The only medically documented and proven PCT I have seen on any board

By Praetorian

In response to a question I posted some time ago.

If you haven't used hcg during your cycle...which is hard to believe these days as it is almost a given..I would hope anyway..and if you dont know why then you might want to rethink your complete AAS cycle. As well, a cycle this long (60+ months) without hcg will have definitely reduced testicular mass and clomid and nolva will do just about nothing in returning that mass to precycle size in time to save any gains you have made. Nolva and clomid are SERMs and act in similar ways..clomid somehwat better at stimulating LH. But running both concurrently with hcg will not hamper recover as they will counter any aromitization of endo test that hcg produces. HCG is necessary to give the testes the boost back to regain the lost mass...without that you are in for a long rough ride. Keep in mind this is also individualistic and depends on a variety of things ie cycle length, AAS used, age, etc. If you are over 30 good luck with clomid and nolva!!!
The best PCT I can think of if you havent run hcg during is hcg, nolva, clomid, hgh, slin in moderation, strict diet(high pro, efas, mod to low carbs) IGF-1 if you like also would help.
I am starting PCT in 2 weeks and will be running this minus the IGF...see study below on returning to eugonadal stae after aas induced hypogodandism.

First thing...get a full blood count from your Dr. Test levels free and bound, liver enzymes, cholesterol etc. This will give you a starting point of which to measure yourself against. Now, for recovery...depending on your age and length of cycle, dosage etc this will determine how long it will take to full eugonadal function.
The standard protocol would be clomid, nolvadex, and HCG taken concurrently for 45 days and then a blood test taken to see how you stand as compared to the first blood work. Most people will tell you not to take more than 500iu HCG per day but sometimes the testes will not respond to this dosage as they have been dormant too long and have lost considerable mass. Remeber, your endo test levels will not rise (normal testicular function) until you have regained most of the lost mass in the testes. This is the reason for taking HCG at 500iu 2-3 days per week while on cycle...thus you never lose much testicular mass.

See below for a study done on men with hypogonadal function due to exogenous testesterone or AAS usage.

Objective:
Although shown to be effective for their intended medical treatment, AAS have been shown to induce hypogonadotropic hypogonadism in adult males. The medical literature is conflicting in the reports of spontaneous return and long-term suppression of gonadal suppression post AAS usage. This observational study documents the treatment protocol of HCG, clomiphene citrate, and tamoxifen in returning hormonal function to normal post AAS usage. Design:
Five HIV-negative males age 27-49, weighing 77-100 kg, with serum total testosterone levels below 240 ng/dL and luteinizing hormone (LH) levels below 1.5 mIU/mL were considered for this observational study. All five patients were administered the treatment protocol.

Methods:
Treatment consisted of combination therapy which included concurrent administration of (a) Human Chorionic Gonadotropin, (b) Clomiphene Citrate and (c) Tamoxifen Citrate for a standard duration of 45 days. This protocol was repeated with every patient until serum LH and total testosterone values reached normal ranges.
Results:
All five patients were considered eugonadal by normal laboratory reference ranges by the conclusion of treatment. Average serum total testosterone rose from 98.2 to 692.8 ng/dL ($p<.001$) while the average serum LH rose from an average undetectable value of less than 1.0 to 7.92 mIU/mL ($p<.0008$).

Conclusions: Although the treatment protocol of HCG, clomiphene citrate, and tamoxifen proved beneficial in reversing AAS induced hypogonadotropic hypogonadism, future controlled studies need to be performed to confirm the beneficial effects of this combined pharmacotherapy in returning HPGA functioning to normal.
Key Words- anabolic-androgenic steroids, clomiphene, HCG, tamoxifen, testosterone, HIV

INTRODUCTION

Testosterone and testosterone analogues, anabolic-androgenic steroids (AAS), have long been used in the athletic community for improving lean muscle tissue and strength. A positive correlation has been shown with testosterone to include:
increased protein synthesis resulting in lean muscle tissue development (Bhasin et al, 1996; 1997; Hervey et al, 1981; Tenover, 1992),
enhanced sexual desire (libido) (Schiavi et al, 1991),
increased muscular strength (Bhasin et al, 1996; 1997; Hervey et al, 1981; Sih et al, 1997),
increased erythropoiesis (Bhasin et al, 1997; Evans & Amerson, 1974; Sih et al, 1997; Tenover, 1992),
a possible positive effect on bone development (Anderson et al, 1996; 1997; Baran et al, 1978; Tenover, 1992),
improved mental cognition and verbal fluency (Alexander et al, 1998), and male masculinizing characteristics (Starr & Taggart, 1992).

Recently, however, clinicians have recognized the potential benefits of their use in the treatment of various disorders and ailments. Numerous studies have discussed the use of AAS in the treatment of HIV-associated conditions (Bhasin et al, 2000; Grinspoon et al, 1998; 1999; 2000; Rabkin et al, 1999; 2000; Sattler et al, 1999; Strawford et al, 1999; Van Loan et al, 1999), hypogonadism (Bhasin et al, 1997; Davidson et al, 1979; Rabkin et al, 1999; Sih et al, 1997; Snyder et al, 2000; Tenover, 1992; Wagner & Rabkin, 1998; Wang et al, 2000), impotence (Carani et al, 1990; Carey et al, 1988; Klepsch et al, 1982; Lawrence et al, 1998; McClure et al, 1991; Morales et al, 1994; 1997; Nankin et al, 1986 Rakic et al, 1997; Schiavi et al, 1997), burn victims (Demling et al, 1997), various anemia's (Doney et al, 1992; Gascon et al, 1999; Hurtado et al, 1993; Stricker et al, 1984), deteriorated myocardium (Tomoda, 1999), glucose uptake (Hobbs et al, 1996), continuous ambulatory peritoneal dialysis (CAPD) (Dombros et al, 1994), alcoholic hepatitis (Bonkovskyet al 1991; Mendenhall et al, 1993), hemochromatosis (Kley et al, 1992)

and prevention of osteoporosis (Anderson et al, 1996; 1997; Baran et al, 1978; Behre et al 1997; Hamdy et al, 1998; Prakasam et al, 1999).

