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NEWSLETTER

*"Promoting the Highest Standards
 of Endocrine Nursing Practice,
 Education and Research"*

Pituitary

Volume 13, Number 1

February, 2003

HYPOPITUITARISM

by Karen Liebert

Hypopituitarism is defined as reduced secretion of one or more hormones secreted by the anterior lobe of the pituitary gland, resulting in a partial or complete loss of function. The hormones that may be affected include: adrenocorticotrophic hormone (ACTH), thyroid-stimulating hormone (TSH), follicle stimulating hormone (FSH), luteinizing hormone (LH), prolactin (PRL) and growth hormone (GH). These hormones are secreted by the pituitary gland and travel to "target glands" where they maintain function of that gland. For example, the target gland for TSH is the thyroid gland and the target gland for LH and FSH is the testes and ovaries. Loss of stimulation of a target gland results in decreased levels of the hormone that is released by that target gland. For example, loss of TSH results in low thyroid hormone levels resulting in hypothyroidism. Some patients may experience total loss of function referred to as panhypopituitarism. More commonly, patients will have a few hormones that are deficient referred to as partial hypopituitarism. Generally, the order in which pituitary hormones are lost is as follows: GH, then LH and FSH, then ACTH and then TSH. However, the pattern of loss may be different in each individual.

CAUSES OF HYPOPITUITARISM

The causes of hypopituitarism can be grouped into two groups: congenital, in which the defects are present at birth, or acquired, in which the defects develop later. This article will focus on acquired hypopituitarism.

By far, the most common cause of acquired hypopituitarism is from a tumor in the pituitary or hypothalamic region. A list of causes of hypopituitarism can be found below:

Causes of Hypopituitarism

Tumors (pituitary adenoma, craniopharyngioma, meningioma, glioma, Rathke's cleft cyst)

Infarction (Sheehan syndrome, pituitary apoplexy)

Radiation (previous radiotherapy to the pituitary, hypothalamus, head and neck region)

Autoimmune (lymphocytic hypophysitis)

Infiltrative disease (Histocytosis X, Sarcoidosis)

Infection (Tuberculosis, Mycoses, Syphilis, Meningitis, Viral encephalitis)

Tumors that arise from the pituitary region cause hypopituitarism by compression of pituitary or hypothalamic tissue,

Continued on page 2



From the Desk of Joanne Swenson

PRESIDENT'S MESSAGE

As I write this I feel as though I am in the middle of a winter that will never end. The endless cold temperatures and snow have grown old for me. Because of this, I look forward to milder temperatures in the months ahead. This also means planning for our annual symposium in June. The ENS board will be meeting soon to put the final touches on what looks to be another exceptional educational opportunity for endocrine nurses. Final details should be available soon. I hope that you are planning to attend this year's meeting in Philadelphia. As always it is a great opportunity to learn the most current information in endocrinology, as well as meeting with other endocrine nurses.

As always, I encourage you to become involved in the activities of the ENS. Any committee would benefit from your input and assistance. Perhaps you might be interested in writing an article for the newsletter or would like to help plan next year's symposium. Participation in any of the ENS activities is a great way to meet and collaborate with other endocrine nurses. Please do not hesitate to contact me or any of the other board members if we can assist you in these endeavors.

Lastly, I would like to take a moment to congratulate ENS research committee chairperson Cathy Kessenich. Cathy was recently appointed as a full professor at the University of Tampa in Florida. This is a great achievement which is well deserved!

Best wishes for a happy and prosperous new year.

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HYPOPITUITARISM

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impair blood flow to the normal pituitary tissue, or interfering with the delivery of hormones released by the hypothalamus. Hypopituitarism caused by a pituitary tumor may be reversible. Surgical removal or medical therapy to shrink the tumor may restore normal pituitary function. However, the chances of recovery of pituitary function are lessened when a majority of the pituitary gland must also be excised with the tumor or when radiotherapy is used to treat the tumor.

Other common causes of hypopituitarism are from radiation therapy or surgery to the pituitary area. As mentioned earlier, surgical resection of a tumor may result in resection of some normal pituitary tissue surrounding the tumor, resulting in damage or loss of the normal tissue. It is important for patients to find an experienced neurosurgeon who will remove the tumor but leave normal tissue. In the process of radiation, the normal pituitary gland may be damaged as it may be in the radiation field. Most patients who receive radiotherapy will go on to develop partial or panhypopituitarism as a result of damage to the normal pituitary tissue. Since radiation effects take many years to manifest, the pituitary gland may lose function slowly.

The clinical manifestations of hypopituitarism depend on which hormone is missing and the severity of the hormone deficiency. Since hormones released by the pituitary gland act on end organs, like the thyroid gland or adrenal gland, the hypofunctioning of the end organ will result in specific signs and symptoms.

GROWTH HORMONE DEFICIENCY

Growth hormone deficiency (GHD) in adults is associated with increased truncal adiposity, abnormal lipid levels, decreased bone density, impaired cardiac function and reduced quality of life. These symptoms do not occur in all patients, making the recognition of GHD in patients very difficult.

