Heart of the Matter: Hypogonadism Management with Testosterone Therapy

Saturday, May 14, 2011
Walter E. Washington Convention Center
Washington, DC
8:00 AM Program
9:45 AM Adjourn

To participate in other CME/CE programs about hypogonadism, please visit TestosteroneUpdate.org

This event is not part of the AUA 2011 Annual Meeting.

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This activity is supported by an independent educational grant provided by
Offers free downloadable CME, tools, and resources on hypogonadism for clinicians and their patients.

TU is a unique CME-certified initiative committed to alleviating the symptoms of patients suffering from hypogonadism, through accurate diagnosis and reestablishment of constant physiologic testosterone levels, for improved overall health and well-being.

Become a TU member and refer a colleague at [TestosteroneUpdate.org/membership](http://TestosteroneUpdate.org/membership) to receive updates on CME activities, tools, and resources on hypogonadism.

The TU initiative includes a total of 50 expert Distinguished Faculty members who collaborate to provide cutting-edge education on hypogonadism.

**Founding Chairpersons**

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  Warren Alpert Medical School of Brown University
  Providence, Rhode Island

- **Ajay Nehra, MD**
  Professor of Urology
  Department of Urology
  Mayo Clinic College of Medicine
  Rochester, Minnesota
Agenda
Saturday, May 14, 2011

7:30 AM Registration and Breakfast

8:00 AM Welcome and Introduction
Allen D. Seftel, MD Chairperson

8:05 AM Hypogonadism Epidemiologic Evidence: The Imperative for Screening and Diagnosis
Martin M. Miner, MD

8:25 AM Association of Hypogonadism With Chronic Comorbid Conditions
Adrian S. Dobs, MD, MHS

8:45 AM Testosterone and Cardiovascular Health: Safety of Treatment for Hypogonadism
Robert A. Kloner, MD, PhD

9:05 AM Therapeutic Options for Hypogonadism: The Importance of Adherence to and Persistence With Testosterone Therapy
Allen D. Seftel, MD

9:25 AM Interactive Q&A and Discussion
All Faculty

9:45 AM Closing Comments and Adjourn

Chairperson
Allen D. Seftel, MD
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Warren Alpert Medical School of Brown University
Providence, Rhode Island
Intended Audience
Urologists, endocrinologists, primary care physicians, and other healthcare professionals interested in the management and treatment of hypogonadism.

Statement of Need
Hypogonadism is an underdiagnosed and therefore undertreated condition of men that can be associated with serious comorbid conditions. In light of these facts, appropriate screening, recognition, and treatment should be encouraged for any provider seeing men who are at risk.

Extrapolation of 2003 census data suggests that 13.8 million men aged ≥45 years visiting primary care physicians in the United States may have low testosterone levels, yet fewer than 10% of these men are being treated. In the Boston Area Community Health (BACH) survey, only 12.2% of all androgen-deficient men were being treated despite adequate access to healthcare.

Reported prevalences vary among studies depending on the population being studied, the definition of hypogonadism used, and the methodology, however, hypogonadism is becoming increasingly prevalent with the aging of the US population. The overall prevalence of hypogonadism was reported to be 6% at baseline in data from the Massachusetts Male Aging Study (MMAS). Of the 1691 men in that study for whom complete testosterone data were available at baseline, 561 were aged 40 to 49 years, 558 were aged 50 to 59, and 572 were aged 60 to 70. During the follow-up phase (n=1087; mean follow-up interval, 8.8 y; range, 7.0-10.4 y), the overall prevalence doubled to 12.3%. In data from the BACH survey, the reported prevalence of hypogonadism was 5.6% (N=1475; mean age, 47.3 y).

