



J. Carl Holowaty
cholowaty@rgare.com

LETTER FROM THE EDITOR

Dear readers:

Welcome back to another year of *ReFlections* articles! In this first edition for 2014 we have included an article by Dr. Sharylee Barnes, which explains the value of a variety of laboratory tests that can be used following treatment for thyroid cancer. The second article, written by Dr. Allan Brodersen, explains the significance of the changes in the latest version of the Diagnostic and Statistical Manual of Mental Disorders. The third article, which I have written, discusses the risks involved with the use of testosterone. Finally, as part of an ongoing series, Sue Wehrman continues to update us on the HITECH Act and its importance to the insurance industry.

As always, I hope you enjoy reading these articles.

J. Carl Holowaty M.D., D.B.I.M.

THYROID TESTS AND CANCER FOLLOW-UP

By Sharylee Barnes M.D., D.B.I.M.

Vice President and Medical Director
RGA Reinsurance Company

Thyroid tests and papillary-follicular thyroid cancer follow-up successfully take advantage of the knowledge of how the normal thyroid gland functions. The purpose of the gland and its various cells is to produce exactly the right amount of two thyroid hormones to maintain a balanced metabolism.

The balance of hormone levels circulating in the blood involves a feedback loop to two areas of the brain. The hypothalamus makes thyroid releasing hormone (TRH), which travels to TRH receptors in the pituitary gland. The hormone then stimulates the pituitary to release another hormone, the thyroid stimulating hormone (TSH), into the circulation. The thyroid gland itself has follicle cell receptors that allow the TSH to stimulate thyroid follicular

cells to produce both thyroxine (T4) and triiodothyronine (T3). Thyroglobulin (Tg), produced only by the thyroid, is a precursor of both hormones T4 and T3.

The two thyroid hormones T4 and T3 then circulate in the blood and inhibit synthesis of the brain hormones, TRH and TSH, as well as the TRH receptor in the pituitary, as part of a negative feedback loop. As the level of one set of hormones rises, the other set decreases.

Thyroid Cancer Typical Treatment

After pathologic diagnosis of cancer, the first step in treatment is usually total thyroidectomy. Surgery results in removal of the tumor, thereby reducing the risk of recurrence and making it easier to treat a smaller number of any remnant cancer cells.

The second step takes advantage of thyroid gland function, and targets only thyroid cells that use iodine to manufacture thyroid hormones. Even after total thyroidectomy, thyroid cells could remain in the thyroid bed, in cervical lymph nodes, or be located in metastases.



Radioactive iodine (I-131) is administered, and taken up only by functioning thyroid cells, whether normal or neoplastic. The functioning cells are killed by the radiation in the iodine, but there is not enough radiation to kill other tissues of the body. Radioactive iodine (I-131) ablation of thyroid cancer cells was the first great success in targeted cancer treatment.

Once there are presumably no more thyroid cells, future scans make detecting a recurrence of cancer possible at an early stage.

The third step of treatment is replacement of the now-missing essential hormones. Thereafter, good follow-up means testing to detect any recurrences.

Thyroxine (T4) is easily monitored and replaced by Synthroid or other formulations which act identically to natural thyroxine. Thyroxine, natural or exogenous, does two things: first, it restores the patient to the euthyroid state; and second, Thyroxine suppresses the pituitary hormone, TSH, by being part of the normal feedback loop from thyroid to hypothalamus to pituitary. Low levels of TSH are advantageous because any remnant cancer cells will not be stimulated to grow. After years of controversy, most writers agree that replacement of triiodothyronine (T3) is not needed. Frequently, the brand name for exogenous T3 seen in attending physicians' reports is Cytomel.

Good follow-up means looking for possible thyroid cancer recurrences, and one key to this is ultrasound scans of the neck. Ultrasound is useful for all pathologic types of recurrent cancer in the neck. Knowledge of cell function, however, can aid in detection of recurrences of papillary/follicular types of cancer in all areas of the body, including the neck. The fact that these thyroid cells take up iodine and/or produce hormones creates another way to look for recurrences.

Thyroglobulin (Tg)

The only cells in the body that produce thyroglobulin are thyroid cells. This is true whether the cells are normal or cancerous. Almost all differentiated papillary, papillary-follicular, or follicular cancer cells make thyroglobulin, and they are by far the commonest type of thyroid cancers. Measuring Tg is an excellent way to test for success of ablation or recurrence of thyroid cancers in the vast majority of patients. Thyroglobulin is a very convenient tumor marker.

Pre-surgical measurement of Tg will show if the patient's cancer cells are producing thyroglobulin and enhances the usefulness of Tg as a tumor marker in that particular patient.

Circulating pretreatment levels of 100 ng/ml of thyroglobulin are usually found in persons with thyroid cancer. Unfortunately, thyroglobulin (Tg) is not produced by medullary, undifferentiated papillary/follicular, or anaplastic thyroid carcinomas and one must fall back on the mainstay of neck ultrasound for follow up.