Confirmation of GHD should be made in patients who are suspected of having GHD based on medical history, clinical assessment of symptoms, and results of all other endocrine testing. Serum insulin-

like growth factor-I (IGF-I) level, a growth hormone dependent peptide that mediates the action of growth hormone, provides an effective way to clinically assess the overall growth hormone status and is often used as a "screening test" for GHD. A low IGF-I level can support a suspected diagnosis of GHD, but should not be considered a definitive confirmation of the diagnosis. Due to the pulsatility of growth hormone levels throughout the day, provocative stimulation testing using various medications (insulin, arginine, growth hormone releasing hormone) is needed to produce a rise in growth hormone levels over a two hour time period. An abnormally low rise in growth hormone levels in response to one or more of these stimulation agents confirms GHD.

As currently approved, growth hormone replacement therapy is given as a daily, subcutaneous injection. It is recommended that patients start with a low dose and gradually titrate to a maintenance dose based upon clinical response (absence of significant side effects) and biochemical parameters (normalization of IGF-I levels). By using low doses of growth hormone in the initial phases of therapy and titrating up slowly side effects can be minimized. The most common side effects include arthralgias, myalgias, peripheral edema, carpal tunnel syndrome, and headache. These symptoms usually resolve with a lowering of the dose.

THYROID DEFICIENCY

Patients with a TSH deficiency suffer from the loss of normal levels of thyroid hormone. They are thyroid deficient just as a person with primary hypothyroidism is due to thyroid gland destruction, for example, Hashimoto's thyroiditis. In thyroid gland failure, thyroid hormone levels in the blood are, of course, low and TSH levels are elevated. When the thyroid gland deficiency is due to a pituitary deficiency, thyroid hormone levels are low but TSH levels (hormone secreted by the pituitary gland) are also low. The initial diagnosis of pituitary hypothyroidism is made by showing thyroid hormone, T3 and T4, levels are low in the presence of a low TSH level.

Symptoms of low thyroid hormone include dry skin, constipation, cold intolerance, and muscle aching. Once a TSH deficiency is documented, patients will be required to take oral thyroid hormone, usually in the form of l-thyroxine. Patients should be started on a very low dose of medication and gradually titrated over many weeks. Because a TSH level will be low in these patients, a serum T4 level may be used to titrate the dose of thyroid replacement.

LH and FSH DEFICIENCY
(see separate article on page 3)

ADRENAL DEFICIENCY
(see separate article on page 4)

Vance, ML. Hypopituitarism. *New England Journal of Medicine*. 1994; 330: 1651-1662.

Pinsone JJ. Hypopituitarism. In: Becker KL, Bilesikiam JP, Bremner WJ, et al (eds). *Principles and Practice of Endocrinology and Metabolism* (3rd edition). Philadelphia: Lippincott Williams & Wilkins, 2001.

Thanks to members of the Pituitary Task Force for their excellent work on this issue of the newsletter.

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CALENDAR

of events

- Feb. 27 - Mar. 1, 2003** Preventive Cardiovascular Nurses Association
9th Annual Symposium and Exposition, San Francisco, CA
- Mar. 24 2003** 22nd Joint Meeting of the British Endocrine Societies
Glasgow, United Kingdom
- May 14-18, 2003** AACE's 12th Annual meeting and Clinical Congress
San Diego
- Jun. 20, 2003** 13th Annual Endocrine Nurses Society Symposium
Philadelphia, PA
- Jun. 19-22, 2003** The Endocrine Society 85th Annual Meeting
Philadelphia, PA

American Association
of
Clinical Endocrinologists



12th Annual Meeting & Clinical Congress
May 14-18, 2003

**PREVENTIVE
ENDOCRINOLOGY**
*San Diego Marriott Hotel & Marina
San Diego, California*

- Earn up to 34 hours of CME credit in category 1 of the AMA Physician's Recognition Award
- Satellite Symposia beginning at noon on Wednesday, May 14
- 16 workshops and 14 plenary sessions beginning on Thursday, May 15
- Exhibit Hall with over 60 companies
- Poster session adjacent to the Exhibit Hall
- ACE Convocation and Induction of Fellows followed by a reception on Saturday, May 17
- Dinner/Dance immediately following the ACE reception

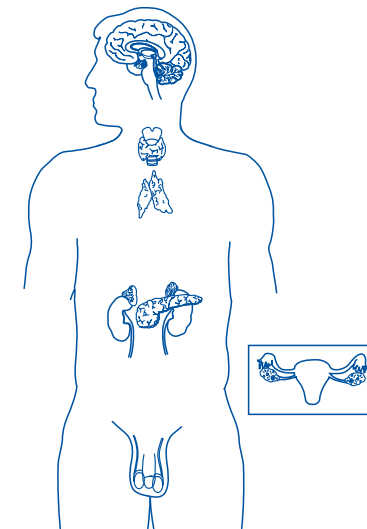
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HYPOGONADISM

by Denise Verity

Physiology

Low gonadal function (hypogonadism) is a hormonal deficit found in males and females resulting in testicular or ovarian under function. In primary hypogonadism, the defect occurs in the target organs, ovary and testis. The cause of secondary hypogonadism is due to damage to the hypothalamus or pituitary. Primary and secondary hypogonadism may be either congenital or acquired. Hormone replacement for hypogonadism is necessary to achieve normal sexual maturation, maintain bone density and muscle mass, and enhance libido.



