irritating C-8 component in the stomach.

This section has been less scientific than the sections on proteins and fats, because I've found that while people are willing to change their protein and fat choices quite readily, they are stubborn about carbohydrates. Since dieters crave carbohydrates more than other foods, trying to stick to precise carbohydrate recommendations can quickly jettison your resolve to stick to the diet. Don't get too anxious over the exact quality of the carbohydrate sources in your diet. Just reducing carbohydrates will make you nervous; don't make it any harder.

CHAPTER 18

PROBLEMS WITH MODERN DIETING

YOU NOW KNOW most of the currently accepted opinions on nutrition for athletes. The diet articles in just about any athletic magazine would advocate eating numerous small low-fat, high-carbohydrate and moderate protein meals, doing aerobics and not trying to lose dramatic amounts of weight each week. Although I have refined this advice, and educated you to reach for high goals, I haven't shaken the earth beneath your feet. No diet revolutions, yet.

In many aspects we've laid out things perfectly. However, there is a lot fundamentally *wrong* with this way of dieting. Its advantages are what make me call it the Athletes' No Tantrum Diet. It is nutritionally correct, considering I'm giving it to a big, irrational baby that constantly craves carbohydrates. Its disadvantages are the subject of this chapter.

Each of the small problems caused by the Modern Diet causes more and more problems, until the diet goes terribly wrong, and you give up before you reach your goals. Even worse, sometimes dieters go out-of-control and get even fatter from binge eating.

The first obstacle to correct dieting is your misjudgment of

your body fat. A bodybuilder will typically estimate his body fat percentage around 10 percent, when it's really 15 percent or more. He picks a contest and gives himself 12 weeks to lose all of the fat and get in contest (winning) shape. Some bodybuilders only give themselves 8 weeks!

It's pretty common for bodybuilders to be deluded. After reading the glamorous success stories in magazine articles and advertisements (aren't they the same thing?), the sub-excellent bodybuilder, with legitimate hopes and dreams, thinks, "I can do it too." However, the top professional bodybuilders in the stories rarely have more than 9 percent body fat off-season and use an arsenal of (not mentioned) steroids, growth hormones and other secret weapons.

Impatience is the next obstacle on the course. You want the fat to *go away faster* than a puny, picayune pound per week. So you throw all of the careful planning, numbers and calculations out the window — you drop calories dramatically or add hours of aerobics every day. Sound familiar? Oh yes, the number on the bathroom scale plummets each week. However, your size and shape and strength are diminishing, too. You say to yourself: "I weigh the same as before I started working out." Yeah, and still have a floppy butt.

The body does have a brain. It knows it's slowly being starved to death. Metabolism slows d-o-w-n. You think, "Gee, at 1500 calories a day and 3 hours of aerobics, I should be losing 2-1/2 (the magic-magic-magic number) pounds a week, but *I'm stuck!*" You're not losing much weight, and what's coming off is muscle. You've come down with pencil-neck-itis. You get discouraged and abandon the diet and the contest. Then you binge to gain the lost muscle (my body really needs this extra peanut butter) and strength back. Oh yeah, the fat goes back on, too.

What do we do about these obstacles? Lectures, illustrations, chidings to just "be better" are pretty lame on my part. I am the *Guru* and am expected to help.

A big rear view mirror would solve 90 percent of the delusions about body fat. The back and glutes hold a lot of fat.

The only thing to do about impatience is to face reality. A pound a week just isn't enough — fat loss must be accelerated without muscle loss or metabolism slowdown.

When you have overcome these obstacles, you will realize all your dieting goals. Conventional modern diets will never be enough. If they were, you wouldn't need this book. I call the next step Isocaloric Dieting.

CHAPTER 19

THE ISOCALORIC DIET

WHILE IT IS POSSIBLE to lose more than one pound per week through further calorie reduction or by increasing aerobics, you will lose some muscle along with the fat. On paper, your body will look as if the diet is working. The mirror, however, will tell a different story. Even a 5 percent change in calories or aerobics will cause muscle loss.

The first way to prevent muscle loss is to change the fat to carbohydrate ratio. In the Modern Diet, the ratios were:

Protein	25%
Fat	10%
Carbohydrate	65%

In the Isocaloric Diet, these are changed to:

Protein	1/3
Fat	1/3
Carbohydrate	1/3

Adjusting these ratios will cause faster *fat* loss. Although

there is a scientific explanation for this phenomenon, I first discovered it the hard way through trial and error. No other combination worked.

At first, it seemed logical to try reducing calories further, but this caused too much muscle loss too quickly. Does increasing protein help maintain muscle mass? Nope, it doesn't. Increasing aerobics looks like a popular choice — after all, gyms have lots of steppers and rowers and bikes, all occupied with well-meaning individuals. Aerobics must be good, right? Wrong! As a matter of fact, increased aerobics causes just as much muscle loss as calorie restriction.

After screwing up in so many other ways, I finally arrived at the 1/3 ratios. Most athletes won't be happy with this. They've been conditioned not to eat dietary fat. Besides, lowering carbohydrates increases hunger and anxiety for a while. However, eating lots of carbohydrates makes your metabolism unable to burn fat efficiently.

If you have patience and not too much fat to lose, you will probably get damn close to your goal with the Modern Diet. The Modern Diet will allow you to be relatively happy, sociable, energetic and feed your carbohydrate addiction.

When "damn close" isn't close enough, you need the Isocaloric Diet. Will you be hungrier? Only at first. Will your strength decrease? Again, only at first. Will you lose muscle? Less than you would with any of the other alternatives.

In the Isocaloric Diet, we are once again concerned with the quality of the body's energy sources, not the quantity. Some people will object to eating a diet that is 1/3 fat. Fat is B-A-A-A-D, isn't it?

Fat isn't perfect, but it's all we have to work with. We can't decrease total calories because we don't want to lose muscle.

The high carbohydrates of the Modern Diet will need to change to either protein or fat. Exchanging the carbohydrates for protein won't work because not all of the amino acids can be converted into energy. Much of the amino acid content is excreted as urea, a waste product. Calorie for calorie, protein will not provide the same energy as carbohydrates. This is why high-protein diets cause faster fat loss than high-carbohydrate diets. Because protein has less usable energy, the body will strip down muscle to scavenge the energy-producing amino acids. Glutamine will be used first, then the branched-chain amino acids, then alanine.

If we can't use protein, all that's left is fat. Eating more fat will also reduce insulin secretion and make the fat-burning energy pathways more efficient. In the presence of insulin, the body will not release stored fat for energy. Therefore, as dieters, we want to reduce insulin secretion.

In the future, there may be better alternatives. The latest nutritional research shows that dietary pyruvate and lactate can activate alternative energy cycles that work better than fat or protein. These future foods will not cause insulin secretion, allowing faster fat loss while preserving more muscle from catabolism. Right now, however, these options are both tantalizing and futile. Currently, both pyruvate and lactate are hard to find, expensive, bad-tasting and boring to eat.

Almost any fat — saturated or not, essential or not — will work in the Isocaloric Diet. Energy-wise, it doesn't matter. You could use MCTs, for that matter, but they're are not as much fun as an additional serving of oily fish, walnuts or avocado.

If you have followed my recommendations for fat choices in the Modern Diet, you are eating mostly essential fats. You would be shocked at how little fat you'll need to eat to increase

fat calories by 23 percent. Salad and cooking oils don't take up much room. It doesn't take many avocados or walnuts to add a lot of calories. How about — dare I say it — peanut butter (no trans, that is)? Surely this isn't diet food!

Some people have asked me: If the Isocaloric Diet is so superior to the Modern Diet, why didn't I recommend it first? Well, most people are used to something like the Modern Diet. It's easy to start, "believe" and follow. Each change in long-standing habits requires more discipline.

Why increase protein by 8 percent? Fatty foods usually contain protein. Eggs, fish, meat, and that slice of cheese you can finally eat all contain protein in addition to fat. Even peanut butter contains protein.

The Isocaloric Diet requires more discipline than the Modern Diet, but if you're impatient, or have an inflexible timetable, the Isocaloric Diet will get you virtually all the way to your goals. The Modern Diet won't. Of course, strength and energy will suffer for about 5 days, but after that, you'll feel even better than before.