It is critical to recognize the signs and symptoms of hypogonadism prior to choosing appropriate interventions. Hypogonadism is associated with an array of signs and symptoms, many of them subtle and nonspecific, and with several serious comorbid conditions, such as diabetes and osteoporosis. In addition, mounting evidence indicates that low testosterone levels are associated with premature cardiovascular disease, cardiovascular events, and cardiac death, as well as increased all-cause mortality. In a well-designed population-based study of men aged 50 to 91 years (mean, 73.6 y), Laughlin et al found a >40% increased risk of mortality among men in the lowest quartile of total testosterone or bioavailable testosterone compared with the highest quartile. Despite compelling evidence, many clinicians are not aware of the connections between testosterone, comorbid conditions, and overall health.

Educational Objectives
At the conclusion of this activity, participants should be better able to:

• Define hypogonadism as a significant, chronic medical condition that is associated with obesity, metabolic syndrome, diabetes, dyslipidemia, hypertension, frailty, and cardiovascular disease and may lead to increased morbidity
• Examine current data on the relationship between cardiovascular disease and serum testosterone concentrations demonstrating that a causal relationship has not been established, and recognize that testosterone therapy may be safely initiated
• Determine appropriate therapy to address patient needs by differentiating between testosterone formulations currently available and in development
• Realize that a critical component of the overall health of men is eugonadal testosterone levels
Accreditation and Certification

This activity has been planned and implemented in accordance with the Essential Areas and policies of the Accreditation Council for Continuing Medical Education (ACCME) through the joint sponsorship of the Annenberg Center for Health Sciences at Eisenhower and CogniMed Inc. The Annenberg Center is accredited by the ACCME to provide continuing medical education for physicians.

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All staff at the Annenberg Center for Health Sciences at Eisenhower have nothing to disclose.

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Adrian S. Dobs, MD, MHS, receives research support from the National Institutes of Health and Takeda Pharmaceutical Company Limited.

Robert A. Kloner, MD, PhD, has consulted in an unpaid capacity for and has served on the speakers bureau of Pfizer Inc.

Martin M. Miner, MD, receives research support from Auxilium Pharmaceuticals, Inc., and Indevus Pharmaceuticals, Inc. He is a consultant for GlaxoSmithKline/Schering-Plough Corporation and Sanofi-Aventis.

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Allen D. Seftel, MD, is Head of the Division of Urology at Cooper University Hospital and Professor of Urology at Robert Wood Johnson School of Medicine, in Camden, New Jersey.

Dr Seftel received a medical degree from the State University of New York Health Science Center at Brooklyn (SUNY Downstate Medical Center). He completed a residency in urology at Case Western Reserve University, in Cleveland, Ohio. Additional postdoctoral training included an American Foundation of Urologic Disease Scholar in Impotence Fellowship at Boston University School of Medicine, in Massachusetts.

The focus of Dr Seftel’s research has been centered on oral therapy for restoring erectile function, including the use of sildenafil and tadalafil; Medicated Urethral System for Erection (MUSE®); vacuum therapy; and penile prostheses. Other areas of research include the association of glycemic control with sexual function in men with type 2 diabetes and sexual aspects of rehabilitation in spinal cord injury. Hypogonadism, ejaculatory dysfunction, and female sexual dysfunction are among his other areas of investigation.

Dr Seftel has written extensively on male sexual dysfunction. He is also actively engaged in continuing medical education at the University Hospitals of Cleveland and other medical institutions. Dr Seftel offered a special instructional course in the evaluation of male sexual function at the American Urological Association (AUA) Annual Meeting. He is a reviewer for *Urology*, *Journal of Urology*, and the AUA Annual Meeting Abstract Committee.

Dr Seftel serves as Editor in Chief of the *International Journal of Impotence Research* and *Journal of Sexual Medicine* and the abstract author of *Journal of Urology*’s Urologic Survey section, “Male and Female Sexual Function and Dysfunction: Andrology.” Dr Seftel is a member of numerous medical organizations, including the AUA, the American Association of Clinical Urologists, and the International Society of Sexual Medicine. He has been listed in *America’s Top Doctors*.

