Anyone who has employed the use of AAS (Anabolic/Androgenic Steroids) as a part of their training regime has certainly experienced the excessive and totally unnecessary post-cycle lean tissue loss (like muscle) that occurs as a result of testicular degradation. In truth it is idiocy to accept that in most cases a gross weight gain of 20-30 pounds of new tissue during the AAS cycle can result in a net gain of only a couple of pounds remaining post-cycle. In some cases, the result is additional lean tissue loss beyond what was AAS derived in a sort of one step forward and two steps back nightmare.

As athletes we each endlessly strive to achieve greater levels of musculature and conditioning in progressive steps. Sadly for most, this is just a dream predominantly due to a lack of understanding HPTA maintenance/regeneration and the seemingly ignorance of the little things that result in an additional pound of muscle here and there. (Think about 10 protocols, AAS or other wise, each providing an additional pound of lean muscle. Okay, go look at 10 lbs of steak at the store and realize that is the amount of additional muscle normally erroneously tossed away. Yikes!) A prime example is the fact that if you want to be huge you have got to pay attention to your balls and not accept raisin status anymore. Once you realize their potential you will enable yourself to achieve a new dimension in progress of the freaky nature. In this article we will discuss one of the protocols that allows athletes to make progress during “off or on periods”, and note some of the more detrimental protocols erroneously endorsed. But first its back to school for some basic physiology.

*Knowledge gives each of us the ability to plan for specific results. Anything else is just guessing.

HPTA?

For the chemically enhanced athlete there is a direct connection between the size of your testes and the size of your arms. No, not the concern of one arm being able to crush a Honda Civic and the other unable to hold a pencil thing from adolescence. We are talking about pre and post-cycle testicular function and natural endogenous androgen production.

The average males body “circulates” about 50mg of testosterone weekly. Of course the body “produces” much more testosterone than just the average weekly circulating 50mg. In truth much of our endogenous testosterone is converted to estrogens, DHT and androstenediol through various metabolic pathways.

HPTA function is relatively easy to understand once one realizes that it is simply a series of chemical messages that act as checks and balances to moderate or control endogenous androgen production.

HPTA FUNCTION

The HPTA refers to the Hypothalamus-Pituitary-Testes-Axis. This is the endocrine systems primary androgen and testosterone making area for males (most readers already know that women do not have testes so they also lack the HPTA).

Under normal conditions testosterone production begins when the hypothalamus senses low circulatory androgen levels such as testosterone. In response to the signal the hypothalamus secretes and releases a hormone called Gonadotropin Releasing Hormone (GnRH) that contacts receptors of the pituitary gland. As you recall, hormones and receptors are simply a method of organs, glands and tissues communicating with one another. GnRH tells the pituitary gland to secrete two gonadotropic hormones called Luteinizing Hormone (LH) and Follicle Stimulating Hormone (FSH). Next, both LH and FSH enter the vascular system and take a trip down south to the testes where the leydig cells (interstitial cells and sertoli cells) are located. The mergence of LH and FSH with interstitial and sertoli cell receptor results in testosterone manufacturing and sperm production.

HPAA FUNCTION?

A small percentage of testosterone and other androgens come from another source called the Hypothalamus-Pituitary-Adrenal-Axis or HPAA. When the pituitary gland secretes adrenocorticotropic

hormone (ACTH) the adrenal glands release a series of adrenalgenic/androgenic hormones. The main one for our point of discussion is dehydroepiandrosterone (DHEA). Through a series of enzymic interactions beginning with DHEA various other hormones are produced. These enzymic interactions are referred to as pathways. Much like a road or pathway one can imagine in life, each can lead to a different goal…or not.

