effects on the heart and palpitations and irregular heart rhythms are frequent symptoms caused by hyperthyroidism. Because of this, hyperthyroidism is associated with increased cardiac problems and can lead to death related to heat disease. This study examines the effect of radioactive iodine therapy and surgery on long-term heart disease outcomes in patients with Graves' disease and toxic multinodular goiter and the relationship of these outcomes to post-treatment thyroid status.

THE FULL ARTICLE TITLE

Essi R et al 2018 Cardiovascular morbidity and mortality after treatment of hyperthyroidism with either radioactive iodine or thyroidectomy. Thyroid. Epub 2018 Jul 23. PMID: 29882483.

SUMMARY OF THE STUDY

This was a study examining a registry of patients who had been treated for hyperthyroidism in Finland from 1986 through 2007. Surgery patients were identified from procedure codes in the Finland Hospital Discharge Registry. Radioactive iodine therapy patients all were treated at Tampere University Hospital. The discharge diagnoses and dates of hospital admissions were obtained from the Finnish Hospital Discharge Registry. The Population Registration Centre provided information regarding causes of death and residency status.

A control group of Finnish residents from the comprehensive national Population Center was formed by random selection of three age- and sex-matched subjects (for each patient) who were alive when the hyperthyroid patients were treated. A total 4334 were in the surgery group (615 men and 3719 women) and 1814 in the radioactive iodine therapy group (329 men and 1485 women). The cause of the hyperthyroidism was Graves' disease in 50% of the patients, toxic multinodular goiter in 33% of patients and "unspecified" in 17% of patients. Men and women were of similar ages at the time of treatment for hyperthyroidism.

About 2 years before patients were treated for hyperthyroidism, their hospitalization rates for heart disease began to increase and was 1.6 times more common than in the control patients. Hypertension, atrial fibrillation, coronary artery disease, strokes, heart failure, and valvular disease and cardiomyopathies all were more common in the hyperthyroid patients with atrial fibrillation and other arrhythmias the most common

There was no difference between surgery and radioactive iodine therapy on subsequent hospitalizations for any or all categories of heart disease providing that radioactive iodine therapy was followed by hypothyroidism. In contrast, if radioactive iodine therapy did not result in hypothyroidism, radioactive iodine therapy was associated with a greater risk of subsequent hospitalization for arrhythmias, including atrial fibrillation, and heart failure.

Death due to heart disease was increased in hyperthyroidism patients treated with radioactive iodine therapy as compared to those treated with surgery, but this difference went away of the patients who did not become hypothyroid after radioactive iodine therapy were removed from the analysis. Death was similar in patients with toxic multinodular goiter as compared to those with Graves' disease.

WHAT ARE THE IMPLICATIONS OF THIS STUDY?

This study shows that the rates of hospitalization for heart disease are increased in patients with hyperthy-

HYPERTHYROIDISM, continued

roidism. These rates were decreased after surgery or radioactive iodine therapy that produced hypothyroidism but persisted at the higher rate in patients treated with radioactive iodine therapy who did not develop hypothyroidism. Death also was increased on patients treated with radioactive iodine therapy who did not develop hypothyroidism but was not increased in patients treated with surgery or patients treated with radioactive iodine therapy that did produce hypothyroidism. These data suggest that the goal of radioactive iodine therapy for hyperthyroidism should be to produce hypothyroidism.

— Alan P. Farwell, MD, FACE

ATA THYROID BROCHURE LINKS

Hyperthyroidism (Overactive): https://www.thyroid.org/hyperthyroidism/
Graves' Disease: https://www.thyroid.org/graves-disease/
Thyroid Surgery: https://www.thyroid.org/thyroid-surgery/
Radioactive Iodine: https://www.thyroid.org/radioactive-iodine/

ABBREVIATIONS & DEFINITIONS

Hyperthyroidism: a condition where the thyroid gland is overactive and produces too much thyroid hormone. Hyperthyroidism may be treated with antithyroid meds (Methimazole, Propylthiouracil), radioactive iodine or surgery.

Graves' disease: the most common cause of hyperthyroidism in the United States. It is caused by antibodies that attack the thyroid and turn it on.

Toxic nodular goiter: characterized by one or more nodules or lumps in the thyroid that may gradually grow and increase their activity so that the total output of thyroid hormone in the blood is greater than normal.

