Clinical THYROIDOLOGY FOR PATIENTS

AMERICAN THYROID ASSOCIATION
FOUNDED 1923

VOLUME 5 • ISSUE 4 • 2012

www.thyroid.org

A publication of the American Thyroid Association

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EDITOR'S COMMENTS

Welcome to Clinical Thyroidology for Patients, bringing to you, the patients, the most up-to-date, cutting edge thyroid research. What you read here as research studies will likely become the accepted practice in the future. *Clinical Thyroidology for Patients* is published on a monthly basis and includes summaries of research studies that were discussed in a recent issue of *Clinical Thyroidology*, a publication of the American Thyroid Association for physicians. This means that you, the patients, are getting the latest information on thyroid research and treatment almost as soon as your physicians.

The Calendar of Events highlights educational forums and support groups that are organized around the country by members of the Alliance for Thyroid Patient Education. The Alliance member groups consist of: the *American Thyroid Association*, the *Graves' Disease and Thyroid Foundation*, the *Light of Life Foundation* and *ThyCa: Thyroid Cancer Survivors Association*.

In this issue, the studies ask the following questions:

- How effective are antithyroid drugs in treating Graves' Disease in children?
- Do thyroid disorders cause postpartum psychosis?
- Does subclinical hypothyroidism in the mother affect the baby's IQ scores?
- Can medical treatment shrink thyroid nodules?
- Do suppressive levothyroxine doses in thyroid cancer patients cause heart disease?
- Does RAI use in thyroid cancer patients depend on the hospital where the patient is treated?

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

HOW TO NAVIGATE THIS DOCUMENT: The Table of Contents and the Bookmarks are linked to the articles. To navigate, move your cursor over the article title you wish to see (either in the Contents or in the Bookmarks panel) and the hand will show a pointing finger, indicating a link. Left-click the title and the article will instantly appear. To return to the Contents, move the cursor to the bottom of the page and left-click Back to Table of Contents.

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HYPERTHYROIDISM

Prolonged therapy with antithyroid medication leads to a 50% remission rate for Graves' Disease in children

BACKGROUND

Graves' Disease is the most common cause of hyperthyroidism in children. Graves' Disease is caused by the patient's immune system producing an antibody that attacks and turns on the thyroid. One of the goals of treating patients with antithyroid medications is the hope that these drugs may cause the immune system to stop producing the antibody, resulting in the Graves' Disease going into remission. The recommended anthithyroid medication in the United States is Methimazole while in Europe Carbimazole is frequently used. Carboimazole is actually converted into Methimazole in the body. Unfortunately, an initial course of antithyroid medication for up to two years is effective in producing a remission in only ~30% of patients. The other two treatment option for Graves' Disease, destruction of the thyroid gland by radioactive iodine treatment and surgical removal of the gland, usually cause permanent hypothyroidism requiring lifelong thyroid hormone therapy. Since all of these therapies have good and bad points, the recommended treatment varies among pediatric endocrinologists and institutions. The goal of this study was to determine the effect prolonged treatment with Carbimazole on remission of Graves' Disease in children.

THE FULL ARTICLE TITLE

Léger J et al.. French Childhood Graves' Disease Study Group: Positive impact of long-term antithyroid drug treatment on the outcome of children with Graves' disease: national long-term cohort study. J Clin Endocrinol Metab. October 26, 2011 [Epub ahead of print].

SUMMARY OF THE STUDY

A total of 154 patients with Graves' Disease age 18 or younger were followed at multiple centers in France. The patients were initially treated with Carbimazole for 2 years before discontinuation of the drug. The patients were followed for at least 18 months to evaluate whether they remained in remission or relapsed. In case of relapse, they were given an option of another course of Carbimazole or definitive therapy with either radioactive iodine or surgery. This was repeated for up to 3 treatment cycles of Carbimaziole. The average age of the patients was ~12 years and they were followed for an average of 10.4 years. The remission rates after the discontinuation of Carbimazole increased progressively to 20%, 37%, 45%, and 49% after 4, 6, 8, and 10 years of follow-up, respectively. After 10 years of follow up, over 50% of patients were in remission while 11% of patients were still receiving Carbimazole and 36% opted for definitive therapy with either radioactive iodine or surgery. Only 3 patients developed a serious adverse reaction that required stopping the medication.