Hypogonadism in Women

Secondary hypogonadism results from the subnormal secretion of follicular stimulating hormone (FSH) and luteinizing hormone (LH.) from the pituitary. Causes of primary hypogonadism in women include Turner's syndrome, congenital adrenal insufficiency, chemotherapy and radiation. The interference of gonadotropin releasing hormone (GnRH) from the hypothalamus and communication with the pituitary gland causes central hypogonadism. Some of these conditions include benign and malignant pituitary tumors, pituitary stalk syndrome, Kallman's syndrome and pituitary trauma. Secondary hypogonadism is the second most common hormonal deficiency after pituitary surgery. Manifestations of female hypogonadism

include dyspareunia, ammenorhea, delayed puberty, osteoporosis, anovulation and decrease in libido. Synthetic estrogen replacement in young women is necessary for induction of puberty and sexual maturation while progesterone is advisable for normal uterine functioning. In adults physiologic doses of estrogen and progesterone are given in combination to prevent dysfunctional bleeding, endometrial hyperplasia and uterine cancer. Estrogen preparations include oral tablets, transdermal patches, and intravaginal preparations. Determination of the dose and mode of administration depends on the cause of the disorder, age, stage of pubertal development, rationale for replacement and patient preference.

Hypogonadism in Men

Similar to female hypogonadism, testosterone deficiency results from the abnormal secretion of follicular stimulating hormone (FSH) and luteinizing hormone (LH.) from the pituitary. Causes of primary hypogonadism in men include Klinefelter's syndrome, (XXY chromosome), cryptorchidism, androgen receptor defect, congenital adrenal hyperplasia, chemotherapy and radiation. Secondary hypogonadism is due to hypothalamic or pituitary injury leading to low LH and FSH. Manifestations of male hypogonadism, both primary and secondary, include gynecomastia, small testis, micropenis, decrease in libido, delay in pubertal development, lack of energy, and decrease in bone density. Some of these problems depend on the age of onset of the loss of the sex steroids, testosterone and estrogen. Congenital forms of male hypogonadism may also cause infertility, erectile dysfunction, infertility, ambiguous genitalia and a decrease in sperm count. Testosterone replacement can be administered as an intramuscular injection, or a patch or gel preparation applied transdermally. Other forms include oral preparations and subcutaneous pellets, however their absorption and efficacy are questionable. The intramuscular route may cause discomfort at the injection site. When using the patch, the patient may experience

adhesion difficulty and localized irritation to the skin. The gel, a third viable option can be messy, leaving an unpleasant odor in the area of application. The benefits of testosterone replacement however outweigh the risk of potential side effects of hypogonadism regardless of the original source of the disorder.

Summary

Hormone replacement in male and female hypogonadism is essential to sustain quality of life, physical health and maintain emotional stability. The availability of estrogen and testosterone supplementation encourages its use in the hypogonadal deficient adult and pediatric population. The variety of available formulations improves compliance, maximize benefits of therapy and provide patients with alternatives to suit their individual needs. Reducing the risk of cardiovascular complications, strengthening bone density, and decreasing morbidity reinforce the importance of hormone replacement in hypogonadal patients. Before beginning replacement, health care providers should determine the cause of the disorder.

Bibliography:

1. Blevins, L.S. (2002). Management of Gonadotropin Deficiency. In A. Becker, L.S. Blevins, Jr., (Eds.) Management of Hypopituitarism (pp. 38 - 43) London, UK: Pharmacia Corporation.
2. Stocker, D. J. & Vigerski, R.A. (2002). Male Hypogonadism. In M. McDermott (Ed.) Endocrine Secrets (4th ed., pp. 348 - 355). Philadelphia, PA: Hanley & Belfus, Inc.

The 2003 edition of the Membership Directory has been published. If you haven't received your copy or have changes to your membership profile, please contact Rubi Defensor at rdefensor@nih.gov

ADRENAL INSUFFICIENCY

by Marie Cook

Adrenal Insufficiency is a lack of cortisol production by the adrenal gland. It is either primary or secondary in etiology. Primary adrenal insufficiency is failure of the adrenal gland to produce cortisol. Secondary adrenal insufficiency is failure of the pituitary gland to produce adrenocorticotropic hormone, ACTH.

The most common causes of primary adrenal insufficiency are: 1) autoimmune adrenal destruction and 2) bilateral adrenalectomy. Secondary adrenal insufficiency can result from suppression of the ACTH secreting cells of the pituitary by chronic administration of glucocorticoids (to treat asthma, for example) or damage to the pituitary by a pituitary tumor, surgery or pituitary irradiation.