Thyroglobulin and TSH

If no TSH is being produced (due to replacement and/or suppressive therapy), any cancer cells that exist will not be stimulated to make thyroglobulin. This presents a dilemma, as the patient needs and gets thyroid hormone (usually Synthroid), but then there may be no thyroglobulin available to act as a tumor marker.

TSH stimulation is needed for thyroid cells to begin functioning again. Then serum thyroglobulin levels can be used to detect cancer cell recurrence in the most prevalent types of thyroid carcinoma. Serial thyroglobulin levels are more useful, as a change in the Tg level over time compensates for problems intrinsic to the test itself. It is also best to use the same laboratory when comparing results because of the technical difficulty faced by the laboratories.

Another result in a patient who is not producing TSH is that any existing thyroid cells will not be taking up

iodine to make thyroglobulin. If cells are not taking up the iodine: first, doctors cannot use a radioactive iodine tracer (I-123) for a total body scan; and second, nor will the cells take up the iodine of I-131 to achieve radiation therapy.

Obviously, to follow patients by scanning or to treat recurrences with I-131, they must regain the TSH that was suppressed by needed therapy.

The I-123 or the radioactive iodine uptake (RAIU) test was the only method of detecting recurrent carcinoma outside the neck before tests for thyroglobulin became available. Though it is an older test it is still very effective. Nuclear scanning takes advantage of iodine uptake by functioning thyroid cells and can detect even a small cluster of cells. It is useful when thyroglobulin levels are obscured by anti-thyroglobulin antibodies or when there was a sub-total thyroidectomy performed that allows continued production of thyroglobulin. I-123 has a shorter half-life than I-131 (a half day vs. 8.1 days), so use of I-123 exposes the body to less radiation for less time and the type of radiation differs.

There are two ways to increase TSH in a suppressed patient – an old and a new method.

The old choice was to withdraw thyroxine therapy to make patients produce their own TSH in response to a fairly sudden hypothyroid state. A sudden hypothyroid state is not akin to natural hypothyroidism. Acute T4 withdrawal makes patients feel much sicker than gradual hypothyroid disease and they could be ill for weeks from sudden lack of T4. If very severe it may be termed 'Myxedema Madness'.

The newer choice is simply to give the patient exogenous Recombinant Human TSH (rhTSH), which has been available since the late 1990s. Thyrotropin alpha (rhTSH) has an amino acid sequence that is identical to human pituitary thyroid stimulating hormone. Commercially, Thyrogen®, a highly purified rhTSH, is synthesized by recombinant DNA technology. When given rhTSH, the patients are not ill; they are not hypothyroid; they keep taking their replacement hormone; but they have lots of circulating TSH from rhTSH. If a patient has any differentiated thyroid cells, the cells will produce thyroglobulin and take up iodine to do so. Using rhTSH is much faster, with far less morbidity than the old method of waiting for the patient's body to respond to iatrogenic hypothyroidism.

Recombinant TSH nicely circumvents the thyroid hormone- hypothalamic- pituitary negative feedback loop.

Thyrogen (rhTSH) is administered intramuscularly, usually in two doses 24 hours apart. Thyroglobulin (Tg) may be produced if recurrent cancer cells exist; then Tg can be measured 72 hours after the last dose. If needed, imaging or further ablation may be done in two stages during the same time period.

Anti-thyroglobulin antibodies (ATA) are not uncommon and unfortunately interfere with accurate measurement of thyroglobulin, making this valuable tool more difficult to assess. Anti-thyroglobulin antibodies occur with Hashimoto's thyroiditis or Graves' disease and Tg-specific antibodies aid in the diagnosis of these diseases. Anti-thyroglobulin antibodies may also be present in apparently healthy euthyroid individuals (~12.5%). Additionally, ATAs will be present in approximately 25% of thyroid cancer patients. As treatment succeeds in thyroid cancer, thyroiditis, or autoimmune thyroid disease, the autoantibodies diminish with time. For the most accurate use of the thyroglobulin level, it is advisable that both Tg and ANA should be tested each time and the pattern analyzed by an endocrinologist or head and neck surgeon.

Medullary thyroid cancer, undifferentiated papillary/ follicular and anaplastic thyroid cancers do not produce thyroglobulin or thyroid hormones, so they do not take up iodine. These characteristics exclude most of the follow-up tools and treatments discussed above. Fortunately, ultrasound of the neck is sensitive for finding residual local disease in medullary and anaplastic cancers. As well, serum Calcitonin is a good marker for Medullary thyroid cancer and useful for detection of metastases. After total thyroidectomy calcitonin should be below the normal range. ■



Sharylee Barnes M.D., D.B.I.M.
sbarnes@rgare.com

Dr. Sharylee Barnes is Vice President and Medical Director with RGA Reinsurance Company, where she is responsible for medical underwriting, education, researching new medical practices and literature, and assisting in developing RGA's underwriting guidelines. She is a member of the American Academy of Insurance Medicine and the Canadian Life Insurance Medical Officers Association.