WHAT ARE THE IMPLICATIONS OF THIS STUDY?

This study showed that Graves' Disease remission rates increase with the duration of antithyroid drug treatment and may be up to 50%. The prolonged treatment with Carbimazole was well tolerated with only 3 adverse reactions. Continuous long-term treatment rather than several 2 year treatment cycles may have more beneficial effects on Graves' Disease remission and should be studied further.

— Alina Gavrila, MD

ATA THYROID BROCHURE LINKS

Hyperthyroidism: http://thyroid.org/patients/patient_brochures/hyperthyroidism.html

Graves' disease: http://thyroid.org/patients/patient_brochures/graves.html

Radioactive Iodine Therapy: http://thyroid.org/patients/patient_brochures/radioactive.html

Thyroid Surgery: http://thyroid.org/patients/patient_brochures/surgery.html

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HYPERTHYROIDISM, continued

ABBREVIATIONS & DEFINITIONS

Hyperthyroidism: a condition where the thyroid gland is overactive and produces too much thyroid hormone. Hyperthyroidism may be treated with antithyroid medications (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.

Antibodies: proteins that are produced by the body's immune cells that attack and destroy bacteria and viruses that cause infections. Occasionally the antibodies get confused and attack the body's own tissues, causing autoimmune disease.

Immune system: a system of organs, tissues, and cells in our body that has the role to recognize potentially harmful foreign substances and organisms as well as abnormal body cells and produce antibodies to destroy these factors.

Antithyroid treatment: medications that block the thyroid from making thyroid hormone. Methimazole and Propylthiouracil (PTU) have been approved to treat hyperthyroidism in the United States especially when it is caused by Graves' disease. Carbimazole, which is converted to Methimazole in the body, is also used outside the US.

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

Hypothyroidism: a condition where the thyroid gland is underactive and does not produce enough thyroid hormone. Treatment requires taking thyroid hormone pills.

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THYROID AND PREGNANCY

Postpartum psychosis is more prevalent in women with autoimmune thyroid disease

BACKGROUND

After delivering a baby, some women may develop psychiatric issues including postpartum depression. The most extreme of these psychiatric problems is postpartum psychosis and is a psychiatric emergency. Postpartum psychosis is characterized by mood changes with delusions and hallucinations, agitation, insomnia and cognitive impairment, which may be accompanied by thoughts of suicide and infanticide. It occurs in 0.1% of new mothers, often without prior warning signs or prior history of a psychiatric disorder. The cause of postpartum psychosis is unknown. Because they can also cause marked changes in mood and are relatively common in the postpartum period, thyroid disorders have been suggested as either causing or contributing to postpaturm psychosis. Most thyroid disorders in the postpartum period are caused by patients developing antibodies that attack the thyroid and either turn it on (hyperthyroidism) or turn it off (hypothytroidism). This is called autoimmune thyroid disease and is characterized by positive TPO antibodies. The aim of this study was to see if autoimmune thyroid disease is associated with postpartum psychosis.

THE FULL ARTICLE TITLE

Bergink V et al. Prevalence of autoimmune thyroid dysfunction in postpartum psychosis. Br J Psychiatry 2011;198:264-8. Epub February 22, 2011.

SUMMARY OF THE STUDY

All patients attending the mother and inpatient unit of the Department of Psychiatry at the Erasmus Medical Centre in the Netherlands between August 2005 and November 2008 were evaluated for postpartum psychosis. Overall, 31 patients were identified as having psychosis following the delivery of their first child. None of the women had a prior psychiatric history. A total of 23 patients presented with manic psychosis, 5 with a mixed episode of mania and depression and 3 with psychotic depression. The thyroid status in these patients was compared to 117 women who had delivered their first baby and were followed for the first year after delivery. Blood levels of TPO antibodies, TSH and free T_4 were measured at various times in the patients with postpartum psychosis and at 4 weeks and 9 months postpartum in the control women.