The Isocaloric Diet begins to solve a major problem in dieting (aside from hunger and anxiety): impatience. While the problem of impatience has not been completely solved, we've appeased it for now. Although there is a better diet plan to come, it is illuminating to examine the preliminary solutions to the dilemmas I've encountered in my years as a professional body confidante.

CHAPTER 20

MUSCLE CATABOLISM WHILE DIETING

AS ATHLETES, we want to make sure that the weight we lose is fat, not catabolized muscle. While "catabolism" technically means the breaking down of any tissue, we will focus on muscle catabolism exclusively.

Ultimately, the chemical that allows us to move, create heat, think and live is Adenosine Triphosphate (ATP). As you move your eyeballs to read this page, you are using the same energy source that lights up a firefly. All substances used for energy are eventually converted to ATP. All of the carbohydrates we eat (unless we eat too much) will be converted to glucose and then to ATP. Although glucose is converted the fastest, ATP is not picky. Fats, either dietary or stored, can also be converted to ATP. When you "lose" fat, your body is converting stored fat to ATP.

Many people assume that once your blood glucose and liver glycogen are low, your body automatically switches over to stored body fat. Voilà — the stored fat is shed and all of us can live happily ... Of course, the truth is much different.

The body can also convert protein to ATP. This conversion can happen locally; an exercising muscle will use the amino acid

leucine right in the cell. However, most of the amino acids are leaked out of the muscle cell into general circulation. They are then picked up by the liver, which disassembles them into usable energy substrates. This process is called gluconeogenesis, an unwieldy word meaning the creation of glucose from amino acids (or other substrates). Supplementation with branched-chain amino acids, glutamine or alpha-ketoglutarate is supposed to prevent this process.

Gluconeogenesis can use dietary protein or protein from catabolized muscle tissue. Do we have any say which it is?

There are a number of strategies to prevent muscle wasting. Beyond good genetics, how clever you are will determine how much muscle you lose. Sometimes a potentially wonderful anti-catabolic trick will interfere with fat loss. Sure, you could just use steroids, but there are other options. Let's take a look at a few.

CHAPTER 21

ANTI-CATABOLIC STRATEGIES

THESE RECOMMENDATIONS for anti-catabolic strategies start simple and get more daring as the list progresses. Dieters usually stop at some point on the list because they find the substance difficult to acquire or somehow morally repugnant. This is your personal choice.

PRESERVE GLUCOSE

Since glucose is the fastest substrate for ATP, a high-carbohydrate diet (which is easily converted to glucose), will keep the raiding of muscle amino acid stores to a minimum. This anti-catabolic strategy only works when calorie debits are less than 10 percent. High-carbohydrate diets with calorie debits greater than 10 percent will actually cause more muscle loss than a higher fat diet would.

MINIMIZE INSULIN SECRETION

This is the fix-it remedy for the shortcomings of a high-carbohydrate diet. Carbohydrate quality influences insulin secretion. Simple sugars are fast and concentrated, causing high insulin secretion. Even fructose, the noninsulinergic sugar,

should be avoided because it impairs insulin sensitivity. Refer back to the chapter on carbohydrates if you need to refresh your memory.

SUPPLY KEY AMINO ACIDS IN DIETARY FORM

The solution bodybuilders first came up with was just to eat more protein. However, not every amino acid can be used directly to produce ATP. Many of the amino acids in whole proteins are broken down to urea and excreted in urine. Since glutamine is the main amino acid used to create glucose, proteins high in glutamine would be ideal. Unfortunately, the two best choices, beet and wheat protein (which are 50 percent glutamine), are not commercially available yet.

Since these proteins are hard to get, the next choice would be supplemental amino acids. Sports nutritionists have recommended the branched-chain amino acids (BCAAs), and more recently, free-form glutamine. Interestingly, most ingested free-form glutamine doesn't get into general circulation because the intestines use it up first. This is called the glutamine paradox.

Better than free-form glutamine is its analogue, alpha-ketoglutarate, which survives digestion intact. Like many supplements, all forms of glutamine — free-form, peptide-bound and alpha-ketoglutarate — are really bad tasting. In all of the interesting research, they were taken intravenously or by intubation. Will you want to swallow these foul substances? Currently, not many people do. This is why I think whey protein, with its high BCAA content, is so important. Food should be enjoyed. Although whey is bland, the other choices taste awful.

Will dietary pyruvate and lactate take the place of amino acids in the future? According to the ATP flow chart, pyruvate and lactate seem to be preferable to fatty acids and amino acids

for ATP production. Unfortunately, I have no idea how to make them even innocuously bland, other than loading them all into capsules. Oh swell, another bunch of pills to swallow.

LOWER INSULIN BY QUASI-NUTRITIONAL MEANS

Chromium and vanadyl sulfate both reduce the secretion of insulin. How do they work? Beats me. These two minerals either support (chromium) or mimic (vanadyl) insulin and cause the transport of blood glucose into muscle cells preferentially over fat cells. Although there is some research showing these effects, scientists haven't found all of the mechanisms at a cellular level. Remember, vanadyl sulfate is not on the FDA GRAS list and could easily be yanked at any time. Curiously, the more potent of the chromiums, chromium picolinate, is not on the GRAS list either. However, chromium nicotinate is.

USE INSULIN REPLACEMENTS

Chromium and vanadyl sulfate are readily available, but they are not the best insulin mimics. Metformin and phenformin, which I call "insulin agonists," work much better. I've used metformin (available in Mexico and Europe), while life extensionists prefer the more potent phenformin. Metformin is now a prescription drug in America, under the trade name Glucophage.

DON'T OVERTRAIN

This should be obvious, but most athletes overtrain anyway. People sneak aerobics more than anything else. The fat-burning zone of aerobic activity is only 60 to 65 percent of your maximum heart rate. To figure your maximum heart rate quickly, subtract your age from 220. During aerobics, your heart rate should be just slightly above *half* that. Most dieters do aer-

obics at about 70 percent (usually higher) of maximum heart rate, which forces too much reliance on anaerobic energy substrates. The only way to sustain a high aerobic workload and still keep your heart rate between 60 and 65 percent of maximum is to be tremendously aerobically fit, which you should accomplish before the diet.

Weight training can be overtraining too. Each workout causes microtrauma to the muscle cells — inflammation, tears and strains. To recuperate, you need to replace amino acids and glycogen. Research illustrates that the classic 10 sets of 10 repetitions of squats raise cortisol significantly. Consider this carefully if you are trying to train every day.

Even with these nutritional fortifications, muscle catabolism still happens. Catabolism is not a passive process; once amino acids are in the muscle cell, an active messenger is needed to get them out.

The messenger is a hormone called cortisol, secreted by the adrenal glands. Some cortisol is necessary for life, but too much will interfere with athletic performance. Cortisol is a natural anti-inflammatory that combats stress and starvation. Cortisol is a white hat kind of hormone in these extreme situations, but *excess* cortisol is the Black Bart of the metabolism.

The most potent strategy against catabolism is to thwart cortisol. Exotic nutrients are only a passive defense against catabolism. Replacing lost amino acids repairs damage that is already done. Active anti-catabolics catch the damage before it happens.

Reduce The Inflammation

Some of the cortisol secretion is a response to tissue inflammation from the physical stress of exercise. To stop tissue

inflammation, you can use anti-inflammatory drugs before you work out. Any of a wide variety of over-the-counter medications — aspirin, acetaminophen, ibuprofen and naproxen — will work. Remember that all of these medications have side effects, so you may want to rotate them or not use them every day. In addition, 500 mg of Vitamin C taken right before your workout will also prevent inflammation.

Use Anabolic Steroids

Cortisol antagonists block the cortisol receptor on the muscle cell, the "parking space" where the hormone attaches to the cell. There are no perfect drugs specifically designed for this purpose, but we will make do with what we have. Anabolic steroids are the best cortisol antagonists currently available. Anabolic steroids and cortisol (which is another type of steroid) are so close in chemical structure that steroids will block the cortisol receptor. In fact, most of the "anabolism" from anabolic steroids comes from this anti-catabolic effect. Some anabolic steroids are also anti-inflammatory.