Thyroidectomy: surgery to remove the entire thyroid gland. When the entire thyroid is removed it is termed a total thyroidectomy. When less is removed, such as in removal of a lobe, it is termed a partial thyroidectomy.

Radioactive iodine (RAI) : this plays a valuable role in diagnosing and treating thyroid problems since it is taken up only by the thyroid gland. I-131 is the destructive form used to destroy thyroid tissue in the treatment of thyroid cancer and with an overactive thyroid. I-123 is the non-destructive form that does not damage the thyroid and is used in scans to take pictures of the thyroid (Thyroid Scan) or to take pictures of the whole body to look for thyroid cancer (Whole Body Scan).

Hypothyroidism: a condition where the thyroid gland is underactive and doesn't produce enough thyroid hormone. Treatment requires taking thyroid hormone pills.

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HYPERTHYROIDISM

Quantitative uptake of sestamibi differentiates Type 1 from Type 2 amiodarone-induced thyrotoxicosis

BACKGROUND

Amiodarone is a medication commonly used to treat patients with irregular heart rhythms. It contains considerable amounts of iodine that is normally used by the thyroid gland to make thyroid hormone. In some patients taking amiodarone, thyrotoxicosis develops, releasing high levels of thyroid hormone into the blood. There are two types of amiodarone-induced thyrotoxicosis. Type 1 occurs in patients with an underlying thyroid condition, such as Graves' disease, and leads to formation of new thyroid hormones. This is treated with antithyroid medications. Type 2 occurs due to a destructive process in the thyroid, leading to leakage of thyroid hormones from the gland. This is treated with steroids. Mixed forms are treated with a combination of these medications.

In order to differentiate the two types of amiodarone-induced thyrotoxicosis and treat them appropriately, a scan using sestamibi can be used. A sestamibi scan uses a tracer that shows good uptake by the thyroid gland in type 1 amiodarone-induced thyrotoxicosis (AIT-1) but almost no uptake in type 2 (AIT-2). If there are components of both types, then a faint but definite uptake can be seen. The aim of this study was to analyze the usefulness of sestamibi in differentiating between the types of amiodarone-induced thyrotoxicosis.

THE FULL ARTICLE TITLE

Censi S et al 2018 Amiodarone-induced thyrotoxicosis: differential diagnosis using 99mTc-sestamibi and target-to-background ratio (TBR). Clin Nucl Med 43:655–662. PMID: 30036255.

SUMMARY OF THE STUDY

The study authors reviewed the records of 30 patients with amiodarone-induced thyrotoxicosis who were followed by several tests and response to therapy. All patients had thyroid function tests every 15 days during follow up and also had thyroid ultrasounds to estimate thyroid blood flow. Patients also underwent sestamibi scans with calculation of the quantity of tracer uptake by the thyroid in reference to background, resulting in the target-to-background ratio (TBR).

The authors found that according to response to treatment, 14 out of 30 patients had AIT-1, 12 out of 30 had AIT-2 and 4 had mixed forms. Of the 14, 12 AIT-1 patients had clear diffuse uptake of the sestamibi tracer, one had increased uptake in a nodule, and one had minimal uptake, suggesting a mixed form. A total of 6 of the 12 AIT-2 patients had no thyroid uptake and 6 had minimal uptake, suggesting a mixed form. The visual results of the sestamibi scans in the four patients with mixed forms were inconsistent. When comparing the quantitative TBR of the sestamibi scans with the final clinical diagnosis, the authors were able to successfully differentiate patients with AIT-1 from those with AIT-2. However, the method was not reliable for the mixed form.

WHAT ARE THE IMPLICATIONS OF THIS STUDY?

Amiodarone-induced thyrotoxicosis is often treated with both antithyroid and steroid medications due to either unclear etiology or insufficient time for diagnostic studies to determine the type. This study demonstrates that quantifying the sestamibi uptake using TBR is very helpful in differentiating AIT-1 from AIT-2. This is important for both physicians and patients as early correct diagnosis ensures appropriate treatment and avoids overtreatment.

