

EDITOR'S COMMENTS2

THYROID CANCER.....3

Should the noninvasive encapsulated follicular variant of papillary thyroid cancer be re-classified as benign?

The likelihood of low risk thyroid cancer causing death or recurring is very low and some researchers have debated whether some of these tumors should still be called cancer. The current study goal was to define a subgroup of noninvasive papillary thyroid cancer that is associated with a very low risk of causing death or recurring in the future, after being removed after surgery.

Nikiforov YE et al. Nomenclature revision for encapsulated follicular variant of papillary thyroid carcinoma: a paradigm shift to reduce overtreatment of indolent tumors. JAMA Oncol. April 14, 2016 [Epub ahead of print].

THYROID NODULES5

Reclassification of noninvasive follicular variant of papillary thyroid carcinoma as a benign condition will reduce the incidence of cancer in indeterminate thyroid biopsies

Thyroid biopsy results are characterized into 6 diagnostic categories (the Bethesda system) according to the risk of cancer. Recently, the noninvasive encapsulated follicular variant of papillary thyroid cancer has been suggested to be re-named from a cancer to a benign tumor (NIFTP). The aim of this study is to assess how this reclassification will impact the risk of thyroid cancer for each of the categories in the Bethesda system.

Faquin WC et al. Impact of reclassifying noninvasive follicular variant of papillary thyroid carcinoma on the risk of malignancy in the Bethesda System for Reporting Thyroid Cytopathology. Cancer Cytopathol 2016;124:181-7. Epub October 12, 2015.

THYROID NODULES7

Ultrasound and molecular marker analysis for diagnosing cancer in indeterminate thyroid nodules

Some thyroid biopsies come back as “indeterminate”, meaning that they cannot be diagnosed as benign or cancerous on the basis of the cells alone. Testing for molecular markers can be used to diagnose cancer or to determine that the nodule is benign and can be . Ultrasound is also an important and useful tool to try and determine a nodule's risk of being cancer. This study looked at using a combination of ultrasound characteristics and checking for the 2 most common molecular mutations in thyroid nodules.

De Napoli L et al. Indeterminate single thyroid nodule: synergistic impact of mutational markers and sonographic features in triaging patients to appropriate surgery. Thyroid 2016;26:390-4. Epub December 9, 2015.

HYPERTHYROIDISM9

Risk factors for developing eye disease in patients with Graves' hyperthyroidism

Approximately 25% of patients who develop Graves' hyperthyroidism will also have clinically apparent thyroid eye disease. The aim of this study is to evaluate risk factors for developing eye disease in a large multicenter study of patients with Graves' hyperthyroidism.

Khong JJ et al. Risk factors for Graves' Orbitopathy; the Australian Thyroid-associated Orbitopathy Research (ATOR) Study. J Clin Endocrinol Metab. April 7, 2016 [Epub ahead of print].

HYPOTHYROIDISM11

Thyroid-associated eye disease occurs in 6% of patients with Hashimoto's Thyroiditis

Thyroid-associated eye disease is more common in patients with Graves' disease and may affect ~25% of these patients. However, patients with Hashimoto's may also be affected by thyroid-associated eye disease. This study examined the types of antibodies that are seen in Graves' disease and Hashimoto's thyroiditis, what antibodies are associated with thyroid-associated eye disease and how common is thyroid-associated eye disease in patients with Hashimoto's thyroiditis.

Kahaly GJ et al Thyroid stimulating antibodies are highly prevalent in Hashimoto's thyroiditis and associated orbitopathy. J Clin Endocrinol Metab. March 10, 2016 [Epub ahead of print].

HYPOTHYROIDISM13

Thyroxine reduces epicardial fat tissue mass in subclinical hypothyroidism

Increased thickness of the epicardial fat tissue may lead to heart disease. Some studies have shown that the thickness of epicardial fat tissue is increased in subclinical hypothyroidism. The aim of this study was to determine whether epicardial fat tissue thickness is increased in subclinical hypothyroidism and whether treatment with levothyroxine in these patients will reduce this thickness.

Sayin I et al. Thickening of the epicardial adipose tissue can be alleviated by thyroid hormone replacement therapy in patients with subclinical hypothyroidism. Kardiol Pol. April 26, 2016 [Epub ahead of print].



AMERICAN
THYROID
ASSOCIATION

FOUNDED 1923

www.thyroid.org

Editor

Alan P. Farwell, MD, FACE
Boston Medical Center
Boston University School of Medicine
88 East Newton St., Boston, MA 02115
American Thyroid Association
e-mail: thyroid@thyroid.org
www.thyroid.org/patients/ct/index.html

Editorial Board

Jessie Block-Galaraza, MD, Albany, NY
Gary Bloom, New York, NY
Jamshid Farahati, MD, Bottrop, Germany
Alina Gavril-Filip, MD, Boston, MA
Melanie Goldfarb, MD, MS, FACS, FACE,
Santa Monica, CA
Shirin Haddady, MD, MPH, Boston, MA
Julie E. Hallanger Johnson, MD, Fargo, ND
Ronald Koppersmith, MD, College Station, TX
Angela Leung, MD, Los Angeles, CA
Maria Papaleontiou, MD, Ann Arbor, MI
Liuska Pesce, MD, Iowa City, Iowa
Wendy Sacks, MD, Los Angeles, CA
Anna M. Sawka, MD, Toronto, ON, Canada
Phillip Segal, MD, Toronto, ON, Canada
Vibhavs Sharma, MD, Albany, NY
Whitney Woodmansee, MD, Boston, MA

American Thyroid Association

President

Antonio C. Bianco, MD, PhD

Secretary/Chief Operating Officer

Victor J. Bernet, MD

Treasurer

David H. Sarne, MD

President-Elect

John C. Morris, III, MD

Past-President

Robert C. Smallridge, MD

Executive Director

Barbara R. Smith, CAE
American Thyroid Association
6066 Leesburg Pike, Suite 550
Falls Church, VA 22041
Telephone: 703-998-8890
Fax: 703-998-8893
Email: thyroid@thyroid.org

Designed by

Karen Durland, kdurland@gmail.com

Clinical Thyroidology for the Public

Copyright © 2016 by the American Thyroid Association, Inc. All rights reserved.



