HIGH-DOSE TESTOSTERONE REPLACEMENT THERAPY

In 1997, I first began to prescribe testosterone replacement therapy (TRT) for prostate cancer (CaP) patients who wanted to try to improve their quality of life. Before prescribing TRT for men with CaP, I had done extensive research and medical literature reviews. After a careful and thorough evaluation of all available information, I concluded that testosterone (T) might have some beneficial effects for select patients with CaP. This view led to some rather nasty and unflattering criticisms from a number of sources.

At the June 2004 American Society of Clinical Oncology meeting, Abstract #4560 is reported in Proceedings; ASCO; Volume 23; 2004; by Morris, M.J., et al., from Memorial-Sloan Kettering Hospital in New York. The authors found in preclinical studies, growth of selected prostate cancer cells following prolonged androgen blockade can be repressed by reintroducing high-dose testosterone. Their clinical study involved using TRT to treat patients with metastatic hormone refractory prostate cancer. All patients had castrate testosterone levels. Men were treated with three times the standard dosing of testosterone using Testoderm patches or AndroGel lotion. The study’s intent was to maximize serum testosterone levels. Twelve patients were treated. No patient had to be taken off study for tumor flare. “Therapy was well tolerated.” The median total testosterone level achieved by all patients during week 1 was 409, range 95–908. Median treatment duration for the third cohort of patients treated was 59 days, with a range of 27–124 days. One patient had his PSA decline by greater than 50%. Two additional patients had stable PSA’s. The conclusion of the study was “administration of testosterone to patients with advanced prostate cancer for one week, four weeks, or until disease progression is safe and not limited by tumor flare. The authors further stated that “PSA declines were seen, but were rare and short-lived, and suggested that combination therapy may be more productive.” This study is especially noteworthy because it did not conclude that TRT was contraindicated. Their suggestion is to consider using TRT in combination therapy. Compassionate Oncology has been using combination therapy for their TRT patients since 1997.

As Jackie Gleason would say, “How sweet it is.” Finally other doctors are questioning and challenging the prejudice that you must never give testosterone to anyone with prostate cancer.
Patients with metastatic hormone refractory prostate cancer (HRPC) in relapse require more than just high-dose testosterone in order to have any significant chance for a beneficial and sustained response. Some of our HRPC patients have been able to remain on high-dose TRT for years. I strongly believe that most patients require much higher levels of testosterone than achieved in the 2004 ASCO Abstract #4562. I believe the authors used far too low a dose of testosterone. And, they did not use simultaneous 5-alpha-reductase inhibitors. All of our patients on high-dose TRT are treated with Avodart with or without daily Proscar. Most take both. All of our metastatic, hormone refractory prostate cancer patients are also treated with my prostate cancer antiangiogenic cocktail (AAC). Our target testosterone level is 1,200-2,500+, which is a much higher level than this study utilized. We very rarely lower testosterone doses, even with testosterone levels of 2,500-4,000, as long as the PSA is stable or declining. I believe that our very different approach for treating metastatic hormone refractory prostate cancer patients explains our superior response rate and duration of response.

In my opinion, the only indication for using testosterone replacement therapy in any patient with prostate cancer (CaP) is to improve their quality of life, not to treat CaP. TRT should only be given to carefully screened patients after they have been treated with (preferably triple) hormone blockade or some type of radical local therapy. I believe it is first necessary to debulk the body of most prostate cancer cells before you should consider TRT. Triple hormone blockade (THB) can be quite effective at debulking prostate cancer.

Hormone blockade is not usually believed to be capable of killing all of the prostate cancer cells present in a patient. Since 80% of men in their 80's have CaP but only 2-3% die from it, cure is not necessary for the vast majority of men. Most of us die with CaP, not from it. There are some prostate cancer experts who believe hormone blockade may cure some patients. I never made this claim, but some studies support this possibility.