While AAS have proven effective in cases of lean muscle wasting conditions (HIV/AIDS), this class of medicines is not without their inherent problems. AAS have been shown to induce hypogonadotropic hypogonadism (Alen et al, 1987; Bhasin et al, 1996; Bijlsma et al, 1982; Clerico et al, 1981; Jarow & Lipshultz, 1990; Strawford et al, 1999; Stromme et al, 1974). This condition typically results from an abnormality in the normal functioning of the hypothalamic-pituitary-gonadal axis (HPGA), usually from a negative feedback inhibition of one of the hormone secreting glands, causing a cascading unbalance in the rest of the axis. Possibly resulting from a physiological abnormality (i.e. mumps orchitis, Klinefelters syndrome, pituitary tumor) or as an acquired result of exogenous factors (i.e. androgen therapy, AAS administration). Clerico et al (1981) found a dramatic suppression of serum gonadotropin levels in athletes given methandrostenelone, suggesting a direct action of AAS on the hypothalamus. Similar results of suppressed gonadotropins have been found in patients supplementing solely testosterone (Bhasin et al, 1996; Marynick et al, 1979; Strawford et al, 1999; Tenover, 1992). Case report studies discussed a 36-year old male competitive bodybuilder and a 39-year old father, each using various AAS regimens over extended periods of time, who showed a blunted response to GnRH stimulation tests (Jarow & Lipshultz, 1990). One particular study administered 600 mg of nandrolone decanoate to 30 HIV-positive males over twelve weeks (Sattler et al, 1999). The results made no reference to LH or testosterone levels. The lack of gonadotropin measurement is puzzling as the data showed 12 of 30 subjects experienced testicular shrinkage, implying Leydig cell dysfunction and suppressed testosterone levels. Other studies using AAS have also shown no reference to LH or FSH levels but suppressed values are expected in each case (Bagatell et al, 1994; Behre et al, 1997; Sheffield-Moore et al, 1999; Tricker et al, 1996).
Declining, or suppressed, circulating testosterone levels as a result of either pathophysiological or induced hypogonadal conditions can have many negative consequences in males. Declining levels of testosterone have been directly linked to a progressive decrease in muscle mass (Mauras et al, 1998), loss of libido (Schiavi et al, 1991), decrease in muscular strength (Balagopal et al, 1997; Mauras et al, 1998) impotence (Rakic et al, 1997), oligospermia or azoospermia (Vermeulen & Kaufman, 1995), increase in adiposity (Mauras et al, 1998) and an increased risk of osteoporosis (Wishart et al, 1995).

While some research suggests that the hormonal axis will spontaneously return to normal shortly after cessation of testosterone administration (Knuth et al, 1989), documented cases have taken up to 2 ½ years to return to normal (Jarow & Lipshultz, 1990). This case of a 39-year old male who previously used AAS was found to have low serum testosterone levels (6nmol/L, range 14 to 28 nmol/L) 2 ½ years after his last administration of the drugs (Jarow & Lipshultz, 1990). For most men, suffering with diminished libido, impotence, depression, fatigue, muscle atrophy, and infertility for 2 ½ years is not a pleasant option. Other androgen or anabolic steroid induced cases of hypogonadotropic hypogonadism have taken 6 months (Gazvani et al, 1997; Wu et al, 1996), 8 months (Gazvani et al, 1997), 10 months (Boyadjiev et al, 2000), 12 months (Schurmeyer et al, 1984), and 18 months (Gazvani et al, 1997) to finally return to eugonadal status.
The individual use of human chorionic gonadotropin (HCG), clomiphene citrate, and tamoxifen citrate in the treatment of testicular sub-function and gonadotropin suppression, respectively, is well documented. HCG has been shown to significantly improve gonadal function in hypogonadotropic hypogonadal adult males (Barrio et al,

1999; Burgess & Calderon, 1997; Cisternino et al, 1998; D'Agata et al, 1982; 1984; Dunkel et al, 1985; Kelly et al, 1982; Ley & Leonard, 1985; Liu et al, 1988; Martikainen et al, 1986; Okuyama et al, 1986; Ulloa-Aguirre et al, 1985; Vicari et al, 1992). Studies using clomiphene citrate to induce endogenous gonadotropin production in males found significant improvements in LH and FSH values after treatment (Bjork et al, 1977; Burge et al, 1997; Guay et al, 1995; Landefeld et al, 1983; Lim & Fang, 1976; Ross et al, 1980; Spijkstra et al, 1988). Tamoxifen citrate has also been found to produce a profound increase in serum LH levels as well as improved semen and sperm quality (Gazvani et al, 1997; Krause et al, 1985; Lewis-Jones et al, 1987; Wu et al, 1996).

As HCG's effect is centralized at the Leydig cells of the testicles, clomiphene citrate and tamoxifen citrate act upon the hypothalamic-pituitary region in stimulating gonadotropin production. Tamoxifen, a nonsteroidal antiestrogen, and clomiphene citrate, a nonsteroidal ovulatory stimulant, compete with estrogen for estrogen receptor binding sites, thus eliminating excess estrogen circulation at the level of the hypothalamus and pituitary and allowing gonadotropin production to resume normally. The normal operation of both the testicular and hypothalamic-pituitary regions is crucial in returning HPGA function to normal. Returning one component of the axis to normal without concurrently returning the other would sabotage and inhibit the operation of the entire HPGaxis. It was with this understanding that HCG was eventually combined with clomiphene citrate and tamoxifen as attempted therapy to reverse gonada function in hypogonadotropic hypogonadal males.
In accordance with previous studies, each medication was used individually, and along with HCG, in initial trials. The simultaneous use of clomiphene citrate and tamoxifen was determined through preliminary use of clomiphene citrate and tamoxifen individually. It was discovered that although both clomiphene citrate and tamoxifen met with some success, when combined together they achieved a more significant increase in gonadotropin production. This clinical outcome resulted in the combination therapy of HCG, clomiphene citrate and tamoxifen.

Following is a clinical evaluation of the combined, simultaneous use of HCG, clomiphene citrate, and tamoxifen citrate as a treatment option in suppressed testosterone and gonadotropin levels in hypogonadotropic hypogonadal adult males. This observational analysis of the aforementioned treatment protocol assessed the efficacy of these medicines under non-controlled conditions.

METHODS

An observational study was done on the medical records of 5 adult male patients presenting to a clinic with induced hypogonadotropic hypogonadism. Patients were monitored and treatment recorded for the purposes of this observational study.

SUBJECTS

The medical records of five males age 27-49, mean 35.2, weighing 77-100 kg, mean 89.8 kg, with serum total testosterone levels below 240 ng/dL and serum luteinizing hormone (LH) levels below 1.5 mIU/mL were examined. Average presenting testosterone level was 98.2 ng/dL (normal= 240-827 ng/dL) while average LH level was undetectable at <1.0 mIU/mL (normal= 1.5-9.3 mIU/mL). The 5 patients had a history of AAS usage ranging from 9-60 months prior to presentation. All patients had ceased any testosterone therapy or AAS usage prior to initiation of treatment. Initial laboratory values confirmed that all patients had discontinued AAS long enough for endogenous lab values to fall below normal reference ranges. All patients were muscular in nature with an average BMI less than 27 at presentation. Table 1

presents the patient characteristics, anabolic history, and side effects upon presentation of the 5 patients.