CLINICAL **THYROIDOLOGY** FOR THE **PUBLIC**

A publication of the American Thyroid Association

VOLUME 9 • ISSUE 7 • JULY 2016

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 will be providing summaries of research studies that were discussed in a recent issue of *Clinical Thyroidology*, a publication of the American Thyroid Association for physicians. These summaries are present in lay language to allow the rapid dissemination of thyroid research to the widest possible audience. This means that you are getting the latest information on thyroid research and treatment almost as soon as your physicians. As always, we are happy to entertain any suggestions to improve *Clinical Thyroidology for the Public* so let us know what you want to see.

We also provide even faster updates of late-breaking thyroid news through **Twitter** at [@thyroidfriends](https://twitter.com/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* and *Thyroid Federation International*.

July is [Graves' Disease Awareness Month](#).

In this issue, the studies ask the following questions:

1. Is the non-invasive encapsulated variant of papillary thyroid cancer actually a cancer?
2. Does the risk of thyroid cancer change in indeterminate thyroid biopsies with the reclassification of the non-invasive encapsulated variant of papillary thyroid cancer to a benign tumor?
3. Can ultrasound findings combined with limited molecular marker analysis clarify the diagnosis of cancer in indeterminate thyroid biopsies?
4. What are the factors for developing thyroid eye disease in patients with Graves' disease?
5. How common is thyroid eye disease in patients with Hashimotos thyroiditis?
6. Is the thickness of the epicardial fat pad affected by levothyroxine therapy in patients with subclinical hypothyroidism?

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



**THYROID CANCER****Should the noninvasive encapsulated follicular variant of papillary thyroid cancer be re-classified as benign?****BACKGROUND**

The number of patients with thyroid cancer has been rising and more low risk papillary thyroid cancers are being diagnosed in recent years. The likelihood of low risk thyroid cancer causing death or coming back after initial treatment (cancer recurrence) is very low and some researchers have debated whether some of these tumors should still be called cancer. The current study goal was to define a subgroup of noninvasive papillary thyroid cancer that is associated with a very low risk of causing death or recurring in the future, after being removed after surgery. The noninvasive follicular variant thyroid cancers in this study were confined within a tumor capsule in the thyroid gland and did not spread to lymph nodes or other organs.

THE FULL ARTICLE TITLE

Nikiforov YE et al. Nomenclature revision for encapsulated follicular variant of papillary thyroid carcinoma: a paradigm shift to reduce overtreatment of indolent tumors. JAMA Oncol. April 14, 2016 [Epub ahead of print].

SUMMARY OF THE STUDY

In this study, the authors reviewed select thyroid surgical specimens from multiple institutions. The review included specimens from 109 patients with noninvasive encapsulated follicular variant papillary thyroid carcinoma that was confined to a thyroid capsule and who were not treated with radioactive iodine and observed for 10 to 26 years. These were compared to specimens from 101 patients with invasive follicular variant papillary thyroid carcinoma (presence of invasion of blood vessels and or thyroid capsular invasion) observed for 1 to 18 years. The patients were selected on a historical review of pathology records from 13 institutions. A total of 24 expert pathologists reviewed all of the specimen slides. The long-term outcomes of the two groups, based on chart review, was compared. In another phase of the study, 30 of the above cases were subject to molecular marker testing using ThyroSeq v2 panel and 23 pathologists who did not know the molecular results (blinded) reviewed the slides to develop a scoring system for evaluating the nucleus of the

cells. These data were compared to results from benign hyperplastic nodules. Furthermore, the newly developed nuclear scoring system was tested by 22 pathologists who reviewed tumors from 26 patients (validation phase).

This study showed that 12% (12/101) of the patients with invasive follicular variant papillary thyroid carcinoma had a recurrence of their cancer. However, none of the 109 patients with noninvasive encapsulated follicular variant papillary thyroid carcinoma died or had cancer recurrence. This led to the proposal to rename this latter cancer as noninvasive follicular thyroid neoplasm with papillary-like nuclear features (NIFTP). NIFTP diagnostic criteria included all of the following: 1) the tumor being encapsulated or clear demarcated, 2) follicular growth pattern with specific features, 3) a nuclear score of 2 to 3, 4) no invasion of blood vessels in the thyroid, 5) no break-down (necrosis) of the tumor, and 6) no high mitotic (proliferative) activity.

WHAT ARE THE IMPLICATIONS OF THIS STUDY?

The authors concluded that noninvasive encapsulated follicular variant papillary thyroid carcinoma should be re-named as the non-cancer NIFTP in the future. It is important to know that at present, a NIFTP diagnosis cannot be made before thyroid surgery, as the tumor needs to be completely removed for thorough examination. If this is confirmed, then such patients will require much less monitoring and testing after surgery than current cancer patients. More research is needed to confirm the results of this study, to evaluate the performance of the new classification system and to determine the implications on patient's long-term outcomes.

— Anna Sawka, MD

ATA THYROID BROCHURE LINKS

Thyroid Nodules: <http://www.thyroid.org/thyroid-nodules/>

Thyroid Cancer: <http://www.thyroid.org/thyroid-cancer/>

Thyroid Surgery: <http://www.thyroid.org/thyroid-surgery/>

**THYROID CANCER**, continued**ABBREVIATIONS & DEFINITIONS**

Thyroid nodule: an abnormal growth of thyroid cells that forms a lump within the thyroid. While most thyroid nodules are non-cancerous (Benign), ~5% are cancerous.

Papillary thyroid cancer: the most common type of thyroid cancer. There are three variants of papillary thyroid cancer: classic, follicular and tall-cell.

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

Lymph node: bean-shaped organ that plays a role in removing what the body considers harmful, such as infections and cancer cells.

Lobectomy: surgery to remove one lobe of the thyroid.

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).

Molecular markers: genes and microRNAs that are expressed in benign or cancerous cells. Molecular markers can be used in thyroid biopsy specimens to either to diagnose cancer or to determine that the nodule is benign.



