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Creatine: Physiology, Supplementation, and Exercise

By Christie Engesser

Introduction. The intense competitiveness of today's athletes compels them to seek any tactics yielding improvement, including supplements and ergogenic aids. Most athletes will not consider banned substances, but will turn to any and every "wonder drug" that appears in the market. Unfortunately the information on which most athletes' decisions are based is likely to be found in popular fitness magazines, not refereed science journals. Anytime a substance is ingested into the body, there is equal potential for positive and negative results. Thus, before any kind of ergogenic aid is added to a training regime, a thorough investigation of the supplement should be undertaken to ascertain the specific purposes of and functions of the supplement. Users should be aware of proper ingestion guidelines and potential side effects.

A relatively new ergogenic aid called creatine is surfacing among collegiate and elite level athletes. Creatine is advertised as an energy enhancing molecule. Yet the athletes who supplement their diets with it are likely to possess limited information regarding the reasons for supplementation. After a brief review of creatine's physiological function, supplementation with creatine monohydrate will be discussed, focusing on its specific muscular and exercise effects. Finally, recommendations will be made for the athlete.

The Physiology of Creatine and Phosphocreatine. Creatine, first discovered in 1832, is a naturally-occurring molecule produced from the amino acids glycine, arginine, and methionine in the liver, kidneys, and pancreas. Blood transport shuttles the creatine to its useful site in the skeletal muscle fibers, where 95 percent of the total creatine in the body is located. Once inside the body, creatine is stored, predominantly in skeletal muscles. The body maintains a storage pool of approximately 120g, two thirds of which is stored in the phosphocreatine form and one third in the free creatine form (1). As might be expected, the body does not store all of the ingested creatine; rather the excess is converted to creatinine and lost in urine. In fact, the average male has a turnover rate of approximately 2g/day, which is counterbalanced by an intake close to 1g/day of creatine from various animal products present in the diet.

Creatine functions in two ways, as a storage site for high energy phosphates to buffer shifts in the adenosine triphosophate (ATP) dephosphorylation reaction and as a phosphocreatine shuttle (2). The removal of this phosphate from an ATP molecule produces the energy required for muscular contraction. Even more important is the reverse reaction in which phosphocreatine rejoins a phosphate with adenosine diphosphate (ADP), thereby yielding another ATP molecule available for energy production. Creatine stores the high energy phosphate and helps regulate the equilibrium of the reaction by phosphorylating an ADP when it is thermodynamically favorable. Greater stores of phosphocreatine allow for phosphorylation of ADP at an increased rate and thus improved physical performance.

From the biochemical perspective, creatine is critical to the ATP supply (2, 3). Creatine is often specified as the main energy transport mechanism for the muscle, a statement supported by the ATP/creatine energy production reaction. Creatine's major role in energy production is its ability to continually phosphorylate ADP. Thus phosphocreatine is the critical form of the molecule. Interestingly, Meyer et al. point out that 50 percent of the phosphocreatine store is used in the same time period that 0.5 percent of the ATP concentration is used (2). This suggests that at the onset of activity, ADP is being phosphorylated faster than ATP is being depleted, which is beneficial for muscle contraction. Hence the idea that creatine and phosphocreatine act as a shuttle is supported in the shuttling of energy (in the form of phosphates) between molecules. Further evidence from Meyer et al. includes the finding of a significant decrease of ATP stores once

creatine kinase, the catalysis for the reaction, is removed. Too, a greater ATP:ADP ratio produced by the increased phosphocreatine stores results in muscle fiber relaxation, and relaxed muscle has a greater potential to generate force via contraction. Lastly, phosphocreatine depletion in rats has been shown to increase fatigue when compared to controls in anaerobic stimulation.

Supplementation and Muscular Effects. Before investigating the possible muscular effects of creatine supplementation for performance athletes, it is necessary to establish that the human body will absorb excess quantities of the substance. A 1992 study examined the effects of ingesting 5g of creatine monohydrate, four to six times daily, for periods ranging from 4.5 days to every other day for three weeks (4). (Five grams of creatine monohydrate is comparable to the amount of creatine in a 2.4 lb. uncooked steak.) In addition to the various supplementation schedules, 5 of the 17 participants performed 60 minutes of exercise with one leg each day. Results from the study show that creatine supplementation in the pattern described above does in fact increase the total amount of creatine stored in the muscle. This dosage is currently used as the standard ingestion schedule for supplementing with creatine monohydrate. This study and similar work report 1) an average of up to 20 percent increase in creatine levels, 20 percent of which is in the phosphocreatine form, 2) higher uptake of creatine in the initial days of supplementation, 3) exercise as facilitator to creatine uptake, and 4) no negative side effects (5, 6).

Nonetheless, supplementing with creatine serves no purpose if the increased stores of creatine do not positively affect the resynthesis rate of phosphocreatine, the essential molecule for the ADP/ ATP conversion reaction. Research undertaken by Greenhaff and associates (5, 6) indicated increased rates of phosphocreatine resynthesis after creatine ingestion of 20g/day for five days. Both studies used electrically stimulated contractions for periods of 1.6 seconds with equal rest intervals. Muscle creatine levels were accessed at intervals of 0, 20, 60, and 120 seconds following the contractions. Specifically, results indicate that phosphocreatine resynthesis increased one minute after activity. They found a positive correlation between total creatine content and increased rate of phosphocreatine resynthesis, indicating that muscles with higher creatine stores are likely to have an increased rate of phosphocreatine resynthesis.

Other findings indicate creatine has potential for use as an ergogenic aid, but also suggest limits to its application. Supplementation has been reported to lower muscle lactate levels (7), but does not benefit blood lactate levels or oxygen consumption (8, 9, 10). The literature reports no health risk with short-term high dosage ingestion schedules (5, 6). Long term effects are unknown, however, particularly with regard to long-term, high-dosage patterns of use.

Because high-intensity exercise needs greater amounts of ATP, increased phosphocreatine stores, necessary to phosphorylate ADP molecules, should positively affect the work done by the muscle fibers. Therefore, it has been theorized that creatine will benefit short duration high-intensity exercise bouts such as an 800-meter track race or a 200-meter freestyle swimming event. One reason for this idea is the fact that high-intensity exercise, often anaerobic in nature, can deplete phosphocreatine stores 10 seconds after onset of activity. Stores are depleted rapidly because anaerobic exercise is largely produced by Type II or fast twitch muscles, which Balsom reports have a higher level of phosphocreatine than Type I fibers (1). This is a key finding with regard to potential benefits of increasing creatine stores to prepare for high-intensity exercise or Type II muscle action. Because the presence of creatine is critical to the release of energy from ATP, creatine levels are considered to be limiting factors in muscular force and energy production.

Supplementation and Exercise Effects. It is clear that creatine plays a crucial role in energy production for muscle contraction and that supplementation with creatine monohydrate supplementation can increase creatine stores in the body. To determine the effectiveness of the substance as an ergogenic aid, however, it is necessary to observe its effects on exercising athletes. Four studies help clarify the possible benefits and consequences of increased creatine consumption among athletes. Although this is a relatively small research pool from which to make recommendations, our understanding of creatine is still developing and applied research is

currently advancing.

An investigation of the effects of creatine stores on five sets of 30 repetitions of maximal isokinetic knee extensions (6, 10) was one of the first studies to be conducted involving oral creatine supplementation and exercise. Twelve participants were randomly placed in placebo and experimental groups. During the ten days between the pre- and post-tests, each participant ingested either 6g glucose or 5g creatine monohydrate plus 1g glucose four times a day for five days. Measurements of skeletal muscle isokinetic torque from this double blind study indicate that oral supplementation benefits torque production. Peak muscle torque is maintained at higher levels when 20g of creatine is ingested for five days. Increased ATP resynthesis, possible because of the increased phosphocreatine stores, also causes a decreased accumulation of plasma ammonia, which contributes to the reduced decline in rates of peak muscle torque.

The discovery that short duration high-intensity repetitions were facilitated by creatine supplementation was a springboard for further research. Half of the eight subjects in a 1995 double-blind study ingested creatine monohydrate in the same manner as documented by Greenhaff et al. (6, 10) and Harris et al. (4). After 14 days, anaerobic power was measured and found to have increased in all three trials in the experimental group. No differences were observed in the placebo group. In addition, body composition and bench press ability were assessed after 28 days. Interestingly, body weight was found to have increased significantly in the supplementing group. Volume of lifting significantly increased, due to a substantial increase in the number of repetitions. (Although this evidence appears to speak for itself, it should be noted that the company supplying the creatine monohydrate supplement for the investigation also financed the research.)