None of the patients or controls had a prior history of thyroid or autoimmune disease. At 4 weeks postpartum, 5% of the control group had positive autoimmune thyroid disease, while 19% of the patients with postpartum psychosis had autoimmune thyroid disease on admission to the hospital and half exhibited clinical thyroid dysfunction. At 9 months, 13% of the controls and 29% of the women with postpartum psychosis had autoimmune thyroid disease. Patients with postpartum psychosis and autoimmune thyroid disease had a higher rate of progression to clinical thyroid dysfunction (67% in the postpartum psychosis group versus 20% in the control group). Lithium treatment may have played a role in the worsening of thyroid disease in the women with postpartum psychosis and autoimmune thyroid disease because lithium is known to alter thyroid function in patients with autoimmune thyroid disease.

WHAT ARE THE IMPLICATIONS OF THIS STUDY?

Women with postpartum psychosis are at higher risk for autoimmune thyroid disease and clinical thyroid disorders, and autoimmune thyroid disease may play a role in the development of postpartum psychosis. These results indicate that patients presenting with postpartum psychosis should be screened by measurement of blood levels of TPO antibodies. Also, patients known to have autoimmune thyroid disease either before pregnancy or developing postpartum may be at higher risk for developing postpartum psychosis. The connection between autoimmune thyroid disease and postpartum psychosis clearly requires further study.

— Glenn Braunstein, MD

ATA THYROID BROCHURE LINKS

Postpartum Thyroiditis: http://thyroid.org/patients/patient_brochures/postpartum.html

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THYROID AND PREGNANCY, continued

ABBREVIATIONS & DEFINITIONS

Antibodies: proteins that are produced by the body's immune cells that attack and destroy bacteria and viruses that cause infections. Occasionally the antibodies get confused and attack the body's own tissues, causing autoimmune disease.

Autoimmune thyroid disease (AITD): 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).

Thyroxine (T_4): the major hormone secreted by the thyroid gland. Thyroxine is broken down to produce Triiodothyronine which causes most of the effects of the thyroid hormones. Free T_4 refers to the biologically active form that is not bound to proteins in the blood, and, therefore, can enter the cells throughout the body.

Postpartum thyroiditis: an inflammation of the thyroid in women who have just delivered a baby. The inflammation first causes mild hyperthyroidism for 1-3 months after delivery. This is followed by hypothyroidism starting 4-6 months after delivery. The hypothyroidism resolves and normal thyroid function returns 12-18 months after delivery in most women. While many women have both the hyperthyroid and the hypothyroid phase, some women may only have one or the other.

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.

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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THYROID AND PREGNANCY

Subclinical hypothyroidism during pregnancy and baby IQ scores

BACKGROUND

Thyroid hormone is needed for normal brain development. The absence of thyroid hormone during development leads to irreversible mental retardation. Babies born to mothers with severe hypothyroidism that is either unrecognized or untreated during pregnancy may have lower IQ scores. Current guidelines by the American Thyroid Association recommend keeping the TSH <2.5 in hypothyroid women during pregnancy. However, it is unclear if babies born to mothers with only very mild hypothyroidism (subclinical hypothyroidism, where the TSH is increased but the other thyroid hormones are normal) during pregnancy will have the same problems. This study compared the IQ scores between children whose mothers had subclinical hypothyroidism during pregnancy and children whose mothers had normal thyroid function during pregnancy.

THE FULL ARTICLE TITLE

Behrooz HG et al. IQ scores of children evaluated between ages 4 and 14.5 years born to women with subclinical hypothyroidism were similar to the IQs of children born to euthyroid treated women. Subclinical hypothyroidism in pregnancy: intellectual development of offspring. Thyroid 2011;21:1143-7.

SUMMARY OF THE STUDY

This was a study of 44 pregnant Iranian hypothyroid mothers who had their babies born between 1991-2003. The researchers performed IQ testing on the mothers' children when they reached 4-15 years of age. All of the women had been receiving thyroid hormone medication for their hypothyroidism before becoming pregnant, but some mothers remained mildly hypothyroid at the beginning of pregnancy. The authors found no difference in any of the childrens' IQ scores. The 25 children born to mothers with mild hypothyroidism at the beginning of pregnancy had similar IQs compared to the 19 children born to mothers who had normal thyroid function.

WHAT ARE THE IMPLICATIONS OF THIS STUDY?

This study suggests that children's IQ scores are not affected by mothers' mild hypothyroidism at the beginning of pregnancy. These results agree with some, but not all, of the other studies that have been done on this topic. Since the potential effects of hypothyroidism on brain development can be devastating, further research is urgently needed. Until then the recommendation to keep the TSH <2.5 in pregnant hypothyroid women remains the standard of care.