**THYROID NODULES**

Reclassification of noninvasive follicular variant of papillary thyroid carcinoma as a benign condition will reduce the incidence of cancer in indeterminate thyroid biopsies

BACKGROUND

Thyroid biopsy is an important test in the evaluation and management of thyroid nodules. Biopsy results are categorized according to 6 main categories (the Bethesda System): 1) non diagnostic, 2) benign, 3) atypia of undetermined significance/follicular lesion of undetermined significance, 4) follicular neoplasm/suspicious for follicular neoplasm, 5) suspicious for malignancy and 6) malignant. Each category is associated with a higher risk of thyroid cancer than the previous one. As such, the risk of thyroid cancer for each category influences subsequent management, since nodules with biopsy results in high risk category will need surgery, whereas those that fall into a low risk class are often followed.

The number of patients diagnosed with papillary thyroid cancer has been rising; however, the majority of these cases are from low risk papillary carcinomas. Recently, the noninvasive encapsulated follicular variant of papillary thyroid cancer has been suggested to be re-named from a cancer to a benign “noninvasive follicular thyroid neoplasm with papillary-like nuclear features (NIFTP)”. The aim of this study is to assess how this reclassification will impact the risk of thyroid cancer for each of the 6 diagnostic categories in the Bethesda system.

THE FULL ARTICLE TITLE

Faquin WC et al. Impact of reclassifying noninvasive follicular variant of papillary thyroid carcinoma on the risk of malignancy in the Bethesda System for Reporting Thyroid Cytopathology. *Cancer Cytopathol* 2016;124:181-7. Epub October 12, 2015.

SUMMARY OF THE STUDY

The authors studied 6943 consecutive thyroid biopsies collected between January 1, 2013 and June 30, 2014 from 5 academic institutions. They also examined the final surgical pathology that was available from 1827 of the

biopsy specimens. With this information, they calculated the risk of thyroid cancer for each Bethesda category and presented the result as a range. This analysis was performed twice, before and after reclassifying the NIFTP as a benign tumor rather than a carcinoma.

The initial risk of thyroid cancer ranges for the Bethesda categories were 4.4%-25.3% for nondiagnostic, 0.9%-9.3% for benign, 12.1% to 31.2% for atypia of undetermined significance, 21.8% to 33.2% for follicular neoplasm, 62.1% to 82.6% for suspicious for malignancy and 75.9% to 99.1% for malignant biopsy specimens. A total of 756 (41%) patients that eventually had surgery were diagnosed with papillary thyroid cancer and 174 (23%) of these were characterized as NIFTP. Reclassification of these NIFTPs as a benign tumor resulted in a decrease in risk of thyroid cancer which was most pronounced in the 3 intermediate risk FNAB categories: atypia of undermined significance (5.2% to 13.6% decrease), follicular neoplasm (9.9% to 15.1% decrease) and suspicious for malignancy (17.6% to 23.4% decrease).

WHAT ARE THE IMPLICATIONS OF THIS STUDY?

The results from this study indicate that reclassifying noninvasive encapsulated follicular variant of papillary thyroid cancer to the non-cancerous NIFTP will have a significant impact on the risk of thyroid cancer, especially in the indeterminate biopsy categories. This may decrease further the need to pursue surgery in these categories. Importantly, more studies are needed before the diagnosis of NIFTP can be clearly considered a benign tumor.

— Philip Segal, MD

ATA THYROID BROCHURE LINKS

Thyroid Nodules: <http://www.thyroid.org/thyroid-nodules/>

Thyroid Cancer: <http://www.thyroid.org/thyroid-cancer/>

**THYROID NODULES**, continued**ABBREVIATIONS & DEFINITIONS**

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

Thyroid biopsy: a simple procedure that is done in the doctor's office to determine if a thyroid nodule is benign (non-cancerous) or cancer. The doctor uses a very thin needle to withdraw cells from the thyroid nodule. Patients usually return home or to work after the biopsy without any ill effects.

Thyroid nodule: an abnormal growth of thyroid cells that forms a lump within the thyroid. While most thyroid nodules are non-cancerous (Benign), ~5% are cancerous.

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.





THYROID NODULES

Ultrasound and molecular marker analysis for diagnosing cancer in indeterminate thyroid nodules

BACKGROUND

Thyroid nodules are common. Although thyroid biopsy is the “gold standard” for diagnosing thyroid cancer, some biopsies come back as “indeterminate”, meaning that they cannot be diagnosed as benign or cancerous on the basis of the cells alone. The practice used to be for all patients with an indeterminate nodule to have surgery, but now there are some tests available that can help assess the risk of cancer. Testing for molecular markers (mutations in genes that are expressed in either benign or cancerous cells) can be used in thyroid biopsy specimens to either to diagnose cancer or to determine that the nodule is benign. However, these tests check for many cancer mutations and can be expensive (if you have an insurance that doesn’t cover it). Ultrasound is also an important and useful tool to try and determine a nodule’s risk of being cancer. This study looked at using a combination of ultrasound characteristics and checking for the 2 most common molecular mutations, BRAF and NRAS, for determining thyroid cancer in patients that had a thyroid nodule that was “suspicious for a follicular neoplasm” and then had surgery.

THE FULL ARTICLE TITLE

De Napoli L et al. Indeterminate single thyroid nodule: synergistic impact of mutational markers and sonographic features in triaging patients to appropriate surgery. *Thyroid* 2016;26:390-4. Epub December 9, 2015.

SUMMARY OF THE STUDY

At total of 258 patients with thyroid nodules that on biopsy had lesions “suspicious for a follicular neoplasm” and then had their entire thyroid removed. They recorded ultrasound features of the suspicious nodule and tested the tissue samples for BRAF and NRAS. About 35% of the nodules were cancerous. Of 8 nodules that had a BRAF mutation, all were cancer. Of 31 nodules that had NRAS mutations, 22 were cancer (70%). No nodules had both mutations. Patients that had 2 malignant-type features on ultrasound had cancer. Adding positive ultrasound features to mutation positivity for a nodule increased the prediction for cancer.

WHAT ARE THE IMPLICATIONS OF THIS STUDY?

This study shows that combining ultrasound features and limited molecular marker testing with BRAF and NRAS could help in the diagnosis of thyroid cancer. Further studies are needed to confirm these finding but this may lead to a less expensive way to evaluate whether a nodule needs to be removed by surgery or can be monitored without surgery.