Signs and symptoms of adrenal insufficiency may include hypotension, fatigue, weakness, nausea, anorexia, diarrhea, abdominal pain and weight loss. Skin pigmentation occurs in primary disease only, and is due to the high levels of ACTH which stimulate skin pigmentation.

The diagnosis of adrenal insufficiency is confirmed by performing an ACTH stimulation test. Baseline ACTH and cortisol levels are drawn followed by the IV administration of 1 mcg Cortrosyn. A second cortisol is drawn at 30 minutes. The stimulated value should rise to at least 18 ng/ml. The classic ACTH stimulation test uses a 250mcg dose of Cortrosyn. It is thought by some that the larger dose is misleading in diagnosing subtle secondary adrenal insufficiency. When extensive analysis is performed of the 1 mcg and 250 mcg tests, experts believe the 250mcg test is associated with false negative results, ie; the patient has secondary adrenal insufficiency, but is not identified by the high dose. Conversely, the low (1mcg) dose is associated with occasional false positive diagnosis, ie; they are identified as having secondary adrenal insufficiency, but really don't have the disease. Most endocrine clinicians use the 1mcg dose and use a cut off point of 15 mcg at the 30-minute time point. Secondary adrenal insufficiency is clearly identified using these criteria.

Therapy for adrenal insufficiency

consists of oral replacement of cortisone. The dose of hydrocortisone is usually 10 or 20 mg taken in the morning and for those patients taking Prednisone, the usual dose is 5 mg. For patients who have primary adrenal insufficiency, aldosterone must also be replaced with Fludrocortisone (Florinef). The usual dose is 0.1-0.2 mg/day.

Patients must be carefully counseled regarding the necessity of compliance with their medical regimen.

References:

1. Gabriel Dickstein, Carmela Shechner, Wendell E. Nicholson, Itzhak Rosner, Zila Shen-Orr, Fayad Adawi, Michal Lahav. Adrenocorticotropic Stimulation Test: Effects of Basal Cortisol Level, Time of Day, and suggested New Sensitive Low Dose Test. *J Clin Endocrinol & Metab.* Vol. 72. 773-778. 1991.

2. Tarig AM Abdu, Richard N. Clayton. The Low-Dose Synacthen Test for the Assessment of Secondary Adrenal Insufficiency. *Curr Opin Endocrinol.* Vol. 7. 116-121. 2000.

3. Karen Tordjman, Anat Jaffe, Nilly Grazas, Clara Apter, Naftali Stern. The Role of the Low Dose (1 µg) Adrenocorticotropic Test in the Evaluation of Patients with Pituitary Diseases. *J Clin Endocrinol & Metab.* Vol. 80. 1301-1305. 1995.

GUIDELINES FOR PATIENTS WITH ADRENAL INSUFFICIENCY

- Obtain and wear a medical ID bracelet identifying you as an Addison Disease patient.
- ALWAYS take your medication. It is not an option to miss a dose. Missing your medication may lead to adrenal crisis.
- For simple stress such as a cold, sore throat or flu, no dose change will usually be required. For physical stress such as pneumonia, fracture or surgery, the dose will need to be increased. (The dose is usually doubled, but you should check with your endocrinologist.)
- If you are ill and have vomiting and or diarrhea for 6 hours or more, you will need to go to an emergency room for intravenous cortisone.

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PHILADELPHIA

(continued)

Styles from Empire to Chippendale are highlighted, along with Paul Revere silver, Windsor chairs, needlework, porcelain, and clocks.

The grounds include a 200-acre garden; get a good look on the 45-minute guided tram tour. In the Touch-It room, children can dress in costumes, play games, and guess at the purpose of curious antiques.

Valley Forge

One of the region's most historic areas, Valley Forge is famous for Washington's encampment with his forces in the winter of 1777. See Washington's headquarters and his troops' log cabins. Nearby is the nation's largest retail mall, the Plaza & the Court at King of Prussia.

The following list includes some of the most visited areas of Philadelphia:

Independence National Historic Park

Philadelphia Zoo

Longwood Gardens

The Franklin Institute Science Museum

Farmer's Markets

South Street

Independence Hall

University of Pennsylvania

Please Touch Museum

James A. Michener Art Museum

Fonthill Museum

Cheesesteak Corner

The Philadelphia City Pass is a great way to enjoy Philadelphia. Pay one low price, get admission tickets to five famous attractions and avoid ticket lines! Includes admission to Philadelphia Trolley Tour, Franklin Institute Science Museum, Philadelphia Zoo, Academy of Natural Sciences, and the Independence Seaport Museum.

Food Specialties of Philly

"Where can I find my favorite Philadelphia foods like cheesesteaks, soft pretzels, water ices, Tastykakes and other famous local delicacies?" Find out some answers below!