DHEA > Androstenedione > androstenediol > Testosterone

This is not to say that an elevation in DHEA will result in a corresponding elevation in testosterone. There are many enzymic reactions possible that can lead to DHEA and/or androstenedione being converted or aromatized into estrogens instead. However the ability to increase total androgen production can be done with a little work. (Sorry, but you need to understand this stuff if you want to keep or improve upon your post-cycle lean mass)

Testosterone exists in either a bound or unbound state. Unbound is also called free or active testosterone. The S & M team that bind (deactivate) testosterone and other sex hormones are sex hormone binding globulin (SHBG) and albumin. The average male produces between 6-10 mg of testosterone daily. Of that, 6-10 mg only 1-2% is free or active. Think about that for a minute. Do you recall those kids in school who always seemed to be the most muscular, strongest, and fastest? They were the ones who produced the upper range of testosterone, while the rest fell somewhere below. So obviously a few extra milligrams of naturally circulating testosterone can make a profound difference.

Shrunken Nuts Syndrome

We are all aware of the profound alterations in musculature that occurs when an athlete introduces AAS to the body. Increased anabolism and significant nutrient turnover results in an increase in muscle mass (Gee, do you really think so?). Unfortunately AAS also induce Shrunken Nut Syndrome. (Ya, that looks great in the bedroom comically hanging between a set of 31” legs, huh?)

Many AAS are susceptible to conversion to estrogens through a process called aromatization. That simply means that a nasty enzyme in the body called aromatase can chemically alter androgens like manly testosterone into the female hormone estrogen. Anyone who has a woman in his life knows how anything female can reap havoc upon everything male if it is allowed to have its way unabated, of course. (But they are so much fun!) As such it should be no surprise that significant elevation of circulatory estrogen will shut down the HPTA and manly stuff like a Loraina Bobbit sorority party. This is called a negative feedback loop. Initially the elevation in AAS derived estrogen simply suppresses hypothalamic release of GnRH. Since the pituitary is deprived of the hormone GnRH that tells it to produce and secrete LH and FSH, the testes do not get the make testosterone and sperm messages respectively thus being unemployed. The result is of course Shrunken Nuts Syndrome…unnecessarily to a great degree. Please remember that the hormone controlled pathways leading to testosterone synthesis and release by the testes is a chain of hormonal events delivering messages:

Hypothalamus secretes GnRH
GnRH tells the pituitary gland to release LH and FSH
LH tells the testes to synthesize and release testosterone and FSH tells the testes to produce sperm.

There are many post cycle strategies employed by chemically enhanced athletes intended to regenerate HPTA function and therefore result in normal or above testosterone production. Of course this should include a return to normal sized testes and sperm production as well. Some of these strategies work quite well thus allowing for a significant amount of post-cycle lean mass retention and progress. And of course, others only cause more problems.

Before we continue, it is important that reader realize that the body is a complex organism that is closely regulated by Action/Reaction Factors intended to hold us in homeostasis. Homeostasis is simply a condition of no change and Action/Reaction Factors are the metabolic events that attempt to maintain it. As example, consider what happens when we introduce exogenous testosterone in a dosage that significantly exceeds what is considered normal by our bodies. We have introduced a substance that will trigger a significant anabolic action to which the body will counter-act with its own reaction. The action is an increase in lean tissue protein synthesis with a repartitioning

effect that drives calories away from fat cells and toward muscle tissue. Since this is a deviation from the normal perceived homeostasis the body begins its counter attack as a reaction once it catches on. In most situations the body requires about two weeks to realize that a significant alteration in a hormone level has occurred. This is the point of initial significant reaction as well. The increase in circulatory estrogens acts as a messenger that tells the hypothalamus to reduce or stop the release of GnRH. As you know by now, this is a negative feedback loop that shuts down the whole “make testosterone, sperm and let’s party” HPTA pathway beginning at the very source. Naturally the use of anti-estrogens and non-aromatizing AAS dramatically decrease the negative feedback loop during an AAS cycle, but there is another issue to consider as well. Through neuronet input the HPTA is able to sense androgenic activity to some extent. In short this simply means that when androgen levels are high the over stimulation of various receptors in the brain results in a message from the nervous system that tells the HPTA to decrease androgen production as well. Since the body cannot differentiate between androgens it makes itself and those administered, it readily believes that any elevation is controllable by shutting down the HPTA and does just that. Of course after a prolonged period of shut down the result is Shrunken Nuts Syndrome.