LABORATORY STUDIES

Initial blood screening consisted of:

AST, ALT, GGT, TOTAL CHOLESTEROL, LH, FSH, TESTOSTERONE, GLUCOSE, PROLACTIN, PSA TOTAL, TSH, T3 UPTAKE, T4 TOTAL, T4 FREE, HEMOGLOBIN, HEMATOCRIT

Table 2 shows all baseline serum blood levels at presentation. Baseline blood screening excluded any form of hyperprolactinemia or hypothyroidism as causes of hypogonadism in most patients. After physician examination and history and physical evaluation, it was determined that a history of AAS usage was present and most likely the cause of the patients' hypogonadotropic hypogonadal lab values; not hyperprolactinemia or hypothyroidism.

Laboratory testing was performed by Quest Diagnostics Inc., (Houston, TX) and SmithKline Beecham Clinical Laboratories, (Houston, TX). Repeat serum LH & testosterone samples were measured by immunoassay using chiron reagant kits on an ACS-180 instrument.

METHODS

A review of patients' medical records showed a treatment intervention of (a) human chorionic gonadotropin (HCG) (Ferring Pharmaceuticals), (b) clomiphene citrate (Teva Pharmaceuticals), and (c) tamoxifen (AstraZeneca). Typical dosage of HCG consisted of 2500 units every other day for 16 days.

All HCG injections were self-administered intramuscularly. Starting dosages of clomiphene citrate and tamoxifen were 50mg and 20 mg daily, respectively. Patients started all three medications simultaneously and reported for the first follow-up blood work after completion of HCG, 16 days later. The post HCG blood analysis assessed testosterone-total response only. If testicular stimulation, i.e. testosterone production, was inadequate, additional HCG was administered at this stage of therapy rather than waiting an additional 30-45 days before the protocol completion. If the testicular response to the HCG demonstrated sufficient testicular stimulation (typically a blood serum level of >300 ng/dL), clomiphene citrate and tamoxifen were continued for 15 and 30 days, respectively. The arbitrary cut-off level of 300 ng/dL was used as a general assessment where sufficient Leydig cell stimulation was taking place even in light of artificial stimulation from HCG. A repeat blood sample was then taken at day 45 to assess hypothalamic-pituitary-gonadal axis status via luteinizing hormone and total testosterone levels. Because of the varying cessation times of the medications, the concluding blood sample was taken after a 30 and 15-day washout period of HCG and clomiphene citrate, respectively. For HPGA function to be considered normal, both LH and testosterone values had to fall within the normal reference ranges. For the purposes of patient treatment, if LH and testosterone values were still below normal limits at the conclusion of 45 days of treatment, a repeat protocol administration of HCG, clomiphene citrate, and tamoxifen was given. This protocol was repeated with every patient until LH and testosterone values reached normal ranges.

RESULTS

All five patients were considered eugonadal by normal laboratory reference ranges by the conclusion of treatment. Average serum total testosterone rose from 98.2 to 692.8 ng/dL. Average serum LH rose from <1.0 to 7.92 mIU/mL. An average of 48,974 U of HCG (five 10,000 Unit boxes), 3412.5 mg of clomiphene citrate (68.25 50mg tablets), and 968.71 mg of tamoxifen (48.44 20mg tablets) were used to treat all patients to eugonadal. Total treatment time ranged from 43-120 days. Mean

elapsed time from initiation of treatment to eugonadal was 68.6 days. Statistical analysis was performed using repeated measures ANOVA. Pre and post treatment testosterone values were significantly ($p<.001$) different as were the LH values ($p<.0008$). Table 3 demonstrates the hormone changes during the treatment period and the duration to eugonadal.

ADVERSE EVENTS

None of the study subjects had any serious or treatment-terminating effects as a result of the multi-drug protocol. No problems were noted with regards to parameters of normal urologic function or treatment causing gynecomastia. Any side effects documented at presentation were reversed by the conclusion of treatment.

DISCUSSION

This observational study demonstrates the possible efficacy of HCG, clomiphene citrate, and tamoxifen citrate in returning the HPGA to normal physiological function in adult males suffering from androgen induced hypogonadotropic hypogonadism. In the case of decreased testicular function manifested by low testosterone levels, it is of primary importance to first return the normal function of the testicular cells. The initial lack of response to HCG should not immediately be a cause for the initiation of testosterone replacement therapy, as with the current accepted therapy modality by many physicians. Blood analysis confirmed that no exogenous testosterone was administered during the treatment period, as exogenous androgens would have had a suppressive effect on endogenous gonadotropin production. Therefore, because of the corresponding normal gonadotropin and testosterone values, it is accepted that gonadotropin and testicular function were normal by the conclusion of treatment. The standard treatment of HIV-related muscle wasting, AAS therapy, may involve decades of treatment and the attendant problems with any therapy of a prolonged nature. Polycythemia vera, elevated hepatic enzymes, and prolonged negative alterations in lipid profile are a few of the dangers experienced by HIV patients administered AAS for extended periods. Of greatest concern is the increasing numbers of individuals who are currently being treated with AAS to increase muscle mass either for medicinal or recreational means without attention being given to periodically returning the HPGA to normal. With roughly 4 million men in the U.S. being considered hypogonadal (Lacayo R., 2000; Sheffield-Moore et al, 1999; Shelton DL, 2000), an estimated 200,000 men are currently receiving testosterone treatment for the condition (Shelton DL, 2000). As stated earlier, AAS are being prescribed to HIV & AIDS sufferers to combat progressive muscle loss. The Centers for Disease Control and Prevention (CDC) reported an estimated 635,000+ men diagnosed with AIDS through December 2000 while an estimated 97,700 have been reported with HIV (Centers for Disease Control, vol.12, No. 2, table 5; Centers for Disease Control, vol. 12, No. 2, table 6). In 2000 alone over 31,000 men were diagnosed with the AIDS virus (Centers for Disease Control, vol. 12, No. 2, figure 3). Between hypogonadal, AIDS, & HIV males, potentially over 900,000 men are being administered AAS therapy.