— Maria Papaleontiou, MD

ATA THYROID BROCHURE LINKS

Hyperthyroidism (Overactive): https://www.thyroid.org/hyperthyroidism/

HYPERTHYROIDISM, continued

ABBREVIATIONS & DEFINITIONS

Amiodarone: an iodine-rich drug that is commonly used for the treatment of irregular heart rhythms. Amiodarone can cause thyroid problems, including both hypothyroidism and hyperthyroidism.

Hyperthyroidism: a condition where the thyroid gland is overactive and produces too much thyroid hormone. Hyperthyroidism may be treated with antithyroid meds (Methimazole, Propylthiouracil), radioactive iodine or surgery.

Amiodarone induced Thyrotoxicosis: elevated thyroid hormone levels that can occur as a result of excessive iodine from amiodarone resulting in increased thyroid hormone production and secretion or to destruction of thyroid cells with release of thyroid hormone into the blood.

Sestamibi: a pharmaceutical agent used in nuclear medicine imaging.

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HYPOTHYROIDISM

A novel, futuristic method of thyroid hormone treatment

BACKGROUND

The thyroid gland is responsible for making the thyroid hormones, T_3 and T_4. Hypothyroidism, or an underactive thyroid, is a common medical condition that requires replacement of the thyroid hormones. Levothyroxine (synthetic T_4) is the most commonly prescribed form of thyroid hormone treatment. However, there has been growing interest to understand the risks and benefits of also replacing T_3, along with T_4, in patients with hypothyroidism as some patients do not feel well on T_4 alone. Although more data are needed regarding the potential concerns of T_3, it is thought that approximately 5–7% of T_3 and 93–95% of T_4 may be a safe ratio for those who desire T_3. Currently, T_3 and T_4 treatments are available as either synthetic or animal-derived formulations. The animal-derived products (desiccated thyroid extract) contain approximately 25% T_3 and 75% T_4 in a single pill. Synthetic T_3 and T_4 are only available as two separate prescription pills.

Thermal inkjet printing is a form of 2D printing which may have interesting uses within the pharmaceutical industry. It can deliver very precise amounts of a one or more substances through the use of a special type of printing. Thus, it can potentially produce a highly personalized combined single pill of synthetic T_4 and T_3 (in any desired ratio of the two thyroid hormones). This study reports the potential use of 2D-printed T_4 and/or T_3 gel films as a novel, futuristic form of personalized thyroid replacement therapy.

THE FULL ARTICLE TITLE

Alomari M et al. Printing T_3 and T_4 oral drug combinations as a novel strategy for hypothyroidism. Int J Pharm 2018 Jul 29;549(1-2):363-369.

SUMMARY OF THE STUDY

This article describes that commonly-used dosages of T_4 and/or T_3 were able to be successfully printed onto gel films. The amounts of actual T_4 and T_3 in the gels were fairly close to those being delivered from the inkjets. The gels contained minimal water, and they were stable up to a very high temperature.

WHAT ARE THE IMPLICATIONS OF THIS STUDY?

This is the first study to report how thyroid hormones may be synthetically produced into a single combined T_4+T_3 pill using 2D printing. This technology has been reported also for a few other medications in the research setting only. This technology is not yet FDA-approved and has yet to be used commercially on a large scale. However, it is an interesting concept and may offer the ability to titrate thyroid hormone replacement therapy very precisely and in a highly-personalized form of medical therapy.

— Angela M. Leung, MD, MSc

ATA THYROID BROCHURE LINKS

Hypothyroidism (Underactive): https://www.thyroid.org/hypothyroidism/
Thyroid Hormone Treatment: https://www.thyroid.org/thyroid-hormone-treatment/

HYPOTHYROIDISM, continued

ABBREVIATIONS & DEFINITIONS

Hypothyroidism: A condition where the thyroid gland is underactive and doesn't produce enough thyroid hormone. Treatment requires taking thyroid hormone pills.

Levothyroxine (T_4): the major hormone produced by the thyroid gland and available in pill form as Synthroid™, Levoxyl™, Tyrosint™ and generic preparations.

Thyroid hormone therapy: patients with hypothyroidism are most often treated with Levothyroxine in order to return their thyroid hormone levels to normal. Replacement therapy means the goal is a TSH in the normal range and is the usual therapy. Suppressive therapy means that the goal is a TSH below the normal range and is used in thyroid cancer patients to prevent growth of any remaining cancer cells.