Essentially all of the patients initially treated by me with TRT were in remission from their prostate cancer, and also had low testosterone levels. Each patient received a comprehensive discussion regarding TRT and CaP, including all risks, benefits, and alternatives. Each patient was aware that 99.9+% of physicians would advise against using testosterone
replacement therapy in anyone with CaP. Now that the June 2004 ASCO Abstract #4560 has been published, perhaps more doctors will prescribe TRT. Patients are aware that conventional teachings claim that testosterone would cause their prostate cancer to grow, metastasize, and even cause their death.

Currently, many experts use intermittent androgen blockade (IAB) to treat prostate cancer patients, even for those with metastatic disease. More patients are now demanding that they be treated with IAB. At the American Urological Association (AUA) May 2004 annual meeting, Abstract #1458 reported results from a prospective multinational Phase III trial which compared IAB to continuous hormone blockade for men with a rising PSA after radical prostatectomy. Estimated progression free survival (PFS) on IAB was 1,234 days versus 1,010 days for continuous HB. Quality of life is clearly improved during off hormone blockade cycles and many studies are reporting intermittent androgen blockade appears to be at least as effective as continuous hormone blockade. Some of us believe strongly that IAB prolongs survival compared to continuous HB. If testosterone were harmful for all men with prostate cancer, then intermittent hormone blockade should never be allowed. When a man goes off hormone blockade, his own endogenous testosterone recovers. In our series of 185 men treated with triple hormone blockade® followed by finasteride maintenance® as sole therapy for clinically localized prostate cancer, testosterone levels average about 530 after THB. Prior to THB, the average T level was 350. Proscar raises T levels. Is a testosterone of 530 better or worse than a T of 1,600? No one has ever studied this.

As of May 2004, Compassionate Oncology has used high-dose testosterone replacement therapy to treat more than 100, but fewer than 200 prostate cancer patients. We have used TRT on patients with all stages of CaP. In the lab, increasing levels of testosterone added to a prostate cancer cell line (LNCaP) results in a bell-shaped growth curve. High levels of T inhibit the growth of CaP cells, and the higher the level, the greater the inhibitory effect, whereas low levels of T stimulate LNCaP cells to grow. LNCaP refers to a cell line grown from the lymph node (hence LN) of a patient with CaP, therefore LNCaP (lymph node from cancer of the prostate). We have all been indoctrinated (?)brainwashed?) to accept with absolute certainty that giving T to any patient previously treated for CaP is like throwing gasoline on a fire. We are told that T is always contraindicated for all men with CaP
(that means it is the exact wrong thing to do and will essentially always cause harm). The package inserts for every TRT product tell you that patients with CaP must not ever use T. Period. No exceptions.

An article in Breast Cancer Research and Treatment, Volume 67, pages 111-116, 2001 by Per Lonning, et al. They report on prior studies showing that estrogen in high concentrations inhibits the growth of MCF-7 cells in vitro, and induces apoptosis in estrogen-deprived cells. The MCF-7 cell line is a breast cancer cell line, analogous to the LNCaP prostate cancer cell line. Both cell lines show a bell-shaped growth curve when exposed to increasing doses of hormone. The MCF-7 cells, when grown in a medium containing very low concentrations of estrogen, achieve maximum growth stimulation. A CaP patient on hormone blockade has low levels of testosterone (rather than a zero level), in spite of being treated with every known hormone-blocking agent. These low testosterone levels stimulate prostate cancer cells to grow. The higher the level of estrogen in the breast cancer cell line, and the higher the level of testosterone in the prostate cancer cell line, the greater the inhibition of cancer growth. These identical responses in breast and CaP are not a coincidence (in my opinion). Instead, we believe this reflects some innate biological characteristic or behavior that results in these inhibitory responses to high levels of T and/or estrogen for hormone-dependent cancers. I summarize this information in my May 2003 video and my March 2004 video, which can be ordered by calling our office at (310) 229-3555).