— Ronald Kuppersmith, MD

ATA THYROID BROCHURE LINKS

Thyroid Cancer: <http://www.thyroid.org/thyroid-cancer/>

Thyroid Nodules: <http://www.thyroid.org/thyroid-nodules/>

ABBREVIATIONS & DEFINITIONS

Thyroid nodule: an abnormal growth of thyroid cells that forms a lump within the thyroid. While most thyroid nodules are non-cancerous (Benign), ~5% are cancerous.

Thyroid Ultrasound: a common imaging test used to evaluate the structure of the thyroid gland. Ultrasound uses soundwaves to create a picture of the structure of the thyroid gland and accurately identify and characterize nodules within the thyroid. Ultrasound is

also frequently used to guide the needle into a nodule during a thyroid nodule biopsy.

Thyroid biopsy: a simple procedure that is done in the doctor’s office to determine if a thyroid nodule is benign (non-cancerous) or cancer. The doctor uses a very thin needle to withdraw cells from the thyroid nodule. Patients usually return home or to work after the biopsy without any ill effects.

**THYROID NODULES**, continued

Indeterminate thyroid biopsy: this happens usually when the diagnosis is a follicular or hurthle cell lesion. Follicular and hurthle cells are normal cells found in the thyroid. Current analysis of thyroid biopsy results cannot differentiate between follicular or hurthle cell cancer from noncancerous adenomas. This occurs in 15-20% of biopsies and often results in the need for surgery to remove the nodule.

Molecular markers: genes and microRNAs that are expressed in benign or cancerous cells. Molecular markers can be used in thyroid biopsy specimens to either to diagnose cancer or to determine that the nodule is benign.

Cancer-associated genes: these are genes that are normally expressed in cells. Cancer cells frequently have mutations in these genes. It is unclear whether mutations in these genes cause the cancer or are just associated with the cancer cells. The cancer-associated genes important in thyroid cancer are BRAF, RET/PTC and RAS.

BRAF gene: this is gene that codes for a protein that is involved in a signaling pathway and is important for cell growth. Mutations in the BRAF gene in adults appear to cause cancer.

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 July is **Graves' Disease Awareness Month** and a bracelet is available through the [ATA Marketplace](#) to support thyroid cancer awareness and education related to thyroid disease.



**HYPERTHYROIDISM****Risk factors for developing eye disease in patients with Graves' hyperthyroidism****BACKGROUND**

Graves' disease is the most common cause of hyperthyroidism. Approximately 25% of patients who develop Graves' hyperthyroidism will also have clinically apparent thyroid eye disease. While thyroid eye disease is most often seen in patients with Graves' disease, it also can be seen with Hashimoto's thyroiditis and includes inflammation of the eyes, eye muscles and the surrounding tissues. Symptoms include dry eyes, red eyes, bulging of the eyes and double vision. Most patients with thyroid eye disease have mild cases; severe cases can be very disfiguring and disabling and there are limited treatment options. Several prior smaller studies have reported that smoking, older age, male gender, exposure to radioactive iodine treatment, and hypothyroidism are predisposing factors for Graves' eye disease. The reason for these associations is not known. The aim of this study is to evaluate risk factors for developing eye disease in a large multicenter study of patients with Graves' hyperthyroidism.

THE FULL ARTICLE TITLE

Khong JJ et al. Risk factors for Graves' Orbitopathy; the Australian Thyroid-associated Orbitopathy Research (ATOR) Study. *J Clin Endocrinol Metab.* April 7, 2016 [Epub ahead of print].

SUMMARY OF THE STUDY

A total of 1042 patients with history of Graves' disease were recruited from endocrine and ophthalmology clinics in Southern Australia between 2009 and 2013. The patients' age, gender, duration since the onset of Graves' hyperthyroidism as well as eye disease, family history of Graves' disease, ethnicity and smoking status were recorded at the time of the study participation. Prior treatment for hyperthyroidism, including antithyroid medications, radioactive iodine and thyroid surgery, was recorded. The eye disease was diagnosed based on presence of symptoms and signs such as stare, red eyes, lid swelling, eye protrusion and blurred vision. An ophthalmologist performed an eye exam in all patients to assess for presence of eye disease and its severity. Several eye and vision parameters were measured, including visual acuity, inflammatory score, lid retraction and eye protrusion.

Among the study patients, 604 (58%) had Graves' eye disease (cases), while 438 patients did not have eye disease (controls). The proportion of women to men was similar in both cases and controls (4:1). The mean age of onset of Graves' hyperthyroidism was about 2.5 years later (43 years vs. 40.6 years) and the mean duration of Graves' hyperthyroidism was longer by almost 4 years (8.8 years vs. 5.0 years) in patients with eye disease as compared to those without eye disease. For every 10-year increase in age at diagnosis of Graves' disease, the risk for eye disease increased by 17% and for each 1-year increase in Graves' disease duration, the risk for eye disease increased by 7%.

The mean age at diagnosis of Graves' eye disease was 45 years. Only 4.8% of patients had onset of eye disease prior to the diagnosis of Graves' hyperthyroidism and most patients were diagnosed with eye disease within the same year as the diagnosis of Graves' hyperthyroidism. A higher proportion of cases were Caucasians (80% vs. 65%) and smokers (current and ex-smokers) (59% vs. 37%) as compared to controls. The risk of having Graves' eye disease was two times higher in smokers, as compared with non-smokers. The presence of a family history of Graves' disease and of serum TSH receptor antibodies did not differ between cases and controls.

A greater proportion of cases than controls underwent treatment for Graves' hyperthyroidism with radioactive iodine (31% vs. 16%) and thyroid surgery (23% vs. 5%), while a lesser proportion of cases used antithyroid medications (87% vs. 99%). The risk of Graves' eye disease was 7 times lower in patients treated with antithyroid medications than those not receiving antithyroid treatment.

Among the patients with Graves' eye disease, 51 (8%) developed impairment of the optic nerve function. These patients had more advanced age (mean age of 55 years) with more severe eye inflammation and restricted extraocular muscle movement. Smoking was not a risk factor for optic nerve involvement.