Desiccated thyroid extract: thyroid hormone pill made from animal thyroid glands. Currently desiccated thyroid extract is made from pig thyroids and is available as Armour Thyroid™ and Nature-Throid™.

Thyroxine (T_4): the major hormone produced by the thyroid gland. T_4 gets converted to the active hormone T_3 in various tissues in the body.

Triiodothyronine (T_3): the active thyroid hormone, usually produced from thyroxine.

Triiodothyronine (T_3): the active thyroid hormone, usually produced from thyroxine, available in pill form as Cytomel™.

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THYROID CANCER

Thyroglobulin levels do not predict recurrence after lobectomy for low-risk papillary thyroid cancer

BACKGROUND

Thyroid cancer is the fastest rising cancer in women. Fortunately, most patients do well after initial treatment. Much of thyroid cancer management currently is based on the risk of cancer recurrence. Indeed, as we have recognized that patients with low-risk thyroid cancer have excellent survival, we are being less aggressive with treatments in order to minimize any long-term complications. Therefore, patients with smaller cancers confined to the thyroid are more frequently being treated with a thyroid lobecotmy (removal of half the thyroid) rather than the traditional total thyroidectomy. After surgery and, if needed, radioactive iodine therapy, patients are monitored for cancer recurrence by ultrasound imaging and by a blood test (thyroglobulin), which is accurate after a total thyroidectomy, but there are few large studies looking at how thyroglobulin predicts recurrence after a lobectomy. This study examines the role of thyroglobulin in predicting cancer recurrence after thyroid lobectomy.

THE FULL ARTICLE TITLE

Park S et al 2018 Changes in serum thyroglobulin levels after lobectomy in patients with low-risk papillary thyroid cancer. Thyroid. Epub 2018 May 30. PMID: 29845894.

SUMMARY OF THE STUDY

The follow-up (measurement of thyroglobulin levels and documentation of cancer recurrence) of 208 patients at a single hospital in Korea were examined. Most patients had a microcarcinoma (<1cm). A total of 15 patients with cancer recurrence were added to look for predictive factors. Only patients that did not need thyroid hormone supplementation, had normal TSH levels, and no thyroglobulin antibodies, were included. They looked at the thyroglobulin:TSH ratio (since TSH changes can affect the value of thyroglobulin), and assessed if there was a >20% increase in thyroglobulin level in 2 consecutive thyroglobulin levels and in whom there was a biopsy proven cancer recurrence.

Only 4 patients (2%) had a structural recurrence over an average follow-up of 7 years, with no deaths. Of the 19 total patients with cancer recurrence, 13 recurred with cancer in the remaining thyroid lobe and 6 in the lymph nodes. In general, the serum thyroglobulin as well as the thyroglobulin:TSH ratio increased by about 10% per year for all patients. However, both patients with and without recurrence had increases, and decreases in their thyroglobulin levels – there was no pattern or association and changes in thyroglobulin were therefore not predictive of recurrence.

WHAT ARE THE IMPLICATIONS OF THIS STUDY?

Following serum thyroglobulin after thyroid lobectomy may not be accurate in predicting recurrence. This may mean that patients either should only have ultrasound surveillance, or, due to the low rate of recurrence and non-existent deaths, maybe no surveillance at all is needed, especially for microcarcinomas.

— Melanie Goldfarb, MD, MSc, FACS, FACE

ATA THYROID BROCHURE LINKS

Papillary and Follicular Thyroid Cancer: https://www.thyroid.org/thyroid-cancer/

THYROID CANCER, continued

ABBREVIATIONS & DEFINITIONS

Papillary thyroid cancer: the most common type of thyroid cancer. There are 4 variants of papillary thyroid cancer: classic, follicular, tall-cell and noninvasive follicular thyroid neoplasm with papillary-like nuclear features (NIFTP).

Papillary microcarcinoma: a papillary thyroid cancer smaller than 1 cm in diameter.

Lobectomy: surgery to remove one lobe of the thyroid.

Thyroglobulin antibodies: these are antibodies that attack the thyroid instead of bacteria and viruses, they are a marker for autoimmune thyroid disease, which is the main underlying cause for hypothyroidism and hyperthyroidism in the United States.