As I read, studied, and reviewed the medical literature, I found particularly relevant some articles that were published from 1947 through the 1960's. I reached the conclusion that the statement that TRT was always contraindicated was nothing more than opinion and/or prejudice. Apparently, no one challenged this opinion. Here was an example in medicine where we are taught to do things one way because that is how it has always been done. You do not and may not question or challenge this view. We are never told this view represents an opinion rather than proven scientific fact. Since androgen blockade initially results in remission for most men with metastatic CaP, it was only logical to conclude that adding T must be harmful, and therefore contraindicated. However, in medicine, what seems logical may not be factual -- or, what is logical at one stage of CaP treatment may not be logical at a different time or stage of disease. In medicine there are many
“paradoxical” responses. Later, when new insights are learned, what seemed paradoxical is often reclassified as logical or even obvious.

Two articles published in 1981 and 1982 by Fowler and Whitmore are the major medical publications that convinced urologists and others that T was always harmful to men with CaP. Careful scrutiny of these “landmark” articles reveals that the authors never stated T was contraindicated. They concluded that T should be “used with caution” in men with metastatic CaP. We absolutely agree with their conclusion. However, we disagree with the legacy that followed these articles and has left us with, until now, the unchallenged categorical dictum to avoid T for any CaP patient at any time and at any stage of illness.

These two articles cite prior medical publications as references to support their conclusion to avoid TRT. However, when I studied these referenced articles, I found that many of these articles showed testosterone replacement therapy could induce remissions in some men with metastatic CaP, even if they were “terminal,” hormone refractory, and/or bedridden. These responses were always described using adjectives like “unexpected” or “paradoxical” or against “conventional wisdom.” These articles usually concluded that studies should be done to try to understand the relationship between testosterone and prostate cancer. One such article was published in 1947 in a urology journal, but today we still know very little about the relationship between T and CaP. Details of these studies along with the exact medical references can be found in my video lectures from May 2003 and March 2004. The two videos are complementary to each other rather than repetitive.

My impression from reviewing TRT medical literature is that following the publication of the two Fowler and Whitmore articles, physicians concluded the relationship between T and CaP was so obvious, known, and certain that it must not be challenged. The articles reporting beneficial results for some patients treated with testosterone replacement therapy seemed to have been quickly forgotten. Surely I was not the first doctor who questioned how T and CaP interact. Is it possible that this relationship has not been exhaustively studied with confirmed, reproducible results? The totally unexpected answer is that it had not. Even today, testosterone and prostate cancer cell interactions, especially in patients previously treated for prostate cancer, remain uninvestigated and in need of immediate research and understanding.
Treatment interventions for a substantial number and variety of illnesses often resemble a pendulum. When I was a medical student, it was contraindicated to give nitroglycerine (NTG) to a patient with a heart attack. We were taught that although it helped angina, it would decrease the circulation to the coronary arteries, and could increase the workload on the heart. We now know it improves coronary artery circulation, and reduces the workload on the heart. Today, we often give NTG intravenously. As we learn and understand more, our standards of practice change. The first time I heard that you could treat coronary artery disease with diet and exercise (Pritikin type program), I was probably amused. Many cardiologists were angry and felt it their responsibility to discredit and ostracize any doctor who believed in this treatment. Everyone “knew” the only way to treat coronary artery disease was with surgery. The Pritikin treatment approach was strange and must have threatened or challenged conventional “wisdom.” Very few of us can change our opinions regarding how we treat various diseases until our medical literature literally forces us to adopt a new approach. Too many of us (doctors) prefer to criticize or ridicule a new or novel approach.

Dr. Robert Atkins died before his diet became the method used by tens of millions of Americans. Doctors dismissed and ridiculed his approach. In the May 18, 2004, Annals of Internal Medicine, an editorial by Dr. Walter Willet from the Harvard School of Public Health concludes: “We can no longer dismiss very-low-carbohydrate diets....Dr. Atkins deserves credit for observing that many persons can control their weight by greatly reducing carbohydrate intake.” His editorial also notes that the Atkins Diet markedly decreased triglyceride levels with some favorable effects on HDL, LDL, and total cholesterol levels, even though dieters consumed unlimited amounts of fats on the Atkins Diet.