Studies recently published on patients suffering from various tissuedepleting conditions and HIV affliction (Bhasin et al, 2000; Grinspoon et al, 1998; 1999; 2000; Rabkin et al, 1999; 2000; Sattler et al, 1999; Strawford et al, 1999;1999; Van Loan et al, 1999) have not identified what should be done to restore normal endocrine status post-treatment. Considering the dosages and compounds administered in many studies, there is no question that subjects were left hypogonadal after therapy. In the cases where the periodic use of testosterone or AAS are necessary, intervention to return the HPGA to normal should be initiated as soon as possible

after the cessation of the AAS. As described herein, a possible treatment modality may be the combined regimen of HCG, clomiphene citrate, and tamoxifen. Medical history has demonstrated examples of physician-induced complications resulting from treatment. Iatrogenic hyperthyroidism (Bartsch & Scheiber, 1981) and iatrogenic Cushing's syndrome (Cihak & Beary, 1977; Kimmerle & Rolla, 1985; Smidt & Johnston, 1975; Tuel et al, 1990) are cases were administered medications or treatments provoked abnormalities in patients' normal physiology. The administration of testosterone as a treatment for hypogonadotropic hypogonadism falls into this same category of causing endocrine related abnormalities (Bhasin et al, 1996; Marynick et al, 1979; Strawford et al, 1999; Tenover, 1992). Testosterone replacement therapy has proven to be very effective in reversing the symptoms of suppressed testosterone production, but does not treat the underlying cause of the deficiency. Positive effects of testosterone treatment; i.e. improved sex drive, improved sense of well-being, lean body mass; are all transient in light of plummeting gonadotropin levels. Upon cessation of testosterone treatment patients can expect a complete reversal of positive benefits as exogenously influenced testosterone levels metabolize and decline rapidly. Further controlled studies need to be performed showing the combined effects of HCG, clomiphene citrate, and tamoxifen in returning HPGA functioning to normal. Long-term follow-up on these patients returning to normal will be necessary to ensure permanent reversal of hypogonadotropic hypogonadal conditions. In addition, studies documenting dose-response curves for pituitary inhibition and reversal due to AAS administration are critical in determining the correct dose, duration, and form of treatment that is optimal without causing permanent damage. When the need for long-term androgen use presents, using moderately supraphysiologic doses of androgens as suggested by Strawford and colleagues (1999) coupled with post-treatment HPGA restoration as demonstrated here, may be a more effective means over high-dose protocols used to offset negative alterations in lean body mass. Unfortunately current studies have yet to adequately address a standard of patient care post-androgen therapy. Because of the negative impact of the hypogonadal state on physical and mental well- being, pharmacotherapy that restores HPGA function more rapidly than current modalities would greatly benefit men with hypogonadotropic hypogonadism.

While we believe that the treatment protocol was effective in returning normal hormonal function to these men, the lack of randomization or a control group leaves room for speculation. Although cases of spontaneous return to eugonadism with no medicinal intervention have been published, these reports documented durations anywhere from 6-18 months before normal hormone status was achieved (Gazvani et al, 1997; Wu et al, 1996). If the alternative treatment modality described herein can reverse suppressed gonadotropin production and AAS associated side effects much sooner than non-treatment, further evaluation of this therapy should continue.

REFERENCES

Alen M, Rahkila P, Reinila M, Vihko R. Androgenic-Anabolic Steroid Effects on Serum Thyroid, Pituitary and Steroid Hormones in Athletes. American Journal of Sports Medicine. 1987; 15: 357-361.

Alexander GM, Swerdloff RS, Wang C, Davidson T, McDonald V, Steiner B, Hines M. April Androgen-behavior Correlations in Hypogonadal Men and Eugonadal Men. II. Cognitive Abilities. Hormones and Behavior. 1998; 33(2): 85-94.

Anderson FH, Francis RM, Faulkner K. Androgen Supplementation in Eugonadal Men with Osteoporosis: Effects of Six Months of Treatment on Bone Mineral Density and Cardiovascular Risk Factors. Bone. 1996 Feb; 18(2): 171-177.

Anderson FH, Francis RM, Peaston RT, Wastell HJ. Androgen Supplementation in

Eugonadal Men With Osteoporosis: Effects of Six Months' Treatment on Markers of Bone Formation and Resorption. Journal of Bone and Mineral Research. 1997 Mar;12(3): 472-478.
Bagatell CJ, Heiman JR, Matsumoto AM, Rivier JE, Bremner WJ. Metabolic and Behavioral Effects of High-Dose, Exogenous Testosterone in Healthy Men. Journal of Clinical Endocrinology and Metabolism. 1994 Aug; 79(2): 561-567.
Bagatell CJ, Matsumoto AM, Christensen RB, Rivier JE, Bremner WJ. Comparison of a gonadotropin releasing –hormone antagonist plus testosterone (T) versus T alone as potential male contraceptive regimens. Journal of Clinical Endocrinology and Metabolism. 1993 Aug; 77(2): 427-32.
Balagopal P, Rooyackers OE, Adey DB, Ades PA, Nair KS. Effects of Aging on In Vivo Synthesis of Skeletal Muscle Myosin Heavy-Chain and Sarcoplasmic Protein in Humans. American Journal of Physiology. 1997; 273 (4 pt 1): E790-800.
Baran DT, Bergfeld MA, Teitelbaum SL, Avioli LV. Effect of Testosterone Therapy on Bone Formation in an Osteoporotic Hypogonadal Male. Calcified Tissue Research. 1978 Dec; 26(2): 103-106.
Barrio R, de Luis D, Alonso M, Lamas A, Moreno JC. Induction of Puberty with Human Chorionic Gonadotropin and Follicle-Stimulating Hormone in Adolescent Males With Hypogonadotrophic Hypogonadism. Fertility and Sterility. 1999 Feb; 71(2): 244-248.
Bartsch G, Scheiber K. Tamoxifen Treatment in Oligozoospermia. European Urology. 1981; 7(5): 283-287.
Behre HM, Kliesch S, Leifke E, Link TM, Nieschlag E. Long-Term Effect of Testosterone Therapy on Bone Mineral Density in Hypogonadal Men. Journal of Clinical Endocrinology and Metabolism. 1997 Aug; 82(8): 2386- 2390.
Bhasin S, Storer TW, Berman N, Callegari C, Clevenger B, Phillips J, Bunnell TJ, Tricker R, Shirazi A, Casaburi R. The Effects of Supraphysiologic Doses of Testosterone on Muscle Size and Strength in Normal Men. New England Journal of Medicine. 1996 July 4; 335: 1-7.
Bhasin S, Storer TW, Berman N, Yarasheski KE, Clevenger B, Phillips J, Lee WP, Bunnell TJ, Casaburi R. Testosterone Replacement Increases Fat-Free Mass and Muscle Size in Hypogonadal Men. Journal of Clinical Endocrinology and Metabolism. 1997; 82(2): 407-413.
Bhasin S, Storer TW, Javanbakht M, Berman N, Yarasheski KE, Phillips J, Dike M, Sinha-Hikim I, Shen R, Hays RD, Beall G. Testosterone Replacement and Resistance Exercise in HIV-Infected Men With Weight Loss and Low Testosterone Levels. JAMA. 2000 Feb 9; 283(6): 763-770.
Bijlsma JWJ, Duursma SA, Thijssen JHH, Huber O. Influence of Nandrolondecanoate on the Pituitary-Gonadal Axis in Males. Acta Endocrinologica. 1982 Sep; 101: 108-112.
Bjork JT, Varma RR, Borkowf HI. Clomiphene Citrate Therapy in a Patient with Laennec's Cirrhosis. Gastroenterology. 1977 Jun; 72(6): 1308-1311.
Bonkovsky HL, Singh RH, Jafri IH, Fiellin DA, Smith GS, Simon D, Cotsonis GA, Slaker DP. A Randomized, Controlled Trial of Treatment of Alcoholic Hepatitis with Parental Nutrition and Oxandrolone. II. Short-term Effects on Nitrogen Metabolism, Metabolic Balance, and Nutrition. American Journal of Gastroenterology. 1991 Sep; 86(9): 1209-1218.
Boyadjiev NP, Georgieva KN, Massaldjieva RI, Gueorguiev SI. Reversible Hypogonadism and Azoospermia as a Result of Anabolic-Androgenic Steroid Use in a Body Builder With Personality Disorder. A Case Report. Journal of Sports Medicine and Physical Fitness. 2000 Sep; 40(3): 271-274.
Burge MR, Lanzi RA, Skarda ST, Eaton RP. Idiopathic Hypogonadotropic Hypogonadism in a Male Runner is Reversed by Clomiphene Citrate. Fertility and Sterility. 1997 April; 67(4): 783-785.