Thyroglobulin: a protein made only by thyroid cells, both normal and cancerous. When all normal thyroid tissue is destroyed after radioactive iodine therapy in patients with thyroid cancer, thyroglobulin can be used as a thyroid cancer marker in patients that do not have thyroglobulin antibodies.

Cancer recurrence: this occurs when the cancer comes back after an initial treatment that was successful in destroying all detectable cancer at some point.

Thyroid Awareness Monthly Campaigns

The ATA will be highlighting a distinct thyroid disorder each month and a portion of the sales for Bravelets™ will be donated to the ATA. The month of **November** is **Hyperthyroidism Awareness Month** and a bracelet is available through the **ATA Marketplace** to support thyroid cancer awareness and education related to thyroid disease.

AMERICAN THYROID ASSOCIATION
ATA | Founded 1923

Clinical Thyroidology® for the Public

VOLUME 11 | ISSUE 11 | NOVEMBER 2018

ATA Alliance for Thyroid Patient Education

GOAL

The goal of our organizations is to provide accurate and reliable information for patients about the diagnosis, evaluation and treatment of thyroid diseases.

We look forward to future collaborations and continuing to work together toward the improvement of thyroid education and resources for patients.

WHO WE ARE (in alphabetical order)

AMERICAN THYROID ASSOCIATION
www.thyroid.org
ATA Patient Resources:
http://www.thyroid.org/thyroid-information/
Find a Thyroid Specialist: www.thyroid.org
(Toll-free): 1-800-THYROID
thyroid@thyroid.org

BITE ME CANCER
http://www.bitemecancer.org
info@bitemecancer.org

GRAVES' DISEASE AND THYROID FOUNDATION
www.gdatf.org
(Toll-free): 877-643-3123
info@ngdf.org

LIGHT OF LIFE FOUNDATION
www.checkyourneck.com
info@checkyourneck.com

THYCA: THYROID CANCER SURVIVORS' ASSOCIATION, INC.
www.thyca.org
(Toll-free): 877-588-7904
thyca@thyca.org

THYROID CANCER ALLIANCE
www.thyroidcanceralliance.org
www.thyroidcancerpatientinfo.org
Rotterdam, The Netherlands

THYROID CANCER CANADA
www.thyroidcancercanada.org
416-487-8267
info@thyroidcancercanada.org

THYROID FEDERATION INTERNATIONAL
www.thyroid-fed.org
tfi@thyroid-fed.org

AMERICAN THYROID ASSOCIATION
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Light of Life Foundation
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BITE ME CANCER!

Thyroid Cancer Canada
Cancer de la thyroïde Canada

Thyroid Federation International

ThyCa: Thyroid Cancer Survivors' Association, Inc.℠
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THYROID CANCER ALLIANCE

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Friends of the ATA

FOUNDED 2005

Get the latest thyroid health information. You'll be among the first to know the latest cutting-edge thyroid research that is important to you and your family.

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By subscribing to *Friends of the ATA Newsletter*, you will receive:

- *Friends of the ATA e-news*, providing up-to-date information on thyroid issues, summaries of recently published articles from the medical literature that covers the broad spectrum of thyroid disorders., and invitations to upcoming patient events
- Updates on the latest patient resources through the ATA website and elsewhere on the world wide web
- Special e-mail alerts about thyroid topics of special interest to you and your family

We will use your email address to send you *Friends of the ATA e-news* and occasional email updates. We won't share your email address with anyone, and you can unsubscribe at any time.

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Donate Now!

JOIN US

"The ATA was a valuable resource for our family when my dad was diagnosed with Anaplastic Thyroid Cancer. When you're faced with a detrimental diagnosis where even a few days can make the difference in life or death, understanding your options quickly is critical. The ATA website offers a one-stop shop for patients and caregivers to find specialists, current clinical trials, general thyroid cancer information, and links to other patient support groups and information."

Mary Catherine Petermann
- Father who was diagnosed with Anaplastic Thyroid Cancer in 2006
- He was treated at Mayo Clinic
- He has clean scans as of October 2016

PLEASE JOIN OUR JOURNEY TO ADVANCED DISCOVERIES AND TREATMENT FOR THYROID DISEASE AND THYROID CANCER

As patients with thyroid disease navigate the challenges to their quality of life and researchers and physicians look for more effective directions, we at the ATA have our own destination– **funding for critical thyroid research, prevention, and treatment.** For 94 years, the ATA has led the way in thyroidology. It's a daily obstacle course to find new drugs, better treatments, advanced surgical methods, and more rapid diagnoses for the 20 million Americans who have some form of thyroid disease.