Use Other Cortisol Antagonists

There are some cortisol antagonists, but they would be more aptly named lame cortisol antagonists. In a disease called Cushing's Syndrome, the adrenal glands secrete too much cortisol. To help these patients, scientists have sought specific cortisol antagonists. The one you will be most familiar with is the "abortion" pill, RU486, which is primarily a progesterone antagonist, but also affects the cortisol receptor. A new Italian quasi-steroid that has some promise is Decylroxibone, which has no overt anabolic action, no androgenic side effects, but potent anti-catabolic activity. However, none of these non-anabolic

steroid drugs are as effective or as free of side effects as anabolic steroids themselves. In many instances, using a cortisol antagonist will increase the number of receptors or raise cortisol secretion.

REDUCE THE NUMBER OF CORTISOL RECEPTORS

A small amount of research shows that some anabolic steroids can cause a reduction in the number of cortisol receptors. For decades, researchers have been fixated on androgen receptors. The new research explains why steroids work so well. Although cortisol receptors are pretty abundant, cortisol receptor attenuation has exciting implications.

REDUCE ADRENAL CORTISOL SECRETION

This is the most basic attack on Cushing's Syndrome. The most popular of these drugs is Cytadren, which unfortunately has been virtually unusable for athletes and dieters. Too much Cytadren will raise adrenocorticotropic hormone (ACTH), the hormone that the pituitary secretes when cortisol is too high. Even a moderate amount of Cytadren reduces cortisol enough to cause joint pains. Perhaps Cytadren plus anabolic steroids or over-the-counter anti-inflammatories will be the happy medium. So far, Cytadren hasn't fulfilled its promise as a true alternative to anabolic steroids.

USE PHANTOM ZONE ANTI-CATABOLICS

Some drugs impart a perceptible anti-catabolic effect, although there isn't any substantial research (or any research at all) on this effect. Bodybuilders are not afraid to fool around with drugs that don't seem applicable. For example, clenbuterol has quite remarkable anti-catabolic effects, which were verified

in human studies long after bodybuilders started using it. Nolvadex, an anti-estrogen, has no studies proving that it causes muscle gains, but it does. Studies of cortisol reduction from morphine use inspired some athletes to use Nubain and Stadol. Visually, you can see that *something* anabolic or anti-catabolic is happening with these drugs. Can we measure these effects in a laboratory? No one has bothered to do it yet.

Until now, anabolic steroids have covered up most catabolic blunders so well that coaches (including myself) didn't have to address them. Anabolic steroids are no-brain solutions. Working without them is dieting without a net. I hope that ultimately these alternative methods will achieve an effectiveness similar to steroids.

For more detailed information on particular anti-catabolic substances, turn to Chapter 30, which contains the top 50 dieting drugs. Chapter 48, the Special Section on Diuretics, has additional comments on anabolic steroids while dieting.

CHAPTER 22

ANTI-CATABOLICS IN THE REAL WORLD

PROFESSIONAL BODYBUILDERS use virtually every anti-catabolic strategy except cutting back on training. I'd like to comment on the actual implications of these anti-catabolic strategies for the rest of us.

First, I urge you to supplement your diet with both chromium and vanadyl sulfate. Although we don't completely understand how these two minerals work, the effects are discernible.

Secondly, evaluate your weekly training schedule. You may benefit from purchasing a heart rate monitor to ensure that your aerobics are in the fat-burning zone between 60 and 65 percent of maximum heart rate.

In addition, most weight-lifters work out too much. After seeing and trying every imaginable workout routine, I have reached the conclusion that no bodybuilder needs to exercise each body part more than once a week. The following schedule has been the most productive for all of the athletes I've trained:

Day 1	Shoulders and Arms
Day 2	Legs and Abdominals

Day 3 and 4	Off
Day 5	Chest and Back
Day 6 and 7	Off

Although this routine can be started on any day, it fits nicely into a regular week, working out between Monday and Friday, with the weekends off. Although some people think it's hard-core to split the body parts up into two workouts over the day, this is not necessary nor productive. While dieting, you don't build up enough glycogen for a second workout. In addition, you'll secrete less cortisol with just one workout.

Many anti-catabolic supplements look appealing in magazine advertisements, but the problems of cost and palatability make them too cumbersome for sustained use. For example, ornithine alpha-keto-glutarate (OKG), like all amino acid supplements, is a foul-tasting powder. Manufacturers tried creating a salt of OKG to reduce the bad taste, but it didn't work. The challenge to nutritionists and supplement designers is to make these wonder substrates at least bland.

Although it involves some sleuthing, learning how to import non-FDA-approved foreign drugs is a worthwhile pursuit. In the future, substances like vanadyl sulfate and OKG may be restricted.

I just can't ignore anabolic steroids. It seems odd to me that when so many dieters are losing muscle from catabolism, the FDA won't recognize this as a medical condition. It is currently a felony for American doctors to prescribe anabolic steroids for this purpose. However, an American citizen can have a physician in a foreign country write and fill a prescription for anabolic steroids (up to a 90-day supply), which you can then bring back into the United States. There are many Mexican doctors

who would be happy to write and fill a steroid prescription for you on your vacation. By the way, you can also do this with any Customs Alert drug that is usually disallowed if imported by mail. Anabolic steroid prescriptions are not any more illegal than the foreign prescriptions of Valium, Xanax and Codeine.

Are phantom zone anti-catabolics worth pursuing? Clenbuterol, at least, is definitely worth the effort. It has both thermogenic and anti-catabolic properties. Is the bother of traveling to Mexico (or Italy or Germany) and finding a doctor worth the effort? Yes. Besides you might need the practice. In the future, these circuitous routes may be the only way to acquire amino acids, vitamins and herbal preparations. It may seem extreme now, but in the future, there may be anti-catabolic vacation tour groups. Life extensionists currently sponsor growth hormone excursions.

Is there such a thing as a diet fringe? Of course. When people with AIDS (PWAs) couldn't get the drugs they needed because of FDA interference, they made them in underground laboratories. RU486 is being manufactured openly in New York State and is legal to use as long as the drug does not cross a state line. I've published a "recipe" for gamma-hydroxybutyrate (GHB). Is this militant? Yes, indeed. Illegal? Definitely not!

Over the years, I've seen a lot of magic pills and wish-fulfillment supplements. I've sampled arginine, ornithine, L-Dopa, Catapres, GABA, GHB, OKG, creatine and more. They all claim to have some anabolic or anti-catabolic action. I play with these substances because it's my job, my hobby, and at one time, my obsession.

I've been wishy-washy about many of these anti-catabolics because I've spent so many years working with anabolic steroids, which are the best anti-catabolics in the world. It

reminds me of my 1976 Corvette. It was heavy, slow, with big ugly bumpers and a gutless engine. At the time, it was the fastest new car I could buy. After I drove it, I said, "This car s-u-c-k-s!" because I remembered my pre-smog cars. But the laws had changed.

Anabolic steroids and clenbuterol are the only effective anti-catabolics! All of the others are shit! A-h-h-h-h. Now I feel *much better!* There's not a whole bunch you can do about these drugs. They're damned inconvenient to get and use legally. I'm not going to break any more laws, nor do I advocate such reckless behavior. It's just been frustrating that none of these anti-catabolic compounds are quite good enough.

CHAPTER 23

HOW DIETING SLOWS DOWN THE METABOLISM

EVERY DIETER DREADS the awful day when he stops losing weight. Suddenly the lowered calories have become the new maintenance level. Although many people expect it, and a whole diet book (*The Rotation Diet*) was written to avoid it, it's still a shock when your metabolism slows down.

I have some happy, happy news to tell you. Metabolic slowdown doesn't have to happen. Once you understand why it happens, you can take a number of steps to avoid it.

Regulating thyroid hormone levels is the cornerstone to keeping the metabolism humming along. You might be saying: "Uh-oh, don't be monkeying around with my thyroid, that's b-a-a-d." Granted, most of the following information is, well, radical. Most physicians will start with the "No, no, no" litany about the idea of even touching your thyroid, like it's sacred or something. Surprise! Bodybuilders and life extensionists have been adjusting their thyroids for years.

Thyroid is *the* regulator of your overall metabolism. It sets your basal body temperature after the hypothalamus commands, "Make it so." Thyroid hormone also adjusts the rate of other

metabolic processes, including protein synthesis. The thyroid gland secretes two types of thyroid hormones, T4 and T3, in a ratio of 80 percent T4 to 20 percent T3. Although most of the thyroid hormone in general circulation is T4, T3 is the real dynamo. At the cellular level, T3 is the one that hooks up to the receptors. It's curious that most active T3 comes from the deconstruction of T4 to T3, not the dollop that the thyroid gland produces.