Burgess S, Calderon MD. Subcutaneous Self-Administration of Highly Purified Follicle Stimulating Hormone and Human Chorionic Gonadotrophin for the Treatment of Male Hypogonadotrophic Hypogonadism. Spanish Collaborative Group on Male Hypogonadotropic Hypogonadism. Human Reproduction. 1997 May; 12(5): 980-986.
Carani C, Zini D, Baldini A, Della Casa L, Ghizzani A, Marrama P. Effects of Androgen Treatment in Impotent Men with Normal and Low Levels of Free Testosterone. Archives of Sexual Behavior. 1990 Jun; 19(3): 223-234.
Carey PO, Howards SS, Vance ML. Transdermal Testosterone Treatment of Hypogonadal Men. Journal of Urology. 1988 Jul; 140(1): 76-79.
Centers for Disease Control. Division of HIV/AIDS Prevention. Survey Report vol. 12, No. 2. Table 6. www.cdc.gov
Centers for Disease Control. Division of HIV/AIDS Prevention. Survey Report vol. 12, No. 2. Figure 3. www.cdc.gov
Centers for Disease Control. Division of HIV/AIDS Prevention. Survey Report vol. 12, No. 2. Table 5. www.cdc.gov
Cihak RW, Beary FD. Elevated Triiodothyronine and Dextrothyroxine Levels: A Potential Cause of Iatrogenic Hyperthyroidism. Southern Medical Journal. 1977 Feb; 70(2): 256-257.
Cisternino M, Manzoni SM, Coslovich E, Autelli M. Hormonal Replacement Therapy with HCG and HU-FSH in Thalassaemic Patients Affected by Hypogonadotropic Hypogonadism. Journal of Pediatric Endocrinology and Metabolism. 1998; 11 Suppl 3: 885-890.
Clerico A, Ferdeghini M, Palombo C, Leoncini R, Del Chicca MG, Sardano G, Mariani G. Effect of Anabolic Treatment on the Serum Levels of Gonadotropins, Testosterone, Prolactin, Thyroid Hormones and Myoglobin of Male Athletes Under Physical Training. Journal of Nuclear Medicine and Allied Science. 1981 July-Sep; 25(3): 79-88.
Cook LH, Freinkel RK, Zugerman C, Levin DL, Radtke R. Iatrogenic Hyperadrenocorticism During Topical Steroid Therapy: Assessment of Systemic Effects by Metabolic Criteria. Journal of American Academy of Dermatology. 1982 Jun; 6(6): 1054-1060.
D'Agata R, Heindel JJ, Vicari E, Aliffi A, Gulizia S, Polosa P. HCG-Induced Maturation of the Seminiferous Epithelium in Hypogonadotropic Men. Hormone Research. 1984; 19(1): 23-32.
D'Agata R, Vicari E, Aliffi A, Maugeri G, Mongioi A, Gulizia S. Testicular Responsiveness to Chronic Human Chorionic Gonadotropin Administration in Hypogonadotropic Hypogonadism. Journal of Clinical Endocrinology and Metabolism. 1982 Jul; 55(1): 76-80.
Davidson JM, Camargo CA, Smith ER. Effects of Androgen on Sexual Behavior in Hypogonadal Men. Journal of Clinical Endocrinology and Metabolism. 1979 Jun; 48(6): 955-958.
Demling RH, DeSanti L. Oxandrolone, an Anabolic Steroid, Significantly Increases the Rate of Weight Gain in the Recovery Phase After Major Burns. The Journal of Trauma. 1997 Jul; 43(1): 47-51.
Dombros NV, Digenis GE, Soliman G, Oreopoulos DG. Anabolic Steroids in the Treatment of Malnourished CAPD Patients: a Retrospective Study. Peritoneal Dialysis International. 1994; 14(4): 344-347.
Donega P, Gallerani M, Vigna GB, Fellin R. Reversible Hyperthyroidism and Cardiomyopathy Caused by Consumption of Iodocasein. American Journal of Medical Science. 2000 Aug; 320(2): 148-150.
Doney K, Pepe M, Storb R, Bryant E, Anasetti C, Appelbaum FR, Buckner CD, Sanders J, Singer J, Sullivan K, et al. Immunosuppressive Therapy of Aplastic Anemia: Results of a Prospective, Randomized Trial of Antithymocyte Globulin (ATG), Methylprednisolone, and Oxymetholone to ATG, Very High-Dose Methylprednisolone,