Cheesesteaks

- Campo's Deli @ Market Street, 214 Market Street, (215) 923-1000
- Jim's Steaks, 401 South Street, (215) 928-1911

Hoagies

- Campo's Deli @ Market Street, 214 Market Street, (215) 923-1000
- Primo Hoagies, 21st and Chestnut Streets, (215) 463-8488
- Salumeria, 45 N. 12th Street, (215) 592-8150
- Tony Luke's Old Philly Style Sandwiches, 118 S. 18th Street, (215) 568-4630

Ice Cream & Water Ice

- Rita's Water Ice, 235 South Street, (215) 629-3910
- John's Water Ice, 702 Christian Street, (215) 925-6955

Tastykakes - a Philadelphia Tradition

- The Tastykake Baking Company, 29th & Allegheny Avenue, (800) 33-TASTY

Soft Pretzels

- Philadelphia Soft Pretzels Inc., 4315 N 3rd Street, (215) 324-4315
- Federal Pretzel Baking Company, 638 Federal Street, (215) 467-0505

And the Philadelphia Oddities

They can be viewed as simple oddities, quirky objects or places with little significance or meaning. Or they can be seen as delightful rarities - little gems that spark wonder, curiosity, bemusement. Perhaps, no other city has such an abundance of little-known, off-beat fascinating gems as Philadelphia.

After visiting the historic shrines, museums, colonial-era houses, monuments, art and architect, there is still an endless supply of Philadelphia oddities to amuse and delight.

Here is my list of Philadelphia's most interesting, quirky (at times macabre) curiosities:

James Hayes' Tumor	Harriet
The Grindstone Church	Peter the Mint Eagle
Old Baldy	Sparks Shot Tower
Cave of Kelpius	The Gibbet

Grumblethorpe Blood Stains	Biopond
The Whispering Bench	Giant Slide
"Painless Parker's" Bucket of Teeth	Dedication Stone
Mother and Twins Monument	Wistar Brain Collection

(Details for the curiosities can be viewed through <http://www.ushistory.org/oddities/index.htm>)

And finally, a special place that I have located for all you medical people. Someplace you cannot find in just any city...the award winning -

Mutter Museum

Phone: (215) 563-3737

Part freak show, part scientific, historical vault, the Mutter Museum is one of Philadelphia's truly original - and truly bizarre—treasures. Housed in the College of Physicians of Philadelphia, the Mutter was first established in 1849 to preserve scientific material for research. The collection has since grown to include such artifacts as the livers of Siamese twins Chang and Eng, bladder stones and Florence Nightingale's sewing kit!

The Museum's 20,000 displayed objects include anatomical and pathological specimens, models, illustrations, slides and photographs, and scientist and doctor memorabilia from 1750 to the present. Special exhibits about historical events in medical history and contemporary health issues are on display alongside the permanent collection.

For an unforgettable gift that just keeps on giving, nothing beats the Mutter Museum's calendar of 12 gorgeously photographed images of human distortions.

As you can see there is something for everybody ranging from the cultured to the curious. I hope to see you on the streets of Philadelphia - and of course at the meeting too!

March 24-26

Glasgow, UK
22nd joint meeting of the British Endocrine Societies
www.endocrinology.org

April 24-25

Paris, France
46th Journées Internationales d'Endocrinologie
Clinique: Endocrinology of menopause and 'andropause'
Email: klotz@ulb.ac.be

April 26-30

Lyon, France
6th European Congress of Endocrinology
www.endocrinology2003.com

May 1-4

Athens, Greece
2nd European Consensus Conference Menopause: State of the Art Toward Consensus
www.eska.fr

May 29-31

Riga, Latvia
10th European Federation of Endocrine Societies
Postgraduate Course in Clinical Endocrinology
Email: pirags@latnet.lv

Eating new foods, site seeing, and discovering a new city are some of the extra benefits of attending the Endocrine Nurses Society meeting this June. The historic city of Brotherly Love is rich with culture - so much so that it can sometimes be overwhelming. This article attempts to familiarize you with Philadelphia and consolidate a "wish list" for you during your stay.

Avenue of the Arts

Avenue of the Arts is Philadelphia's premier entertainment and arts district. Stretching 4.5 miles, from Lehigh Avenue (North) to Washington Avenue (South), it showcases internationally acclaimed theaters, museums and concert halls, and lets visitors explore a wide variety of shopping and fine dining experiences.

Benjamin Franklin Parkway

A mile-long cultural strip of museums, libraries and fountains, the tree-lined Parkway starts at City Hall and ends at the Philadelphia Museum of Art, one of the nation's most comprehensive art museums.



Chestnut Hill/Germantown

Cobblestone streets and 19th-century stone edifices grace Chestnut Hill, a community of shops, cafes, and parks just 15 minutes from Center City. In neighboring Germantown, the site of the first documented protest against slavery and scene of the Battle of Germantown, historic attractions, churches and parks mix with an eclectic array of locally owned businesses.