Adrian Sandra Dobs, MD, MHS, is Professor of Medicine and Oncology and Vice Chair for Faculty Development in the Department of Medicine of the Johns Hopkins University School of Medicine, in Baltimore, Maryland, and Director of the Johns Hopkins Clinical Research Network of the Johns Hopkins Institute for Clinical and Translational Research. Dr Dobs is also Co-Director of the Johns Hopkins Center for the Reduction of Cancer Disparities of the Johns Hopkins Bloomberg School of Public Health.

Dr Dobs received a medical degree from Albany Medical College, in New York, and completed an internship in internal medicine at Montefiore Hospital, Albert Einstein College of Medicine, in the Bronx, New York. She held a fellowship in endocrinology from the Johns Hopkins University School of Medicine and earned a master in health sciences degree in cardiovascular epidemiology at the Johns Hopkins University Bloomberg School of Public Health.

Dr Dobs is an investigator on two multi-center AIDS cohort studies and is studying sex hormones and cardiovascular risk and the reduction of cancer disparities. She lectures in the United States and internationally in these areas, as well as aging and testosterone therapy and testosterone and cardiovascular disease.

With book chapters, monographs, and journal articles, as well as television and Web contributions, Dr Dobs has published extensively on topics that include hormonal and other changes with aging and skeletal muscle decline and other effects of HIV and injection drug use. Journals publishing her research include *Journal of Clinical Endocrinology and Metabolism*, *JAIDS*, and *Journal of Andrology*. She co-chairs the International Registry for Hypogonadal Men.

Dr Dobs is very active in mentoring medical students and postdoctoral fellows and was honored by the Johns Hopkins University School of Medicine with the 2009 David M. Levine Excellence in Mentoring Award.
Robert A. Kloner, MD, PhD, is Professor of Medicine in the Cardiovascular Division of the Keck School of Medicine at the University of Southern California, in Los Angeles. He is also Director of Research at the Heart Institute of Good Samaritan Hospital, in Los Angeles, and Attending Cardiologist at Los Angeles County/University of Southern California Medical Center.

Dr Kloner received medical and doctor of philosophy degrees from Northwestern University Medical School, in Chicago, Illinois. He completed an internship and a residency in internal medicine at Peter Bent Brigham Hospital, in Boston, Massachusetts. Additional training includes clinical and research fellowships in medicine and cardiology at Harvard Medical School and Brigham and Women’s Hospital, both in Boston. He served as Assistant and then Associate Professor of Medicine at Harvard Medical School and as an attending cardiologist at Brigham and Women’s Hospital. There he received an Established Investigator Award of the American Heart Association.

Among Dr Kloner’s major research interests are cardiac cell transplantation, protection of ischemic myocardium, cardiac function following coronary artery occlusion, the effect of toxins on the heart, preventive cardiology, hypertension, and phosphodiesterase type 5 inhibition. He has been funded by the National Institutes of Health (NIH) for studies on cardiac cell transplantation, doxorubicin cardiomyopathy, functional analysis of cardiac grafts, and stem cells. He has served on the NIH Cardiovascular Study Section A and has participated in a number of NIH Workshops.

Dr Kloner has contributed more than 600 original papers, 210 chapters or monographs, and 450 abstracts. He is the author and editor of 18 medical texts, including Cardiovascular Trials Reviews, The Guide to Cardiology, Stunned Myocardium, Ischemic Preconditioning, and VIAGRA, and 3 novels, The Beta Virus, Mind Cure, and The Deity Genes. Dr Kloner serves as Editor in Chief of Journal of Cardiovascular Pharmacology and Therapeutics and on the editorial boards of Circulation, American Journal of Cardiology, Heart, Basic Research in Cardiology, and International Journal of Impotence Research.

Among his many career distinctions, Dr Kloner has been listed in Who’s Who in America and The Best Doctors in America and was identified as one of the world’s most highly cited authors by the Institute for Scientific Information. Dr Kloner is a Fellow of the American College of Cardiology and an Inaugural Fellow of the Council on Basic Cardiovascular Sciences of the AHA and was elected to the American Society of Clinical Investigation.

Martin M. Miner, MD, is Director of the Men’s Health Center of Miriam Hospital, in Providence, Rhode Island, and is in practice with Swansea Family Practice, in Massachusetts. He is also Clinical Associate Professor of Family Medicine and Urology at Warren Alpert Medical School of Brown University, in Providence.