DSM-5 – WHAT'S THE FUSS?

Changes in the New Edition of the Diagnostic and Statistical Manual of Mental Disorders

By Allan Brodersen M.D.

Vice President and Medical Director

RGA Reinsurance Company

The publication in May 2013 of the *Diagnostic and Statistical Manual of Mental Disorders* Fifth edition (DSM-5)¹ engendered significant controversy over some of the changes from the fourth edition (DSM-IV)². Indeed, one editorialist recommended that DSM-5 be used either cautiously or not at all³. But a side-by-side review of both DSM-IV and DSM-5 does not show substantial changes or justify the fuss that has been raised. In this article, we will review many of the major changes that will be of interest in underwriting, comparing DSM-IV directly with DSM-5 and discuss what impact, if any, we can expect from these changes.

History

Efforts began as early as the 1840s to provide a standard classification for mental disorders⁴ in institutionalized individuals. By 1880, there were seven classifications: mania (agitation), melancholia (depression), monomania (psychosis), paresis, dementia, dipsomania (alcoholism) and epilepsy. The first DSM was published in 1952. Subsequent editions were revised and expanded and a multiaxial assessment system was added in 1980. DSM-IV was published in 1994. DSM-5 comprises nearly 1000 pages.

Purpose of DSM-5

The preface to DSM-5 states that the purpose of the publication, in addition to updating and streamlining diagnostic categories, is to harmonize these classifications with the upcoming *International Classification of Diseases* Eleventh edition (ICD-11) due to be released by the World Health Organization (WHO) in 2015. As a result, many of the changes are more organizational than substantive as will be discussed below. The multiaxial system of assessment has been eliminated and the Global Assessment of Function (GAF) has been replaced by the WHO's Disability Assessment Schedule (WHODAS).

Major Changes in Diagnostic Categories

One of the most controversial changes is the removal of the so-called "bereavement exclusion". In the DSM-IV criteria for Major Depressive Episode there is a caveat to consider if the symptoms were not better accounted for by bereavement within the previous two months, though this caveat did not specifically exclude a diagnosis of Major Depressive Episode. This caveat was eliminated in DSM-5 noting that, although symptoms may be attributed to a loss, the presence of a coexisting depressive disorder should also be considered. Substantial criteria are given in a footnote to help distinguish normal grief from a major depressive disorder. Critics say this will result in normal grief being "medicalized" and inappropriately treated with psychotropic drugs⁵, but when one places DSM-IV side-by-side with DSM-5 there are more robust criteria for distinguishing between grief and depression in DSM-5. If the criteria are applied appropriately there should be no increase in bereaved people being diagnosed with major depressive episodes. If the criteria are not applied properly, it is not the fault of the criteria. Insofar as underwriting is concerned it is incumbent on us to understand these criteria so we classify the risk appropriately.

In DSM-IV, substance-related disorders including alcohol-related disorders were divided into abuse and dependence, with specific criteria for each. In DSM-5 these are combined as Substance Use Disorder. However, almost all of the DSM-IV criteria for both substance abuse and substance dependence are included in the DSM-5 criteria for Substance Use Disorder with minimal changes. It is unlikely, therefore, that any significant increase will occur in the diagnosis of these disorders and the impact on underwriting should be minimal.

Another change of interest to underwriting is in the Feeding and Eating disorders chapter. In DSM-5 under Bulimia there is no longer a distinction between purging

and non-purging types as there was in DSM-IV. Underwriters should be aware of this change as the risk classification in these individuals may depend on this distinction. Also, a new diagnostic category is presented in this section: Binge-Eating Disorder, distinct from bulimia. This diagnostic category was a diagnosis for further study in DSM-IV and criteria include recurrent episodes at least weekly over three months of significant overeating with associated problems and distress, but without the inappropriate compensatory behaviors seen in bulimia.

Autistic disorder, Asperger's disorder and the other pervasive developmental disorders are combined in DSM-5 under Autism Spectrum Disorder. Qualifiers for specific genetic disorders such as Rett syndrome are classified as Autism Spectrum Disorder with Rett syndrome. Since these criteria are reorganized rather than changed, once again there would not be any expected impact on diagnosis or underwriting.

The dementias are now classified under Major Cognitive Disorder. Again, specifiers of subtypes are used such as "Major Cognitive Disorder due to Alzheimer's disease". The criteria for these remain mostly the same as in DSM-IV; however, there is a new category of Minor Cognitive Disorder. Minor Cognitive Disorder is distinguished from Major by having "modest" as opposed to "significant" cognitive decline and does not involve impairments in the instrumental activities of daily living (IADLs). This addition is not groundbreaking; clinicians for some time have been using the term "mild cognitive impairment" to describe individuals with mild changes but not full-blown dementia. Despite this addition, the challenge for both clinicians and underwriters remains how to distinguish between normal aging and Minor Cognitive Disorder as we recognize that there is a continuum from minor to major cognitive disorders and mortality is significantly increased with the latter. This change is not likely to resolve this uncertainty, so therefore it is unlikely it will have much impact on the underwriting of this impairment.