Festival Pier and Camden Waterfront

The center of Philly's maritime and commercial activity, Festival Pier is the scene of special events - fairs and live music in summer, outdoor ice skating in winter - throughout the year. Cross over to the Camden Waterfront for concerts, minor league baseball and a splendid aquarium.

Independence Mall

The heart of America's most historic square mile includes the Liberty Bell and Independence Hall, the site where the Declaration of Independence was drafted and signed and the U.S. Constitution was written.

Northern Liberties

Adjacent to Old City, another of Philly's vibrant areas has great bars, live music, casual dining and late Victorian-period architectural buildings with original marble fireplaces and stained glass windows.

Old City

America's first commercial district combines historic charm and urban chic with galleries, theaters, shops and restaurants. The First Friday evening of every month is a gallery "happening."

Rittenhouse Square

One of founder William Penn's four original public squares, this is an area of beautiful boutiques, salons and restaurants, including alfresco brasseries that look out onto the greenery of the park.

South Philadelphia

South Philly is home to the city's oldest Italian restaurants, the outdoors Italian Market and Philly cheesesteaks.

COUNTRYSIDE PLACES

Brandywine Valley

The lush hills of Chester County are home to the Brandywine River Museum, famous for three generations of Wyeth paintings; the Brandywine Battlefield, where the largest engagement of the Revolutionary War took place, and 1050-acre Longwood Gardens, one of the world's premier horticultural displays year round.

Winterthur Museum, Garden and Library Phone: (800) 448-3883 is the gem of the Brandywine Valley, this outstanding museum is a nine-story mansion with 175 rooms furnished with more than 89,000 articles made or used in America in the 17th, 18th, and 19th centuries. Built in 1839, it was the home of Henry Francis du Pont, a passionate and scholarly collector of antiques whose aim was to amass examples of American interior and decorative arts.

Continued on page 9

Acromegaly is the constellation of clinical signs and symptoms resulting from the hypersecretion of growth hormone. The relationship of elevated growth hormone levels to increased morbidity and mortality in acromegaly is becoming increasingly clear. This appreciation has led to new, tougher standards for biochemical cure, as well as to new treatment guidelines for clinicians. Here, we review the revised therapeutic goals in acromegaly, the new consensus treatment algorithm, and discuss guidelines for monitoring the effectiveness of treatment.

New Treatment Goals in Acromegaly

There are multiple goals of therapy in acromegaly. Since acromegaly most often results from a pituitary tumor, the first goal is to remove or reduce the tumor mass, or to restrain its growth. Controlling tumor size reduces or prevents signs and symptoms due to the tumor mass, and may also lead to a reduction in growth hormone, the second important goal of therapy. Recent consensus criteria for cure acknowledge the importance of GH control, and state that GH levels are the single greatest determinant of mortality in acromegaly. The criteria set a new, more stringent guideline for GH levels, and suggest that the post-glucose GH nadir should be below 1 ug/l.

The new consensus criteria for cure also include goals for IGF-I control. The addition of IGF-I criteria reflects evidence that GH and IGF-I levels are not always concordant. Though there is little epidemiological data yet on the consequence of isolated elevations in IGF-I levels, the fact that clinically active acromegaly can occur with elevated IGF-I levels alone suggests that IGF-I should be normalized. The guidelines thus advise that IGF-I levels should be returned to within the age and sex adjusted normal range. However, care must be taken to avoid a level of IGF-I normalization that could result in growth hormone deficiency, since up to 50% of adults with GHD can have normal IGF-I levels.

The last goal of therapy is to relieve symptoms and control comorbidities. Many signs and symptoms of acromegaly correlate with GH levels, and will be alleviated as GH levels fall. However, associated co-morbidities are not always completely reversed, and these should be aggressively co-treated.

Consensus Treatment Guidelines

A consensus treatment algorithm for acromegaly was recently published in JCEM (Figure 1). This document reflects the consensus on treatment that was reached by the Acromegaly Treatment Consensus Workshop, a gathering of 68 leading neuro-endocrinologists and neurosurgeons worldwide, and is designed as a guideline to help clinicians choose amongst the available treatment options. Transsphenoidal surgery remains the treatment

small macroadenomas without cavernous sinus invasion, or in the presence of visual field loss. Primary medical therapy with a somatostatin analogue (SRL) can be considered in patients for whom surgery is contra-indicated, or in selected patients who are less likely to be cured with surgery. This group includes those who have macroadenomas with cavernous sinus invasion but without impingement on the optic chiasm. Therapy with an SRL can be considered pre-op to reduce tumor bulk, though the potential benefits are not yet known.

Adjunctive medical therapy should be instituted if acromegaly is uncontrolled post-surgery. The adjunctive therapy of choice is a somatostatin analogue, because of their proven efficacy at normalizing GH and IGF-I in 65-70% of patients, and their long term safety record. Their tendency to restrain tumor growth is also a clear benefit. One recent review showed that less than 1% of GH secreting pituitary tumors grew during somatostatin analogue therapy. A dopamine agonist may be added if biochemical control is not achieved, particularly in tumors that secrete both prolactin and GH. If acromegaly is uncontrolled after addition of an SRL +/- a dopamine agonist, XRT can be considered, or no further action taken, depending on the degree of clinical disease activity and the severity of the persistent biochemical abnormalities. If XRT is chosen, medical therapy should be used to obtain biochemical control while waiting for the XRT to take effect.