**HYPERTHYROIDISM, continued****WHAT ARE THE IMPLICATIONS OF THIS STUDY?**

Older age at onset and longer duration of Graves' hyperthyroidism as well as smoking were associated with a higher risk of developing Graves' eye disease. This study confirms the predisposing factors for Graves' eye disease reported by prior studies, with the exception of male gender, which was not a risk factor in this study. Importantly, smoking is a modifiable risk factor consistently associated with Graves' eye disease in all studies.

Patients with Graves' disease should be counseled not to smoke.

— Alina Gavrilă, MD, MMSC

ATA THYROID BROCHURE LINKS

Hyperthyroidism: <http://www.thyroid.org/hyperthyroidism/>

Graves' Disease: <http://www.thyroid.org/graves-disease/>

ABBREVIATIONS & DEFINITIONS

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

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

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

Antithyroid medications: block the thyroid from making thyroid hormone. Methimazole, carbimazole and propylthiouracil are used to treat hyperthyroidism, especially when it is caused by Graves' disease.

Radioactive iodine (RAI) treatment: iodine-131 is the destructive form used to destroy thyroid tissue in the treatment of thyroid cancer and with an overactive thyroid.

TSR receptor antibodies: antibodies often present in the serum of patients with Graves' disease that are directed against the TSH receptor located on the thyroid cell surface, often causing stimulation of this receptor with resulting hyperthyroidism.



HYPOTHYROIDISM

Thyroid-associated eye disease occurs in 6% of patients with Hashimoto's Thyroiditis

BACKGROUND

Autoimmune thyroid diseases comprise two main clinical presentations: Graves' disease (hyperthyroidism) and Hashimoto's thyroiditis (hypothyroidism), both characterized by antibodies that attack the thyroid. Indeed, these disorders are the opposite ends of the same underlying cause. Thyroid-associated eye disease is more common in patients with Graves' disease and may affect ~25% of these patients. However, patients with Hashimoto's may also be affected by thyroid-associated eye disease. This study examined the types of antibodies that are seen in Graves' disease and Hashimoto's thyroiditis, what antibodies are associated with thyroid-associated eye disease and how common is thyroid-associated eye disease in patients with Hashimoto's thyroiditis.

THE FULL ARTICLE TITLE

Kahaly GJ et al Thyroid stimulating antibodies are highly prevalent in Hashimoto's thyroiditis and associated orbitopathy. J Clin Endocrinol Metab. March 10, 2016 [Epub ahead of print].

SUMMARY OF THE STUDY

The records of 700 consecutive patients followed at the endocrine outpatient clinic and at the joint thyroid–eye clinic of the Johannes Gutenberg University Medical Center in Mainz, Germany with a diagnosis of Hashimoto's thyroiditis were reviewed. All patients were screened for thyroid-associated eye disease, which was classified as clinically active or inactive, and as mild, moderate-to-severe, or sight-threatening. For comparison, a group of 53 patients with Graves' disease (26 without and 27 with eye disease) and 302 healthy control subjects were also included in this study.

Thyroid-associated eye disease was present in 44 (6%) of the 700 patients with Hashimoto's thyroiditis. Compared to the patients without thyroid-associated eye disease, those with thyroid-associated eye disease tended to be older (49.3 yr vs. 35.2 yr); have a longer duration of

Hashimoto's thyroiditis (2.4 yr vs. 0.9 yr), were heavier smokers, and were less likely to present with another associated autoimmune disease (only thyroid disease: 75% vs. 52.6%; associated type 1 diabetes, 2.3% vs. 16.3%). Thyroid-associated eye disease was mild and inactive in two thirds of the patients. Eye disease activity/severity was independent of sex, age, thyroid function, and smoking. No differences were noted in the prevalence of hypothyroidism or rate of levothyroxine replacement.

TSAb was positive in 5.5% in the patients with Hashimoto's and thyroid-associated eye disease and 68.2% in those with Hashimoto's but not eye disease. However, the levels of TSAb were higher in the patients with Hashimoto's and thyroid-associated eye disease.

WHAT ARE THE IMPLICATIONS OF THIS STUDY?

This study reports that the prevalence of thyroid-associated eye disease was 6%. In 2/3 of the 44 cases, the eye disease was inactive (i.e., noninflammatory) and mild (not requiring glucocorticoid or radiation therapy). Patients with Hashimoto's thyroiditis and thyroid-associated eye disease were somewhat older, had Hashimoto's for a longer time, were less likely to have another associated autoimmune disease, had higher levels of TSAb and were more likely to be smokers. The study confirms the link of thyroid-associated eye disease with TSAb, the levels of which are strongly correlated with the occurrence and activity/severity of thyroid-associated eye disease.

— Alan P. Farwell, ND, FACE

ATA THYROID BROCHURE LINKS

Hyperthyroidism: <http://www.thyroid.org/hyperthyroidism/>

Hypothyroidism: <http://www.thyroid.org/hypothyroidism/>

Graves' Disease: <http://www.thyroid.org/graves-disease/>

**HYPOTHYROIDISM, continued****ABBREVIATIONS & DEFINITIONS**

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).

Hashimoto's thyroiditis: the most common cause of hypothyroidism in the United States. It is caused by antibodies that attack the thyroid and destroy it.

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.

September 21-25, 2016



86th Annual Meeting of the American Thyroid Association

DENVER, COLORADO
www.thyroid.org



HYPOTHYROIDISM

Thyroxine reduces epicardial fat tissue mass in subclinical hypothyroidism

BACKGROUND

Epicardial fat tissue is a fat deposit surrounding the heart, which is in direct contact with the coronary arteries (responsible for the heart's blood supply). Increased thickness of the epicardial fat tissue may lead to heart disease. Some studies have shown that the thickness of epicardial fat tissue is increased in subclinical hypothyroidism. Similarly, other cardiac risk factors, including high cholesterol levels, are increased in patients with subclinical hypothyroidism. The aim of this study was to determine whether epicardial fat tissue thickness is increased in subclinical hypothyroidism and whether treatment with levothyroxine in these patients will reduce this thickness.

THE FULL ARTICLE TITLE

Sayin I et al. Thickening of the epicardial adipose tissue can be alleviated by thyroid hormone replacement therapy in patients with subclinical hypothyroidism. *Kardiol Pol.* April 26, 2016 [Epub ahead of print].