Other Changes

Bipolar disorder is given its own chapter in DSM-5, but there are no significant changes in the criteria. The criteria for personality disorders are unchanged, but in section III of the manual an alternative model for understanding these problems is offered that focuses on personality function and differential diagnosis. Post-traumatic stress disorder (PTSD) is now separated from the anxiety disorders and included with the Adjustment Disorders in a new chapter called Trauma and Stress-Related Disorders. The criteria are reorganized but not substantially changed although additional diagnostic information for children aged six and under are added.

Other New Diagnostic Categories

Hoarding Disorder and Premenstrual Dysphoric Disorder are new categories that are certainly familiar through the popular media; an ongoing television series focuses on individuals with the former. Illness Anxiety Disorder and Somatic Symptom Disorder are new categories, but actually cover what were previously categorized as hypochondriasis, somatization disorder and undifferentiated somatoform disorder in DSM-IV. Disruptive Mood Dysregulation Disorder describes individuals aged 6-18 years with severe temper outbursts grossly out of proportion to the situation occurring at least three times a week for 12 months, inconsistent to the developmental level of the individual and in multiple settings. Meeting all of these criteria should distinguish this disorder from an occasional temper tantrum.

Conclusion

While there are some changes in DSM-5, on close review and comparison with DSM-IV, the changes are not likely to have any substantial impact on underwriting or on practice and diagnosis. In summary, it is difficult to see what all the fuss is about. Diagnostic uncertainty is a part of all medicine, not just psychiatry as one observer noted⁶. Likewise in underwriting, certainty is unachievable but understanding and familiarity with the concepts of and criteria for these diagnoses enable us to make the best decisions possible regarding the risk classification of individuals with these problems. ■

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Allan Brodersen M.D.
abrodersen@rgare.com

Dr. Allan Brodersen is a Vice President and Medical Director of RGA Reinsurance Company. In this role, he is responsible for reviewing life underwriting facultative cases and for educating RGA underwriters and clients on a variety of medical topics. In addition, he researches selected topics for the development of underwriting guidelines.

TESTOSTERONE DEFICIENCY

By J. Carl Holowaty M.D., D.B.I.M.

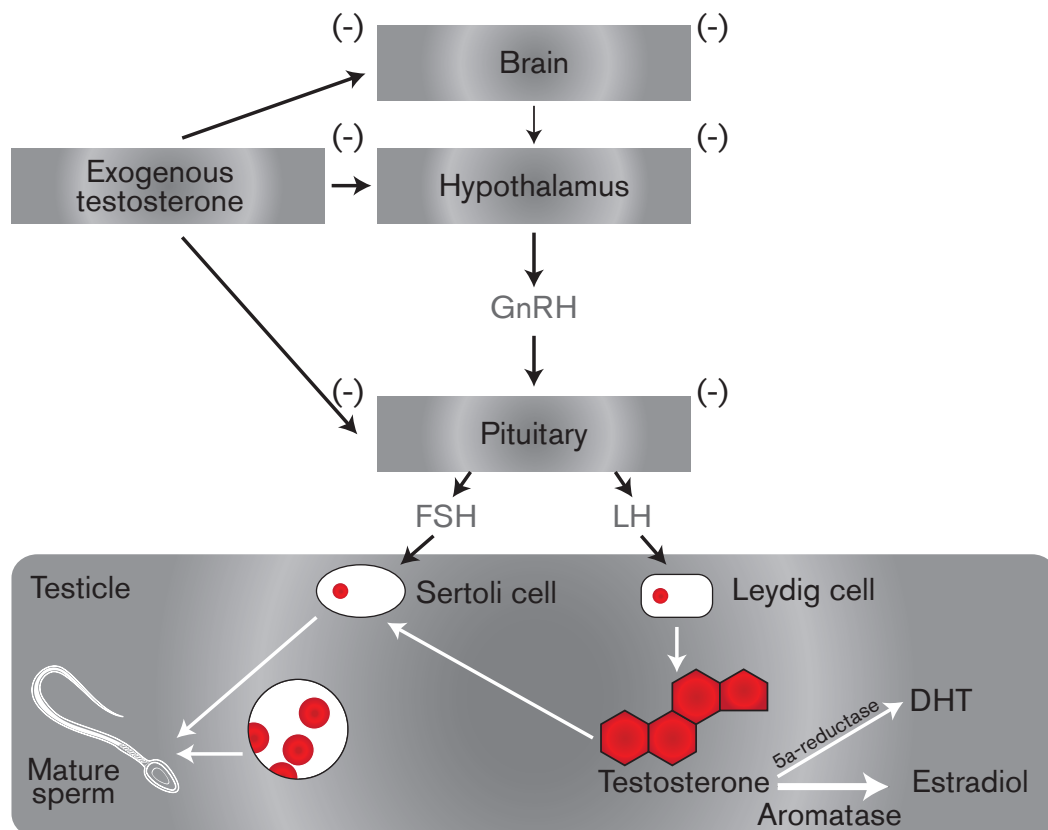
Senior Vice President and Chief Medical Officer