The main cause of metabolic slowdown is a decrease in active thyroid hormone. There are three major reasons why thyroid hormone attenuates. First, thyroid levels adjust to the amount of muscle in the body. When you lose muscle, the thyroid gland secretes less. Therefore, the factors that cause muscle loss — calorie restriction, protein deficiency, amino acid substrate deficiency in the muscle cell and (yes) training too much on lowered calories — also cause decreased production thyroid hormone.

If the body didn't lower thyroid hormone secretion, and thereby lower body temperature, body temperature would ... rise! To help you understand this, imagine that you are eating maintenance calories, but you suddenly lose 10 pounds of muscle. Make up some lurid reason why. If your thyroid levels didn't decrease, the same amount of thyroid would be supplying less muscle to move and heat. It's not nice for dieters, but if thyroid levels stayed the same with less meat to cook, the too-high thyroid levels would burn off more muscle.

It's pretty obvious that maintaining muscle mass while dieting is important. Less heat-producing muscle means less thyroid hormone. Less thyroid hormone means you can't eat as much. Not only does muscle wasting cause a decrease both T4 and T3, but T4 does not convert as well into T3. Many doctors

are not aware that on low-calorie diets, when insulin levels are lower (from lowered carbohydrate intake), T4 doesn't convert to T3 very well.

The second cause of thyroid hormone attenuation is cortisol. When the cortisol receptors on the pituitary gland are activated, they reduce production of thyroid stimulating hormone (TSH), which is the messenger between the hypothalamus (in the brain) and the thyroid gland. Many physicians are not aware of this new area because they don't have physically stressed athletes as patients.

Cortisol also affects the T4 to T3 conversion process. A specific enzyme called 5'-deiodinase is impaired in the presence of cortisol. Overtrained athletes experience this phenomenon more than is commonly suspected. A variety of factors will contribute to this reduction in T3 — overtraining, stress, insulin levels and other drugs (thermogenic agents in particular). Often, T4 levels are appear to be within the normal range, while T3 is very low, but still "normal." If T3 is too low, body temperature is lower than before, then (ipso facto), fewer calories are burned.

T3 impairment is called Euthyroid Sick Syndrome (ESS). There is quite a controversy among thyroid specialists about how to treat it. One camp proclaims that T3 lowers as part of a natural and body-preserving action. The opposing side recognizes that lowered metabolism should be treated like any other disease.

This is the first totally new concept in this book. Damn it, finally! Most of the metabolic slowdown from dieting is from ESS. Overtraining and interference from thermogenic drugs will cause ESS, even at relatively high calorie levels.

The first thing to do to avoid ESS is not to decrease calories too much, which we discussed way back in the beginning.

It's also important make sure you do not overtrain. Besides these two "safe" choices, there's only one thing left to do: Regulate thyroid levels by supplementing the (lowered) thyroid hormone with a daily oral medication. This would be a *big step* for many of you. Thyroid is a prescription medication; to put it bluntly, most doctors will not look kindly on your self-therapy.

For a minute forget about dieting and consider that most normal, sedentary people's thyroid levels are not *ideal*. Average, yes; optimal, no! To a doctor, good health is normal. My job is to optimize the metabolism to create maximum performance. We don't just want good health, but superior health.

Although thyroid problems are most noticeable while dieting, I've learned (from countless body temperatures and thyroid blood tests) that many athletes who appear healthy have suboptimal thyroid levels. Dieting only exacerbates it. So what do we do?

CHAPTER 24

OPTIMIZING YOUR METABOLIC RATE

MANY INDICATORS of metabolic rate are easy to observe: body temperature, heart rate, blood pressure and respiration rate. Even a layperson with a handshake and a finger on the wrist can make a rough judgment of your metabolism. In this modern medical age, we can use blood tests to even more accurately determine thyroid hormone levels.

For many decades, physicians didn't have such fancy (and expensive) blood tests. The most common method of determining thyroid levels was basal body temperature. Early morning resting body temperature reading was (and probably still is) the first method to test thyroid status.

Most people think that body temperature is pretty close to 98.6°F, which is considered normal. It is — you guessed it — an average. However, body temperature fluctuates throughout the day. To get the most accurate and consistent reading, you should take your temperature at a specific time each day.

What follows is the step-by-step procedure my athletes have used to regulate their thyroid thermostat. Most of these adjustments should be made before dieting. Why? Although most dieters have some degree of Euthyroid Sick Syndrome

(ESS) some people have a subnormal amount of both thyroid hormones (T4 and T3) even when they think they're healthy. By optimizing your thyroid, you will be able to eat more calories, store less body fat and — a little-known bonus — achieve better protein synthesis. Optimizing your metabolism through thyroid regulation is the most important non-nutrition, non-training thing an athlete can do. Hardworking dieters with ideal nutrition and training will achieve only mediocre results if they have sub-optimal thyroid hormone levels.

Doctors agreed years ago that the best time to take temperature readings is right when you wake up from overnight sleeping, while you are still lying down in bed. This important initial reading should be done before dieting. Of course, monitoring temperature while dieting is also wise, but this reading will be our reference point. Don't take any drugs that will affect this reading: no caffeine, thermogenic drugs (including herbs) or aspirin. Needless to say, a cold, flu or any kind of fever will make the initial readings worthless for our purpose.

I've tried to use old-fashioned mercury thermometers, but they drive me crazy because I can never read the damn things. Digital oral thermometers (which can be purchased for under $10) are usually accurate, but I check them initially against a mercury one (yes, I do use a magnifying glass) to make sure. After much trial and error, I now use the Thermoscan inner ear infrared scanner thermometer, which costs about $100. It's accurate, and I don't have to wait for the reading.

After reviewing my client's daily temperature diaries, I've established a range of ideal body temperatures to strive for. Good to great bodybuilders who maintain a lean physique, eat lots of food, and don't yo-yo diet have the best readings.

Why bodybuilders? They're the healthiest. Many endurance

athletes' heart rate, blood pressure, temperature and overall metabolism are unhealthily low. They also don't have much strength (in body and mind) after training. In my mind, these athletes are sick.

The morning temperature range I've determined to be optimal is different than doctors'. For optimal performance, your reading should be between 97.8°F and 98.2°F. Most women, even female bodybuilders, are below this range. By the way, 9 out of 10 thyroid prescriptions are written for women.

Some very rare individuals have a temperature above 98.2°F. If this reading can't be explained, it is a potential medical problem and should be addressed by thyroid specialists.

If your temperature is outside (on the downside) of the normal range, there are a few ways to fix it, none of them "natural." Supplements won't work. People tried taking supplemental iodine, but it actually slowed down the pituitary's output of TSH.

To boost your body temperature to 98.2°F, you will need to increase the level of T3. T4 is only used to store the hormone in the blood for eventual conversion to T3; it has no metabolic purpose. Although T3 is the real locomotive of the metabolism, if you care to look up "thyroid preparations" in the *Physicians' Desk Reference*, you'll be confronted with a bewildering array of prescription drugs.

Taking the giant step, optimizing your body temperature with thyroid, will be frustrating. Most doctors won't embrace the concept of optimizing anything in your metabolism. Sure, an MD will order various tests to make sure that everything is — you guessed it — normal. In most cases, you won't get a prescription written. The practical solution is to find a source for thyroid medication from outside of the country and have it

mailed in. Unfortunately, foreign mail-order companies are in business one month and gone the next. I advise you to stock up to tide yourself over while locating another source.

Which is the best thyroid medication? The quick and dirty solution is to use straight T3. T3 is what we need, right? It's quick because you don't have to worry about any underlying cause for the thyroid insufficiency. You also avoid all sorts of expensive blood tests and doctor visits. All you need is a bottle of Cytomel and a $10 thermometer and you're rolling.

Any physicians reading this would be all near-apoplectic at this point. Reckless! Dangerous! Perhaps you could fill in a more alarming adjective: _____! However, in reality, virtually all of the bodybuilders who use thyroid hormone, especially while dieting, use T3 with no blood tests and no thermometer, either.