SUMMARY OF THE STUDY

The study included 44 patients with subclinical hypothyroidism and a control group with 42 healthy patients of the same age and sex. Subclinical hypothyroidism was defined as a persistent TSH > 10 mIU/L or increased TSH after 3 months from baseline (TSH > 5 mIU/L). The group of patients with subclinical hypothyroidism was then treated with levothyroxine at doses to achieve a normal TSH. An echocardiogram was performed to measure the thickness of the epicardial fat tissue at diagnosis and after treatment.

The study found that epicardial fat tissue thickness was significantly greater in patients with subclinical hypothyroidism as compared to controls. Also, treatment with thyroxine to normalize TSH was associated with a decrease in the epicardial fat tissue thickness in the majority of patients with subclinical hypothyroidism. The decrease of the epicardial fat tissue thickness correlated with the extent of the decrease in TSH.

WHAT ARE THE IMPLICATIONS OF THIS STUDY?

This study showed that increased epicardial fat tissue thickness may be seen in patients with subclinical hypothyroidism and may contribute to a possible increase in heart disease risk in these patients. Importantly, treatment with Levothyroxine results in a decrease in the thickness of the epicardial fat tissue. This suggests another reason to consider treating patients with subclinical hypothyroidism. However, the findings of this study should be cautiously interpreted, as measurement of epicardial fat tissue thickness is not currently used in clinical practice because it is not as sensitive of a test as standard tests for coronary artery disease.

— Maria Papaleontiou, MD

ATA THYROID BROCHURE LINKS

Hypothyroidism: <http://www.thyroid.org/hypothyroidism/>

ABBREVIATIONS & DEFINITIONS

Subclinical Hypothyroidism: a mild form of hypothyroidism where the only abnormal hormone level is an increased TSH. There is controversy as to whether this should be treated or not.

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 (T₄): the major hormone produced by the thyroid gland. T₄ gets converted to the active hormone T₃ in various tissues in the body.



HYPOTHYROIDISM, continued

Triiodothyronine (T₃): the active thyroid hormone, usually produced from thyroxine.

Levothyroxine (T₄): the major hormone produced by the thyroid gland and available in pill form as Synthroid™, Levoxyl™, Tyrosint™ and generic preparations.

Coronary artery disease: it develops when the major blood vessels that supply your heart with blood, oxygen and nutrients (coronary arteries) become damaged or diseased.

**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 towards the improvement of thyroid education and resources for patients.

WHO WE ARE (in alphabetical order)

- **American Thyroid Association**
- **Bite Me Cancer**
- **Graves' Disease and Thyroid Foundation**
- **Light of Life Foundation**
- **ThyCa: Thyroid Cancer Survivors' Association, Inc.**
- **Thyroid Cancer Canada**
- **Thyroid Federation International**

AMERICAN THYROID ASSOCIATION

www.thyroid.org

ATA Patient Resources: <http://www.thyroid.org/patients-portal/>

Find a Thyroid Specialist: www.thyroid.org

Phone (toll-free): 1-800-THYROID

e-mail: thyroid@thyroid.org

ATA Mission: The ATA leads in promoting thyroid health and understanding thyroid biology.

ATA Vision: The ATA is the leading organization focused on thyroid biology and the prevention and treatment of thyroid disorders through excellence and innovation in research, clinical care, education, and public health.

ATA Values: The ATA values scientific inquiry, clinical excellence, public service, education, collaboration, and collegiality.

To further our mission, vision and values the ATA sponsors "Friends of the ATA" online to advance the information provided to patients and the public such as this publication, *Clinical Thyroidology for the Public*. We welcome your support.

continued on next page

AMERICAN
THYROID
ASSOCIATION
FOUNDED 1923

ThyCa: Thyroid Cancer
Survivors' Association, Inc.SM
www.thyca.org



**ATA Alliance for Thyroid Patient Education****Continued...****BITE ME CANCER**<http://www.bitemecancer.org>

Bite Me Cancer was formed as a nonprofit foundation in September, 2010, by Nikki Ferraro, who was 17-years old at the time. Nikki was diagnosed with a rare form of thyroid cancer in April 2010 when she was a junior at Chantilly HS in Virginia. Nikki was determined to lead a Relay for Life team just two weeks after her diagnosis. She named the team Bite Me Cancer and experienced immediate success. When Nikki decided to create a foundation a few months later, she wanted to continue the legacy of her team name and thus her foundation became the Bite Me Cancer Foundation.

e-mail: info@bitemecancer.org**GRAVES' DISEASE AND THYROID FOUNDATION**www.gdatf.org

Phone (toll-free): 1-877-NGDF-123 or 643-3123

e-mail: Gravesdiseasefd@gmail.com

Founded in 1990, the Graves' Disease Foundation offers support and resources to Graves' disease patients, their families, and health care professionals. Their mission is to find the cause of and the cure for Graves' thyroid disease through research, to improve the quality of life for persons with Graves' disease and their caregivers and to educate persons with Graves' disease, their caregivers, healthcare professionals, and the general public about Graves' disease and its treatment. The web site features a monitored bulletin board.

LIGHT OF LIFE FOUNDATIONwww.checkyourneck.comemail: info@checkyourneck.com

The Light of Life Foundation, founded in 1997, is a nonprofit organization that strives to improve the quality of life for thyroid cancer patients, educate the public and professionals about thyroid cancer, and promote research and development to improve thyroid cancer care.

continued on next pageAMERICAN
THYROID
ASSOCIATION
FOUNDED 1923ThyCa: Thyroid Cancer
Survivors' Association, Inc.SM
www.thyca.org



AMERICAN
THYROID
ASSOCIATION
FOUNDED 1923

www.thyroid.org

ATA Alliance for Thyroid Patient Education

Continued...

THYCA: THYROID CANCER SURVIVORS' ASSOCIATION, INC.

www.thyca.org

Phone (toll-free): 877 588-7904

e-mail: thyca@thyca.org

ThyCa: Thyroid Cancer Survivors' Association, Inc., founded in 1995, is an international nonprofit organization, guided by a medical advisory council of renowned thyroid cancer specialists, offering support and information to thyroid cancer survivors, families, and health care professionals worldwide.