RGA Reinsurance Company

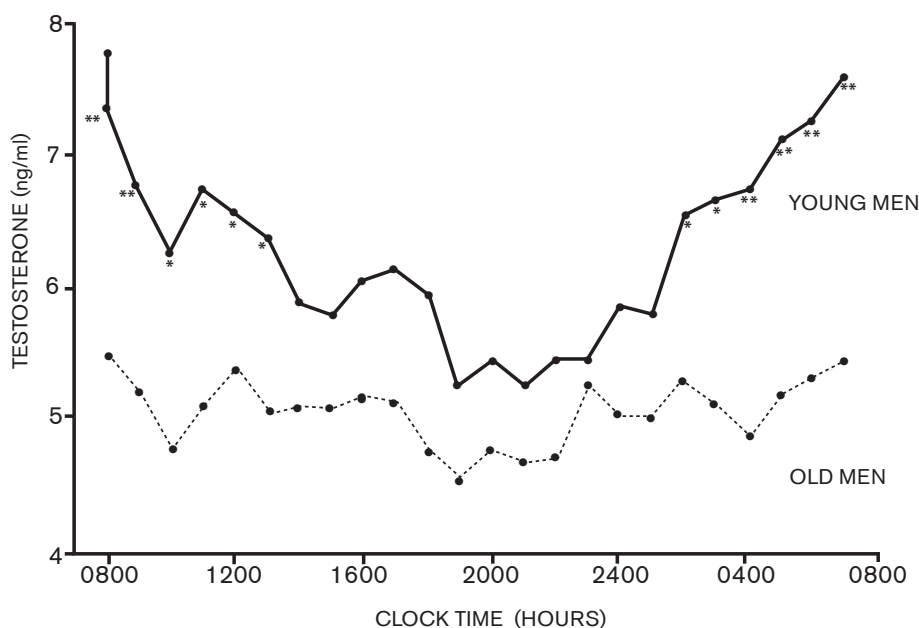
Testosterone testing is on the rise in the U.S. and United Kingdom.¹ Some of this rise is probably related to the increasing awareness of the symptoms of testosterone deficiency (TD) among males. These symptoms include fatigue, decreasing libido, depression, sleep disturbances, decreasing strength and possibly cognitive changes. Informal surveys suggest that these symptoms are very common as men age.² 'Direct-to-consumer' advertising on television and other forms of media suggests that the detection and treatment of TD may restore masculine vitality to those men with symptoms of 'low T'. Furthermore, specialty male hormone clinics cater to this 'need'.

The symptoms of TD are very common in men, although many men with these same symptoms do not have TD. The actual incidence of low testosterone (levels < 300 ng/dL) in men above the age of 45 has been estimated to approach 40% of the population in a primary care setting.³ The incidence of TD is even higher in those who are obese and have Type 2 diabetes. It is important to consider the mortality implications of abnormal serum testosterone levels and the possible impact of exogenous testosterone therapy.

Testosterone is an anabolic hormone naturally produced in the testicles. It is responsible for the maintenance of muscle mass, plays a role in the production of red blood cells, and is necessary for the development of male sexual characteristics. It reduces fat mass and improves insulin sensitivity. In addition to the symptoms of TD noted in the previous paragraph, TD may cause a reduction in muscle mass (sarcopenia), increase body fat, decrease serum HDL cholesterol, decrease in hemoglobin, and lead to osteoporosis, and erectile dysfunction.



The level of testosterone normally varies throughout the day, with a peak reached at approximately 8 a.m. As men age, not only do the basal levels of testosterone gradually diminish, but the morning peak also gradually disappears. This decline is estimated to be as much as 2% per year.⁴



Measurement of serum testosterone is usually performed in the morning, in order to measure the peak levels of this hormone. Obesity can affect serum testosterone levels. It is not unusual to have low total serum testosterone levels in obese males, whereas the free serum testosterone levels in the same individuals can be in the normal range. It is therefore recommended that free testosterone should also be measured in obese men with low total testosterone.

Lower testosterone levels are associated with a higher risk of all-cause and cardiovascular mortality among men.⁵ This relationship also exists for dihydrotestosterone, a metabolite of testosterone, but not for estradiol, another metabolite of testosterone. Some studies show that, in older subjects, the optimal all-cause survival is demonstrated in those people with mid-range levels of testosterone, suggesting that there may be a U-shaped relationship between testosterone levels and mortality.⁶

Testosterone therapy often results in increase in muscle mass and strength, as well as reduction in fat mass. Bone mineral density usually increases as well. The effect on erectile dysfunction is less clear. Testosterone administration also reduces insulin resistance and can improve the lipid profile. While these benefits are usually desirable, the balance between the risk and benefit of therapy is still being debated.⁷

The study submitted by Vigen et al. to JAMA in 2013 demonstrated that, among testosterone-deficient entrants from the Veterans Affairs medical centers, the hazard ratio for the combined outcome of MI stroke and overall mortality was 1.29 in the group that was given testosterone therapy vs. those who were not treated. The increased risk was present regardless of which testosterone preparation was used. This outcome certainly merits further study. It suggests that, while testosterone deficiency is related to additional mortality risk, simply treating the deficiency is not entirely beneficial, and may in itself entail considerable risk. It is this risk that is important for insurance medical directors and medical underwriters to fully appreciate. Its significance is compounded by the high prevalence of low testosterone levels among aging males, and the increasing prescription of testosterone replacement products.