As I've matured in this human performance business I've gotten more precise. I recommend frequent thyroid blood tests so you can see what's going on. Only with a blood test can you see why thyroid hormone levels are normal but not optimal.

I like to look at 3 tests. You should be familiar with them (and their prices) so that you can be specific with your doctor. Even though you probably can't wrangle a thyroid prescription out of him, you can use him for the testing.

THYROID STIMULATING HORMONE (TSH) TEST

This recent advancement in thyroid tests is called a "sensitive thyrotropin assay," because the older TSH tests were not sensitive enough. The test costs about $25.

Normal	.3 – 5 µU/ml
Subclinical	5.1 – 20 µU/ml
High	20 µU/ml
Lowest sensitivity	.1 µU/ml

TSH is produced in the pituitary. Most hypothyroidism, which is a deficiency of both T4 and T3, is usually indicated by an *overproduction* of TSH. The older TSH tests only went down to 1 µU/ml. This new test is nice to have, because if you overmedicate with thyroid hormone, T4 and T3 levels can look normal because of the medicine. However, if TSH is too low, you will know that too much medication is being used and TSH and natural T4 and T3 are being suppressed.

Unfortunately, athletes who are optimizing their body temperature will unfortunately suppress TSH when increasing T3 to optimal levels. With a normal person a thyroid specialist will try to balance the hormones so that T4 levels are as normal as possible and TSH is still above .3 µU/ml or above. However, the dosage required to achieve body temperatures between 97.8°F and 98.2°F will be often so high that it will suppress TSH. It can take 8 weeks after stopping the thyroid medication to get TSH levels back to normal.

TSH is not the only important test. Although a reading above 20 µU/ml will usually predict low T4 and T3, T4 and T3 tests are also necessary.

FREE T4 TEST

Technically, this test is called an "equilibrium dialysis." This is the *only* T4 test you want to do. There are others, such as Total T4 and Free T4 Index, but they aren't accurate enough. In fact, Free T4 by Immunometic is only 89 percent accurate! At $65, this test is the most expensive T4 test, and not every blood testing lab offers it. Normal Free T4 ranges from 4.5 to 12.5

µg/dl. Athletes are rarely below this range. If body temperature is below 97.8°F and T4 is low but not out of the normal range, a synthetic T4 thyroid drug like Synthroid may be the best choice. Don't be surprised, though, that raising T4 will not raise T3 appreciably. Remember, unlike classic hypothyroidism in sedentary people, most Euthyroid Sick Syndrome (ESS) in athletes is caused by faulty T4 to T3 conversion. Most T4 daily dosages are in the 100 to 150 mcg range, and Synthroid is available in dosages that vary from 25 mcg to 300 mcg.

RADIOIMMUNOASSAY FREE T3 TEST

This is where the action is, athletically and metabolically speaking. Although commercial T3 drugs like Cytomel and Triostat have been the most popular choice with bodybuilders, most thyroid specialists work predominantly with T4. T3 is a great thyroid drug, but is difficult to work with for most people. It is hard to get consistent T3 readings because most of the T3 is unbound (as opposed to T4, which is 99.95 percent bound). T3 levels will peak within 4 hours of ingestion and rapidly attenuate after 60 hours. Most doctors don't like to work within these variables.

As long as you always have the blood drawn at the same time (usually in the morning) and you don't take the T3 medication before the test, then reliable T3 values should not be a problem.

The Free T3 blood test values should be somewhere between 80 and 175 ng/dl. Pay close attention to the this amount; when low-calorie diets stop working, it's usually a problem with T4 to T3 conversion. If you've been tracking your temperature each morning, (what, you haven't; why not?), this number will only confirm your suspicions.

ESS occurs when TSH and T4 are both somewhere in the normal range, but T3 is low. T3 could be below 80 ng/dl or, more commonly, T3 will be toward the low end of normal while T4 is normal or high. What's happening? T4 is normally converted to T3; if something goes wrong, it is diverted to into non-active *reverse* T3. Is this confusing? Ha-ha-ha — have a GP explain it to you!

When you have low T3 and normal T4, supplementing with Synthroid will not help very much because it will not correct the enzyme inhibition. The ideal solution, most doctors would say, would be to take a break from the low-calorie diet. Yeah, right.

Another solution would be to use Cytomel (synthetic T3). This is the fork in the road. Prevailing medical opinion is that non-medical cases of ESS should not be treated with exogenous thyroid hormone. Most physicians think that a lowered body temperature is not a sickness. I, not being a doctor, differ in opinion. The true guide is the original body temperature measurement, which should be between 97.2°F and 98.2°F even while dieting — especially while dieting. Some thermogenic drugs that have tremendous fat-burning properties will block conversion of T4 to T3.

Let's condense what we've learned so far. Thyroid optimization should start before you begin dieting. You will want to adjust your body temperature with oral thyroid medication so your morning temperature will be between 97.8°F and 98.2°F.

If you skip the blood tests and just start using Cytomel, you will never know why your body temperature was low in the first place. If you use T3 with only a thermometer as a guide, you may overdose and suppress production of TSH. Once suppressed, it takes 8 weeks to get TSH functioning again. Unfor-

tunately, the amount of T3 needed to boost body temperature into the preferred range will usually stop TSH production. Most doctors would rather see a patient with TSH, T4 and T3 in the normal range, even if each measurement is not ideal.

To get the blood tests, you can go to a doctor, or in some states, a chiropractor. The 3 tests will total about $125 plus the office visit. The initial tests should be done when on maintenance calories and no thyroid medication. If you have a low body temperature, the TSH, T4 and T3 readings will indicate why. If the values show classic hypothyroidism, which is high TSH and low both T4 and T3, then Synthroid is a good choice.

If you have ESS, which is low T3 with normal TSH and T4, use Cytomel. Find a daily dosage that sets your temperature between 97.8°F and 98.2°F. Once you determine your ideal temperature and daily dosage of Cytomel, do the T3 blood test again to establish the Free T3 value that corresponds to the ideal temperature.

Why redo the T3 blood test? Whenever you use thermogenic agents, your body temperature will rise. Use of these agents will not allow you to see a *true normal* body temperature reading. While dieting, Free T3 tests are the only correct indicator of T3 levels, *not* body temperature. For example, using the thermogenic agent clenbuterol will raise body temperature and *lower T3 levels.*

Whenever you redo the Free T3 test, you should do another TSH test. It should not surprise you if your TSH is suppressed. Unfortunately, this is the price of admission for an optimal body temperature, not forever, but as long as you want the optimal body temperature. What? You thought that low body temperature somehow heals itself after a while? I wish. Many times, getting the right Cytomel or Synthroid dosage will

shut down TSH so that if you stop taking the medication "cold turkey," you will have low levels of natural thyroid hormone for 2 months. All of these things must be considered carefully before you "monkey around" with your thyroid.

By the way, although it is rare, I have encountered a few athletes who have normal to high T3 levels who still have a problem achieving optimal body temperature. Although thyroid is the chief regulator of body temperature, the adrenal system (adrenaline and noradrenaline) also has an effect. A faulty adrenal system can lower body temperature even when thyroid levels are high. You will find more information on the adrenal system in Chapter 26, "Thermogenic Agents and Body Temperature."

CHAPTER 25

(UGLY) LOWER BODY FAT

YOU'VE LEARNED A LOT about fat loss in the last 24 chapters. You've wrestled with catabolism and metabolic slowdown. You've taken a crash course in thyroid management. Many of you could jump into the diet right now and lose a good amount of weight, and might be able to reach your goal of a single-digit body fat percentage. However, many of you (especially women) would still encounter problems. In fact, you may already have discovered that there is a point when the stubborn fat stops melting away like the rest of the fat on the body. Some people have an alarming discrepancy between their upper and lower body fat, as if they had two different bodies glued together at the waist.

Bad things usually happen while chasing down the last of the sub-equator fat. Muscle wasting accelerates, especially in the face. You may become so disheartened that you abandon the diet and start a rampage of binge eating, which puts the fat back on. In the worst-case scenario, the fat from bingeing lands precisely where you don't want it: on your hips, thighs and buttocks.