We have observed that high-dose TRT can lower PSA levels in a significant percentage of our patients. This observation is fact, not opinion. Some men have even been treated with intermittent cycles of high-dose TRT. The improvement in quality of life when T levels are over 1,200-1,800 is truly astonishing. We have a list of volunteer patients who have agreed to speak to men interested in this.

Back to the two articles (by Fowler and Whitmore). These articles describe the same population of patients, but reported
their findings in two different medical journals. The method utilized by the authors was a retrospective chart review (this type of study is always subject to reviewer bias since the author is hoping to find evidence to support his hypothesis or bias). The men were treated between 1949 and 1967. The articles were not published until 1981 and 1982. This means that all of the patients were treated from 14 to 23 years prior to the publication of these “landmark” articles. They relied on the patients’ medical records to provide all necessary and relevant information. It is highly unlikely that this approach could identify and address all the necessary and pertinent details. During much of that time period, all medical record entries were handwritten, rather than dictated. The admission history and physical, as well as the doctor’s discharge summary, had to be handwritten by the doctor. Perhaps some important information was inadvertently omitted from the medical records because of time constraints and/or a doctor’s tired writing hand. Neither patients nor family members were interviewed. Remember the doctors did not know that 15 years later someone would review these charts and try to reach meaningful and appropriate conclusions that when published would provide the major rationale for limiting treatment options for treating prostate cancer for the next 30-40 plus years.

The best medical studies are prospectively randomized and double blinded. This means that neither the doctor nor the patient knows what treatment they are receiving and all patients are prospectively randomized to treatment A or treatment B. All known prognostic factors must be used to randomize patients equally to each treatment. Otherwise, a better outcome on one arm could be due to better baseline prognostic factors rather than the treatment. The Fowler studies were retrospective, not prospective. Patients were not treated uniformly; some got oral testosterone; some shots; some received testosterone every two to three days, others at longer intervals, and very importantly, testosterone levels were not even measured. In spite of these major shortcomings, these articles resulted in setting unchallenged medical standards for treating CaP. Surely we should question whether new insights and/or knowledge have been identified some 22+ years after these articles were published. The PSA test did not even become commercially available until 1986.

If you remove the men in these two studies who had progressive, metastatic, hormone refractory CaP in relapse, nine out of 18
of the remaining patients stable or improved when treated with testosterone replacement therapy. One of these men did not progress for 420 days, even though all of them had metastatic CaP. Four patients were previously untreated; the rest were on hormone blockade. One man with metastatic, previously untreated prostate cancer remained on T for 310 days. Only 36% of men who appeared to be in remission on hormone blockade experienced an unfavorable response to TRT within 30 days of starting treatment, and no men had any significant negative reaction to TRT. Five men remained on T for 120 to 420 days. Four others were on T for 50 to 70 days. Thus, nine out of 18 men who were not clinically progressing with hormone refractory prostate cancer at the time TRT was started were able to enjoy benefits from testosterone replacement therapy.

In the Fowler and Whitmore articles, the definition of favorable responses to TRT include improved appetite, weight gain, improved overall sense of well-being, and/or resolution of hot flashes. Many men reported being able to be sexually active on TRT. These responses improve the patient’s quality of life. Other articles cited by these authors report resolution of bone pain for some patients when they were treated with testosterone replacement therapy after progressing on estrogen. These patients enjoyed “paradoxical” benefit from TRT. The articles that are quoted as confirming the belief you should never give TRT to any patient with CaP actually show that some patients improved on TRT.

For the Fowler and Whitmore series of men who progressed while on TRT, the authors were able to identify 33 men whose response to testosterone withdrawal could be evaluated after their TRT was stopped. There was prompt regression of all subjective symptoms for all but two of these 33 patients. All elevated prostatic acid phosphatase levels (an earlier blood test similar to PSA) reverted to pretreatment levels, usually within days.