The previous studies involved anaerobic exercise, meaning exercise using energy produced with insufficient oxygen. This type of exercise, primarily powered by Type II muscle fibers, is not limited to muscular contractions and weightlifting. In a third study, interval running workouts performed both pre- and post-intervention included one workout of four 300m runs with three minutes rest between repetitions and a second workout of four 1000m runs with four minutes rest (12). The group supplementing with creatine monohydrate improved significantly more than the placebo group. The single blind study resulted in lower times for the final 300m repetition and for all four 1000m trials in the test group. This evidence indicates that sustained anaerobic activities can benefit from creatine supplementation.

An obvious question remains. Where does aerobic, or endurance, activity fit into this picture? Balsom et al. (8) attempted to answer this question by examining effects of creatine supplementation on a treadmill test at 120 percent max VO2 and a 6,000m terrain run. Eighteen male participants completed the two pre-tests and then, in a double blind fashion, were divided into a creatine and a placebo group. After six days, the tests were repeated. Treadmill tests produced no differences in either group. Over the 6,000m terrain run the placebo group showed no change, but the creatine group showed a significant change in performance. Interestingly, the change was negative, indicating that performance time was actually worse after creatine supplementation. Other findings include statistically significant increases in blood lactate levels and body mass in the creatine group. Possible explanations for the lower performance level with supplementation of creatine are tied to the increases in body mass. That is, the weight gain that accompanies supplementation may actually slow runners.

In addition, during endurance activity phosphocreatine stores do not decrease as rapidly because primarily Type I muscle fibers are used in such activity. This circumstance produces a diminished demand for ADP phosphorylation, so phosphocreatine stores are not the limiting factor for this type of exercise (2). Although producing unfavorable results FOR ENDURANCE SPORTS, the investigation helped to validate the present theory that creatine supplementation primarily benefits short duration high-intensity exercise.

Practical Applications. For competitive athletes, there is vital information to be drawn from the

research. The first step, as with all supplements, is to ask oneself, "What will supplementation do for me?" The type of exercise undertaken has been shown to be crucial for predicting the benefits of creatine. An athlete engaged in endurance activities should think twice about using creatine, especially if the inevitable increase in body mass may be a hindrance to performance. For instance, a cross country runner would not be a likely candidate for creatine monohydrate supplementation, but the same runner competing in the 800m or even 1500m on the track may benefit due to the increased ability to complete maximal effort interval workouts. In general, it is recommended that athletes primarily engaging in high-intensity, short duration performances consider creatine as an ergogenic aid.

On the shelf, the supplement will likely be labeled creatine monohydrate, which is a white powder that may be dissolved in a warm liquid. The elevated temperature is necessary to decrease the amount of creatine that is directly converted to creatinine and subsequently lost from the body (4). The standard ingestion schedule begins with a 5-day loading phase, when doses of 5g are ingested four times each day. Following the loading phase is a maintenance period during which 1-2g/day are ingested. Because the body has a maximal creatine storage level near 150-160mmol/(kg dm), taking 20g/day past the first five to six days only costs money. The maximal creatine storage level can vary with age, but not necessarily with gender or training (7), so athletes training at higher levels do not need to increase the dosage. Another point crucial to the loading schedule is the lack of information regarding the washout time. Maughan reports that muscle creatine content will remain high several weeks after the loading phase, but no specific time period is known.

The increased amount of creatine that each person will store with supplementation is related to the initial level of creatine stores in the muscle. The greater the creatine level, the less potential there will be for large increases in creatine stores. Conversely, muscle tissues with lower initial creatine levels have a greater potential for large increases in creatine stores. Athletes on vegetarian diets may have no external source of creatine and must rely solely on internal synthesis, which is usually not adequate to cover the body's turnover. As a result, they may especially benefit from supplementation (1, 4, 6, 10).

Of course, each athlete needs to consider the ethical and health consequences of supplementation. Creatine monohydrate, at the present moment, is not a banned substance. Because it is considered an ergogenic aid, there is a possibility that some people will attach a negative stigma to its use. Nonetheless, it is a substance that is synthesized by the body and found in the diet in animal products. The idiom "more is better" does not apply as the body will only retain a fixed amount of creatine in the muscles. And for vegetarians, the supplement is a tool allowing them to step up to the starting line with the same stores as their meat-eating competitors. From the health standpoint, no negative side effects have been noted, but long term studies have yet to be reported. Although the body reduces its creatine synthesis during periods of supplementation, normal levels do return upon cessation of creatine monohydrate ingestion. Hence, creatine supplementation can be argued to be a safe and ethical boost to performance.

Clearly, more research is needed to determine creatine's most advantageous use as an ergogenic aid. Questions concerning benefits in more specific settings need to be addressed. For instance, what is the correct recommendation for the basketball player who plays 36 minutes of the 40 minute game, which is aerobic in duration, but is filled with anaerobic efforts? In addition, the supplementation schedule should be examined for optimal timing during an athlete's season. Should the loading phase be initiated in the pre-season or closer to the end? If it is initiated in the beginning of the season, is it necessary or beneficial to complete another loading phase after a period of time? What is this time period?

Long term benefits and consequences also should be thoroughly examined. The results do not just concern athletes, as uses for creatine to counteract muscular diseases are also currently being investigated. Long term effects are particularly important in this setting because it is conceivable that supplementation could be a lifelong practice. With more research, safe and effective

guidelines for creatine monohydrate supplementation can be confidently expanded.

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Endogenous Opioids: The Body's Natural Painkillers

By Stephanie Miller

Introduction. Pain is one of the body's most important adaptive mechanisms, with its primary function being protection. The brain is quickly notified that tissue damage is occurring or that it is about to occur so that behavioral responses may intervene to avoid or minimize that damage. If tissue damage has occurred, pain plays a part in hindering activity in order to make the environment more conducive to healing. In a "fight or flight" survival situation, however, pain would have negative effects. Therefore, the body produces substances, endogenous opioids, that have analgesic properties to make us better able to tolerate pain. These substances -- endorphins, enkephalins, and dynorphins -- also have behavioral effects and neurotransmitter and neuromodulator functions (1). Endorphins were named by combining the words endogenous and morphine (2) and their discovery occurred when a substance exhibiting the analgesic characteristics of morphine was extracted from the brain. This discovery was confirmed when the action of the substance was blocked by the morphine antagonist naloxone (3).

The pain response begins with an undesirable stimulus activating one or more types of peripheral pain receptors known as mechanical, thermal, and polymodal nociceptors (4). Information about the stimulus travels along these afferent nerves to the spinal cord, where the neurotransmitter substance P is released. Without the transmission of substance P, no pain perception information can travel to the brain to be processed. Substance P binds to spinal nerve receptors to allow the noxious information to travel to sites where it can be processed, including the brain stem, thalamus, limbic system, and somatosensory cortex.

Two descending pathways from the brain regulate pain. A pathway stretching from the locus caeruleus of the medulla to the dorsal horn acts as an analgesic by releasing norepinepherine, which inhibits the release of substance P. The second pathway, which begins in both the periaqaductal periaqueductal gray matter in the midbrain and the nucleus raphe magnus of the medulla, releases serotonin in the dorsal horn. The latter pathway is vital to the understanding of the actions of endogenous opioids because the areas along the pathway are rich in opioid peptides and opioid receptors, and serotonin induces the release of opioid peptides. After descending from the brain, these substances prevent the transmission of the pain signal by inhibiting the release of substance P (4).

Classifications. The body contains three types of endogenous opioids: beta-endorphins, enkephalins, and dynorphin. Beta-endorphin cell bodies are primarily found in the arcuate nucleus of the hypothalamus, a structure in the head that regulates biochemicals produced by the body. Beta-endorphin activity also has been found in the pituitary gland, a feature that distinguishes this group from the enkephalins, which are not present in that area (5).

Enkephalins may be broken down into two types, methionoine enkephalin (met-enkephalin) and leucine enkephalin (leu-enkephalin), and their ratio is 4:1 respectively (6). They are more widely distributed in the brain than beta-endorphins, being present in several areas including hypothalamic nuclei, limbic structures, caudate-putamen, the brain stem, several layers of the dorsal horn, peripheral nerves, and the adrenal medulla. They were the first endogenous opioids extracted. The most powerful of the opioids, dynorphins, are found throughout the central and peripheral nervous systems. Some research supports the theory that they regulate pain at the spinal cord level, influence feeding behavior at the hypothalamic level, and function with other endogenous opioids to regulate the cardiovascular system. Dynorphins also may be involved in inhibiting intestinal motility, a phenomena that occurs when the body perceives pain. The presence

of a large precursor to this opioid in the anterior pituitary suggests that it has many peripheral targets (7). Another opioid called neo-endorphin also is classified in the dynorphin group.