Your doctor will say that it's "genetic," or "a female thing,"

and you can't "spot reduce." For about 12 years, I've pursued this lower body fat problem in female bodybuilders. I've tried every technique, from traditional to harebrained — more exercise in the area, fancy French rub-on creams and injections, anti-estrogens and steroids. Most of them had absolutely no success at mobilizing fat out of these areas. Money mobilizers, yes. Fat, no.

Well, if I didn't keep pursuing this problem, then the chapter would end right here with, "Lower body fat? You're fucked." However, you will see that this chapter is gloriously exciting. Lower body fat *can* be dieted off (without liposuction). Lower body fat does not just have more fat cells. It responds differently to weight loss because its response to certain hormones differs markedly from most of the other fat on the body. These crazy fat cells are not only the last to reduce, but just as importantly, they can cause further metabolic slowdown and catabolic action in areas far away from your butt.

Although this chapter specifically discusses how to target lower body fat, you can think of it as a bridge. It's a continuation of the previous chapter's discussion of thyroid and a prequel to the upcoming chapter on thermogenic agents. This chapter contains elements common to all three areas.

I'll warn you before we get started that this chapter contains the most complex (and worthwhile) information about fat. As usual, I will try to simplify the scientific jargon, condense the information and skip over the unimportant stuff.

You've read the headline: "Mother Lifts Car to Free Trapped Child!" Perhaps friends have told you about the "rush" from jumping out of an airplane. Or perhaps you've been involved in a near-miss auto accident, after which you have uncontrollable shaking and nausea. All of these events have one thing in common: adrenaline, the "fight or flight" hormone. Think of it as an octane booster to the body whenever you encounter a ***BIG*** stress.

In America, most physicians call this hormone epinephrine, and its close-acting sibling norepinephrine. Europeans and most non-doctors call them adrenaline and noradrenaline. I'll use the adrenaline tag because it's easier to remember. (I'm one of the syllabically impaired.)

Adrenaline and noradrenaline are hormones, naturally produced chemical messengers that impart instructions to various cells in the body. Adrenaline is produced by the two adrenal glands located over each kidney, which weigh about 5 to 7 grams each (for reference, an ounce is 28 grams). Noradrenaline, which is slightly different in chemical structure, is generated at the nerve endings.

Why are there two sources for this hormone? Because noradrenaline can only be generated in the nerves of certain cells. Cells that don't get much blood circulation (like fat cells, for example) aren't candidates for adrenaline, which is circulated systemically.

Some people think that adrenaline is only released under extreme conditions, because that's when it's most noticeable. In reality, both hormones are being produced and consumed continuously. The adrenaline siblings affect many of the same systems as thyroid hormone — body temperature, blood pressure, respiration and heart rate. In fact, the adrenalines and thyroid hormone have synergistic effects. Daily temperature fluctuations and fat distribution in part controlled by the adrenalines, which are properly called catecholamines.

Hormone receptors on cells are like assigned parking spaces, tailored in size and shape to each type of hormone.

Receptors for both adrenaline and noradrenaline are called adrenoreceptors. Adrenoreceptors are almost everywhere: in the blood, the organs, the muscles, and *the fat cells.*

Now it gets either interesting or too damn complicated, depending on what mood you're in. There are 4 types of these receptors (well, maybe 4-1/2), and each one communicates a different message to the cell. Sometimes a fat cell gets completely different messages from its receptor sites. Lower body fat has very screwy adrenoreceptors, much like my crazy old granny in my basement. (Remember, she's in the freezer now.)

Although adrenaline was identified in 1895, it wasn't until 1948 that scientists figured out that they were dealing with Sibyl receptors. At first, they found two receptors and named them alpha and beta. Over the years, they discovered more receptors, which they named alpha-1 (A1), alpha-2 (A2), beta-1 (B1), beta-2 (B2) and the very shy beta-3 (B3). The A2s are bad, bad characters.

Fat cells have both B1 and A2 receptors. Fat cells don't get much blood circulation, so it is noradrenaline that attaches to these receptors.

B1 receptors send good messages. They activate lipase, the enzyme that breaks down fat. Lipase causes the fat cell to disassemble itself, breaking down stored triglycerides into fatty acids and glycerol, which are used for energy throughout the body. B1s are the good guys, and it is noradrenaline that lights them up. Regular adrenaline would do the same thing if it could reach the receptor. However, fat doesn't have any major arteries or veins, only capillaries.

A2s are the bad guys. They block lipase in the fat cell. Worse, A2s also encourage the formation of triglycerides in the cell. A2s also (this was included at no extra charge) decrease the

generation of noradrenaline at the nerve sites. Less noradrenaline means that the good B1s don't light up as brightly. Oh well, lower body fat doesn't have many B1s anyway. The picture looks pretty grim: fat cell disassembly is blocked, more fat is stored, and — I almost forgot — body temperature is slightly reduced. Did I leave anything out? Ummm, yes. Low-calorie diets cause an increase in the number of the A2 receptors.

Let's envision how your lower body fat works. Lower body fat cells have very few B1 receptors, so they do not release much stored fat. They have a vast number of those pesky A2 receptors. When you go on a low-calorie diet, the following things will happen:

1. Fat is lost first and fastest at the cells with lots of B1 receptors.
2. Very little fat is lost in the fat cells that have lots of A2 receptors.
3. Eventually, your noradrenaline levels drop, reducing your body temperature.
4. The number of A2 receptors increases. The last of the fat becomes so hard to mobilize that the body will have to use more amino acids (from muscle) for fuel.
5. When you finally give up on the diet, even so-called normal eating will cause new fat accumulation right in the fat cells that have just increased their number of A2 receptors.

At first glance, it looks like you're screwed. It's not your thyroid. It's not an estrogen problem. Neither steroids nor antiestrogens will help. Ephedrine doesn't fit too well into the B1 receptor. Besides, there aren't very many B1s in lower body fat. It would be perfect if only we could get rid of the A2s or maybe

move them around a little ...

Wait a minute. With all of the anti-this and anti-that drugs available, isn't there something that would block A2s from accepting noradrenaline? Perhaps there's something that would leave the rest of the adrenoreceptors alone. If there is, gimme, gimme, gimme! I gotta have it! NOW!

This wonderful drug, this A2 antagonist, already exists. It's been around a long time, and it's not even high tech. It's a natural herb that even has FDA approval! It's yohimbe, the herb from the African tree bark, the male erection pill. Why hasn't it been used in the past for fat reduction? There isn't much scientific research on A2 receptors or their relationship with fat cells. Even though this herbal product has monumental implications for dieters, its entry in the *Physicians' Desk Reference* doesn't mention fat reduction at all.

I would like to say that all you have to do is take a magic pill like yohimbe (actually, 4 to 5 pills) and voilà, your lower body fat would take care of itself. I hate to rain on the parade, but yohimbe is not perfect in oral form.

Let me remind you that fat cells don't have very good blood circulation — what little there is comes from capillaries. Unfortunately, to adequately saturate the lower body fat receptors with yohimbe, you would be overdosing the rest of the receptors in the body. Yohimbe is a wonderful idea, but just taking a pill won't produce optimal results.

Yohimbe's effect needs to be *localized*. Doctors pooh-pooh spot reduction as a bogus marketing scam, like fat massage or cellulite wraps. On the contrary, new research shows that lower body fat reduction can be achieved with creams or direct injection into the fat.

You may have heard of aminophylline cream, an over-the-

counter asthma medication that appears to measurably reduce body fat. We don't quite know if this is caused by true lipid reduction or just a reduction of the water in and around the fat cell. Technically, aminophylline is supposed to be potentiating B1 receptors. However, stubborn fat doesn't have many B1 receptors. Direct local injection of yohimbe would work better.

Now that you understand adrenoreceptors, I can explain their interaction with thyroid hormone. Thyroid hormone stimulates muscle cells to burn more energy. Through the B2 receptor, both adrenaline and noradrenaline also send a heat message to the muscle cell. On a low-calorie diet, heat production from thyroid hormone decreases, which causes an increase of A2 activity and an attenuation of the B2 receptors. Sure, you could maintain body temperature by increasing thyroid medication, but you'd have to take so much that you would suppress natural TSH and thyroid hormone production. An additional hazard is that more thyroid hormone would speed up processes that shouldn't be accelerated, like heart rate, respiration and blood pressure.