While all-cause mortality is of paramount importance to life insurers, specific causes of increased mortality or morbidity risk can make a difference in assessment of specific cases. The primary complications of testosterone

therapy are increased cardiovascular risk, worsening of sleep apnea and an increase in red blood cell counts (with possible polycythemia in some men). Other factors to consider are mild peripheral edema, prostatism (with elevations of PSA), hypogonadism, increased aggression and mood disorder, infertility, acne, and gynecomastia. The presence of gynecomastia is related to metabolites of testosterone that cause breast development, specifically estradiols. This is the reason that some men being treated with testosterone replacement therapy are also being treated with medications such as Arimidex (i.e., to block the estrogenic properties of the estradiols).

Although the primary cause of TD is related to aging and 'andropause', there are a number of other causes that should be mentioned. Two important causes for underwriters are obesity/diabetes and chronic alcoholism. Some of the other less prevalent causes are:

- Past hx of anabolic steroid abuse
- Disease or loss of testicles
- Chemotherapy or radiation therapy
- Medications (e.g., drugs used to treat prostate cancer, corticosteroids)
- Genetic conditions such as Klinefelter's Syndrome XXY
- Hemochromatosis
- Sarcoidosis
- Pituitary gland disease
- Chronic Illness
- Chronic kidney failure
- Liver cirrhosis
- Prolonged stress

Testosterone therapy can be administered in a variety of ways. Intramuscular injections can be given every 1 – 2 weeks. These rapidly elevate serum testosterone levels, but tend to lead to very high peak levels. Testosterone patches (e.g., Androderm) require nightly application. Several different formulations in a cream or gel can be applied topically to the skin (e.g., AndroGel, Testim and Fortesta). Gels are currently the most common method of testosterone administration. Other formulations include mucoadhesive preparations that are applied to the buccal mucosa and sublingual troches or lozenges. One of the newer preparations (Axiron) is a testosterone 'stick' applied to axillary skin. The last of the available options is a long-acting subcutaneous implant, which needs to be renewed every six months.

At present, testosterone replacement therapy is contraindicated in males with a history of prostate cancer (since it can elevate PSA levels), as well as those with a history of breast cancer and severe obstructive uropathy.

The importance of testosterone replacement therapy for the insurance market has not yet been fully realized; however, the increasing use replacement therapy is likely to present challenges for underwriters. Testosterone deficiency in aging males is very common, but it is not entirely clear how replacement therapy will affect mortality outcome. While it is known that TD is associated with excess mortality, it is also suspected that supplementation will increase risk as well.

Current treatment guidelines suggest that testosterone supplementation should be initiated in patients with symptomatic, unequivocally low testosterone levels confirmed by repeated laboratory tests.⁸ Physicians are further discouraged from routine testosterone replacement therapy based on only one low testosterone level. This advice, however, is not always meticulously adhered to. Even the accepted range of low testosterone is not universally accepted. It ranges from 200 – 350 ng/dL. Additionally, this lower level of 'normal' is probably most relevant in younger, healthy males. A 'normal' lower limit in older men could be significantly lower.

Layton et al.¹ have indicated that it is not only testosterone-deficient men who are being treated. It is not uncommon for men with normal serum testosterone to seek testing and to receive supplementation. His study showed that, in the U.S., between 4 – 9% of testosterone treatment is being given to men with normal or even high testosterone levels.

At times, supplementation may result in levels of serum testosterone far in excess of the upper limits of normal. Should this be considered a form of steroid abuse? If so, what underwriting action would be reasonable? Consideration is also necessary to determine whether supplementation raises the overall mortality risk to the point that Preferred mortality expectations are exceeded. ■

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J. Carl Holowaty M.D., D.B.I.M.

cholowaty@rgare.com

Dr. J. Carl Holowaty is Senior Vice President and Chief Medical Director of RGA Reinsurance Company. He is responsible for the management of the medical department; research, development and maintenance of RGA's underwriting manual; and editing RGA's medical newsletter, ReFlections. In addition to his responsibilities at RGA, Dr. Holowaty serves as the Deputy Medical Director of the Longer Life Foundation. Dr. Holowaty earned his medical degree and a BSc in biochemistry from the University of British Columbia. He is a member of business and insurance industry organizations AAIM, CLIMOA and MMDA.

EHRs – THE STATE OF MEANINGFUL USE IN THE U.S.