THYROID CANCER CANADA

www.thyroidcancercanada.org

Phone: 416-487-8267

Fax: 416-487-0601

e-mail: info@thyroidcancercanada.org

Thyroid Cancer Canada is a non-profit organization founded in 2000. The organization works towards creating an environment in which people who are dealing with thyroid cancer, especially the newly diagnosed, are met with support and information. Their goals & objectives include facilitating communication among thyroid cancer patients, providing credible information about the disease, providing emotional support, and assisting thyroid cancer patients with voicing their needs to health care professionals and those who are responsible for health care policy.

THYROID FEDERATION INTERNATIONAL

www.thyroid-fed.org

e-mail: tfi@thyroid-fed.org

Thyroid Federation International (TFI) was established in Toronto in 1995. Thyroid Federation International aims to work for the benefit of those affected by thyroid disorders throughout the world by providing a network of patient support organizations.



AMERICAN
THYROID
ASSOCIATION
FOUNDED 1923



ThyCa: Thyroid Cancer
Survivors' Association, Inc.SM
www.thyca.org



Join us for the

September 21-25, 2016

86th Annual Meeting of the American Thyroid Association

DENVER, COLORADO
www.thyroid.org



JOIN EXPERTS AND THOUGHT LEADERS IN FIELD OF THYROIDOLOGY TO HEAR INNOVATIVE TALKS, participate in interactive sessions, and network with friends and colleagues at the ATA Annual Meeting. Held at the Sheraton Denver Downtown Hotel in Denver, Colorado, the ATA meeting is open to all health care professionals interested in broadening their knowledge of the thyroid gland and its disorders. The ATA Program Committee led, by Co-Chairs Peter Arvan and Stephanie Fish, have developed a scientific program to satisfy the interests of all audiences.

The Ridgway Trainee Conference, the full-day satellite ultrasound course and focused discussion debate, will be available to accent the robust meeting agenda. Don't miss your opportunity to earn CME credits, develop professionally and foster long lasting connections.

ATA 2016 CALL FOR ABSTRACT SUBMISSIONS

Regular Call:

Site Closed – Wednesday, May 25, 2016

Short Call:

Site Opens – Wednesday, July 27, 2016

Site Closes – Wednesday, August 10, 2016

REGISTRATION and HOUSING OPEN NOW AT WWW.THYROID.ORG

Agenda, meeting updates, exhibitor and sponsor opportunities available online.

SAVE THE DATE FOR THESE UPCOMING ATA MEETINGS:

87th Annual Meeting of the American Thyroid Association – October 18-22, 2017
The Fairmont Empress and Victoria Conference Center, Victoria, BC, Canada

88th Annual Meeting of the American Thyroid Association – October 3-7, 2018
Marriott Marquis, Washington, DC

89th Annual Meeting of the American Thyroid Association – October 30-November 3, 2019
Sheraton Grand Chicago, Chicago, IL

Spring Meeting of the American Thyroid Association – May 28-30, 2020
Westin New York at Times Square, New York, NY



AMERICAN
THYROID
ASSOCIATION
FOUNDED 1923

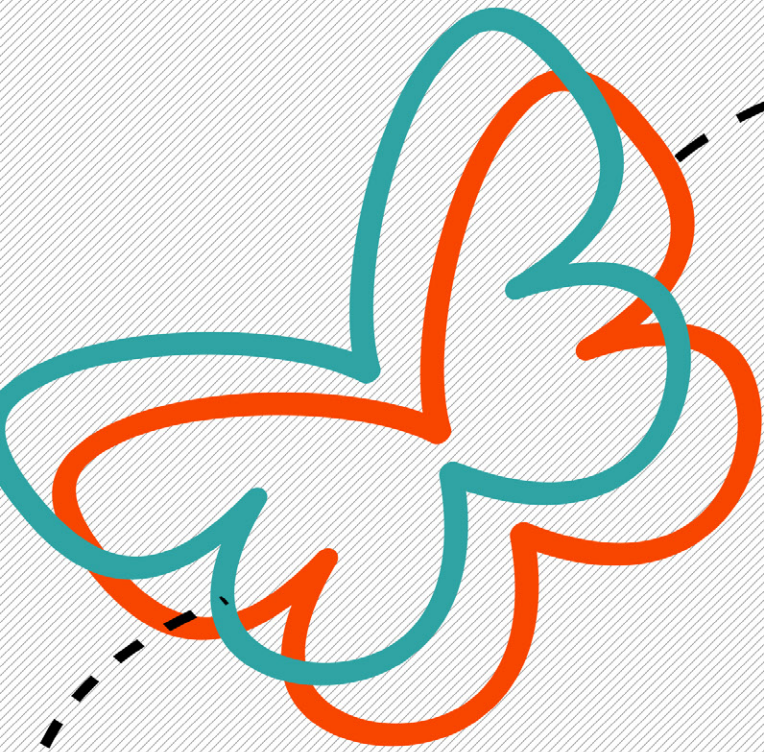
6066 Leesburg Pike, Suite 550, Falls Church VA 22041 USA | 703.998.8890
thyroid@thyroid.org | www.thyroid.org

American Thyroid Association

Dedicated to scientific inquiry, clinical excellence, public service, education and collaboration

Reasons to #GIVE2THYROID

www.thyroid.org/donate



Reason

1

Public & Thyroid Patients

The American Thyroid Association® is dedicated to serving as an educational resource for the public by supporting thyroid research and promoting the prevention, treatment and cure of thyroid-related diseases and thyroid cancer. Help support the continuation of our patient/public education programs and resources including:

- +thyroid brochures
- +summarized medical literature
- +endocrinologist referral
- +monthly newsletters
- +support links
- +patient alliance community
- +health and education forums

Thyroid Physicians, Scientists & Professionals

The American Thyroid Association® provides outstanding leadership in thyroidology by promoting excellence and innovation in clinical management, research, education, and patient care. Help support thyroid specialists and the development of resources that advance our understanding of thyroid disorders and cancer including:

- +clinical practice guidelines
- +position statements
- +early career training
- +research and education grants
- +leadership & service awards
- +community for collaboration
- +continuing education programs
- +peer-review biomedical journals
- +summarized medical literature
- +up to date thyroid news & publications
- +patient education

American Thyroid Association

#GIVE2THYROID

www.thyroid.org/donate



Graves' Disease

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 GRAVES' DISEASE?