Receptors Five distinct types of opioid receptors are found in the body: mu (with subtypes mu-1 and mu-2), kappa, delta, sigma, and epsilon. Each receptor type binds differently with the various opioids.

The mu receptors are primarily located at sites above the spine including the medial thalamus and brain stem areas. Some are also found in the spinal cord. Research has found that the mu-1 receptors are primarily responsible for analgesia and euphoria, and bind with opiates, beta-endorphins, and enkephalins. Mu-2 receptors, on the other hand, have been associated with the respiratory depression associated with morphine (8,9). More is known about the mu-receptor than any of the other types because the morphine antagonist naloxone has a much higher affinity for this receptor type (10). Using this substance has helped researchers learn more about the actions of substances produced by the body. In addition, this drug is used to treat morphine addiction and study withdrawal symptoms. In this use, repeated doses or continuous infusion is required for total reversal of drug effects because morphine outlasts the effects of a single dose of naloxone.

Kappa receptors are located primarily in the dorsal horn of the spinal cord and have a great affinity for dynorphins and neo-endorphins. These receptors also are found in the medullary reticular formation of the brain stem, which allows kappa-specific drugs to produce abnormal contraction of the eye's pupils (miosis) and sedation. Some work indicates that kappa receptors are distributed in the cortex (11). These receptors are not responsible for the euphoria, physical dependence, and respiratory depression associated with morphine but function primarily in spinal analgesia (3). Because no kappa antagonist exists at this time, the actions of dynorphins and neo-endorphins are not fully understood.

Sigma receptors are concentrated in the amygdala and hippocampus and the limbic system brain (8). Hallucinations, sensations of discomfort, and feelings of anxiety result when opioids activate these structures.

Delta receptors are poorly understood, but are known to play a role in analgesia as well as in reinforcing the actions of morphine. They appear to be enkephalin receptors (3,12) and are distributed primarily in the limbic system (11). Both enkephalins and beta-endorphins have equal affinities at these receptors (9).

Some research indicates that the epsilon receptor is specific to beta-endorphins (5,12), which are primarily distributed in the hypothalamus.

Actions. Enkephalins have been associated with addiction to and withdrawal from morphine. Morphine intensely stimulates the enkephalin receptors which causes a negative feedback mechanism suppressing the activity of the enkephalin neurons. This decrease in enkephalin neural activity necessitates an increase in the amount of morphine in order to maintain the same level of analgesia. This biological phenomenon is responsible for the drug tolerance and addiction associated with morphine.

One theory of morphine addiction states that when morphine administration is discontinued, neither morphine nor enkephalin is present, so withdrawal symptoms occur until enkephalin activity resumes (3). Several studies, however, offer conflicting views. Some researchers believe it is possible to be addicted to endogenous opioids, but there is no proof that chronic administration of morphine results in the down regulation of endogenous opioids (13).

The most popular action of endogenous opioids is their creation of a feeling of euphoria during exercise, which endurance athletes frequently refer to as "runner's high." Numerous investigators

have observed an increase in blood plasma levels of beta-endorphin following physical activity (14). However, plasma levels of this substance have not been found to increase proportionally with exercise intensity.

The endogenous opioid system has been used to treat pain through a technique called neuroaugmentation. This technique involves electrical stimulation of specific areas of the brain or brain stem to increase the quantity and reactivity of endogenous opioids. Partial or complete pain relief has been noted in 27% to 76% of the patients treated with neuroaugmentation; lower levels of efficacy were observed in severely ill cancer patients (15). Spinal cord stimulation was found to be successful in treating chronic pain not associated with malignancy.

In addition to analgesia, endogenous opioids have also been found to maintain feeding behavior (16). This finding is supported by research indicating enhanced feeding responses following beta-endorphin injections and work noting that naloxone inhibited these responses. Endogenous opioids also have been linked to moderation of drinking behavior (17) and cough suppression (12).

Endogenous opioids have a great variety of functions throughout the body. Their distribution and the distribution of the opioid receptors ensure a highly effective pain management system. This distribution also indicates that these substances possess many other functions that merit further investigation.

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How Well Are We? A National Profile of Wellness Finds Good and Bad News

By Carolyn Petersen

Americans have been hearing about the need to eat less fat, quit smoking, and exercise more often for more than 20 years. Yet a glance around any busy shopping mall makes it clear that many of us are not heeding these messages, at least not to the degree health officials tell us we should. So how much progress have we made in improving our health?

Wellsource, a Clackamas, Ore.-based developer of information management systems for wellness programs, has surveyed more than 200,000 people to find out how those with access to worksite-based wellness programs are doing. The survey is part of an ongoing effort to establish a national database on wellness practices. The firm used self-reported health and lifestyle information from wellness program participants to evaluate the nutritional status, exercise habits, and overall health of the population in a variety of professions. The result: Americans are doing some things well, but have ample room for inprovement.

Eating Habits. A significant number of participants report efforts to limit their intake of dietary fat. About 45% eat a combination of red meats, poultry, and fish, and 48% prepare meals using low-fat cooking methods such as baking. Forty percent opt for low-fat dairy products when available. About 31% select low-fat desserts, but an equal number consume both low-fat and high-fat sweets. Approximately 55% eat snack regularly, while 43% snack only occasionally.

At first glance, these figures seem positive. But fruit and vegetable consumption patterns highlight the difficulties many Americans experience in eating a balanced diet. Just 6% of the survey participants report eating the five or more servings recommended, and 83% said they eat three or fewer servings. Fruit and vegetable consumption is an important indicator of nutritional health because produce contains little fat, many vitamins, and plenty of fiber - which has been linked to lower rates of cancer and heart disease.

With Westerners' year-round access to high quality fresh foods at moderate cost, it was no surprise that they lead the U.S. in fruit and vegetable consumption. But like confirmed junk food junkies, healthy eaters too stay with their diets, regardless of where they live. Seven percent of Westerners reported eating 5+ servings of fruits and vegetables each day, but rates were nearly as high in the South (6.5%) and the Northeast and Midwest (5.4%). Those over 40 consumed more servings than the under-40 age group, perhaps due to the fact that young people have grown up eating sugary snacks that were unknown before the 1950s and 1960s.

Exercise. Despite their healthy eating patterns, Americans living in Western states reported the lowest level of physical activity, with just 27% getting some type of aerobic exercise three or more times a week. Midwesterners had the highest participation rate, 36%, followed by Southerners (32%) and Northeasterners (29%).

The group also was questioned about stretching and strength training participation. Nationally, 34% indicated they perform some strength-building exercises each week, while 47% reported involvement in a weekly stretching activity.

Stress Management. The efficacy of stress management programs has been questioned as long as employers have provided classes in dealing with life's challenges. Although the survey was not

designed to evaluate the effectiveness of such educational efforts, survey participants indicated a strong need for stress management training. Overall, men and women between the ages of 30 and 50 reported the highest levels of stress. Women felt stress more strongly than men during their 30s, while men's stress levels outdistanced women's levels during their 40s.

Nationally, 22% reported that they were seldom stressed or were coping very well. By region, the rates of comfort were nearly equal, with Southerners the most relaxed (23%), followed by Northeasterners (22%), Midwesterners (21%), and Westerners (20%). Nearly 12% rated their emotional states as "not too happy" or "very unhappy."

Medical Data and Health Status. Health assessment surveys typically contain questions that evaluate an individual's risk for specific diseases or conditions by looking for risk factors such as high blood cholesterol and obesity. In general, few individuals have no risk factors or very high numbers of these indicators. This trend was true in Wellsource's wellness profile. About 7% of respondents reported the presence of 6 or more health risks, while just under 4% reported no risk factors. Of greatest concern was the large number of individuals reporting multiple risk factors: Nearly a quarter of those surveyed have 4 or 5 risk factors, while 46% have 2 or 3 factors.

Overall health status can be difficult to measure, since the presence of one risk factor (e.g., slightly elevated blood pressure) may not hamper the respondent in daily activities. Survey questions addressing general health issues indicated several statistics about Americans' health:

- Sick days: 41.5% took no sick days in the past year, while 20% took three or more days off
- Hospitalization: 6.5% reported spending at least one day in a hospital during the past year
- Wellness: 33% rated good to excellent in terms of overall wellness;
- Fitness: 26.5% rated good to excellent in terms of overall fitness, while 53% rated poor.
- Coronary risk: 53.7% were rated at low risk for heart disease, and 25.7% were rated at high risk.