The ATA has paved the way with management guidelines for clinicians who diagnose and treat thyroid disease. For physicians treating pregnant women diagnosed with thyroid disease, our recent publication presents 97 evidence-based recommendations making sure that best practices are implemented with the latest, most effective treatment.

Through your generous support and donations, research takes the lead and hope is on the horizon. **Will you join us** in our campaign to raise **$1.5 million** for thyroid research, prevention, and treatment? Your compassionate, tax-deductible gift will provide funds for:

- Research grants that pave the way for 1,700 ATA physicians and scientists who have devoted their careers to understanding the biology of and caring for patients affected by thyroid disease.
- Patient education for individuals and families looking for life-changing clinical trials, the best thyroid specialists, and cutting edge treatment and drugs.
- Professional education that offers a wealth of knowledge and leading-edge research for trainees and practitioners.
- A website that is the go-to resource for thyroid information for patients and practitioners alike. In 2016 alone, there were more than 3,700,000 website views of ATA's library of online thyroid information patient brochures.

Donations **of all sizes** will change the future for thyroid patients. You will make a direct impact on patients like Mary Catherine's father as he deals with Anaplastic Thyroid Cancer. You will help scientists like ATA Associate Member Julia Rodiger, Ph.D., a scientist at the National Institutes of Health, as she analyzes thyroid hormones for intestinal stem cell development.

Hyperthyroidism

WHAT IS THE THYROID GLAND?

The thyroid gland is a butterfly-shaped endocrine gland that is normally located in the lower front of the neck. The thyroid's job is to make thyroid hormones, which are secreted into the blood and then carried to every tissue in the body. Thyroid hormone helps the body use energy, stay warm and keep the brain, heart, muscles, and other organs working as they should.

WHAT IS HYPERTHYROIDISM?

The term hyperthyroidism refers to any condition in which there is too much thyroid hormone produced in the body. In other words, the thyroid gland is overactive. Another term that you might hear for this problem is thyrotoxicosis, which refers to high thyroid hormone levels in the blood stream, irrespective of their source.

WHAT ARE THE SYMPTOMS OF HYPERTHYROIDISM?

Thyroid hormone plays a significant role in the pace of many processes in the body. These processes are called your metabolism. If there is too much thyroid hormone, every function of the body tends to speed up. It is not surprising then that some of the symptoms of hyperthyroidism are nervousness, irritability, increased sweating, heart racing, hand tremors, anxiety, difficulty sleeping, thinning of your skin, fine brittle hair and weakness in your muscles—especially in the upper arms and thighs. You may have more frequent bowel movements, but diarrhea is uncommon. You may lose weight despite a good appetite and, for women, menstrual flow may lighten and menstrual periods may occur less often. Since hyperthyroidism increases your metabolism, many individuals initially have a lot of energy. However, as the hyperthyroidism continues, the body tends to break down, so being tired is very common.

Hyperthyroidism usually begins slowly but in some young patients these changes can be very abrupt. At first, the symptoms may be mistaken for simple nervousness due to stress. If you have been trying to lose weight by dieting, you may be pleased with your success until the hyperthyroidism, which has quickened the weight loss, causes other problems.

In *Graves' Disease* (also known as *Basedow's Disease*), which is the most common form of hyperthyroidism, the eyes may look enlarged because the upper lids are elevated. Sometimes, one or both eyes may bulge. Some patients have swelling of the front of the neck from an enlarged thyroid gland (a goiter).

WHAT CAUSES HYPERTHYROIDISM?