If all other options have failed, a growth hormone receptor antagonist (GHRA) can be considered. Growth hormone receptor antagonists (GHRAs) are a new class of agents that will soon be commercially available. These agents block the interaction of GH with its receptor, and thus act to suppress the GH stimulated increase in peripheral production of IGF-1. Initial studies show that GHRAs suppress

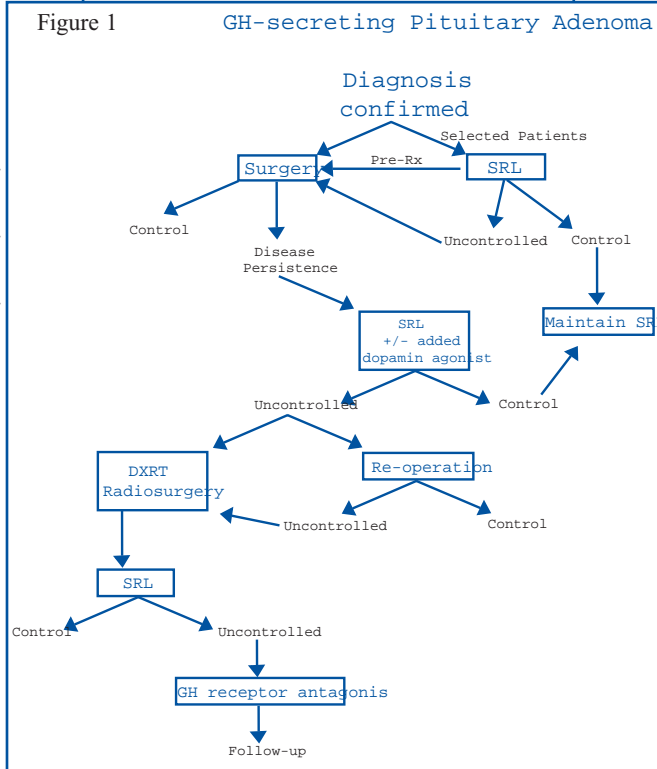


Figure 1: Treatment Algorithm for GH-secreting Pituitary Adenomas
Adapt from Melmed, S., F. F. Casanueva, et al. (2002). "Consensus Guidelines for acromegaly management." J Clin Endocrinol Metab 87(9): 4054-8. Figure 1, page 4055.

of choice for the initial management of acromegaly, since it results in prompt reduction in tumor mass, and offers the possibility of permanent cure. Surgery is particularly indicated for microadenomas,

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IGF-1 in up to 90-95% of patients. However, GHRAs are not effective in reducing GH levels; in fact, they actually go up. Of additional concern is that significant tumor growth has occurred during therapy with GHRAs. Elevated transaminases have also been noted, so long term safety may be an issue.

Guidelines for Monitoring the Effectiveness of Treatment

After treatment, a physical exam, a nadir post-glucose GH level and a random IGF-1 should be done every three months until biochemical control is obtained. Biochemical control has recently been redefined by new consensus criteria for the cure of acromegaly. These consensus criteria were developed by an international workshop group and published in JCEM. According to these criteria, acromegaly is considered to be controlled when nadir post-glucose GH is below 1.0 ug/l and IGF-1 is normalized in a patient with no clinical activity. In this instance, no change in therapy is needed. If there is no residual tumor, well-controlled patients can then be seen every 6 months. Nadir post-glucose GH, IGF-1 and anterior pituitary function should be monitored, as should

symptom relief and quality of life. Residual tumor should be followed with periodic MRI.

Acromegaly is categorized as inadequately controlled when the post-glucose GH nadir is normalized, but IGF-1 is persistently elevated. In this setting, the physician should consider the degree of IGF-1 elevation, and assess the patient for the presence of complications. The clinician must weigh the possible benefit of instituting additional therapy against the possible risks. Acromegaly is considered poorly controlled when both the GH nadir and IGF-1 are elevated, along with clinically active disease. In this instance, the patient is at clear risk of enhanced mortality due to long-term exposure to excess circulating growth hormone, and intensification or change in treatment is recommended.

Conclusion

Acromegaly is a serious illness that is associated with excess morbidity and mortality. Treatment should be aimed at reducing excess mortality by suppressing GH hypersecretion. Additional treatment goals include normalizing IGF-1, controlling tumor growth, alleviating clinical signs and symptoms, and reducing co-morbidities.

Surgery, medical therapy and radiation can be used together or in tandem to meet these therapeutic goals. These treatments hold out new hope for the treatment of this unusual disorder.