Although we never recommend testosterone replacement therapy as sole therapy for men with metastatic hormone refractory prostate cancer (HRPC) in relapse, we do treat many HRPC patients with my prostate cancer antiangiogenic cocktail (PCAAC), and with concurrent TRT. (You can request a copy of my video lecture on PCAAC for details of AAC.) There are approximately eight different medicines in the cocktail. Over time, some medicines and doses change. Men with HRPC consider themselves “sentenced.” They believe they must remain on
hormone blockade for the rest of their lives, and suffer from the symptoms related to androgen deprivation syndrome. Fowler and Whitmore reported that 33 out of 34 men with metastatic HRPC in relapse, when treated with TRT, had an unfavorable response. They reported four deaths (only one of which appears to me to be clearly documented and clearly caused by TRT). One other patient had a horrible complication when he developed permanent paralysis. This latter patient presented with diffuse incapacitating bone pain before TRT. Clearly men with bone pain from metastatic HRPC should never be treated with TRT.

For men with hormone refractory prostate cancer, we first use our most effective hormone blockade protocol (typically including ketoconazole or aminoglutethimide), and add in 15 doses of weekly low-dose Taxotere/Emcyt/carboplatinum chemotherapy (very well tolerated). We switch to AAC after the 15 doses of chemotherapy have been administered. This low-dose chemotherapy protocol that Compassionate Oncology developed and pioneered does not cause nausea or vomiting; only causes significant and always reversible hair loss in about one out of seven men, and amazes our patients at how well they tolerate it. We have a list of patient volunteers who are available to discuss their experiences on chemotherapy treatment. Some of these HRPC men are later able to be successfully treated with testosterone replacement therapy. These patients must be carefully selected and must continue on AAC. We have a number of patients in this category who have been on TRT for many years and have testosterone levels of 1,200 to 3,000. Their quality of life is superb and markedly improved from prior to hormone blockade.

Do not try to be your own doctor and/or try to convince your doctor to prescribe TRT for you. There is a risk of permanent paralysis or even death. The men who have done best have their T levels very quickly rise to over 1,200-1,800 and remain there. If a patient’s testosterone rises to 2,000-3,000, but their PSA is stable or lower, we usually do not decrease their testosterone dose. This target goal for testosterone is not some arbitrary level chosen at random. When I first began to use testosterone replacement therapy in 1997, I was very conservative and tried to achieve T levels of only 200-300. So-called “normal” T levels are reported from 300-800 in some labs and 300-1,400 in other labs. This wide a range of “normal” TRT shows us how little we actually understand about T levels even in men without CaP.
After starting with this 200-300 target, I was pleased to see that PSA levels never “exploded.” This is the same experience that is reported in Abstract #4560 from the ASCO proceedings. But since my goal was to improve quality of life, I was disappointed that most men did not feel much better at this level of testosterone. This initial experience allowed me to aim for a higher target of 300-500. Once again, no patient had their PSA level explode. A lot of my patients felt better with this level of testosterone, but most did not. The ASCO abstract reports a median T level of 409 after one week of treatment. Even this “low” level of testosterone resulted in some favorable PSA responses. I believe the ASCO abstract study did not have a higher response rate and only had fairly short responses to TRT because their target T level was far too low.

By early 2000, my new T target was 600-700. Some men initially noticed significant improvement at this level of T. But usually within one to two months most men reported they no longer felt as good as they had when first starting TRT. Most importantly to me, I again found that PSA levels did not explode. ASCO Abstract #4560, 2004, confirmed this observation. While trying different doses of TRT, some men achieved T levels of over 1,000. When this happened, men usually volunteered that they felt “great.” If their levels fell back to 600-700, they reported they could notice the difference. Most men could accurately predict whether there had been a significant rise or decline in their testosterone levels between 700 and 1,800. I was surprised that many men could even distinguish a T level of 1,000 compared to 1,500. Almost all men are able to consistently enjoy a marked improvement in quality of life when T levels are between 1,200 and 2,000. My target T as of May 2004 is 1,200-2,500 or even higher. On many occasions, T levels up to 4,000 or higher have occurred. I only lower the dose of testosterone if the PSA is rising, even in men who have T levels of 3,000+. One particular response influenced me enormously. A patient with metastatic prostate cancer (not hormone refractory), but previously treated with radical prostatectomy, radiation therapy, and hormone blockade, accidentally raised his T dose when his level of T was just over 2,000. He was supposed to lower his dose. On his next scheduled blood test, his T had risen to over 3,500. His PSA decreased by 0.3 that month.