— Angela Leung, MD

ATA THYROID BROCHURE LINKS

Hypothyroidism: http://thyroid.org/patients/patient_brochures/hypothyroidism.html

Thyroid and Pregnancy: http://thyroid.org/patients/patient_brochures/pregnancy.html

Thyroid Function Tests: http://thyroid.org/patients/patient_brochures/function_tests.html

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ABBREVIATIONS & DEFINITIONS

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

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.

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GOITER

Treatment of goiter and nodules is successful with levothyroxine plus iodine

BACKGROUND

Thyroid nodules (single and multiple) are common. In some European countries with moderate iodine deficiency, goiters (enlarged thyroid glands) and/or thyroid nodules can be found in a third of adults. In the past, patients with nodular goiters whose nodules are benign (noncancerous) had 3 options: 1) no treatment, observation only, 2) surgery, especially with large goiters that cause symptoms and 3) levothyroxine suppression therapy producing a low TSH and a decrease in the size of the goiter. Levothyroxine therapy is not only unpredictable and highly variable, but side effects are frequent, including irregular heart rhythms (atrial fibrillation) and a tendency toward bone loss and osteoporosis in postmenopausal women. Because of this, levothyroxine therapy is no longer recommended as a treatment option. However, a few studies have indicated that levothyroxine therapy that keeps the TSH in the normal range was still able to reduce thyroid or nodule size to some extent in some patients. Some physicians have recommended the addition of iodine for a decrease in goiter and nodule size, especially in Europe. In this study in a modestly iodine-deficient population, the effects of levothyroxine and iodine together on decreasing the size of goiters was examined.

THE FULL ARTICLE TITLE

Grussendorf M et al. Reduction of thyroid nodule volume by levothyroxine and iodine alone and in combination: a randomized, placebo-controlled trial. J Clin Endocrinol Metab. June 29, 2011 [Epub ahead of print]. doi: 10.1210/jc.2011-0356.

SUMMARY OF THE STUDY

In more than 60 centers, a total of 1013 patients were studied for 12 months. The size of nodules and goiters were followed by serial ultrasound studies. There were 4 groups: 1) levothyroxine only, 2) iodide only, 3) levothyroxine plus iodine and 4) no therapy. The TSH target in patients treated with levothyroxine was in the low normal range.

A decrease in the total thyroid size was significant in all of the treatment groups as compared to the group that was not treated. The best response was seen in the levothyroxine plus iodide group and was ~10%. All treatment groups caused a decrease in size of individual nodules, with the levothyroxine plus iodine group the most effective with ~17% decrease in size. However, the response of the individual cases was quite variable with 26% of the goiters actually increased in size even under treatment. There were no significant side effects in the patients that received levothyroxine.

WHAT ARE THE IMPLICATIONS OF THIS STUDY?

In a moderately iodine-deficient area, 12 months of treatment with levothyroxine plus iodine significantly reduced thyroid nodule size. However, 26% of the nodules actually increased in size with levothyroxine plus iodine treatment. It is unclear if similar results would be obtained in regions with sufficient iodine intake, such as the United States. While these results are interesting and require further study, levothyroxine therapy is still not generally recommended to shrink thyroid nodules or goiters.

— Alan Farwell, MD

ATA THYROID BROCHURE LINKS

Thyroid Nodules: http://thyroid.org/patients/patient_brochures/nodules.html

Goiter: http://thyroid.org/patients/patient_brochures/goiter.html

Iodine Deficiency: http://thyroid.org/patients/patient_brochures/iodine_deficiency.html

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GOITER, continued

ABBREVIATIONS & DEFINITIONS

Goiter: a thyroid gland that is enlarged for any reason is called a goiter. A goiter can be seen when the thyroid is overactive, underactive or functioning normally. If there are nodules in the goiter it is called a nodular goiter; if there is more than one nodule it is called a multinodular goiter.

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.

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.

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.

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.

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

Iodine: an element found naturally in various foods that is important for making thyroid hormones and for normal thyroid function. Common foods high in iodine include iodized salt, dairy products, seafood and some breads.