Dr Miner graduated Phi Beta Kappa from Oberlin College and received a Doctor of Medicine degree from the University of Cincinnati College of Medicine, both in Ohio. He completed a residency at Brown University and worked with the Indian Health Service Corps and the Public Health Service. Active on several journal editorial boards, Dr Miner also serves as a reviewer and has published extensively in the areas of erectile dysfunction, cardiovascular disease, benign prostatic hyperplasia, lower urinary tract symptoms, male sexuality, and hormone therapy for men. He has given numerous presentations in the United States and internationally, is active in several research studies on men’s health, and is a consultant to the International Society of Sexual Medicine Consensus Panel.

Dr Miner holds memberships in the American Academy of Family Physicians and the American Urological Association and was elected a Fellow of the Sexual Medical Society of North America. He was chosen the Brown Teacher of the Year in 2003 and 2007.
Reported prevalences vary among studies depending on the population being studied, the definition of hypogonadism used, and the methodology, however, hypogonadism is becoming increasingly prevalent with the aging of the US population. According to the Hypogonadism in Males (HIM) study, an extrapolation from 2003 census data, 13.8 million men aged ≥45 years visiting primary care physicians in the United States may have low testosterone levels.\(^1\)

The overall prevalence of hypogonadism was reported to be 6% at baseline in data from the Massachusetts Male Aging Study (MMAS).\(^2\) Of the 1691 men in that study for whom complete testosterone data were available at baseline, 561 were aged 40 to 49 years, 558 were aged 50 to 59, and 572 were aged 60 to 70. During the follow-up phase (n=1087; mean follow-up interval, 8.8 y; range, 7.0-10.4 y), the overall prevalence doubled to 12.3%. Recognizing that hypogonadism should be defined by serum testosterone levels and clinical symptomatology—and not solely low serum testosterone levels—the Boston Area Community Health (BACH) survey estimated the prevalence of symptomatic hypogonadism as 5.6% (N=1475; mean age, 47.3 y).

Recently, Wu and colleagues sought evidence-based criteria for identifying late-onset hypogonadism in the general population by surveying a random population sample of 3369 men between the ages of 40 and 79 years at 8 European centers.\(^3\) Based on a reductive analytic approach to produce parsimonious clinical and biochemical criteria for diagnosing late-onset hypogonadism, only 3 sexual symptoms were found to have a syndromic association with decreased testosterone levels, and the prevalence of late-onset hypogonadism was determined as 2.1%.

Mounting epidemiologic evidence supports that low testosterone levels are associated with premature cardiovascular disease (CVD), cardiovascular events, and cardiac death, as well as increased all-cause mortality. Furthermore, declines in testosterone have been associated with comorbid conditions and hemoglobin levels that may portend increased frailty,\(^4\) and both longitudinal and cross-sectional studies have linked low testosterone levels with poor mobility, balance impairment, and higher risk of falls.\(^5,6\)

An analysis of data from the landmark Rancho Bernardo population-based study of men aged 50 to 91 years (mean, 73.6 y) found a >40% increased risk of mortality among men in the lowest quartile of total testosterone or bioavailable testosterone compared with the highest quartile.\(^7\)

In a study of male veterans, Shores et al evaluated whether low testosterone levels are a risk factor for mortality.\(^8\) After adjusting for age, medical morbidity, and other clinical covariates, low testosterone levels were associated with increased mortality.

The European Prospective Investigation Into Cancer in Norfolk (EPIC-Norfolk) prospective population study, investigating the relationship between endogenous testosterone levels and mortality, also showed that testosterone concentrations are related to all-cause and CVD-related mortality.\(^9\) Increasing quartiles of testosterone were protective, such that men in the highest quartile had a 30% lower risk of death than did those in the lowest quartile. Even after excluding deaths during the first 2 years of follow-up and adjusting for cardiovascular risk factors and sex hormone-binding globulin, this inverse relationship was maintained.