Trying to keep body temperature optimal on a low-calorie diet by taking too much thyroid hormone will cause hyperthyroidism, even though your temperature is in the optimal range. Thyroid dosage should be optimized before you diet. The free T3 levels you found while at maintenance calories should not be increased while dieting. The interplay of low calories, thermogenic drugs and thyroid hormones can make maintaining optimal body temperature can be a real juggling act.

Although we've discussed lower body fat exclusively, which is usually a "woman thing," A2 receptors are present in other areas. Future research may find that fat distribution is intimately tied into A2 receptor distribution. Many male body-

builders, who have no lower body fat problems, have had a hard time stripping away body fat in the lower back area. Would yohimbe work in these areas? Or are androgen steroid receptors making the fat stubborn? We don't know, but the grand experiment is continuing.

CHAPTER 26

THERMOGENIC AGENTS AND BODY TEMPERATURE

THIS IS ONE OF THOSE heavy, scientific chapters, but cheer up — now that you know the essentials of thyroid hormone and the catecholamines, this information will be much easier to understand. Most thermogenic action, other than what we discussed in the thyroid chapter, is caused by the effect of noradrenaline on B2 adrenoreceptors. Increasing natural adrenaline will raise body temperature, but natural adrenaline is not specific. Some of its effects help you lose weight, but it may cause unwelcome changes too.

Rev up your imagination. Let's dream up a way to burn more calories, not from extra exercise but from non-shivering thermogenesis. The most natural way to do this is to encounter the right environmental stress — for example, you could immerse yourself in cold water. Your natural noradrenaline would be stimulated, and both the fat-mobilizing receptors (B1) and the muscle heat-producing ones (B2) would get the message. Studies have shown this technique to burn fat effectively while preserving muscle. It causes less catabolism than aerobic exercise.

However, when you think of thermogenic agents, you probably aren't thinking of a nice tank of freezing water, but pills. "Magic" pills, while non-natural, are more convenient and don't take up as much room. The ideal magic pill would activate B2 receptors specifically, with perhaps a side effect of B1 fat mobilization. It would raise body temperature without the need to reduce calories or add more exercise. It would help you burn fat without hunger or anxiety. These magic pills do in fact exist — they're called asthma medications.

Technically, these drugs are called beta-adrenergic agonists. For asthma sufferers, the main function of these drugs is to dilate the bronchial tubes to allow breathing. B2 agonists cause dilation of the smooth muscle that lines the lungs and other organs. In a separate action, B1 agonists dilate the blood vessels.

EPHEDRINE

Ephedrine, which is over-the-counter in America, is easy to acquire. It is a refinement of the Chinese herb Ma Huang (Ephedra vulgaris), which has been used in America since the turn of the century.

I have no idea when Western scientists first realized that ephedrine promoted thermogenesis. Chinese herbalists have known about the body-toning properties of Ma Huang for a very long time. For the rest of us, ephedrine became popular in the early 1970s in the Danish "Elsinore Slimming Pill." Athletes became aware of it in the late 1980s.

Studies on obese volunteers showed significant fat loss on a daily regimen of 50 mg of ephedrine 3 times per day. I first encountered ephedrine in late 1989 when the owner of AST Research was kind enough to forward some journal references to me. Since then, ephedrine has been embraced by the athletic

community, and its use in bodybuilding become more refined.

In America, over-the-counter ephedrine comes in 25 mg doses in either tablets or capsules. Ephedrine hydrochloride is cheap and widely available, although the more expensive ephedrine sulfate is easier to assimilate. The generic and brand-name versions of ephedrine hydrochloride are identical.

Research shows that ephedrine works synergistically with other drugs; both caffeine and theophylline will dramatically increase its fat-burning effects. Theophylline is the only drug that the FDA has approved to be "stacked" with ephedrine in the same pill. Primatene tablets and Bronchaid tablets, the two most popular brands of ephedrine, are ephedrine and theophylline combinations. For fat burning, caffeine works just as well as theophylline (and has less of an effect on the lungs), but this dieting duo was not able to get a license to cohabitate from the FDA.

Plain old aspirin has also been proven to extend the effects of ephedrine and caffeine. It's this menage à trois that causes the most exciting thermogenic effects. By 1993, scientists had created a real fat-burning orgy by adding grapefruit to the mix. The bitter component of grapefruit, naringin, prolongs the effects of caffeine by slowing down its breakdown in the liver. That old grapefruit-and-coffee diet wasn't completely bonkers.

The following dosages are the result of my work with athletes, not research studies. Do this combination 3 times per day — on awakening, at noon and no later than 4:00 p.m.:

Ephedrine	50 mg (two 25 mg OTC tablets) Most people prefer ephedrine sulfate, but ephedrine hydrochloride is cheaper. Ephedrine is also available in 50 mg tablets by prescription.

Caffeine	No-Doz, Vivarin or Pep-Back (between 100 mg and 200 mg) The research studies used 10 times as much caffeine as ephedrine, but this is much more than most athletes take. 500 mg of caffeine is a damn big honking jolt of caffeine. A cup of espresso or Middle Eastern coffee made from Robusta beans would be more sociable, but doesn't have enough caffeine to do the trick.
Aspirin	Half of an adult tablet or 1 children's aspirin
Grapefruit	It doesn't matter if it's whole or juice. Naringin is highly concentrated in grapefruit flowers and fruit peels. Refined, canned juices will not be as potent as the peel of the fruit.

The FDA, prude that it is, takes a dim view of this thermogenic orgy. You won't find an all-in-one pill. Some companies have circumvented the regulations by combining the herbal sources of the drugs.

In the place of ephedrine, you'll see ephedra or Ma Huang. Unfortunately, it's hard to tell until you sample the product how potent the herbal concentration is. For caffeine, the label will show South American guarana seeds or kola nuts. Aspirin is a refinement of white willow bark, and, of course, desiccated grapefruit flowers are actually a better choice than the fruit.

There's lotta love out there over ephedrine. It's legal, cheap and doesn't require a visit to the doctor. However, like all drugs, ephedrine does have some side effects. Ephedrine mimics some of the actions of adrenaline, but it does not fit completely into the

B2 receptor. In technical language, it has low receptor affinity.

Ephedrine's low receptor affinity has advantages and disadvantages. The B2 receptor stays "open," and the effects last longer than with more accurately matched molecules, like clenbuterol. Remember, ephedrine is not a pure B2 agonist. Ephedrine also hooks up with the other alpha and beta receptors, and affects noradrenaline generation.

Athletes don't like to talk about ephedrine's dirty big cousin, amphetamine. However, ephedrine's effects — increased heart rate, higher blood pressure and smooth muscle dilation — are like amphetamine on a small scale. Ephedrine has an annoying tendency to relax the bladder and dilate the smooth muscle in the prostate. This causes an urge to urinate although the urine flow is restricted from the slightly enlarged prostate. These side effects are not usually life-threatening, and seem small when contrasted with relief from asthma. However, the FDA is considering changing ephedrine's over-the-counter status because of what they consider frivolous non-medical use of the drug. Herbal concentrates would be yanked too. Today's legal diet trick may be tomorrow's prescription drug.

If you've never tried this ephedrine combination, I'll warn you that the effects are not subtle. You won't just feel warmer from the body heat; ephedrine and caffeine cause central nervous system stimulation. This is not like your morning coffee. It's more like ping-*ping*-*PING*-*PING*-RICOCHET-RRRABBITTT!! in a bottle.

CLENBUTEROL

Although ephedrine works, it isn't elegant. It is an agonist for all of the other adrenoreceptors in addition to the B2 receptor. Biochemists have pursued pure B2 agonists that have no B1,

A1 or A2 agonistic effects. The most popular, albuterol, has been around since the early 1970s. It's better than ephedrine, but it's not a perfect B2 agonist.

The B2 agonist that has gained worldwide attention is clenbuterol. An excellent oral asthma medication, it also has tremendous fat-burning and anti-catabolic properties. It is sold over-the-counter in many countries and is considered safe enough for use in liquid preparations for children and geriatrics.

Clenbuterol uses a very unusual thermogenic pathway: brown adipose thermogenesis (BAT). There is much controversy among scientists about how much BAT happens, and at what stage of human life.