References

- Ben-Shlomo, A. and S. Melmed (2001). "Acromegaly." *Endocrinol Metab Clin North Am* 30(3): 565-83.
- Freda, P. U. (2002). "Somatostatin analogs in acromegaly." *J Clin Endocrinol Metab* 87(7): 3013-8.
- Giustina, A., A. Barkan, et al. (2000). "Criteria for cure of acromegaly: a consensus statement." *J Clin Endocrinol Metab* 85(2): 526-9.
- Kopchick, J. J., C. Parkinson, et al. (2002). "Growth hormone receptor antagonists: discovery, development, and use in patients with acromegaly." *Endocr Rev* 23(5): 623-46.
- Melmed, S., F. F. Casanueva, et al. (2002). "Guidelines for acromegaly management." *J Clin Endocrinol Metab* 87(9): 4054-8.
- Melmed, S., I. Jackson, et al. (1998). "Current treatment guidelines for acromegaly." *J Clin Endocrinol Metab* 83(8): 2646-52.

TIPS FOR ADMINISTERING SANDOSTATIN LAR DEPOT

by Teresa Kidder

Sandostatin LAR is an injectable medication available to acromegalic patients to lower growth hormone levels. It is supplied in an administration kit containing a vial of Sandostatin LAR (10, 20 or 30mg), diluent, (2cc), a syringe and 2 needles (one for mixing and drawing up the medication, another for injecting the medication). A step by step instruction sheet is also included.

Mixing and administration of this product is often a source of frustration for the nurse or doctor, but if you follow the instructions provided in the injection kit and the following tips, you should be able to administer the medication successfully!

- It is very important to allow the vials to reach room temperature, approximately 30 to 60 minutes. In my experience and that of my colleagues, 60 minutes is best.
- Make sure that the Sandostatin LAR

powder has settled to the bottom of the vial prior to adding the diluent.

- Slowly inject the diluent down the side of the Sandostatin LAR vial.
- Let the vial of Sandostatin LAR sit at least 5 minutes to allow adequate saturation of the powder. Towards the end of this period, prepare the injection site, either the right or left gluteus.
- After 5 minutes, check for dry spots. Do not invert the vial.
- If there are no dry spots, swirl the vial to mix. Do not shake the vial. The suspension should look milky when mixed. Proceed immediately to the next step.
- Do not invert the vial. With the vial tipped at approximately a 45-degree angle, draw the suspension into the syringe. The vial contains overfill and some residual will remain in the vial.

- Draw approximately 1 cc of air into the syringe and continue to gently rock the syringe to maintain the mixed suspension.

- Immediately before changing the needle, clear the syringe of air and attach the second needle. Always use a 19 gauge, 1 1/2" needle to inject the medication.

- Proceed with the intramuscular injection.
- If a blood vessel has been penetrated, change the needle and the injection site.

- If the needle clogs, gently rock the syringe to maintain the suspension, attach another needle and proceed with the injection.

Novartis has provided the following resources for help with mixing and administration issues: www.us.Sandostatin.com or call 1-877-LAR-HELP

ENDOCRINE NURSES SOCIETY 13TH ANNUAL SYMPOSIUM Marriott Hotel Philadelphia, PA June 20-21, 2003

Saturday, June 21, 2003

7:00 a.m. - 7:55 a.m.	Breakfast Session	
7:10 a.m. - 7:15 a.m.	Welcome and Introduction	Joanne Swenson, ENS Pres.
7:15 a.m. - 7:55 a.m.	Giving an Effective Presentation	Matt Kim, MD
8:00 a.m. - 8:50 a.m.	Parathyroid Hormone in the Treatment of Osteoporosis	Clifford Rosen, MD
8:50 a.m. - 9:40 a.m.	Vitamin D	Michael Holick, MD
9:40 a.m. - 10:00 a.m.	BREAK	
10:00 a.m. - 10:50 p.m.	New Developments in Thyroid Cancer Treatment	R. Michael Tuttle, MD Rebecca Qualey, RN
10:50 p.m. - 11:30 p.m.	Cushings Disease	Beverly Biller, MD Karen Szczesiul, RN
12:15 p.m. - 2:30 p.m.	LUNCH and Business Meeting	
	GRAND ROUNDS 2:30 p.m. - 4:30 p.m.	
2:30 p.m. - 3:30 p.m.	Treatment of Type 2 Diabetes & Metabolic Syndrome	Beth LaLande, MD Linda Pachuki-Hyde, RN, CDE
3:30 p.m. - 4:30 p.m.	Polycystic Ovarian Syndrome	John Nestler, MD Linda Thurby-Hay, RN, MSN, CS, CDE
4:30 - 5:00 p.m.	Evaluation/Raffle/Wrap Up	

BOARD POSITIONS TO BE FILLED FOR 2003

The following board positions will be announced at the June 2003 Annual Meeting.

President Elect

Treasurer

Member at Large

Names may be submitted to the nominations committee prior to April, 2003.

Contact Marie Cook via email at: MarieCook@Earthlink.net