The average age of survey participants was 40, with most between 20 and 60 years old. About 52% of respondents were female. Approximately 78% of those surveyed were Caucasian, with the remainder being of African-American (7%), Hispanic and Asian (2.5% each), and Native American (1%) descent. Southerners accounted for 49% of the responses, while the rest of the surveys were distributed fairly evenly among Westerners (19%), Northeasterners (17%), and Midwesterners (14%).

Book Review

Westcott, W. 1995. *Strength Fitness: Physiological Principles and Training Techniques (4th ed.).* Dubuque, IA: Wm. C. Brown Communications, Inc.

Although strength training has long been associated with improvements in physique, it is now credited with such health benefits as increased bone density and reduced body fat. Wayne L. Westcott, Ph.D., who is one of the country's leading authorities on these topics, presents the specifics of these concepts in the fourth edition of his book Strength Fitness.

The 13 chapters of *Strength Fitness* were written for both beginning and advanced strength trainees, and they include recommendations for youth and senior exercise participants as well as fundamental concepts in nutrition, exercise physiology, and biomechanics. Other excellent sections of this book include a glossary of common (but often forgotten) fitness terms, six handy appendices that help trainees estimate muscle length and fiber type, and gauge progress with a strength training logbook.

A key feature of this book is the last chapter, which provide readers with a selection of 32 recommended exercises. Many of the exercises presented are appropriate for beginning strength trainees and can be adapted to both free weights and machines. Other featured exercises would probably better suit participants with intermediate or advanced skills. For example, it is not likely that most beginners would need some of the calf or neck exercises as part of their basic workout. With the inclusion of these exercises however, the book appeals to a broader audience. A strength of this chapter lies in the black and white photo-illustrated pages, which show starting positions for each exercise. Below each illustration is a description of the exercise including an assessment of joint and muscle action as well as bulleted procedures for performing the movement.

No attempt is made to foist a "method" upon the trainee, and the author makes a point to emphasize the basic scientific principles that underlie sound strength training programs. The five-page bibliography is a treasure trove of useful references from well respected researchers in exercise science. Overall, *Strength Fitness* is a timely, engaging text and is an appropriate reference for strength trainees of every ability.

Christopher Berger

Role of Non-Steroidal Anti-inflammatory drugs (NSAIDs) in Sports Medicine

By Christopher Berger

Introduction. Non-steroidal anti-inflammatory drugs (NSAIDs) are widely used medications taken by athletes of every ability. Although NSAIDs are commonplace in sports medicine, their role is not well understood. Despite the marketing finesse of pharmaceutical companies, it is often difficult for exercise participants to discriminate among the various over-the-counter products available. In the case of stronger prescription NSAIDs, the athlete or athletic trainer may not anticipate the serious side effects of these drugs. It is the latter situation that is most often studied by NSAID researchers because although comparative efficacy has not been definitively studied, a growing body of research documents substantial differences in toxicity among drugs (1). This paper explores the role of both non-prescription and prescription NSAIDs in sports medicine and attempts to provide athletes and those who work with them with an understanding of the beneficial actions and negative effects of these common drugs.

What do NSAIDs do? NSAIDs exhibit analgesic, anti-pyretic (fever reducing), and anti-inflammatory properties. They are taken by athletes for relief from a variety of disorders including acute injuries (e.g. muscle strain) and chronic injuries (e.g. tendinitis) (2). As analgesics, NSAIDs are effective only against pain of low-to-moderate intensity. They are particularly effective on pain arising from inflammation as opposed to more visceral pain (such as that resulting from appendicitis), hence their popularity for the disorders mentioned previously. A major advantage that NSAIDs have over other analgesics (narcotics, opiates, etc.) is that NSAIDs lack undesirable central nervous system effects including the development of physical dependence and respiratory depression (3). As anti-pyretics, NSAIDs are noted for their ability to reduce body temperature from dangerous levels (3). In sports medicine, however, NSAIDs are not commonly used for fever reduction (4). It is an NSAID's ability to reduce pain and inflammation that has given rise to its popularity among exercise participants suffering from musculoskeletal injuries (5).

What is inflammation? In many sports, tissue damage is often unavoidable. However, it is the subsequent acute or chronic soft tissue inflammation that is ultimately responsible for lingering pain and incapacity (6). Inflammation is a complex response of vascular tissue to physiological damage (7) characterized by heat, redness, swelling, and pain. It is not always easy to determine the physiological cause of inflammation. Consider the example of delayed onset muscle soreness (DOMS), which is believed to be the result of one or more of five factors including osmotic pressure changes and alteration in cellular calcium regulation (8). Regardless of the precipitating event, the initial response to soft tissue damage is a local release of inflammatory mediators which are activated by the release of prostaglandins discharged by the damaged tissue and inflammatory cells (6). The inflammatory mediators (which include histamine, bradykinin, and seratonin) promote increased vasodilation and sensitivity to pain.

Subsequent stages of the inflammatory response have been summarized by Guyton and Hall (9):

1. Vasodilation of the local blood vessels with consequent increased local blood flow;
2. Increased permeability of the capillaries with leakage of large quantities of fluid into interstitial spaces;
3. Clotting of the fluid in the interstitial spaces because of excessive amounts of fibrinogen and other proteins leaking from the capillaries;

4. Migration of large numbers of granulocytes and monocytes into the tissue; and
5. Swelling of the tissue cells.

As suggested in the case of DOMS, the above inflammatory response can occur whether precipitated by bacteria, trauma, chemicals, heat, or other phenomena (9).

NSAIDs are believed to block inflammation through several pathways, but one commonly accepted mechanism of action is through the inhibition of the enzyme cyclooxygenase, which acts upon arachidonic acid to produce the prostaglandins mentioned above (10). Arachidonic acid is a fatty acid released from cell membranes in response to injury. It is believed that NSAIDs may also have other effects on the inflammatory response (1,11).

In short, NSAIDs block the production of prostaglandins which, in turn, reduce pain and inflammation.

One final point to integrate the NSAID's action upon the anti-inflammatory response should be noted (4). Most patients will recover from injuries whether or not NSAIDs are given soon afterward. NSAIDs do not seriously delay the healing process; healing has been shown to be slightly more rapid and inflammation slightly decreased with their use.

What kinds of NSAIDs are available? Although a variety of treatments is available for the management of sports injuries, there is considerable variation among individuals with respect to their likelihood or ability to adhere to a particular regimen. It is this individual variation that determines whether or not an athlete should take an NSAID and whether or not the NSAID will produce the desired effect (11). With any drug therapy, the benefits and risks of taking the drug should be weighed by trained professionals in consultation with the athlete.

In 1993 more than 20 NSAIDs were available in the United States (10). At least four NSAIDs (acetylsalicylic acid, ibuprofen, naproxen sodium, and ketaprofen) are available without a prescription and, as a result, frequently are used by athletes.

Acetaminophen (i.e. Tylenol), which was discovered in 1877, is not classified as an NSAID. Like the NSAIDs, it possesses analgesic and antipyretic properties, but is not considered to be an anti-inflammatory because it cannot inhibit the enzyme that acts upon arachidonic acid in the peripheral tissues (12).

Is there a "right" NSAID for me? The major components of clinical effectiveness of a drug are relative drug efficacy and relative drug toxicity (1). Comparative efficacy has not been definitively studied, but several studies have documented substantial differences in toxicity among NSAIDs (1,2,6,13). Does this mean that the exercise participant is left to choose an NSAID based upon its attribute of being the least risky as opposed to the most effective? This question is difficult to answer, but the literature seems to support the former philosophy.

Part of the problem lies in the physiological variation among individuals mentioned earlier, but other problems include trying to figure out what constitutes an effective drug treatment. Fries (1) has summarized what was believed to be the most well-performed studies and has developed lists of NSAIDs more likely than others to achieve desired results depending upon disease, therapeutic objectives, and risk of side effects. However, in so doing, he demonstrated some of the problems previously mentioned. Are subjects adhering to the therapy because the therapy is working? Are subjects representative of others who may base NSAID choice upon similar factors? Are differences in toxicity greater or less than differences in efficacy?

It is tempting to draw definitive conclusions from such findings, but it is noteworthy that these data were collected from 116 patients with rheumatoid arthritis. Although the inflammatory response is believed to be the same in all individuals, to what extent would rheumatoid arthritis patients be typical of most exercising populations? How well such conclusions can be generalized to other populations is unclear. Moreover, how similar is rheumatoid arthritis (a disease) to the injuries that would prompt most exercise participants to take or seek a prescription for an NSAID? Additional rankings in the study support the same conclusion: desired results depend upon disease, therapeutic objectives, and risk of side effects (1).