and Oxymetholone. Blood. 1992 May 15; 79(10): 2566-2571.
Dunkel L, Perheentupa J, Sorva R. Single versus Repeated Dose Human Chorionic Gonadotropin Stimulation in the Differential Diagnosis of Hypogonadotropic Hypogonadism. Journal of Clinical Endocrinology and Metabolism. 1985 Feb; 60(2): 333-337.
Evans RP and Amerson AB. 1974 Androgens and Erythropoiesis. Journal of Clinical Pharmacology. 1974; 14: 94-101.
Gascon A, Belvis JJ, Berisa F, Iglesias E, Estopinan V, Terul JL. Nandrolone Decanoate is a Good Alternative for the Treatment of Anemia in Elderly Male Patients on Hemodialysis. Geriatric Nephrol Urol. 1999; 9(2): 67-72.
Gazvani MR, Buckett W, Luckas MJM, Aird IA, Hipkin LJ, Lewis-Jones DI. Conservative management of azoospermia following steroid abuse. Human Reproduction. 1997; 12(8): 1706-1708.
Grinspoon C, Corcoran C, Askari H, Schoenfeld D, Wolf L, Burrows B, Walsh M, Hayden D, Parlman K, Anderson E, Basgoz N, Klibanski A. Effects of Androgen Administration in Men With the AIDS Wasting Syndrome. A Randomized, Double-Blind, Placebo-Controled Trial. Annals of Internal Medicine. 1998 July 1; 129(1): 18-26.
Grinspoon S, Corcoran C, Anderson E, Hubbard J, Stanley T, Basgoz N, Klibanski A. Sustained Anabolic Effects of Long-Term Androgen Administration in Men With AIDS Wasting. Clinical Infectious Diseases. 1999 Mar; 28(3): 634-636.
Grinspoon S, Corcoran C, Parlman K, Costello M, Rosenthal D, Anderson E, Stanley T, Schoenfeld D, Burrows B, Hayden D, Basgoz N, Klibanski A. Effects of Testosterone and Progressive Resistance Training in Eugonadal Men With AIDS Wasting. A Randomized, Controlled Trial. Annals of Internal Medicine. 2000 Sep 5; 133(5): 348-355.
Grinspoon S, Corcoran C, Stanley T, Baaj A, Basgoz N, Klibanski A. Effects of Hypogonadism and Testosterone Administration on Depression Indices in HIV-Infected Men. Journal of Clinical Endocrinology and Metabolism. 2000 Jan; 85(1): 60-5.
Grubb SR, Cantley LK, Jones DL, Carter WH. Iatrogenic Cushing's Syndrome After Intrapericardial Corticosteroid Therapy. Annals of Internal Medicine. 1981 Dec; 95(6): 706-707.
Guay AT, Bansal S, Heatley GJ. Effect of Raising Endogenous Testosterone Levels in Impotent Men With Secondary Hypogonadism: Double Blind Placebo-Controlled Trial with Clomiphene Citrate. Journal of Clinical Endocrinology and Metabolism. 1995 Dec; 80(12): 3546-3552.
Hamdy RC, Moore SW, Whalen KE, Landy C. Nandrolone Decanoate for Men with Osteoporosis. American Journal of Therapeutics. 1998 Mar; 5(2): 89-95.
Hankins JH, Heise CM, Cowan RJ. Iatrogenic Hyperthyroidism Secondary to Dextrothyroxine Administration. Clinical Nuclear Medicine. 1984 Jan; 9(1): 17-19.
Hervey GR, Knibbs AV, Burkinshaw L, Morgan DB, Jones PRM, Chettle DR, Vartsky D. Effects of Methandienone on the Performance and Body
Composition of Men Undergoing Athletic Training. Clinical Science. 1981; 60(4): 457-461.
Hobbs CJ, Jones RE, Plymate SR. Nandrolone, a 19-Nortestosterone, Enhances Insulin-Independent Glucose Uptake in Normal Men. Journal of Clinical Endocrinology and Metabolism. 1996 Apr; 81(4): 1582-1585.
Hurtado R, Sosa R, Majluf A, Labardini JR. Refractory Anaemia (RA) Type I FAB Treated With Oxymetholone (OXY): Long-Term Results. British Journal of Haematology. 1993 Sep; 85(1): 235-236.
Jarow JP and Lipshultz LI. Anabolic Steroid-Induced Hypogonadotropic Hypogonadism. American Journal of Sports Medicine. 1990 Jul-Aug; 18(4): 429-431.

Kelly WF, Kjeld JM, Mashiter K, Joplin GF. Reassessment of the Human Chorionic Gonadotropin Stimulation Test in Hypogonadal Males. Archives of Andrology. 1982 Feb; 8(1): 53-59.
Kimmerle R, Rolla AR. Iatrogenic Cushing's Syndrome Due to Dexamethasone Nasal Drops. American Journal of Medicine. 1985 Oct;
79(4): 535-537.
Klepsch I, Maicanescu-Georgescu M, Marinescu L. Clinical and Hormonal Effects of Testosterone Undecanoate (TU) in Male Sexual Impotence. Endocrinologie. 1982 Oct-Dec; 20(4): 289-293.
Kley HK, Stremmel W, Kley JB, Schaghecke R. Testosterone Treatment of Men With Idiopathic Hemochromatosis. Clinical Investigation. 1992 Jul; 70(7): 566-572.
Krause W, Hubner HM, Wichmann U. Treatment of Oligozoospermia by Tamoxifen: No Evidence for Direct Testicular Action. Andrologia. 1985 May- June; 17(3): 285-290.
Lacayo R. Are You Man Enough? Time Magazine. 2000 April 24; 155: 58-64.
Landefeld CS, Schambelan M, Kaplan SL, Embury SH. Clomiphene- Responsive Hypogonadism in Sickle Cell Anemia. Annals of Internal Medicine. 1983 Oct; 99(4): 480-483.
Lawrence IG, Price DE, Howlett TA, Harris KP, Feehally J, Walls J. Correcting Impotence in the Male Dialysis Patient: Experience With Testosterone Replacement and Vacuum Tumescence Therapy. American Journal of Kidney Disorders. 1998 Feb; 31(2): 313-319.
Lewis-Jones DI, Lynch RV, Machin DC, Desmond AD. Improvement in Semen Quality in Infertile Males After Treatment with Tamoxifen. Andrologia. 1987 Jan-Feb; 19(1): 86-90.
Ley SB, Leonard JM. Male Hypogonadotropic Hypogonadism: Factors Influencing Response to Human Chorionic Gonadotropin and Human Menopausal Gonadotropin, Including Prior Exogenous Androgens. Journal of Clinical Endocrinology and Metabolism. 1985 Oct; 61(4): 746-752.
Lim VS, Fang VS. Restoration of Plasma Testosterone Levels in Uremic Men With Clomiphene Citrate. Journal of Clinical Endocrinology and Metabolism. 1976 Dec; 43(6): 1370-1377.
Liu L, Banks SM, Barnes KM, Sherins RJ. Two-year Comparison of Testicular Responses to Pulsatile Gonadotropin-Releasing Hormone and Exogenous Gonadotropins from the Inception of Therapy in Men with Isolated Hypogonadotropic Hypogonadism. Journal of Clinical Endocrinology and Metabolism. 1988 Dec; 67(6): 1140-1145.
Martikainen H, Alen M, Rahkila P, Vihko R. Testicular Responsiveness to Human Chorionic Gonadotrophin During Transient Hypogonadotrophic Hypogonadism Induced by Androgenic/Anabolic Steroids in Power Athletes. Journal of Steroid Biochemistry. 1986 July; 25(1): 109-112.
Marynick SP, Loriaux DL, Sherins RJ, Pita JC Jr, Lipsett MB. 1979 Sep Evidence that Testosterone can Suppress Pituitary Gonadotropin Secretion Independently of Peripheral Aromatization. Journal of Clinical Endocrinology and Metabolism. 49(3): 396-398.
Mauras N, Hayes V, Welch S, Rini A, Helgeson K, Dokler M, Veldhuis JD, Urban RJ. Testosterone Deficiency in Young Men: Marked Alterations in Whole Body Protein Kinetics, Strength, and Adiposity. Journal of Clinical Endocrinology and Metabolism. 1998; 83: 1886-1892.
McClure RD, Oses R, Ernest ML. Mar Hypogonadal Impotence Treated by Transdermal Testosterone. Urology. 1991; 37(3): 224-228.
Mendenhall CL, Moritz TE, Roselle GA, Morgan TR, Nemchausky BA, Tamburro CH, Schiff ER, McClain CJ, Marsano LS, Allen JI. A Study of Oral Nutritional Support with