The most common cause (in more than 70% of people) is overproduction of thyroid hormone by the entire thyroid gland. This condition is also known as Graves' disease (see the *Graves' Disease brochure* for details). Graves' disease is caused by antibodies in the blood that turn on the thyroid and cause it to grow and secrete too much thyroid hormone. This type of hyperthyroidism tends to run in families and it occurs more often in young women. Little is known about why specific individuals get this disease. Another type of hyperthyroidism is characterized by one or more nodules or lumps in the thyroid that may gradually grow and increase their activity so that the total output of thyroid hormone into the blood is greater than normal. This condition is known as toxic nodular or multinodular goiter. Also, people may temporarily have symptoms of hyperthyroidism if they have a condition called thyroiditis. This condition is caused by a problem with the immune system or a viral infection that causes the gland to leak stored thyroid hormone. The same symptoms can also be caused by taking too much thyroid hormone in tablet form. In these last two forms, there is excess thyroid hormone but the thyroid is not overactive.

HOW IS HYPERTHYROIDISM DIAGNOSED?

If your physician suspects that you have hyperthyroidism, diagnosis is usually a simple matter. A physical examination usually detects an enlarged thyroid gland and a rapid pulse. The physician will also look for moist, smooth skin and a tremor of your fingers. Your reflexes are likely to be fast, and your eyes may have some abnormalities if you have Graves' disease.

Hyperthyroidism

The diagnosis of hyperthyroidism will be confirmed by laboratory tests that measure the amount of thyroid hormones— thyroxine (T4) and triiodothyronine (T3)—and thyroid-stimulating hormone (TSH) in your blood. A high level of thyroid hormone in the blood plus a low level of TSH is common with an overactive thyroid gland. If blood tests show that your thyroid is overactive, your doctor may want to measure levels of thyrotropin receptor antibodies (TRAbs), which when elevated confirm the diagnosis of Graves disease. Your doctor may also want to obtain a picture of your thyroid (a thyroid scan). The scan will find out if your entire thyroid gland is overactive or whether you have a toxic nodular goiter or thyroiditis (thyroid inflammation). A test that measures the ability of the gland to collect iodine (a thyroid uptake) may be done at the same time.

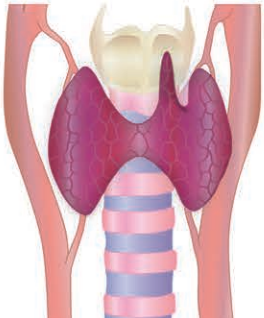
HOW IS HYPERTHYROIDISM TREATED?

No single treatment is best for all patients with hyperthyroidism. The appropriate choice of treatment will be influenced by your age, the type of hyperthyroidism that you have, the severity of your hyperthyroidism, other medical conditions that may be affecting your health, and your own preference. It may be a good idea to consult with an endocrinologist who is experienced in the treatment of hyperthyroid patients. If you are unconvinced or unclear about any thyroid treatment plan, a second opinion is a good idea.

Antithyroid Drugs: Drugs known as antithyroid agents—methimazole (Tapazole®) or in rare instances propylthiouracil (PTU)—may be prescribed if your doctor chooses to treat the hyperthyroidism by blocking the thyroid gland's ability to make new thyroid hormone. Methimazole is presently the preferred one due to less severe side-effects. These drugs work well to control the overactive thyroid, and do not cause permanent damage to the thyroid gland. In about 20% to 30% of patients with Graves' disease, treatment with antithyroid drugs for a period of 12 to 18 months will result in prolonged remission of the disease. For patients with toxic nodular or multinodular goiter, antithyroid drugs are sometimes used in preparation for either radioiodine treatment or surgery.

Antithyroid drugs cause allergic reactions in about 5% of patients who take them. Common minor reactions are red skin rashes, hives, and occasionally fever and joint pains. A rarer (occurring in 1 of 500 patients), but more serious side effect is a decrease in the number of white blood cells. Such a decrease can lower your resistance to infection. Very rarely, these white blood cells disappear completely, producing a condition known as agranulocytosis, a potentially fatal problem if a serious infection occurs. If you are taking one of these drugs and develop a fever or sore throat, you should stop the drug immediately and have a white blood cell count that day. Even if the drug has lowered your white blood cell count, the count will return to normal if the drug is stopped immediately. But if you continue to take one of these drugs in spite of a low white blood cell count, there is a risk of a more serious, even life-threatening infection. Liver damage is another very rare side effect. A very serious liver problem can occur with PTU use which is why this medication should not generally be prescribed. You should stop either methimazole or PTU and call your doctor if you develop yellow eyes, dark urine, severe fatigue, or abdominal pain.