I have seen a number of testosterone levels between 2,000 and 4,000 on more than 100 different occasions, and find PSA levels
rarely increase and often decrease. Once again, even with these supraphysiologic levels of testosterone, PSA’s do not explode. I have also found that men whose FSH or LH levels remain high on TRT can usually tolerate higher doses of T. I often titrate T doses using FSH and/or LH as a marker.

We have a list of patient volunteers who are willing to discuss their testosterone replacement therapy experiences with interested candidates. Even for men with metastatic disease, some are able to remain on TRT for as long as four plus years, others obviously for less.

We favor AndroGel for most; T-Gel for a significant percent of men (compounded at a pharmacy in Colorado), and rarely Androderm (patches). Testim was too sticky and our patients did not like Striant. We have an occasional man whose T levels are difficult to raise to or above 1,000. The highest AndroGel dose we have tried is 25 grams per day. Our highest T-Gel dose has been 1,800 milligrams per day. We advise rubbing these products in, rather than just applying them. You must apply testosterone in the a.m. and never miss a dose. You must wait five minutes after application before putting on your shirt. The testosterone should be applied equally to four specific anatomic areas.

Most men will have a substantial increase in their level of T when the first start on TRT. However, their level of T almost always falls after the first few weeks of treatment so it becomes necessary to increase their dose of TRT to reach and/or maintain our desired T level.

We know of a few other doctors are using testosterone replacement therapy for some of their patients (like Dr. Charles “Snuffy” Myers). And we can add to this list the 2004 Abstract #4560 doctors.

Pioneering triple hormone blockade® with finasteride (Proscar) maintenance® as primary therapy for prostate cancer is a wonderful legacy that I take pride having accomplished and for which I am best known. But discovering that some patients clearly benefit from high-dose testosterone replacement therapy is exhilarating for both patients and me.

All men treated with continuous hormone blockade have at least some prostate cancer cells mutate and evolve in order to learn how to survive without testosterone. Observing favorable
responses to testosterone replacement therapy might appear to challenge the Nobel Prize winning teachings of Huggins and Hodges who won their 1941 Nobel Prize by showing you can induce remissions in some CaP patients by suppressing T (by castration or estrogen).

Since their landmark report, it has been universally accepted that the first commandment of CaP therapy states: “Thou shalt not even think about giving testosterone to any patient with prostate cancer.” I agree with Huggins and Hodges that the initial treatment for metastatic CaP needs to be hormone blockade. I use TAB followed by finasteride maintenance® therapy for men with so-called “early” or clinically localized CaP. Men with metastatic prostate cancer require additional therapy along with 3-drug hormone blockade (not THB/Leibowitz protocol). Updated results as of March 2004 on our first 185 men treated with triple hormone blockade for clinically localized CaP are best described on my March 2004 video. We have over a 99% cause specific survival. Only one patient in our series died from metastatic prostate cancer. Only 13 out of 185 patients have required re-treatment.

After that first cycle of HB, it is my opinion that a new treatment paradigm should be considered, especially since the average duration of response using HB for patients with metastatic CaP is only 18 to 20 months. Some of us believe that HB uncovers dormant genes that lead to the development of HRPC. TRT could lead to CaP cell maturation and eventual cell suicide (apoptosis). These are our beliefs while others feel differently. Clearly this is considered very controversial, but I am convinced that our approach is effective for many men who have prostate cancer, even metastatic.

It is clear that high-dose TRT markedly improves quality of life. We have also seen countless PSA’s decline as testosterone levels increase, at least for some men. This contradicts everything we thought we knew about the relationship between T and CaP. Our results need to be sustained and duplicated by others (and the 2004 Abstract #4560 is the first report to support that TRT may be beneficial for some patients). Dr. Charles “Snuffy” Myers told me that he has seen similar favorable responses for a number of men he has treated with high-dose TRT.