Oxandrolone in Malnourished Patients with Alcoholic Hepatitis: Results of a Department of Veterans Affairs Cooperative Study. Hepatology. 1993 April; 17(4): 564-576.
Morales A, Johnston B, Heaton JP, Lundie M. Testosterone Supplementation for Hypogonadal Impotence: Assessment of Biochemical Measures and Therapeutic Outcomes. Journal of Urology. 1997 Mar; 157(3): 849-854.
Morales A, Johnston B, Heaton JW, Clark A. Oral Androgens in the Treatment of Hypogonadal Impotent Men. Journal of Urology. 1994 Oct; 152(4): 115-1118.
Nankin HR, Lin T, Osterman J. Chronic Testosterone Cypionate Therapy in Men with Secondary Impotence. Fertility and Sterility. 1986 Aug; 46(2): 300- 307.
Noci I, Chelo E, Saltarelli O, Donati CG, Scarselli G. Tamoxifen and Oligospermia. Archives of Andrology. 1985; 15(1): 83-88.
Okuyama A, Nakamura M, Namiki M, Aono T, Matsumoto K, Utsunomiya M, Yoshioka T, Itoh H, Itatani H, Mizutani S, et al. Testicular Responsiveness to Long-Term Administration of HCG and HMG in Patients with Hypogonadotrophic Hypogonadism. Hormone Research. 1986; 23(1): 21-30.
Prakasam G, Yeh JK, Chen MM, Castro-Magana M, Liang CT, Aloia JF. Effects of Growth Hormone and Testosterone on Cortical Bone Formation and Bone Density in Aged Orchiectomized Rats. Bone. 1999 May; 24(5): 491-497.
Rabkin JG, Wagner GJ, Rabkin R. A Double-Blind, Placebo-Controlled Trial of Testosterone Therapy for HIV-Positive Men With Hypogonadal Symptoms. Archives of General Psychiatry. 2000 Feb; 57(2): 141-147.
Rabkin JG, Wagner GJ, Rabkin R. Testosterone Therapy for Human Immunodeficiency Virus-Positive Men With and Without Hypogonadism. Journal of Clinical Psychopharmacology. 1999 Feb; 19(1): 19-27.
Rakic Z, Starcevic V, Starcevic VP, Marinkovic J. Testosterone Treatment in Men with Erectile Disorder and Low Levels of Total Testosterone in Serum. Archives of Sexual Behavior. 1997 Oct; 26(5): 495-504.
Ross LS, Kandel GL, Prinz LM, Auletta F. Clomiphene Treatment of the Idiopathic Hypofertile Male: High-Dose, Alternate-Day Therapy. Fertility and Sterility. 1980 Jun; 33(6): 618-623.
Sattler FR, Jaque SV, Schroeder ET, Olson C, Dube MP, Martinez C, Briggs W, Horton R, Azen S. Effects of Pharmacological Doses of Nandrolone Decanoate and Progressive Resistance Training in Immunodeficient Patients Infected with Human Immunodeficiency Virus. Journal of Clinical Endocrinology and Metabolism. 1999; 84(4): 1268-1276.
Schiavi RC, Schreiner-Engel P, White D, Mandeli J. The Relationship Between Pituitary-Gonadal Function and Sexual Behavior in Healthy Aging Men. Psychosomatic Medicine. 1991 Jul-Aug; 53 (4): 363-374.
Schiavi RC, White D, Mandeli J, Levine AC. Effect of Testosterone Administration on Sexual Behavior and Mood in Men with Erectile Dysfunction. Archives of Sexual Behavior. 1997 Jun; 26 (3): 231-241.
Schurmeyer T, Knuth UA, Belkien E, et al. Reversible Azoospermia Induced by the Anabolic Steroid 19-Nortestosterone. Lancet. 1984; I: 417-420. Sheffield-Moore M, Urban RJ, Wolf SE, Jiang J, Catlin DH, Herndon DN Wolfe RR, Ferrando AA. Short-term Oxandrolone Administration Stimulates Net Muscle Protein Synthesis in Young Men. Journal of Clinical Endocrinology and Metabolism. 1999; 84: 2705-2711.
Shelton DL. 2000 Aug 7 Testosterone Therapy Hype May Be Creating False Hopes. http://www.ama-assn.org/sci-pubs/am...oo/hll20807.htm Sih R, Morley JE, Kaiser FE, Perry III HM, Patrick P, Ross C. Testosterone Replacement in Older Hypogonadal Men: a 12-Month Randomized Controlled Trial. Journal of Clinical Endocrinology and Metabolism. 1997; 82: 1661- 1667.
Smidt KP, Johnston E. Undetected Iatrogenic Hypothyroidism: A Late Complication of