It is clear that vascular health is intrinsically tied to both hypogonadism and erectile dysfunction. Forthcoming guidelines regarding cardiometabolic risks and sexual health will incorporate findings from studies by Thompson et al and Inman et al that demonstrated that erectile dysfunction may be an early manifestation of CVD, preceding and predicting future cardiovascular events.\(^10,11\)

These epidemiologic studies, some of which suggest that hypogonadism is a predictive marker for CVD, support that appropriate screening for hypogonadism may indeed be a clinical imperative.

**References**


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**Testosterone Concentrations Related to All-Cause and CVD Mortality**

![Graph showing the relationship between testosterone concentration quartile and cumulative survival over follow-up years for all-cause and CVD mortality.](image)

- **All-Cause**
  - Cumulative Survival
  - Follow-up, y
  - Testosterone concentration quartile
  - Quartiles 1 and 4 are compared.

- **CVD**
  - Follow-up, y
  - Cumulative Survival

Note: 2,146 men aged 40-78 y.

CVD, cardiovascular disease.


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**ED Predicts Coronary Events: 10-Year Follow-up**

- 1,402 men aged 40 to 70 y with no known CAD
- ED and CAD may share common underlying vascular pathology
- ED in younger men related to marked increase in risk of cardiac events
- ED in older men of little prognostic importance

![Graph showing the incidence per 1,000 person-years of coronary events by age and ED status.](image)

CAD, coronary artery disease; ED, erectile dysfunction.

Due in part to the escalating prevalence rates of obesity, metabolic syndrome, and type 2 diabetes, there is an increasing case burden of hypogonadism in the United States. Population-based studies have demonstrated that hypogonadism contributes to the risk of metabolic syndrome and type 2 diabetes, however, the precise mechanism underlying the development of metabolic syndrome and type 2 diabetes remains to be elucidated.

Testosterone exerts fundamental effects on various metabolic and body composition parameters. Low endogenous testosterone levels, via effects on glucose transport and/or body composition, may contribute to increased cardiometabolic risk and subsequent mortality. Conversely, type 2 diabetes and metabolic syndrome may be risk factors for hypogonadism, possibly through increased body weight and aromatase activity contributing to higher estradiol levels, decreased sex hormone-binding globulin and gonadotropin levels, and suppressed testosterone production by Leydig cells.

Hypogonadism is clearly associated with a higher prevalence of components of metabolic syndrome, particularly visceral adiposity and insulin resistance. Studies suggest that visceral adipose tissue may secrete hormones and proinflammatory factors that adversely affect metabolic parameters and contribute to hypogonadism. Testosterone therapy may positively affect insulin sensitivity and type 2 diabetes risk factors, but more clinical evidence is needed to fully understand the implications of these findings.

Increased clinical vigilance and awareness of hypogonadism’s association with comorbid conditions, including type 2 diabetes, insulin resistance, and metabolic syndrome, may present additional therapeutic options to minimize cardiometabolic risk. Though current Endocrine Society clinical practice guidelines do not advocate widespread screening for hypogonadism in the general population, appropriate evaluation and serum testing is recommended for patients with signs and symptoms suggestive of hypogonadism or with associated comorbid conditions.

Clinicians need to understand the relationship between hypogonadism and comorbid conditions to ensure that all appropriate patient populations are screened and patients with hypogonadism receive proper treatment.

NOTES
Self-perpetuating Pathogenic Circle: Visceral Adiposity Is a Pivotal Component


Improved Glycemic Control in Hypogonadal Men Treated With Testosterone Therapy

HOMA index and A1C improvements with testosterone therapy compared to placebo.

ATC, glycated hemoglobin; HOMA, homeostasis model assessment.
Attention has recently focused on the need to clarify the relationship between testosterone and cardiovascular disease (CVD). Many testosterone therapy trials have shown that the administration of exogenous testosterone improves muscle mass and strength, bone density, and body composition. However, none of these studies were of sufficient size or duration to adequately address the effect of testosterone therapy on cardiovascular risk. Furthermore, though epidemiologic studies suggest that low endogenous testosterone levels increase cardiovascular risk and mortality, a large-scale longitudinal, placebo-controlled study is needed to ascertain whether exogenous testosterone has a therapeutic effect on CVD.