By **Susan L. Wehrman** FLMI, ACS

Vice President, Electronic Health Record Initiatives
RGA Reinsurance Company

February marked the five-year anniversary of the HITECH Act, so I thought this would be a good time to take a look at the status of the health information technology movement in the U.S. and what has been accomplished to date.

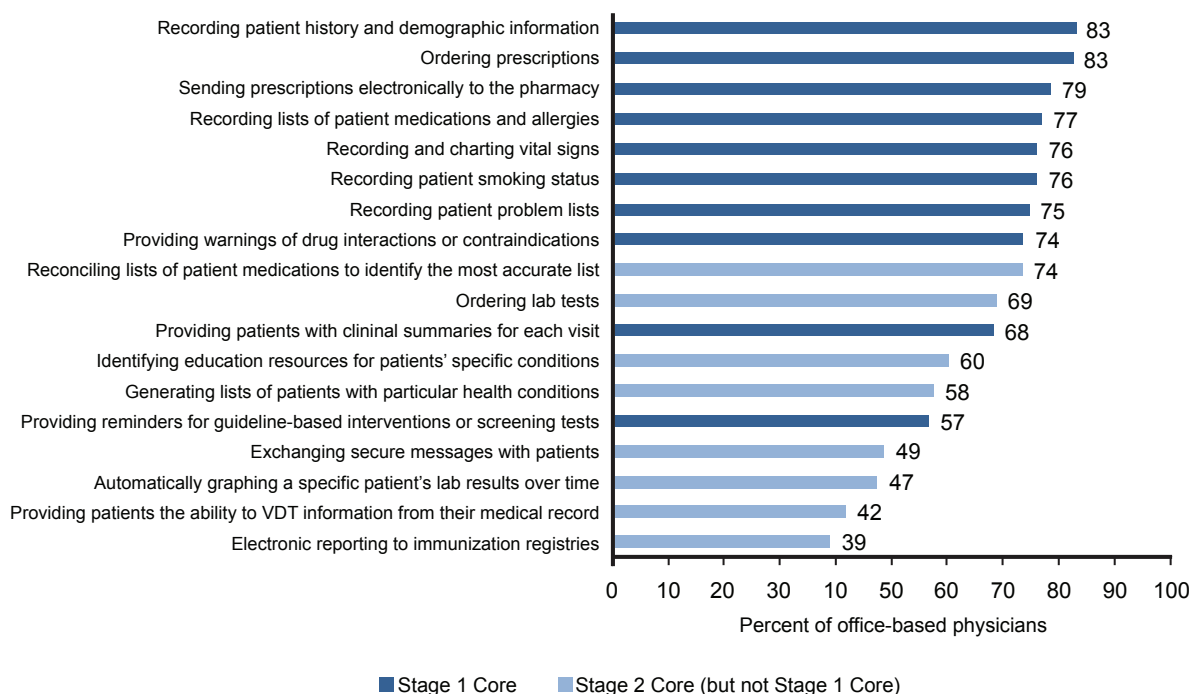
HITECH and Meaningful Use

The Health Information Technology for Economic and Clinical Health (HITECH) Act, which was included in a larger economic stimulus package, was created to promote the adoption of electronic health records (EHR) and supporting technology in the United States. U.S. President Barack Obama signed the act into law on February 17, 2009 as part of the American Recovery and Reinvestment Act of 2009 (ARRA). HITECH earmarked more than \$19 billion in incentives for providers who meet Meaningful Use criteria (to date \$30 billion has been awarded). Meaningful Use consists of three stages; each stipulates criteria for varying degrees of EHR adoption.

Stage 1 focused on data capture and patient access, Stage 2 is geared toward information exchange and care coordination, and Stage 3 aims to improve health outcomes. We are currently in Stage 2 (effective January 2014) and, as a result of pressure from clinician groups and EHR developers who requested more time to improve interoperability, the government extended Stage 2 through 2016. So how are we doing?

Meaningful Use: By the Numbers

Although we still have a long way to go in terms of interoperable systems and access to data, HITECH has definitely influenced adoption rates. The percentage of office-based physicians who have an EHR system increased from 18% in 2001 to 78% in 2013. Moreover, the percentage of physicians with selected computerized capabilities related to Meaningful Use objectives as of 2013 is as follows:



Source: CDC/NCCHS, National Ambulatory Medical Care Survey, Electronic Health Record Survey, 2013

- In 2012, 33% of primary care physicians were able to exchange clinical summaries with other doctors, and 35% could share laboratory or diagnostic tests with doctors outside their practice. Roughly one-third offered electronic access to patients.
- At the end of 2013, 82% of all eligible professionals have registered for the program and 93% of hospitals are participating. Three-quarters have received financial incentives.

As for hospitals, 2013 data show:

- 98.1% of eligible hospitals maintained a medication allergy list;
- 98% kept an active medication list;
- 96.9% recorded patient demographics;
- 95.6% recorded advance directives;
- 95.6% included clinical lab results in EHR data;
- 92.3% recorded vital signs; and
- 83.5% maintained a computerized provider order entry system.