Radio-Iodine Therapy. New Zealand Medical Journal. 1975 Apr 9; 81: 325-328.
Snyder PJ, Peachey H, Berlin JA, Hannoush P, Haddad G, Dlewati A, Santanna J, Loh L, Lenrow DA, Holmes JH, Kapoor SC, Atkinson LE, Strom BL. Effects of Testosterone Replacement in Hypogonadal Men. Journal of Clinical Endocrinology and Metabolism. 2000 Aug; 85(8): 2670-2677. Spijkstra JJ, Spinder T, Gooren L, van Kessel H. Divergent Effects of the Antiestrogen Tamoxifen and of Estrogens on Luteinizing Hormone (LH) Pulse Frequency, but not on Basal LH Levels and LH Pulse Amplitude in Men. Journal of Clinical Endocrinology and Metabolism. 1988 Feb; 66(2): 355-360.
Starr C, Taggart R. Integration and Contol: Endocrine Systems. In: Star C, Taggart R, eds. Biology-The Unity and Diversity of Life. Belmont, California: Wadsworth Publishing Company,1992: 587-590.
Strawford A, Barbieri T, Neese R, Van Loan M, Christiansen M, Hoh R, Sathyan G, Skowronski R, King J, Hellerstein M. Effects of Nandrolone Decanoate Therapy in Borderline Hypogonadal Men With HIV-Associated
Weight Loss. Journal of Acquired Immune Deficiency Syndromes and Human Retrovirology. 1999 Feb 1; 20(2): 137-146.
Strawford A, Barbieri T, Van Loan M, Parks E, Catlin D, Barton N, Neese R, Christiansen M, King J, Hellerstein MK. Resistance Exercise and Supraphysiologic Androgen Therapy in Eugonadal Men With HIV-Related Weight Loss: a Randomized Controlled Trial. JAMA. 1999 April 14; 281(14): 1282-1290.
Stricker RB and Shuman MA. Aplastic Anaemia Complicating Systemic Lupus Erythematosus: Response to Androgens in Two Patients. American Journal of Hematology. 1984 Aug; 17(2): 193-201.
Stromme SB, Meen HD, Aakvaag A. Effects of an Androgenic-Anabolic Steroid on Strength Development and Plasma Testosterone Levels in Normal Males. Medicine and Science in Sports and Exercise. 1974; 6: 203-208.
Tenover JS. Effects of Testosterone Supplementation in the Aging Male. Journal of Clinical Endocrinology and Metabolism. 1992; 75: 1092-1098.
Tomoda H. Effect of Oxymetholone on Left Ventricular Dimensions in Heart Failure Secondary to Idiopathic Dilated Cardiomyopathy or to Mitral or Aortic Regurgitation. American Journal of Cardiology. 1999 Jan 1; 83(1): 123-5.
Tricker R, Casaburi R, Storer TW, Clevenger B, Berman N, Shirazi A, Bhasin S. The Effects of Supraphysiological Doses of Testosterone on Angry Behavior in Healthy Eugonadal Men- A Clinical Research Center Study. Journal of Clinical Endocrinology and Metabolism. 1996 Oct; 81(10): 3754- 3758.
Tuel SM, Meythaler JM, Cross LL. Cushing's Syndrome from Epidural Methylprednisolone. Pain. 1990 Jan; 40(1): 81-84.
Ulloa-Aguirre A, Mendez JP, Diaz-Sanchez V, Altamirano A, Perez-Palacios G. Self-priming Effect of Luteinizing Hormone-Human Chorionic Gonadotropin (HCG) Upon the Biphasic Testicular Response to Exogenous HCG. I. Serum Testosterone Profile. Journal of Clinical Endocrinology and Metabolism. 1985 Nov; 61(5): 926-932.
Valayer-Chaleat E, Calmels P, Giraux P, Fayolle-Minon I. Femoral Fracture and Iatrogenic Hyperthyroidism in Spinal Cord Injury. Spinal Cord. 1998 Aug; 36(8): 593-595.
Van Loan MD, Strawford A, Jacob M, Hellerstein M. Monitoring Changes in Fat-Free Mass in HIV-Positive Men With Hypotestosteronemia and AIDS
Wasting Syndrome Treated With Gonadal Hormone Replacement Therapy. AIDS. 1999 Feb 4; 13(2): 241-248.
Vermeulen A, Kaufman JM. Ageing of the Hypothalamo-Pituitary-Testicular Axis in Men. Hormonal Research. 1995; 43 (1-3): 25-28.
Vicari E, Mongioi A, Calogero AE, Moncada ML, Sidoti G, Polosa P, D'Agata R. Therapy With Human Chorionic Gonadotrophin Alone Induces Spermatogenesis in Men With Isolated Hypogonadotrophic Hypogonadism- Long-Term Follow-Up. International

Journal of Andrology. 1992 Aug; 15(4): 320-329.
Wagner GJ, Rabkin JG. Testosterone Therapy for Clinical Symptoms of Hypogonadism in Eugonadal Men With AIDS. International Journal of STD and AIDS. 1998 Jan; 9(1): 41-44.
Wang C, Swedloff RS, Iranmanesh A, Dobs A, Snyder PJ, Cunningham G, Matsumoto AM, Weber T, Berman N. Transdermal Testosterone Gel Improves Sexual Function, Mood, Muscle Strength, and Body Composition Parameters in Hypogonadal Men. Testosterone Gel Study Group. Journal of Clinical Endocrinology and Metabolism. 2000 Aug; 85(8): 2839-2853.
Watsky JG, Koeniger MA. Prevalence of Iatrogenic Hyperthyroidism in a Community Hospital. Journal of the American Board of Family Practice. 1998 May-June; 11(3): 175-179.
Wishart JM, Need AG, Horowitz M, Morris HA, Nordin BE. Effect of Age on Bone Density and Bone Turnover in Men. Clinical Endocrinology. 1995; 42: 141-146.
Wu FCW, Farley TMM, Peregoudov A, Waites GMH. Effects of testosterone enanthate in normal men: experience from a multicenter contraceptive efficacy study. Fertility and Sterility. 1996 Mar; 65(3): 626-636.
> ABBREVIATIONS
AAS Anabolic-Androgenic Steroids
AIDS Acquired Immunodeficiency Virus
ALT Alanine aminotransferase
AST Aspartate aminotransferase
BMI Body Mass Index
dL deciliter
FSH Follicle Stimulating Hormone
GGT Gamma-glutamyl transferase
GnRH Gonadotropin Releasing Hormone
HCG Human Chorionic Gonadotropin
HIV Human Immunodeficiency Virus
HPGA Hypothalamic Pituitary Gonadal Axis
kg kilogram
LH Luteinizing Hormone
mg milligram
mIU mili International Units
mL milliliter
ng nanogram
PSA Prostate Specific Antigen
T3 Triiodothyronine
T4 Thyroxine
TSH Thyroid Stimulating Hormone