Interest in the relationship between exogenous testosterone and cardiovascular risk was newly generated by the Testosterone in Older Men With Mobility Limitations (TOM) trial, a placebo-controlled, randomized study designed to ascertain the effects of testosterone therapy on lower extremity strength and physical function in a population of older men with hypogonadism and limitations in mobility.

Though the testosterone-treated group had significantly greater improvements in the primary efficacy endpoints (eg, leg-press and chest-press strength and stair-climbing while carrying a load) compared to placebo, hematocrit and hemoglobin levels increased significantly, whereas high-density and low-density lipoprotein levels declined. Provoking further concern, a significantly higher number of cardiovascular adverse events (AEs) were reported in patients receiving testosterone therapy.

Based on the significantly increased incidence of cardiovascular AEs in the treatment arm of the TOM trial, a National Institute on Aging data and safety monitoring board recommended cessation of study enrollment and administration of testosterone therapy, leading to the subsequent early termination of the study.

Though the authors noted that cardiovascular AEs were not formally evaluated and were not projected primary or secondary outcomes, they also reinforced that caution is warranted in extrapolating from these findings to other doses and formulations of testosterone or to other populations, particularly men with hypogonadism without CVD or mobility limitations. It is critical to evaluate the reported cardiovascular AEs in context.

Furthermore, it is important to review recent studies that have investigated the effect of testosterone therapy on cardiovascular parameters as a primary endpoint. In preliminary studies of elderly patients with moderately severe congestive heart failure, testosterone therapy was shown to improve exercise capacity, muscle strength, glucose metabolism, and baroreceptor sensitivity, suggesting that benefits of testosterone are mediated by metabolic and peripheral effects. Small studies have shown a positive effect of testosterone therapy on exercise-induced ischemia, lipid profiles, carotid intima-media thickness, and body composition in hypogonadal men with stable, chronic angina and a protective effect on myocardial ischemia that was maintained without decrement, suggesting a potential effect of testosterone therapy on body composition and cardiovascular parameters.
TOM Trial: Cardiovascular AEs

- Caution is warranted in interpreting and extrapolating from these findings to other doses and formulations of testosterone or to other populations, particularly men with hypogonadism without CVD or mobility limitations
  - Participants had a high prevalence of chronic conditions, including heart disease, obesity, diabetes, and hypertension
  - Cardiovascular AEs were not a planned primary or secondary outcome, and therefore, a structured analysis of cardiovascular AEs was not performed
  - Clinical characteristics of the study population differ from those of most other populations in which testosterone therapy has been administered in a clinical setting or as part of a clinical trial
  - Trials terminated early tend to overestimate treatment differences
  - Lack of a consistent pattern in AEs and a small number of overall AEs suggest the possibility that the differences detected between the two groups may be due to chance

AE, adverse event; CVD, cardiovascular disease; TOM, Testosterone in Older Men With Mobility Limitations.

Relationship Between Changes in Total Testosterone Level and MVC in Testosterone-Treated Group

- In heart failure patients treated with testosterone, there was a significant direct relationship between the increase in plasma levels of testosterone and the increase in maximal isometric muscle strength

$r=0.29;\ P=0.007$.
MVC, maximal voluntary contraction.
Despite the fact that hypogonadism is a significant, chronic medical condition associated with serious comorbid conditions, it remains undertreated. Various studies support the proven benefits of testosterone therapy, which include increased lean body mass, decreased fat mass, and improved sexual desire and function. Furthermore, preliminary studies suggest that testosterone therapy mitigates some risk factors that contribute to metabolic syndrome.

Some physicians erroneously believe that evaluating for hypogonadism and monitoring treatment is complex and prohibitively time-consuming. Physicians unfamiliar with the array of treatment options may expect scheduling constraints to preclude devoting sufficient time to the evaluation and monitoring of hypogonadism or are inexperienced with the preliminary and follow-up evaluations necessary with testosterone therapy.