Other studies of NSAID side effects have been conducted. Clyman (3) also has investigated the relative toxicities of NSAIDs, and states that many common NSAIDs are capable of causing gastrointestinal bleeding (due to the blocking effect they have upon gastric prostaglandins.

Calabrese and Rooney (2) restate several of the key points already made in this paper, but also include an important consideration about the financial and medical costs of taking an NSAID: the average NSAID is more expensive than aspirin, so depending upon the injury, it may not pay to take a newer, more expensive drug. However, if the athlete experiences gastrointestinal distress and needs an antacid to tolerate the therapy, the athlete's overall cost increases.

A stronger view of the medical costs of NSAIDs has been summarized by the Canadian Medical Association (CMA) in a 1996 position paper discussing an evidence-based approach to prescribing NSAIDs for musculoskeletal disease. The CMA stated that no available NSAID lacks the potential for serious toxicity. Thus, long-term use should be avoided whenever possible, particularly in high-risk patients. The CMA also recommended that in patients with gastric risk factors, the lowest effective dose of NSAID should be used. Given the human performance levels expected by many athletes, these considerations must be taken into account.

Future Research. There is much to be done in the area of NSAID research. Weiler (4) has provided some important insights into the strengths and weaknesses of this area with a literature review of more than 50 studies exploring the role of various NSAIDs in sports medicine. A 14-point recommendation is included to better guide NSAID researchers toward conducting studies that examine NSAID efficacy and safety. Problems of current research include studies that have been conducted without double-blind designs (both subject and investigator are unaware of which treatment the subject receives), studies that have been conducted without placebo controls, and studies that lacked control over the interval between injury and the start of treatment. By adhering to more uniform guidelines, researchers should be able to produce results that will enable clinicians to generalize findings to exercise participants and other populations. As already noted, additional research comparing the efficacy between drugs in athletic populations with athletic medical problems needs to be conducted.

Conclusion. NSAIDs are helpful in alleviating the inflammation that results from common injuries experienced by exercise participants. Although it is impossible to state conclusively that certain NSAIDs are "better" than others, it is worth remembering that despite their popularity, they are often powerful drugs and should be used only after more conservative (and possibly more physiological) non-drug therapies such as rest, ice, compression, and elevation (RICE) have been attempted (4,6,10). Simply having plenty of drugs to choose from should not be a predisposing factor in choosing an NSAID therapy.

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Calcium Absorption: A Critical Process in Maintaining Healthy Bones

By September Nelson

Introduction. Everyone knows that calcium is important. Not only the amount consumed, but the source of the calcium is of importance. The amount of calcium that is absorbed from the foods we eat is variable for different sources. The amount of calcium consumed at a time can also affect the absorption.

The importance of dietary calcium in maintaining skeletal strength has frequently been reported (1,2). Calcium is stored in the bones and teeth. It is this mineral component along with phosphorus that maintains the rigidity of the bones. Calcium is also necessary for many physiological processes (3). At the cellular level, calcium helps maintain ion flow through the cellular membrane. Calcium plays an important role in contraction and relaxation of skeletal, cardiac, and smooth muscle. Nerve excitation and conduction also rely on calcium for normal function (4)

Physiological Mechanisms. Because calcium is so important for cellular function, the human body has developed efficient mechanisms to maintain adequate availability. Even when an individual's diet provides little calcium, proper levels can be maintained through hormonal control. Parathyroid hormone stimulates withdrawal of calcium from the stores in bone. This mechanism maintains blood calcium levels, but can lead to calcium-poor bones. When the blood calcium level becomes high, the hormone calcitonin stimulates calcium deposition in the bones, providing a protective effect for the skeleton.

The body also is able to alter the level of absorption of calcium from food. Individuals with low calcium intake are able to absorb a greater fraction of intestinal calcium than those who chronically ingest higher amounts(5). In order to maintain proper calcium levels without depleting bone of its mineral structure, adequate levels of calcium should be included in the diet.

Dietary Recommendations.

The recommended daily allowance (RDA) for adolescents and young adults (19-24 yr.) is 1200-1500 mg of calcium per day, and 800 mg/day for those 25 and over in order to maintain normal levels. The National Institute of Health recommends 1200-1500 mg/day for individuals 11 to 24 years of age, 1000 mg/day for premenopausal and post-menopausal women taking estrogen as well as men 25-65 years of age. The agency suggests that men and women over 65 and postmenopausal women not taking estrogen are suggested to consume 1500 mg of calcium/day (5).

Getting the necessary amount of calcium can be challenging, particularly when the diet contains large quantities of foods that are not rich in calcium. The best way to determine how much calcium is consumed regularly is to read the labels of foods eaten frequently. Some food labels express calcium content in both milligrams and percent of the RDA, while others provide only the percentage. When only the percentage is given, it is important to be sure that this amount was calculated using the RDA for the appropriate age and gender.

Foods vary widely in their calcium content. An 8-ounce cup of nonfat, plain yogurt contains 45% of the RDA for an adult, or about 350 mg. A bag of "light" microwave popcorn, on the other hand, contains less than 2% of the RDA -- making it a very poor snack choice for active adults from the nutritional standpoint.

Dietary calcium is most often consumed via dairy products but is found in many other sources. A study investigating the absorbability of calcium compared various sources for differences in absorption levels (6). Standard servings of whole milk, chocolate milk, yogurt, imitation milk, cheese, and calcium carbonate resulted in similar absorption fractions in healthy, postmenopausal women. Hence, these forms of calcium do not vary significantly in absorption.

Supplementation, Fortification, and Absorption.

When an individual's diet provides inadequate calcium, supplementation with calcium carbonate may be necessary. Adolescents who are experiencing pubertal growth spurts, and pregnant, lactating and post menopausal women may have increased calcium needs. Calcium supplementation has also been suggested for many women to lessen the effects of osteoporosis. When calcium intake is inadequate, the body will remove the needed mineral from the stores found in the bones. Osteoporosis is the resulting weakening of the bones associated with demineralization. Supplementation helps to maintain a positive balance so that adequate calcium remains stored in the bones. Unfortunately, not all of the calcium ingested in food and supplements will be absorbed by the body and utilized. Many factors affect the absorption of calcium into the body.

Other foods may be fortified with calcium as well. Wheat bread fortified with calcium has shown greater absorbability than milk when the two foods are ingested separately (7). This leavened bread also permitted greater calcium absorption than unleavened cookies made with the same fortified wheat flour. The authors suggest that the leavening process positively affects the absorbability of calcium from wheat products by reducing the binding capacity of the wheat fiber (7).

Ingested calcium passes through the stomach and is absorbed by the small intestine. The fraction of calcium absorbed does vary according to the quantity of food ingested. There appears to be an inverse relationship between the amount of calcium taken and the amount absorbed. As the quantity of calcium increases, the percentage that is absorbed decreases (8).

This relationship disappears when calcium in milk is coingested with whole wheat bran. The quantity of calcium ingested, however, does not appear to affect the level of absorption in the presence of wheat bran. Overall, concurrent wheat bran ingestion may negatively affect the absorption of calcium as a result of the high levels of insoluble fiber. The coingestion of wheat bread and milk reduced the absorption of calcium from both products. The levels of phytate (insoluble fiber) are lower in wheat bread than in wheat bran, which is found in cereal. Higher levels of insoluble fiber are associated with a greater decrease in calcium absorption from both bran cereal and coingested milk. Although the specific mechanism is unknown, wheat bran cereal seems to have the ability to bind calcium thus decreasing the availability for absorption in the intestine and increasing its excretion (8).

Vitamin D also plays a role in the absorption of calcium. Although vitamin D is not required for calcium absorption, its presence greatly increases the amount of intestinal absorption by promoting active transport of calcium through the intestinal wall (9).

Vitamin D does not directly cause the increased absorption, but rather begins a series of events which affect intestinal absorption of calcium. The body produces vitamin D with exposure to ultraviolet rays from the sun. Many dairy products are also fortified with vitamin D. One product of vitamin D exhibits a hormonal effect on the intestinal wall by stimulating the production of a calcium-binding protein. This protein aids in the transport of calcium into the cells of the epithelium and its effect can last up to several weeks. The amount of calcium-binding protein is directly proportional to the rate of calcium absorption (4). Appropriate sun exposure and dietary

intake of vitamin D assist in maximizing calcium absorption by the small intestine.