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THYROID AND THE HEART

Can we measure early changes in heart function in thyroid cancer patients taking high dose of thyroid replacement?

BACKGROUND

Thyroid cancer patients are treated after thyroid surgery with suppressive thyroxine therapy (Synthroid, Levothyroxine, Levoxyl, etc.). This means that the dose given is slightly higher than the body usually needs, causing the TSH to be suppressed into the low range. The goal of this therapy is to prevent growth of any remaining thyroid cancer cells. The problem with this treatment is that chronically higher than normal thyroid hormone levels may affect the heart, which can be seen in patients with hyperthyroidism. The earliest change noted in those patients on an echocardiogram is a thickening of the wall of left main chamber of the heart (ventricle). It is unclear if this causes any problems. A new technique at looking at heart function known as speckle tacking echocardiography (STE) can directly examine how the heart works. In this study, the authors looked at the effects of suppressive thyroxine therapy on the heart function using STE.

THE FULL ARTICLE TITLE

Taillard V et al Early detection of Isolated Left Ventricular Diastolic Dysfunction in High-Risk Differentiated Thyroid Carcinoma Patients on TSH-Suppressive Therapy. Clin Endocrinol (Oxf) 2011;75:709-14.

SUMMARY OF THE STUDY

A total of 24 patients with thyroid cancer and without known heart disease, diabetes or high blood pressure were studied. All were treated with thyroxine for 36 months and had a TSH in the low range. The control group had 20 age-matched subjects not on thyroid medication and with normal thyroid function. Their heart function was examined using both echocardiograms and STE.

No difference in cardiac function was detected between the thyroxine-treated and control groups on the echocardiograms. However, with STE, abnormalities in heart relaxation and the size of the left ventricle were seen in the thyroxine-treated group.

WHAT ARE THE IMPLICATIONS OF THIS STUDY?

Thyroid cancer patients with low TSH levels from suppressive thyroxine therapy appear to have minor changes in heart function using a very sensitive test despite having normal results on an echocardiogram. None of these patients had clinical heart problems. The long-term significance of those changes on overall heart function remains to be seen. However, this study suggests that suppressive thyroxine therapy should be reserved to high risk thyroid cancer patients who would benefit the most from this therapy.

— Mona Sabra, MD

ATA THYROID BROCHURE LINKS

Thyroid Hormone Treatment: http://thyroid.org/patients/patient_brochures/hormonetreatment.html

Thyroid cancer: http://thyroid.org/patients/patient_brochures/cancer_of_thyroid.html

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.

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.

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.

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THYROID AND THE HEART, continued

Thyroxine (T_4): the major hormone secreted by the thyroid gland. Thyroxine is broken down to produce Triiodothyronine which causes most of the effects of the thyroid hormones.

Echocardiogram: also known as cardiac echo, is a heart ultrasound that uses sound waves to provide dynamic images of the heart as it beats.

Speckle Tacking Echocardiography (STE): a very sensitive type of echocardiogram where shortening, twisting and thickening of the ventricle wall during heart contraction and relaxation are easily seen and measured.

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THYROID CANCER

Use of radioactive iodine treatment for thyroid cancer has increased and varies among hospitals

BACKGROUND

The initial treatment for thyroid cancer is surgery. Radioactive iodine is often given after thyroidectomy to destroy any remaining thyroid tissue, either normal or cancerous. However, the indications for radioactive iodine treatment have not been strictly defined, leading to considerable variability in its use. The indications for radioactive iodine treatment were re-evaluated in the guidelines published by the American Thyroid Association (ATA) in 2009. In general, more consideration is being given for not using radioactive iodine in low risk patients. This study was done to evaluate changes in practice patterns and the degree of variability among hospitals in the United States in their use of radioactive iodine and the factors that contribute to these variations.

THE FULL ARTICLE TITLE

Haymart MR et al. Use of radioactive iodine for thyroid cancer. JAMA 2011;306:721-8.

SUMMARY OF THE STUDY

The study looked at 189,219 patients with thyroid cancer who had undergone a total thyroidectomy and their records were analyzed for various risk factors. Factors that correlated with use of radioactive iodine were evaluated in the 85,948 patients diagnosed with thyroid cancer between 2004 and 2008. Cancer programs belonged to one of the following four categories: community hospitals, comprehensive community, teaching/research and National Cancer Institute/National Comprehensive Cancer Network.