With that bit of information in mind let’s look at a protocol that accounts for the appropriate time frames and Action/Reaction Factors with a specific intent of lean mass gain and long-term post-cycle lean mass retention. (Get Huge/Stay Huge…and have a set to show as well)

Prevention Phase

Day
1. Testosterone Cypionate 250mg/Methandrostenolone 40mg

2. Methandrostenolone 40mg

3. Testosterone Cypionate 250mg/Methandrostenolone 40mg

4. Methandrostenolone 40mg

5. Testosterone Cypionate 250mg/Methandrostenolone 30mg

6. Methandrostenolone 30mg

7. Testosterone Cypionate 250mg/ Methandrostenolone 30mg

8. Methandrostenolone 30mg

9. Testosterone Cypionate 250mg

10.

11. Testosterone Cypionate 250mg

12.

13. Testosterone Cypionate 250mg

14. Lupron Depot 3.75mg

15. Testosterone Cypionate 150mg/Nandrolone Decanoate 100mg

16.

17. Testosterone Cypionate 100mg/Nandrolone Decanoate 150mg

18.

19. Testosterone Cypionate 50mg/Nandrolone Decanoate 200mg

20.

21. Nandrolone Decanoate 250mg/Fluoxymesterone 20mg

22. Fluoxymesterone 20mg

23. Fluoxymesterone 25mg

24. Fluoxymesterone 25mg

25. Fluoxymesterone 30mg

26. Fluoxymesterone 30mg

27. Fluoxymesterone 35mg

28. Fluoxymesterone 35mg

*Arimidex 0.5mg day 1-28

Testosterone aromatizes heavily and nandrolone has progesterone-like activity. This means both of the long acting injectable AAS employed in this example have the negative reaction of direct HPTA down-regulation. Since methandrostenolone aromatizes to a sort of super-girl estrogen it too inhibits HPTA function, but by adding it to the front of the protocol for a brief period it allows an athlete to create a rapid increase in circulating androgens as the slower acting testosterone cypionate begins to disperse from the administration-site. This allows for a more even circulatory androgen profile with overlapping variations in activity derived from each different AAS. As most are aware the body begins significant adaptation to most any chemical alteration after about two weeks. By employing the AAS progression there is a respectable reduction in adaptation and progress occurs at a greater rate with fewer negative reactions to deal with.

Fluoxymesterone (Halotestin) is a non-aromatizing androstane AAS and derivative of DHT. As such it has no direct effect upon HPTA down-regulation. Though it should be noted that it does create some degree of neuro-net activity. (Ever stood in a long grocery store line with someone on 50mg a day of Halo?) This allows an athlete to employ HPTA stimulating chemistry at this point with less counter productive estrogen to deal with, yet still increase lean tissue mass of a more permanent nature.

Lupron Depot is a synthetic analog of naturally occurring GnRH. As you will recall, GnRH is the hormone the hypothalamus releases to tell the pituitary gland to secrete LH and FSH thus resulting in testosterone and sperm production by the testes. It has an active-life of about 4 weeks. In clinical applications Lupron Depot is used to “shut down” HPTA function. (Huh?) Yup! Please bear with me, as this is one of those important little things. Anytime any gland in the HPTA is continuously stimulated to release a hormone the initial result is an increase in endogenous testosterone and sperm production…for the first 15-18 days. At that point the body begins to react significantly to our chemically induced action by shutting down the receptors that receive the hormone/message. This means that, for a time, the line of communication is shut off. The result is a decrease or total shutdown for natural testosterone and sperm production.

…And The Light Bulb Goes On.