Many factors affect the supply and demand of calcium in the human body. The developmental stage of the individual dictates how much calcium is needed to build and maintain strong bones and in pregnant women, support a growing fetus or child. Intestinal function also plays a role in calcium levels. Digestive disorders may reduce the absorption and availability of calcium to the system. In healthy individuals, absorption may be positively or negative affected by how the calcium is ingested. Dairy products provide calcium that is readily absorbed, but available calcium may be negatively affected if the source foods are taken with wheat bran. The quantity of calcium consumed is also a factor. Small amounts of calcium provide a greater absorption fraction than large amounts. It is important to remember that some calcium is better than none. In order to best utilize what is consumed, smaller quantities over the course of a day may be more beneficial than one large dose.

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Exercise Adherence Improved by a Plan for Behavior Change

By Carolyn Petersen

Introduction. Many would-be exercisers start out with the best of intentions, but quickly find themselves avoiding the gym, the pool, and the bike path in favor of less active pursuits. Although they believe they want to change their lifestyle to include more activity, somehow the shift just doesn't happen.

Although friends and family may be quick to diagnosis the problem as a lack of willpower, research undertaken during the past decade into how people change their behavior indicates otherwise. According to this new theory, known as the Transtheoretical Model of Change (TMC), individuals go through six distinct stages on the way to adopting new habits for good (1). When a person is unsuccessful at keeping that new year's resolution to begin jogging three times a week, that failure most likely results from initiating the action stage too soon.

The six stages of the model include:

Precontemplation - lack of awareness that life can be improved by a change in behavior;

Contemplation - recognition of the problem, initial consideration of behavior change, and information gathering about possible solutions and actions;

Preparation - introspection about the decision, reaffirmation of the need and desire to change behavior, and completion of final pre-action steps;

Action - implementation of the practices needed for successful behavior change (e.g. exercise class attendance);

Maintenance - consolidation of the behaviors initiated during the action stage;

Termination - former problem behaviors are no longer perceived as desirable (e.g. skipping a run results in frustration rather than pleasure).

As is apparent from the names of the stages, most of the work of changing occurs before the individual laces up the running shoes. Less clear, but equally integral to successful change, is the frequent movement of would-be changers among the first four stages before maintenance and termination are reached. During this time, changers use numerous processes of change on the way to lasting success (2). These strategies include, among others, such efforts as social and environmental reevaluation, counterconditioning, and stimulus control.

Putting theory into practice. Investigations into the relevance of the TMC for the adoption of exercise already have been undertaken. In a study of 178 women practicing various levels of physical activity, a Stage of Exercise Scale based on the model was able to differentiate between subjects classified by behavior stage in terms of such physiological variables as peak VO2 and exercise energy expenditure (3).

Researchers also have assessed the applicability of the TMC to exercise adoption and adherence by comparing it with an analysis based on the Seven Day Physical Activity Recall Questionnaire

(4). This method has previously been validated for use within the general population (5). In the worksite-based comparison, 235 employees completed physical activity and stage of change questionnaires with significant correlation among responses.

The big question for exercise professionals, however, is whether the model will work when used as the theoretical basis for exercise programs implemented in the field. The answer from at least one site is a resounding Yes.

Eastern Washington University professor Wendy Repovich used the Stage of Exercise adoption scale to assess student and staff member progress in campus physical education classes. Beginning in the fall of 1996, the surveys of exercise behavior and attitudes toward exercise were given to class enrollees at the beginning and end of the term. More than 350 students have completed surveys to date.

Analysis of the survey responses indicates that students enrolled in walking, aerobic conditioning, and "fast fitness" classes moved forward one stage during the term. Those contemplating regular exercise moved into the preparation stage, and those in the action stage progressed toward maintenance of regular exercise habits.

Repovich believes the TMC approach succeeded in part because the course curriculum included some written exercises designed to help students complete the psychological activities tied to each of the stages. Students were asked to develop their own short-term and long-term goals for exercise, determine what barriers might prevent them from reaching their goals, and develop strategies for handling these difficulties. These efforts have been recognized as critical components of the preparation stage.

Information gathering and awareness of other possible behavior patterns are two activities that usually occur in the precontemplation and contemplation stages of successful changers. To ensure that students became acquainted with the benefits of regular exercise, they are required to read health-related articles. Those taking fitness classes more than once submitted critiques of the materials they read as further reinforcement for exercise adherence.

For some administrators, using the TMC as a basis for exercise program design will require a shift in philosophy. In the Eastern Washington walking classes, for example, the focus was redirected from fitness improvement to awareness of physical benefits. Instead of grading students on how far they could walk in 30 minutes, instructors encouraged students to think about how much more relaxed they felt after a walk or how much more energy they had when exercising on a regular basis. As students became more aware of the ways exercise improved their health, they became more open to enjoying it -- a critical step in making physical activity a habit.

Perhaps the best news for would-be exercisers is that the TMC is equally useful for changing other behaviors. A 1994 analysis of attitudes and behaviors for 12 common problem behaviors (smoking, cocaine use, weight management, high fat consumption, delinquent behavior among adolescents, high-risk sex, condom use, sunscreen use, exposure to radon gas, exercise adherence, regular mammography screening, and smoking prevention activities by physicians) noted that would-be changers proceed through the six stages regardless of the nature of the problem (6). Narrower investigations focusing one a single behavior such as smoking (7,8) or diet management and weight loss (9) support the TMC as an accurate model for understanding health behavior and structuring strategies for improvement. By developing a plan that includes not only the end goal but also all the psychological adjustments needed along the way, would-be changers can -- and have -- taken permanent steps on the road to regular physical activity and better health.

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Book Review

Alter, M. 1996. *Science of Flexibility (2nd ed.).* Champaign, IL: Human Kinetics.

Students of kinesiology, athletic trainers, and coaches will recognize this volume as a new edition of Michael Alter's 1988 text "Science of Stretching." Fortunately, the change in title reflects a broader and more academic view of the field, a change that most readers should find beneficial.

In his presentation of human structure, Alter seeks to make anatomy and function accessible to both athletes and the health care professionals with whom they work. Clear diagrams and tables reinforce discussions in the text, making the book a useful reference for functional anatomy.

The use of stretching as an aid to the development of flexibility has been (and remains) controversial. In this new edition, the author provides a more in-depth look at the body, the physiological processes that regulate performance, and potential strategies for enhancing function through greater flexibility. The descriptions and illustrations should satisfy both beginning and advanced students of anatomy.

Alter covers new ground with a chapter addressing techniques commonly practiced by chiropractors, osteopaths, and massage therapists. He explains the theory behind such techniques as mobilization and manipulation, offers research-based support for their actions, and discusses the risks associated with these therapies. The approach is informational rather than advocatory, so readers can draw their own conclusions.

Also new to this edition is a look at flexibility for special populations (e.g. pregnant women and persons with disabilities), with an emphasis on changes in physical capability related to aging. The review of research literature is thorough and succinct, and the comprehensive reference list makes it possible for readers to locate source material easily.

The book's added depth in anatomy, physiology, and research comes at the expense of specific stretching regimes. The initial text provided a significantly larger array of exercises and variations; readers looking for more are referred to Alter's Sport Stretch for a fuller selection. Illustrations and detailed instructions accompany the 60 stretches presented in this edition.

Both the change in title and content reflect a shift toward a more academic approach. This second edition is detailed enough to serve as a university-level textbook. At the same time, the text's usefulness to athletes, coaches, and clinicians in the field has not been diminished; these readers too will find Science of Flexibility a helpful reference.

Carolyn Petersen

Mitral Valve Prolapse

By Christopher Berger

Introduction. Heart murmurs are abnormal heart sounds occurring due to abnormalities in one or more of the hearts four valves (1). The most common type of heart murmur is a mitral valve prolapse (MVP). Poor closure of the mitral valve and/or subsequent regurgitation of blood from the left ventricle to the left atrium causes a clicking or shuffling sound heard mid- to late-systole. MVPs are reported to be the most common valvular heart disease in industrialized countries, affecting about 3% of adults (2,3,4). MVP occurs in 6-10% of young women and 4% of young men. The prevalence may exceed 10% in women ages 14-30. Following is a brief orientation to characteristics of MVP including methods of detection, associated disorders, and treatment.

Abnormal Heart Sounds (Heart Murmurs). Depending upon when they occur during the normal cardiac cycle, heart murmurs can be classified into three types: systolic, diastolic, and continuous. Brukner and Khan identify eight systolic, two diastolic, and two continuous types of murmurs (5). Fabius identifies six systolic and two diastolic types of murmurs (6). Lillegard and Rucker identify five systolic, two diastolic, and one continuous type of murmur (7). Although sources differ regarding the number of diagnosable murmurs, the classification of when the murmur occurs is critical to an appropriate diagnosis. (Systolic murmurs are typically benign and diastolic murmurs are always pathological.) MVP is classified as a systolic murmur.