According to a readiness report produced by HIMSS (Healthcare Information and Management Systems Society) Analytics, 68% of hospitals reported having 2014 Edition-certified EHR systems, and 60% have already met nine of 16 core objectives mandated for Stage 2. Further, the percentage of acute care hospitals achieving Stages 5, 6 or 7 on the HIMSS Analytics Electronic Medical Records Adoption Model has increased from 11.2% to 37.4% (Leary, HIMSS blog, 2/14).

Stage 2 Requirements

The U.S. Centers for Disease Control (CDC) has reported that 19% of physicians are uncertain whether they will participate in Stage 2, and 12% say they will not participate at all. About 70% say they do plan to participate.

Some of the highlights for Stage 2 include, for example, stipulating that certified EHRs (which must be used by providers to receive meaningful use bonuses) capture occupation, sexual orientation, and disability status under the recommendations. They must also provide the history of patients' medication refills – as obtained from pharmacy benefit managers – and access to Prescription Drug Monitoring Program data, which U.S. states use to track narcotic prescriptions to deter abuse and drug trafficking.

Following is a high-level summary of the requirements for Stage 2:

- **Computerized Physician Order Entry (CPOE):** More than 60% of medication, 30% of laboratory, and 30% of radiology orders must be recorded using CPOE
- **E-Prescribing:** At least 50% of all permissible prescriptions written by providers are compared to at least one drug formulary and transmitted electronically using Certified EHR Technology
- **Demographics:** At least 80% of patients must have demographics recorded as structured data
- **Vital Signs:** At least 80% of all patients must have blood pressure and height/weight recorded as structured data
- **Smoking Status:** At least 80% of patients (>13 years old) must have smoking status recorded as structured data
- **Clinical Decision Support (CDS) Rule:** Providers must implement CDS related to four or more quality measures; functionality for drug-drug and drug-allergy interaction checks
- **Patient Ability To Electronically View, Download and Transmit (VDT) Health Information:**
 - o At least 50% of patients provided online access to their health information within four business days of when it becomes available to the provider
 - o At least 5% of patients must be able to view, download, or transmit their health information to a third party
- **Clinical Summaries:** Provide to patients within one business day for at least 50% of office visits
- **Protect Electronic Health Information:** Providers must conduct or review a security risk analysis according to specific requirements
- **Clinical Lab-Test Results:** At least 55% of lab test results must be incorporated into the EHR as structured data
- **Patient Lists:** Generate patient listings by condition
- **Preventative Care:** EHR provides reminders for

preventive/follow-up care for at least 10% of patients with office visits in the previous two years

- Patient-Specific Education Resources: At least 10% of patients receive approved educational resources
- Medication Reconciliation: Perform medication reconciliation for at least 50% of transitions of care
- Summary Of Care:
 - o Provide for at least 50% of transitions of care and referrals; at least 10% are transmitted electronically directly to the recipient or via an approved Health Information Exchange (HIE).
 - o At least one successful exchange with a recipient whose EHR technology is different from the sender's OR a successful test with an approved CMS test EHR
- Immunization Registries Data Submission: Perform ongoing submissions to immunization registries or centralized systems
- Use Secure Electronic Messaging: Secure electronic messages exchanged with at least 5% of patients

In addition to these core requirements, there are several menu objectives covering family health history, reporting cancer cases, etc., that providers will elect to meet.

Beyond Stage 2 – What's Next?

Recommendations for Stage 3 requirements have not yet been finalized and are still under review. Requirements governing the third and final stage of "meaningful use" of electronic health records are scheduled to become final in the first half of 2015 and become effective in 2017. ■

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Susan L. Wehrman FMLI, ACS
swehrman@rgare.com

Susan L. Wehrman, Vice President, Electronic Health Record Initiatives, heads RGA's newly created Electronic Health Record (EHR) Initiatives area. This function conducts in-depth research and analysis of this evolving segment and monitors all activity in the U.S. and around the world, with the objectives of positioning RGA as an industry thought leader and better assisting clients with EHR-related issues.

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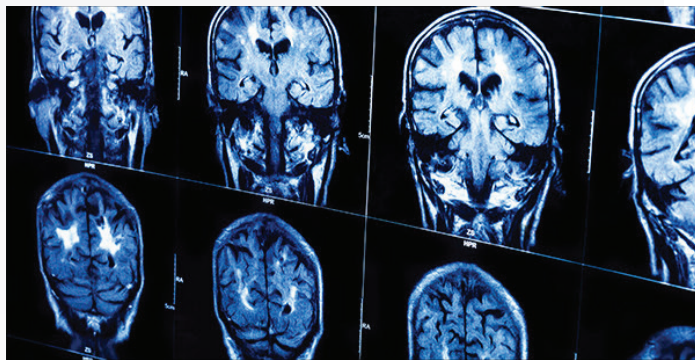


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Moderator: Dr. J. Carl Holowaty
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