So we have the HPTA inhibition from aromatizing AAS use in this example reaching a point of significance at about day 15-18. Due to this the body shuts down natural GnRH production in an attempt to reduce circulatory androgen and estrogen levels. This means that the employment of Lupron Depot (a synthetic GnRH analog) has about a two week delay in action as it takes that period of time for the pituitary to react as a result of the prolonged period it has been in shut down mode. Action/Reaction, remember? For those who hate to do the math the result is a significant increase in endogenous testosterone and sperm production beginning about day 27-29 that will peak at about day 41-43…when the last of the Lupron Depot clears the system. Perfect timing for the next phase!

Support Phase

Day
29. Fluoxymesterone 35mg/Methenolone Enanthate 200mg

30. Fluoxymesterone 35mg

31. Fluoxymesterone 35mg

32. Fluoxymesterone 35mg

33. Fluoxymesterone 35mg/Methenolone Enanthate 200mg

34. Fluoxymesterone 35mg

35. Fluoxymesterone 35mg

36. Fluoxymesterone 20mg/Oxandrolone 25mg

37. Fluoxymesterone 20mg/Oxandrolone 25mg/Methenolone Enanthate 200mg

38. Fluoxymesterone 10mg/Oxandrolone 37.5mg

39. Fluoxymesterone 10mg/Oxandrolone 37.5mg

40. Oxandrolone 50mg

41. Oxandrolone 50mg/Methenolone Enanthate 200mg

42. Oxandrolone 50mg

43. Oxandrolone 50mg

44. Oxandrolone 50mg

45. Oxandrolone 50mg/Methenolone Enanthate 200mg

46. Oxandrolone 50mg/HCG 2000iu

47. Oxandrolone 50mg

48. Oxandrolone 50mg/HCG 2000iu

49. Oxandrolone 50mg

50. Oxandrolone 50mg/HCG 2000iu

51. Oxandrolone 50mg

52. Oxandrolone 50mg/HCG 2000iu/Clomid 50mg 2xd

53. Oxandrolone 50mg/Clomid 50mg 2xd

54. Oxandrolone 50mg/HCG 2000iu/Clomid 50mg 2xd

55. Oxandrolone 50mg/Clomid 50mg 2xd

56. Oxandrolone 50mg/HCG 2000iu/Clomid 50mg 2xd

*Day 57-66 Clomid 50mg daily

*Day 53-56 Proviron 50mg 2 times daily

Neither Methenolone Enanthate (Primobolan Depot) nor Oxandrolone has much effect one way or the other on the HPTA in moderate dosages. Neither aromatizes and neither is very androgenic. However both are very anabolic, which means good solidification of lean tissue gained during the prior phase and good post-cycle retention if the HPTA is functioning normally or above.

HCG maintains a half-life of about 64 hours. This means that the last administration in this example would result in a total activity period of about 14 days. HCG mimics LH in that it

stimulates “just the testes” to produce testosterone and, to a lesser degree, sperm. Since there has been only about a 2-week period of significant shutdown this is rather easily accomplished (when real methenolone is used). By first bringing the testes back into play the period of stimulation to the hypothalamus and pituitary can be greatly reduced. This allows for, again, fewer negative reactions to deal with.

As you will remember, if you did not skip the science geek part of this article, there is an S & M binding team that deactivates androgen molecules in the blood stream. Though they too play an important role in the growth equation, there is a problem that arises during AAS employment. The body increases the amount of SHBG significantly. The result post-cycle is an over-binding effect upon the increased endogenous testosterone production. If left unchecked post-cycle, the result would be additional muscle tissue loss simply due to a much higher percentage of the natural circulating androgens being bound and inactive. Remember; only unbound androgen molecules are active and can induce an anabolic/androgenic response. Proviron is an anti-aromatase that has some androgenic value. It is also a powerful SHBG binder that allows the circulator androgens to run around unabated.

Clomid is an anti-estrogen that has hypothalamic and pituitary stimulating qualities. Since it protects both from circulatory estrogens with a notable GnRH, LH and FSH stimulatory value the result is normal or above production and of course, no Shrunken Nuts Syndrome and comical bedroom moments to not share with your friends and family. Oh ya, and lots of new mass is a plus to consider as well, huh?