Detecting Valvular Abnormalities. The normal mechanical activity of the heart generates a LUB-DUP sound loud enough to be monitored from outside of the body. There are three ways of detecting valvular abnormalities: auscultation (listening using a stethoscope), phonocardiogram, and echocardiogram.

The stethoscope is the most common, least-expensive instrument in use and is the primary means of detecting audibility and when the murmur occurs. A six-point grading system exists for determining the intensity of the murmur (8). The grading system helps the clinician to determine the need for a more sophisticated method of describing the occurrence and audibility of the murmur.

The phonocardiogram is a more sophisticated means of detecting heart murmurs and it consists of a specially designed microphone. When placed on the chest, the microphone records and amplifies sounds which can then be transferred to paper and depicted as waves. The amplitude and frequency of the waves aids the clinician in determining the occurrence and severity of the murmur.

The echocardiogram is particularly useful in diagnosing MVP (9). Also referred to as Doppler echocardiography, the echocardiogram is an ultrasonic procedure which produces a visual depiction of the degree of mitral regurgitation or backflow of blood due to the mitral valves incompetency. The degree to which the mitral valve leaflets prolapse and/or the degree to which blood regurgitates from the left ventricle is the basis of a grading system for echocardiographic measures. This distinction is important because the extension of the mitral valve leaflets above the plane of the atrioventricular junction ventricular systole is a direct indication of the degree to which blood regurgitates from the left ventricle to the left atrium. Severe regurgitation causes a stagnant pool of blood to form which may lead to the formation of blood clots.

MVP Correlates Boudoulas et al report that MVP is not independent of other cardiovascular and systemic disorders (10). Of special interest to the exercise scientist is the relationship between MVP and exercise, particularly since MVPs are so prevalent among younger, physically active

populations. Stoddard et al. hypothesized that patients with documented diagnoses of MVP and no mitral regurgitation at rest would develop mitral regurgitation during exercise (11).

Stoddard et al. also reasoned that since dynamic exercise decreases left ventricular systolic volume, it could aggravate unequal mitral leaflet stresses by increasing left ventricular systolic pressure. Testing 94 patients with MVP, the researchers concluded that in patients having MVP without mitral regurgitation at rest, exercise provoked mitral regurgitation in 32% of patients. However, in a rebuttal, Levine criticizes that work. He argues that, of the subjects studied, their condition only appears to be more severe because it stands in contrast to a benign natural history in the remainder (the population) (12).

In a related area for the exercise scientist, Lax et al. explored the effects of hydration on MVP (13). Ten subjects with documented diagnosis of MVP were examined using echocardiograms before and after oral hydration with 1L of fluid (water or Gatorade). Results indicated that all subjects continued to have MVP after hydration, however, subtle changes upon auscultation were reported (post-treatment). The investigators concluded that mild alterations in a patient's hydration may explain, in part, the variable nature of auscultatory findings in patients with MVP.

Another MVP correlate relates to a potential psychiatric link between mitral valve function and anxiety disorders. Carney et al. explored the interrelationship between major depression, panic disorder, and MVP in patients who complain of non-coronary artery disease chest pain (14). This team studied the first 100 people who agreed to a psychiatric diagnostic interview. They concluded that MVP was significantly associated with major depressive disorder/panic disorder and that the identification and treatment of these psychiatric disorders may alleviate the patients reports of chest discomfort.

Finally, Frederickson (15) and Boudoulas et al. (10) have proposed that the pathogenesis of MVP symptoms is poorly understood and may only be indicative of an MVP syndrome or associated set of symptoms and pathologies that are not only correlated with but are caused by MVP. Frederickson has developed an MVP syndrome support group based upon clinical experience that convinces her that MVP is only one component of a larger disorder. She writes (paraphrased): Mitral valve prolapse syndrome is the most common cardiac condition seen in the general population occurring in up to 20% of the general population. MVP is seen in all ages, races and socioeconomic groups. Symptoms of MVP include: chest pain, shortness of breath, panic and anxiety attacks, migraine headaches, dizziness, fluttering in the chest, abnormal awareness of heartbeat, sleep disturbances, and often irritable bowel syndrome. Symptoms are not usually due to poor cardiac function but rather an imbalance in the nervous system that gives a wide range of symptoms. Patients are frequently accused of being depressed or merely anxious as a source of their symptoms but many studies have shown that MVP patients have no greater incidence of psychiatric problems than the general population. Symptoms are usually controlled by a combination of a diet low in sugar and caffeine, fluid loading, aerobic exercise, and occasionally medication (15).

It should be noted that in Frederickson's account the 3% population prevalence of MVP previously reported is nearly a seven-fold underestimate and that the recommendation was made to increase hydration despite a previous study to the contrary. In addition, the additional somatic disorders are reported in a condition typically described as asymptomatic.

Treatment of MVP Bacterial endocarditis is a rare but serious bacterial infection of the heart valves or tissues lining the heart (17). People with weak or abnormal valves are particularly susceptible to bacterial endocarditis and it is strongly recommended that an antibiotic prophylaxis (prevention treatment) be administered prior to dental/oral/upper respiratory tract procedures. Antibiotic dosages vary according to the patients ability to tolerate a particular regimen. The American Heart Association recommends 3.0 grams of amoxicillin orally one hour before the

procedure and then 1.5 grams six hours after the initial dose. In addition to amoxicillin, ampicillin, clindamycin, erythromycin ethlylsuccinate/stearate, gentamicin, or vancomycin may be substituted (18).

Depending upon the severity of the MVP, the treatment may be more powerful than an occasional dosing of antibiotics. Kinney and Packa suggest that medical treatment include propranolol for chest pain and dysrhythmias, medical therapies for dyspnea and palpitations, barbiturates for hypervagal patients, as well as psychotherapy for patients with panic attacks (19).

For severe MVP, replacement of the valve may be in order. Valve replacement may occur by using biological tissue valves (from cows or pigs) or by using mechanical valves. Mechanical valves have the advantage of durability but the disadvantage of causing blood clots. Biological tissue valves negate the need for anti-coagulant medication but are less durable and more likely to be rejected by the immune system.

MVP Prognosis. There is little likelihood that MVP would cause death. The American Heart Association estimated 15,070 deaths and 70,000 hospitalizations (20) due to valvular disorders and of those, mitral valve disorders accounted for 2,044 deaths (21) and 30,000 hospitalizations (20). Although the statistics compare 1990 with 1993, it is clear that the most commonly diagnosed valvular disorder causes relatively few deaths and hospitalizations. For most people, treatment of an MVP consists of nothing more than the previously recommended administration of antibiotics and occasional rest and stress management. For others, surgical intervention is necessary. In either case, the prognosis is excellent.

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The Piriformis Syndrome

By September Nelson

Introduction. Not all low back, hip, and gluteal (buttock) pain are manifestations of back injury. Pain in any of these areas may indicate injury or irritation of any one of a number of muscles and nerves surrounding the low back and hip. Injury to any of these structures can result in pain and loss of function. A specific muscle that is susceptible to injury and inflammation is the piriformis muscle. Due to the location of this muscle, the sciatic nerve is often involved with piriformis problems. Pain and dysfunction resulting from piriformis injury is referred to as piriformis syndrome. The symptoms of this disorder sometimes mimic those of a bulging lumbar disc, or similar low back injury. Therefore, diagnosis of pain in the low back, gluteal, or hip region should include an evaluation of the piriformis muscle (PM), other hip musculature, and surrounding nerves.

Anatomy and Function. The piriformis muscle is located deep in the gluteal region. This muscle attaches to the sacrum and the lateral portion of the upper part of the femur. It is one part of a group of muscles whose actions include abduction (moving the thigh away from the midline) and external rotation of the thigh (turning the knee and toes outward). These muscles are important in maintaining stability of the hip in all weight bearing activities.

The sciatic nerve passes between the piriformis muscle and a notch in the pelvis as it enters the gluteal region. This large nerve supplies a majority of the nervous innervation to the lower extremity. In some cases, the PM may be split into two bellies, with the sciatic nerve passing in between the two portions. The sciatic nerve also may be split into two trunks which may pass through different portions of the PM (1). In any of these arrangements, the sciatic nerve is susceptible to compression between the piriformis and the pelvis.

The PM is susceptible to hypertrophy, an increase in size, as a result of its high level of activation. With increased use, muscles increase size and strength. During the weight bearing portion of gait, the piriformis is lengthened which initiates a stretch reflex and results in contraction. During the swing phase of gait, the piriformis contracts again to assist with external rotation (1). This double activation may precipitate muscular hypertrophy, thereby decreasing the space available for the sciatic nerve.