To determine treatment efficacy, tolerability, and safety, it is important to routinely monitor patients receiving testosterone therapy at baseline and at follow-up visits, generally at 3 and 6 months after the initiation of therapy and yearly thereafter. Initial assessments should include a digital rectal examination and a serum evaluation of prostate-specific antigen and hemoglobin or hematocrit levels. Patients should also be assessed for voiding symptoms, sleep apnea, gynecomastia, and edema.

An array of testosterone formulations are available and in development. Concerns about formulation-specific adverse events may be minimized and managed by following the labeling protocol. Physicians need to be informed about how safety is ensured for each testosterone formulation and how to communicate with patients about the critical need to adhere to treatment regimens.

Medication nonadherence is a significant issue for patients with chronic conditions. Asking the patient in a nonjudgmental, nonconfrontational way about his medication-taking behavior is a simple way to disclose poor adherence. Adherence should be assessed regularly, with clinicians explaining different routines or behaviors to improve adherence and optimize treatment outcomes. Adherence is enhanced when the patient is involved in therapy selection, understands that the treatment is important, and perceives that the regimen is convenient. Clinicians can optimize adherence by emphasizing to each patient the value of his treatment and by keeping the regimen uncomplicated and customized to fit the patient’s lifestyle. Ultimately, the best outcome will result from collaboration and communication between the patient and the clinician with consideration to individual circumstances.

NOTES
Monitoring During Testosterone Therapy

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<tr>
<th>Parameter</th>
<th>Frequency</th>
<th>Comment</th>
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<tbody>
<tr>
<td>DRE</td>
<td>Baseline, at 3 and 6 mo, and yearly thereafter(^1,2)</td>
<td>Urologic consultation if prostatic abnormality detected(^1); biopsy if abnormal(^2)</td>
</tr>
<tr>
<td>AUAI/PPS</td>
<td>Baseline; prostate-related symptom assessment every 6-12 mo(^2)</td>
<td>Urologic consultation if score &gt;19</td>
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<tr>
<td>PSA</td>
<td>Baseline, at 3 and 6 mo, and yearly thereafter(^1,2)</td>
<td>Biopsy if PSA &gt;4.0 ng/mL(^2)</td>
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<td></td>
<td></td>
<td>Biopsy if PSA increase of ≥1.0 ng/mL in any 12-mo period(^2)</td>
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<tr>
<td></td>
<td></td>
<td>Repeat PSA measurement for PSA increase of 0.7-0.9 ng/mL in 1 y(^2)</td>
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<tr>
<td>Hematocrit(^1,2)</td>
<td>Baseline, at 3 and 6 mo, and yearly thereafter</td>
<td>If &gt;54%, stop until decreases to safe level, evaluate hypoxia and sleep apnea, and reinstitute at reduced dose</td>
</tr>
<tr>
<td>Breast examination(^1,2)</td>
<td>Baseline and follow-up</td>
<td>Detect gynecomastia</td>
</tr>
<tr>
<td>Hypoxia/sleep apnea(^1)</td>
<td>Baseline and as needed clinically</td>
<td>Ask about disordered sleep</td>
</tr>
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AUA, American Urological Association; DRE, digital rectal examination; IPSS, International Prostate Symptom Score; PSA, prostate-specific antigen.


Testosterone Therapy for Hypogonadism: Adherence Considerations

- Chronic condition requiring long-term therapy
  - Patient adherence to and persistence with treatment are critical
  - Patients should be involved in treatment choice
- Ideal testosterone therapy
  - Favorable safety profile
  - Minimal adverse effects
  - Convenient regimen and administration

Goorin LJ, Buncic MC. Drugs. 2004;64(17):1861-1891.
Suggested Reading


This activity will be available online for CME credit. To register to be notified when this activity is available online or to participate in other CME/CE activities about hypogonadism, please visit TestosteroneUpdate.org

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