Most body fat is the type that we see in the meat department in the supermarket. The fat cells vary in size and adreno- or androgen receptor make-up, but other than that, fat is pretty uninteresting stuff — not all that different from the leftover scraps from a steak dinner. Scientists call it white fat, even though it's kind of a pastel yellow color. Fat doesn't have much color because its only blood circulation is from capillaries, which are very small. How small? A micrometer is 1/1000th of 1 millimeter. Capillaries are between 5 and 20 micrometers in diameter.

Oxygen reaches all of the cells of your body through red blood cells (erythrocytes). Red blood cells are about 7 micrometers in diameter. When a 7 micrometer wide red blood cell travels through a 5 to 20 micrometer wide capillary, you can bet they go through one at a time. Fat is whitish largely because very few red blood cells get through it. Most of the liquid circulating through the fat is straw-colored plasma.

Fat that had a larger blood supply, from arterioles (branches of arteries) instead of capillaries, would be a different color.

More red blood cells flowing through the fat would make it appear a dark orange-brownish color. Scientists call this fat *brown fat*. Why don't they call it orange fat? I don't know.

Brown fat is different in many ways from regular fat. Not only does it have more red blood cells (which means more oxygen), but the cells also have mitochondria, which create energy. Let's see ... brown fat has more red blood cells, more oxygen, more mitochondria. Bingo! It's a recipe for heat production. You've just cooked up Brown Adipose Thermogenesis, BAT for short.

During the first few months of human life, brown fat is an important regulator of body heat. Babies have a lot more brown fat than adults. The remaining brown fat is located on your upper back between your shoulder blades.

BAT, like muscle cell thermogenesis, is regulated through B2 adrenoreceptor stimulation by noradrenaline. You can actually feel the thermogenic effect between your shoulder blades. Have someone place a hand there while you are using a thermogenic agent — it will feel warmer than the surrounding areas.

The lingering question for the scientists is this: Is the heat actually produced in the brown fat, or is the effect from hotter blood coursing through an area that has better circulation than the fat around it? Although many people want to think the heat comes from the brown fat, the evidence is beginning to suggest that BAT in adults is caused by hotter blood.

Clenbuterol does more than just cause brown adipose thermogenesis. It is an agonist to all B2 receptors, including heat-producing muscle. Clenbuterol is much more potent than ephedrine because it is has a much better receptor affinity. Clenbuterol burns fat better than both albuterol and metaproterenol.

As an added bonus, it has anabolic and anti-catabolic properties that help to increase muscle mass while *dieting*.

Bang! The initial research on humans contained some amazing body transformations. It looked as if the fat was turning into muscle. The Scottish University study hit the British newspapers in 1988, and I learned of it in time to include it in my *Underground Steroid Handbook II*. Talk about a bomb going off! By the time I re-entered the athletic community in 1990, clenbuterol use was in full swing. A totally fat sedentary slob sitting in an airport lounge in 1988 had turned into a national fitness model by 1990. I could fill another book with stories of such clenbuterol transformations.

Clenbuterol wasn't ever approved by the FDA, but for a while it was easy to come by. It was imported for personal use. It was imported as a raw powdered research chemical and made (underground) into tablets. It was easy to make human versions from the raw chemical. I'd whip up a few bottles of liquid clenbuterol drops for my friends in my own kitchen. As usual, the official party pooper put out Alert Bulletins to stop clenbuterol importation. Nevertheless, a lot of clenbuterol still sneaks into the country.

Clenbuterol is potent. A standard 20 mcg tablet is more thermogenic than a 25 mg tablet of ephedrine. Through almost 5 years of trial and error, we've refined its use as a thermogenic and anabolic agent. Clenbuterol has rattled sports organizations so much that it is often grouped in the same class as anabolic steroids! Currently, it is still legal to own and use in America. Although you cannot use the personal use exemption, you can legally bring it back from a foreign country with a foreign doctor's prescription. Don't be surprised if FDA puts it on the same schedule with steroids, making it much harder to acquire

and use legally.

You might think that because I've helped research, refine and apply clenbuterol, I must be the de facto leader of the Clenbuterol Cult in America. That assumption is incorrect.

I think that the athletic world became so enamored with clenbuterol because, besides anabolic steroids, there isn't another drug with the same bang-for-the-buck, instant-gratification attributes of clenbuterol. To anyone who has seen or felt its effects, they are eerily extraordinary.

I've seen some absolutely remarkable transformations over just a 2-week period. Body fat virtually vanishes and muscles grow bigger and more toned. Even at normal dosages of 2 to 5 tablets per day, clenbuterol has fewer side effects than ephedrine and, of course, much greater potency.

Nevertheless, I'm sorry to say that not everything is hunky-dory with clenbuterol. This roaring thermogenic effect appears to last only 2 to 3 weeks before petering out rapidly. After the body adjusts to clenbuterol through a very complex down-regulation of the B2 receptors, body temperature cools back to near normal.

This rapid attenuation of thermogenic and anabolic effect was noticed almost immediately in laboratory animal experiments. The researchers concluded that the B2 adrenoreceptors were down-regulating. First, the B2 receptors become increasingly insensitive to B2 agonists because of the higher temperature. Then, the receptors "burrow" into the cell so the agonist can't reach them. Next, they are deactivated through phosphorylation of the receptor. Ultimately the actual number of receptors decreases.

This is not earthshaking news. We've known this about natural adrenaline for decades. If there's anything remarkable

about these effects, it's that the attenuation is so rapid. The researchers suggested a dosing schedule of 14 consecutive days of clenbuterol use, followed by an alternating schedule of 2 days off, then 2 days on. This appeared to reinitiate the beneficial effects in the laboratory animals, but I don't know how long this new dosing arrangement succeeded. However, in working with numerous athletes, I found that this strategy didn't work for very long. The remarkable intensity of clenbuterol's initial action doesn't appear to repeat itself.

When reviewing blood test results of bodybuilders using clenbuterol, I noticed a pattern. Serum Free T3 levels would drop very much like classic Euthyroid Sick Syndrome. I theorized that clenbuterol was interfering with the T4 to T3 thyroid conversion. Cytomel works quite well to adjust the T3 level (and body temperature) back to normal. At this point I was pretty happy with what was happening in Clenbuteroland. By combining the researchers' dosing schedule (2 days on, 2 days off), and supplementing with thyroid hormone, we seemed to achieve a continuation of thermogenic effect. It wasn't dazzling, but at least we got a consistent thermogenic effect without escalating the dosage of clenbuterol into the stratosphere. Some athletes were using 400 mcg (that's 20 tablets) each day. Most people would say "case solved" and move onto other things, but I couldn't leave it alone.

Eventually, even with this careful dosage schedule, two things will happen. First, your B2 receptors will down-regulate. In addition, somewhere along the way your natural ability to produce adrenaline and noradrenaline will down-regulate, too. At this point, clenbuterol is not an addition to your body; it replaces normal production.

The problem is that the damage is done already. Increasing

the dosage of clenbuterol will just decrease the number of receptors and the amount of noradrenaline even further. Chasing your plummeting body temperature with additional T3 is initially effective and somewhat benign, but escalating dosages of Cytomel will cause a suppression of natural thyroid production and hyperthyroidism. When you finally stop using clenbuterol, there is a staggering systems crash.

It will take at least 2 weeks (and usually more), for the adrenal system to become fully functional. Until then, alertness, body temperature and energy will be noticeably diminished. When coming off clenbuterol, athletes look and feel like, well, shit. It's not life-threatening and not like the kind of withdrawal caused by other drugs. It's less severe and doesn't last as long as withdrawal from anabolic steroids. Perhaps clenbuterol is best taken in infrequent 2-week dosing periods every 2 months. Unfortunately, clenbuterol's effects are so dramatic and enticing that most people won't leave it alone.

Because of clenbuterol's problems — its limited span of activity and unpleasant withdrawal effects — I have abandoned it. Although it is visibly effective and not entirely dangerous, its problems escalate with continued use. Unfortunately, it still happens to be the only useful anti-catabolic agent besides anabolic steroids.

I now work with another, more potent, thermogenic agent that does not affect the adrenal system at all. As the compound is still under development, it is not discussed in this book.

Researchers have recently isolated another adrenoreceptor that regulates heat production exclusively, the B3 receptor. Drugs that affect this receptor should produce a more tailored thermogenic response. For now, however, B3 agonists are still in the research stage.