Graves' disease is caused by a generalized overactivity of the entire thyroid gland (*hyperthyroidism*). It is named for Robert Graves, an Irish physician, who described this form of hyperthyroidism about 150 years ago.

WHAT ARE THE SYMPTOMS OF GRAVES' DISEASE?

• HYPERTHYROIDISM

The majority of symptoms of Graves' disease are caused by the excessive production of thyroid hormones by the thyroid (*see Hyperthyroidism brochure*).

• EYE DISEASE

Graves' disease is the only kind of hyperthyroidism that can be associated with inflammation of the eyes, swelling of the tissues around the eyes and bulging of the eyes (*called Graves' ophthalmopathy*). Although many patients with Graves' disease have redness and irritation of the eyes at some time, less than five percent ever develop enough inflammation of the eye tissues to cause serious or permanent trouble. Patients who have more than very mild eye symptoms do require an evaluation with an eye doctor (*an ophthalmologist*) as well as their endocrinologist.

Eye symptoms most often begin about six months before or after the diagnosis of Graves' disease has been made. Seldom do eye problems occur long after the disease has been treated. In some patients with eye symptoms, hyperthyroidism never develops and, rarely, patients may be hypothyroid. The severity of the eye symptoms is not related to the

severity of the hyperthyroidism. Early signs of trouble might be red or inflamed eyes, a bulging of the eyes due to inflammation of the tissues behind the eyeball or double vision. Diminished vision or double vision are rare problems that usually occur later if at all. We do not know why, but problems with the eyes occur much more often and are more severe in people with Graves' disease who smoke cigarettes.

• SKIN DISEASE

Rarely, patients with Graves' disease develop a lumpy reddish thickening of the skin in front of the shins known as pretibial myxedema. This skin condition is usually painless and relatively mild, but can be painful. Like the eye trouble of Graves' disease, the skin problem does not necessarily begin precisely when the hyperthyroidism starts. Its severity is not related to the level of thyroid hormone.

WHAT CAUSES GRAVES' DISEASE?

• IMMUNE SYSTEM

Graves' disease is triggered by some process in the body's immune system, which normally protects us from foreign invaders such as bacteria and viruses. The immune system destroys foreign invaders with substances called antibodies produced by blood cells known as lymphocytes. Some people inherit an immune system that can cause problems. Their lymphocytes make antibodies against their own tissues that stimulate or damage them. In Graves' disease, antibodies bind to the surface of thyroid cells and stimulate those cells to overproduce thyroid hormones. This results in an overactive thyroid.

• EYE CHANGES

These same antibodies may also be involved in the eye changes seen in Graves' ophthalmopathy, since the receptors on the thyroid may also be found on the surface of cells behind the eye. Physicians have long suspected that severe emotional stress, such as the death of a loved one, can set off Graves' disease in some patients. Dr. Graves himself commented on stressful events in his patients' lives that came several months before the development of hyperthyroidism. However, most patients who develop Graves' disease report no particular recent stress in their lives.

Graves' Disease

HOW IS THE DIAGNOSIS OF GRAVES' DISEASE MADE?

The diagnosis of hyperthyroidism is made on the basis of your symptoms and findings during a physical exam and it is confirmed by laboratory tests that measure the amount of thyroid hormone (*thyroxine, or T4, and triiodothyronine, or T3*) and thyroid-stimulating hormone (*TSH*) in your blood (*see the Hyperthyroidism brochure*). Sometimes your doctor may want you to have a radioactive image, or scan, of the thyroid to see whether the entire thyroid gland is overactive. Your doctor may also wish to do a blood test to confirm the presence of thyroid-stimulating antibodies (*TSI or TRAb*) that cause Graves' disease, but this test is not usually necessary.

Clues that your hyperthyroidism is caused by Graves' disease are the presence of Graves' eye disease (see above), an enlarged thyroid and a history of other family members with thyroid or autoimmune problems. Some relatives may have had hyperthyroidism or an underactive thyroid; others may have other autoimmune diseases including premature graying of the hair (*beginning in their 20's*). Similarly, there may be a history of related immune problems in the family, including juvenile diabetes, pernicious anemia (*due to lack of vitamin B12*) or painless white patches on the skin known as vitiligo.

HOW IS GRAVES' DISEASE TREATED?

The treatment of hyperthyroidism is described in detail in the Hyperthyroidism brochure. Treatment includes antithyroid drugs (*generally methimazole [Tapazole®], although propylthiouracil [PTU] may be used in rare instances*), radioactive iodine and surgery. Although each treatment has its advantages and disadvantages, most patients will find one that is just right for them. Hyperthyroidism due to Graves' disease is, in general, easily controlled and safely treated and treatment is almost always successful.

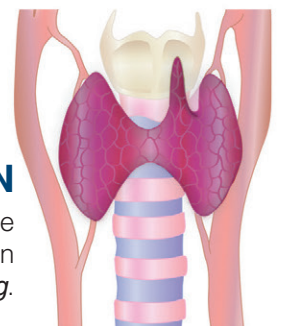
WHAT WILL BE THE OUTCOME OF TREATMENT?

No matter how your hyperthyroidism is controlled, you will probably eventually develop hypothyroidism (*underactive thyroid*). Hypothyroidism will occur sooner if your thyroid has been treated by radioactive iodine or removed in an operation. Even if you are treated with antithyroid drugs alone, hypothyroidism still can occur.

Because of this natural tendency to progress toward hypothyroidism sometime after you have been hyperthyroid, every patient who has ever had hyperthyroidism due to Graves' disease should have blood tests at least once a year to measure thyroid function. When hypothyroidism occurs, a thyroid hormone tablet taken once a day can treat it simply and safely (*see the Hypothyroidism brochure*).

OTHER FAMILY MEMBERS AT RISK

Because Graves' disease is related to a genetic predisposition, examinations of the members of your family may reveal other individuals with thyroid problems.



FURTHER INFORMATION

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