Between 1990 and 2008, the proportion of patients who received radioactive iodine after total thyroidectomy increased from 40.4% to 56%. This increase occurred for both smaller and larger cancers. For the 2004–2008 cases, younger age was associated with a 2-fold increase in the use of radioactive iodine. There was less use for low risk patients as compared to those at higher risk. There was less use of radioactive iodine in hospitals who treated <7 cases per year as compared with those treating >34 per year. For patients of similar risk, there was wide variation between hospitals in the use of radioactive iodine.

WHAT ARE THE IMPLICATIONS OF THIS STUDY?

The use of radioactive iodine increased between 1990 and 2008 and much of the variation in its use was associated with hospital characteristics rather than patient-related factors. The 2009 ATA guideline recommends using radioactive iodine only in select cases of low risk patients, so this trend is likely to decrease in the future. The reason for these recommendations is that recent studies have not found an improvement in survival or cancer recurrence with the use of radioactive iodine in most low risk patients. This is important with a growing concern about possible side effects such as damage to the salivary glands and secondary cancers.

— M. Regina Castro, MD

ATA THYROID BROCHURE LINKS

Thyroid cancer: http://thyroid.org/patients/patient_brochures/cancer_of_thyroid.html

Radioactive Iodine Therapy: http://thyroid.org/patients/patient_brochures/radioactive.html

ABBREVIATIONS & DEFINITIONS

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.

Total thyroidectomy: surgery to remove the entire thyroid gland.

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).

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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.

WHO WE ARE

AMERICAN THYROID ASSOCIATION

www.thyroid.org
ATA Patient Resources: http://www.thyroid.org/patients/
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 Patients*. We welcome your support.

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 FOUNDATION

www.checkyourneck.com
email: 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.

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.

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GRAVES' DISEASE & THYROID FOUNDATION

Light of Life Foundation checkyourneck.com

ThyCa

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ATA Alliance for Thyroid Patient Education
CALENDAR OF EVENTS

Educational forums, patient support groups and other patient-oriented meetings

ATA Conferences www.thyroid.org

Nothing is scheduled at this time. Please visit the website for updates.

Graves' Disease Conferences www.gdatf.org

Nothing is scheduled at this time. Please visit the website for updates.

Light of Life Foundation www.checkyourneck.com

Ongoing — www.checkyourneck.com
Thyroid Cancer Awareness campaign with Cindy Crawford and Brooke Shields

June 12, 2010 — a previous symposium available online at:
http://www.checkyourneck.com/About-Thyroid-Cancer/Thyroid-Cancer-Symposium-Presentations

Thyroid Cancer Symposium Presentations: What's New in Thyroid Cancer?
A Day for Patients and Their Families

Please visit the Light of Life Foundation website to view the Patient Educational Symposium which took place in NYC in 2010. As part of the Patient Educational Program these online presentations provide valuable information in hopes that patients everywhere can gain further information and support about their disease.

ThyCa Conferences www.thyca.org

Every Month
ThyCa Support Group Meetings around the United States and in Canada, Costa Rica, and Philippines.
Complete list of groups, meetings, and contacts at www.thyca.org/sg/local

April 21, 2012 — 8 AM to 4:15 PM. — Lake Regional Hospital, Osage Beach, Missouri
Free Workshop: 8th Annual Midwest Thyroid Cancer Survivors' Workshop with physician speakers
Details at www.thyca.org/conferences

September 2012 — **Thyroid Cancer Awareness Month**
Worldwide observance sponsored by ThyCa: Thyroid Cancer Survivors' Association, Inc., with many partnering organizations. Details at www.thyca.org

October 19–21, 2012 — Chicago, Illinois.
The 15th International Thyroid Cancer Survivors' Conference
Sponsored by ThyCa: Thyroid Cancer Survivors' Association, Inc. Details at www.thyca.org

October 20, 2012 — Chicago, Illinois
The 10th Annual Dinner/Auction Fundraiser for Thyroid Cancer Research, in conjunction with the 15th International Thyroid Cancer Survivors' Conference
Sponsored by ThyCa: Thyroid Cancer Survivors' Association, Inc. Details at www.thyca.org