Radioactive Iodine: Another way to treat hyperthyroidism is to damage or destroy the thyroid cells that make thyroid hormone. Because these cells need iodine to make thyroid hormone, they will take up any form of iodine in your bloodstream, whether it is radioactive or not. The radioactive iodine used in this treatment is administered by mouth, usually in a small capsule that is taken just once. once swallowed, the radioactive iodine gets into your bloodstream and quickly is taken up by the overactive thyroid cells. The radioactive iodine that is not taken up by the thyroid cells disappears from the body within days over a period of several weeks to several months (during which time drug treatment may be used to control hyperthyroid symptoms), radioactive iodine destroys the cells that have taken it up. The result is that the thyroid or thyroid nodules shrink in size, and the level of thyroid hormone in the blood returns to normal. Sometimes patients will remain hyperthyroid, but usually to a lesser degree than before.



FURTHER INFORMATION

Further details on this and other thyroid-related topics are available in the patient thyroid information section on the American Thyroid Association® website at *www.thyroid.org*.

For information on thyroid patient support organizations, please visit the *Patient Support Links* section on the ATA website at *www.thyroid.org*

Hyperthyroidism

For them, a second radioiodine treatment can be given if needed. More often, hypothyroidism (an underactive thyroid) occurs after a few months and lasts lifelong, requiring treatment. In fact, when patients have Graves' disease, a dose of radioactive iodine is chosen with the goal of making the patient hypothyroid so that the hyperthyroidism does not return in the future. Hypothyroidism can easily be treated with a thyroid hormone supplement taken once a day (see *Hypothyroidism brochure*).

Radioactive iodine has been used to treat patients for hyperthyroidism for over 60 years and has been shown to be generally safe. Importantly, there has been no clear increase in cancer in hyperthyroid patients that have been treated with radioactive iodine. As a result, in the United States more than 70% of adults who develop hyperthyroidism are treated with radioactive iodine. More and more children over the age of 5 are also being safely treated with radioiodine.

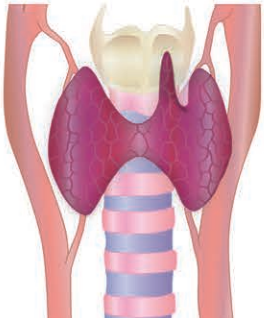
Surgery: Your hyperthyroidism can be permanently cured by surgical removal of all or most of your thyroid gland. This procedure is best performed by a surgeon who has experience in thyroid surgery. An operation could be risky unless your hyperthyroidism is first controlled by an antithyroid drug (see above) or a beta-blocking drug (see below), usually for some days before surgery, your surgeon may want you to take drops of nonradioactive iodine—either Lugol's iodine or supersaturated potassium iodide (SSKI). This extra iodine reduces the blood supply to the thyroid gland and thus makes the surgery easier and safer. Although any surgery is risky, major complications of thyroid surgery occur rarely in patients operated on by an experienced thyroid surgeon. These complications include damage to the parathyroid glands that are next to the thyroid and control your body's calcium levels (causing problems with low calcium levels) and damage to the nerves that control your vocal cords (causing you to have a hoarse voice).

After your thyroid gland is removed, the source of your hyperthyroidism is gone and you will become hypothyroid. As with hypothyroidism that develops after radioiodine treatment, your thyroid hormone levels can be restored to normal by treatment once a day with a thyroid hormone supplement.

Beta-Blockers: No matter which of these three methods of treatment are used for your hyperthyroidism, your physician may prescribe a class of drugs known as beta-blockers that block the action of thyroid hormone on your body. They usually make you feel better within hours to days, even though they do not change the high levels of thyroid hormone in your blood. These drugs may be extremely helpful in slowing down your heart rate and reducing the symptoms of palpitations, shakes, and nervousness until one of the other forms of treatment has a chance to take effect. Propranolol (Inderal®) was the first of these drugs to be developed. Some physicians now prefer related, but longer-acting beta-blocking drugs such as atenolol (Tenormin®), metoprolol (Lopressor®), nadolol (Corgard®), and Inderal-LA® because of their more convenient once- or twice-a-day dosage.

OTHER FAMILY MEMBERS AT RISK

Because hyperthyroidism, especially Graves' disease, may run in families, examinations of the members of your family may reveal other individuals with thyroid problems.



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