Absolute total compliance regarding the frequent, mandatory laboratory blood studies, as well as every six month bone scan
and CT scans of the chest, abdomen, and pelvis with oral and IV contrast are required and essential in order to monitor your progress and to help recognize toxicity or an unfavorable response. Digital rectal exams are regularly done. My May 2003 and October 2004 lectures describe the type of patients most likely to respond to high-dose testosterone replacement therapy. As of May 2004, we have only had three patients on testosterone replacement therapy who reported either bone pain or not feeling well shortly after starting treatment. All three had HRPC with rising PSA’s. We would not treat this type of patient with TRT today. In spite of this, all three had complete relief of their symptoms and reverted to their baseline within one to three days after they stopped their TRT. Only one of them required pain medicines (and for only one or two days), and none had any residual deficit or symptoms.

We have had to restart hormone blockade on a number of men who were treated with TRT, but most had previously failed local therapy and hormone blockade. We have been surprised at the especially rapid and marked declines in PSA when TRT was stopped and hormone blockade restarted. As of May 2004, I advise using about eight to nine months of hormone blockade before restarting TRT. We have not yet had to restart HB on any patient treated with triple hormone blockade® (or Leibowitz protocol) who were then treated with high-dose TRT. Eventually this could change. If your PSA is rising at the time TRT is started, it is unlikely that you will respond favorably. We only have a few exceptions. As our experience and insight evolves and more sophisticated tests become available, including molecular biological analysis, we should be able to better predict which patient would benefit from high-dose testosterone replacement therapy.

A number of our patients have been on high-dose TRT for several years. Some of these men have metastatic disease; some HRPC (the latter are also on our antiangiogenic cocktail). Some previously recurred after local therapy and hormone blockade. Some requested high-dose TRT after triple hormone blockade® (Leibowitz protocol).

The only reason to use testosterone replacement therapy is for quality of life issues. Do not use TRT to try to decrease your PSA. If your PSA declines, it is an extra unexpected “bonus benefit.” While on TRT, most men report some or all of these “side effects:”
I feel better.

I feel stronger.

I have increased libido (desire for sex).

I have morning and/or nocturnal erections.

I have an improved overall sense of well-being.

Fat is turning into muscle. (You need to exercise to accomplish this.)

My fatigue is gone.

I am more motivated.

My depression is better (or gone).

I have improved mental acuity such as memory and/or concentration (approximately two-thirds of men report this improvement. I never expected this benefit, but remember that many men on HB report reduced mental acuity, memory, concentration and/or reduced motivation.)

There is increased body hair, especially on the arms, legs, and/or chest which men seem to like since they had previously lost this hair while on HB. With the regrowth of hair, they feel more “masculine” and restored to their former self-image.

TRT helps to correct or prevent osteoporosis.

Red blood cell counts improve (anemia goes away).

There can be serious side effects as well. You should discuss these with your doctor. Although TRT must be used with extreme caution, as of May 2004, we are convinced that it can and does help many men.

Clearly men on high-dose TRT enjoy a far superior quality of life. Remember, if you are being treated with intermittent hormone blockade, while you are off HB, your endogenous testosterone usually recovers to the 300-400 range. If T is always harmful for all men with CaP, why aren’t you dead? Is
a T of 400 better or worse than a T of 1,200–3,000? Nobody has ever studied this. The risk of prostate cancer goes up as you age, while your level of testosterone goes down. Unraveling the mysteries between T levels and CaP is long overdue. We may be pleasantly surprised with the answers. At least we are asking questions and learning that T may be a two-edged sword as it duels for/against CaP.

As always --

Be happy,
Be well,
Live long and prosper,

DR. BOB

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** None of the above should be construed as medical advice or consultation, and anything discussed in this paper is meant for information only. All medical treatments, consultations, decisions and recommendations can only be made by the patient and his/her treating physician.

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