AN UPDATE TO THE CRISLER HCG PROTOCOL

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In my paper "My Current Best Thoughts on How to Administer TRT for Men", published in A4M's 2004/5 Anti-Aging Clinical Protocols, I introduced a new protocol where small doses of Human Chorionic Gonadotrophin (HCG) are regularly added to traditional TRT (either weekly IM testosterone cypionate or daily cream/gel). The reasons and benefits of this protocol are as follows, along with a new improvement I wish to share:

Any physician who administers TRT will, within the first few months of doing so, field complaints from their patients because they are now experiencing troubling testicular atrophy. Irrespective of the numerous and abundant benefits of TRT, men never enjoy seeing their genitals shrinking! Testicular atrophy occurs because the depressed LH level, secondary to the HPTA suppression TRT induces, no longer supports them. It is well known that HCG—a Luteinizing Hormone (LH) analog—will effectively, and dramatically, restore the testicles to previous form and function. It accomplishes this due to shared moiety between the alpha subunits of both hormones.

So, that satisfies an aesthetic consideration which should not be ignored. Now let's delve into the pharmacodynamics of the TRT medications. For those employing injectable testosterone cypionate, the cypionate ester provides a 5-8 day half-life, depending upon the specific metabolism, activity level, and overall health of the patient. It is now well-established that appropriate TRT using IM injections must be dosed at weekly intervals, in order to avoid seating the patient on a hormonal, and emotional, roller coaster. Adding in some HCG toward the end of the weekly "cycle" compensates for the drop in serum androgen levels by the half-life of the cypionate ester. Certainly the body thrives on regularity, and supplementing the TRT with endogenous testosterone production at just the right time—without inappropriately raising androgen OR estrogen (more on that later)—approximates the excellent performance stability of transdermal testosterone delivery systems for those who, for whatever reason or reasons, prefer test cyp.

But there's another metabolic reason to employ this protocol. The P450 Side Chain Cleavage enzyme, which converts CHOL into pregnenolone at the initiation of all three metabolic pathways CHOL serves as precursor (the sex hormones, glucocorticoids and mineralcorticoids), is actively stimulated, or depressed, by LH concentrations. It is intuitively consistent that during conditions of lowered testosterone levels, commensurate increases in LH production would serve to stimulate this conversion from CHOL into these pathways, thereby feeding more raw material for increased hormone production. And vice versa. Thus the addition of HCG (which also stimulates the P450scc enzyme) helps restore a more natural balance of the hormones within this pathway in patients who are entirely, or even partially, HPTA-suppressed.

It is important that no more than 500IU of HCG be administered on any given day. There is only just so much stimulation possible, and exceeding that not only is wasteful, doing so has important negative consequences. Higher doses overly stimulate testicular aromatase, which inappropriately raises estrogen levels, and brings on the detrimental effects of same. It also causes Leydig cell desentization to LH, and we are therefore inducing primary hypogonadism while perhaps treating secondary hypogonadism. 250IU QD is an effective, and safe, dose. After all, we are merely replacing that which is lost to inhibition.

In my previous report I recommended 250IU of HCG twice per week for all TRT patients, taken the day of, along with the day before, the weekly test cyp injection. After looking at countless lab printouts, listening to subjective reports from patients, and learning more about HCG, I am now shifting that regimen forward one day. In other words, my test cyp TRT patients now take their HCG at 250IU two days before, as well as the day immediately previous to, their IM shot. All administer their HCG subcutaneously, and dosage may be adjusted as necessary (I have yet to see more than 350IU per dose required).

I made this change after realizing that the previous HCG protocol was boosting serum testosterone levels too much, as the test cyp serum concentrations rise, approaching its peak at roughly the 72 hour mark. The original goal of supporting serum androgen levels with HCG had overshot its mark.

Those TRT patients who prefer a transdermal testosterone, or even testosterone pellets (although I am not in favor of same), take their HCG every third day. They needn't concern themselves with diminishing serum androgen levels from their testosterone delivery system. These patients will, of course, notice an increase in serum androgen levels above baseline.

While HCG, as sole TRT, is still considered treatment of choice for hypogonadotrophic hypogonadism by many , my experience is that it just does not bring the same subjective benefits as pure testosterone delivery systems do—even when similar serum androgen levels are produced from comparable baseline values. However, supplementing the more "traditional" TRT of transdermal, or injected, testosterone with HCG stabilizes serum levels, prevents testicular atrophy, helps rebalance expression of other hormones, and brings reports of greatly increased sense of well-being and libido. My patients absolutely love it. As time goes on, we are coming to appreciate HCG as a much more powerful--and wonderful--hormone than previously given credit.

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