Symptoms and Diagnosis. Symptoms of piriformis syndrome often include deep buttock and posterior hip pain. The gluteal discomfort may be accompanied by pain, numbness, and tingling, that radiates into the posterior thigh, leg, and foot. These symptoms are generally associated with a condition known as sciatica. Sciatica pain is often the result of nerve compression in the spinal cord associated with a herniated lumbar disc (2,3). Generally this type of low back injury also will be accompanied by neurological deficits that are absent with piriformis syndrome.

The pain and discomfort of piriformis syndrome are usually exacerbated by standing after prolonged sitting and with activity. Pain is minimized when lying down with the hips slightly flexed, in a curled up position. Tightness and sensitivity may be revealed during palpation of the PM. The PM is stretched when the body is rotated around a planted foot, as in a tennis serve. This motion will reproduce pain. Resisted abduction in the seated position should reproduce discomfort in the PM.

Diagnosis of piriformis syndrome may be delayed or missed completely without a thorough examination. Often, piriformis syndrome is diagnosed after other causes of sciatica have been

ruled out (2,3).

Suggested etiology of piriformis syndrome begins with injury to the PM or the pelvis. Trauma to the PM may result in spasm, edema, contracture, and finally compression and entrapment of the sciatic nerve (1). Hypertrophy or extreme tightness of the piriformis also may compress the sciatic nerve. Piriformis tightness can result from increased lumbar lordosis, swayed low back, and concurrent hip flexor tightness (4). Improper biomechanics, muscle imbalance, and leg length discrepancies may contribute to piriformis problems.

Treatment. Although there appear to be numerous causes of piriformis syndrome, the treatment approach follows a consistent pattern. Treatment of piriformis syndrome should focus on reducing the compression of the sciatic nerve. If compression is the result of inflammation of the PM and or the sciatic nerve, ice can be effective in reducing edema and pain. Ice should be applied directly over the piriformis muscle, which lies just beneath the hip dimple, for 20 to 30 minutes several times per day. Nonsteroidal anti-inflammatory drugs (NSAIDs) such as ibuprofen and naproxen can help control inflammation and pain (4). Rest also may be indicated in order to prevent re-injury.

Passive stretching of tight musculature, too, is important in restoring normal range of motion and function. The correction of biomechanical errors and leg length discrepancies are important (1,4,5). Through gait analysis and physical examination, an athletic trainer, physical therapist, podiatrist, or physician can diagnose gait errors, muscle imbalance, and limb length discrepancies. Muscle strengthening and body mechanics education can correct the biomechanical errors while the prescription of foot orthoses can compensate for leg length differences. Achievement of a normal range of motion should be the first goal. Once adequate range of motion is acquired, strengthening exercises may then be added to correct any muscle imbalances. If these conservative treatments of ice, NSAIDs, stretching, and biomechanical corrections, are ineffective, corticosteroid injections may be prescribed. In extremely resistant cases, surgical release of the piriformis muscle may be indicated.

Stretching protocols involve hip flexion, adduction, and internal rotation applied with slow steady pressure. Once inflammation has been resolved, heat modalities such as ultrasound, whirlpool, and moist heat can be used prior to stretching to improve the elasticity of the tissues being stretched. The application of ultrasound should be supervised by a physical therapist or athletic trainer. Moist heat can be applied over the PM and proximal musculature for 15 minutes. A cloth should be placed between the skin and the heat pack to protect against burning. Maintaining normal range of motion will help alleviate symptoms, as well as prevent progression and recurrence of symptoms.

Correction of biomechanical and training errors are also important in the treatment and prevention as are leg length discrepancies and muscle imbalances. As stated earlier, there are a number of health professionals who can assess the structure and function of the hip and lower extremity. Abnormalities in any of the components of gait: limb length, joint range of motion, muscle function, strength balance, and coordination of movement, can result in added stress on the piriformis muscle. A controlled program of stretching, strengthening, and mechanic education with the correction of structural problems can help to alleviate symptoms and prevent recurrence. Alternative methods of training may also be helpful. Aquatic therapy, changes in the regular running surface, and a change in training volume are options that could be considered.

In determining the diagnosis for low back, gluteal, hip, and lower extremity pain the examination should include an assessment of piriformis health and function. Pain resulting from compression of the sciatic nerve by the PM sciatic nerve can be managed and prevented from recurring. Inflammation can be reduced with ice, NSAIDs, and rest. Further treatment of hypertrophy and tightness includes stretching protocols and correction of biomechanical and structural abnormalities. Education regarding the progression of the disorder also may help the individual

manage and limit the recurrence of piriformis syndrome.

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Women's Health Issues Taking the Spotlight

By Carolyn Petersen

Health issues associated with higher incidence rates in men -- for example, coronary artery disease -- have traditionally been the focus of the majority of clinical research. This situation is changing, however, as women's health advocates and research funding administrators recognize the gaps in our knowledge of best treatment practices.

Concern about breast cancer and efforts to reduce the likelihood of its occurrence may lead women to ignore other equally serious conditions. A study performed by the Washington, D.C.-based Center for Risk Communication found that 61% of the women surveyed were most afraid of cancer, while just 9% were most concerned about heart disease. Heart attacks, however, pose a greater risk of death for women.

The survey, "Myths and Misperceptions About Aging and Women's Health," was conducted for the National Council on Aging (NCA) in collaboration with the Alzheimer's Association, the Older Women's League, and the National Osteoporosis Foundation. More than 1,000 women aged 45 to 64 were asked questions designed to assess their knowledge of health risks and potential therapies.

"If women don't get the right information about their risk for disease, they may make the wrong decisions about their health," says NCA president James Firman. "Health-related decisions that women make in mid-life and earlier years can have a tremendous impact on preventing health problems when they are older."

A related study conducted by CRC, "Women's Perceptions of the Risks of Age-Related Diseases, Including Breast Cancer: A Case Study," explored the factors that contribute to women's' perceptions of health risk. The conclusions of the three-pronged investigation were based on data from focus groups with women aged 45 to 54, a national survey of women of the same age, and analysis of literature covering age-related disease risks and patient decision-making factors.

Confusion about research and treatment procedures as well as an inability to understand the context of medical research reported in the popular media make it difficult for women to make sound decisions, according to the report.

"Actual risk is only one of many factors women use to make health choices," says CRC's Vincent T. Covello. "Often, media reports have a significant impact on women's attitudes. For example, while much has been reported about a perceived link between hormone replacement therapy and breast cancer, far less coverage has been devoted to scientific evidence which shows that women who take hormone replacement therapy live longer than women who don't."

Uneasy subjects. In some situations, women's lack of awareness results from discomfort over discussing health issues with practitioners. A 1996 survey by the research firm Market Facts found that only 12% of medical care providers had brought up the subject of sexually transmitted diseases (STDs) other than HIV/AIDS with new patients aged 18-45. Just 3% of the respondents reported bringing up the issue themselves. Eighty-four percent said they believe STDs are an appropriate subject for discussion, but 64% believe it is the responsibility of medical personnel to initiate such discussions.

This reluctance to talk about reproductive health concerns prevails despite the fact that such diseases can result in permanent infertility and even death.

"Many health providers and women seem to be enacting a 'Don't Ask, Don't Tell' policy when it comes to STDs. The end result is a dangerous 'Don't Know'," says Felicia H. Stewart, M.D.. Stewart is director of reproductive health programs for the Kaiser Family Foundation, one of the organizations that sponsored the survey.

The same code of silence did not necessarily apply to other topics related to reproductive organ health. Discussions about breast self-examinations were initiated by 69% of providers and 4% of patients, Pap smears by 60% of providers and 12% of patients, mammograms by 34% of providers and 7% of patients, and birth control by 33% of providers and 20% of patients.

Clearly, improving women's health will require more than just greater quantities of clinical research. The willingness to discuss clinical findings related to prevention and treatment is another critical factor.

Stepping off on the right foot. Increasing interest in women's health issues is not limited to life-threatening concerns. Bunions, corns, neuromas, calluses, and hammer toes are among the most mundane conditions known, and have been reported up to eight times more frequently in women. The American Orthopaedic Foot and Ankle Society has estimated the annual cost of surgery for these forefoot problems at $2 billion. Time lost from work was estimated to add an additional $1.5 billion to the bill.

High-heeled shoes, considered fashionable for decades, now are coming under fire as researchers finally accumulate the clinical data necessary to support what women have always known: heels may look appealing, but they hold little attraction for the feet.

"We now have very strong evidence that women's shoes are a major cause of these deformities," says orthopedic surgeon Laura Tosi, M.D.. "It has been estimated that 75% of forefoot surgery in this country results from constrictive shoewear."

Lack of knowledge about proper shoe fit may contribute to the problem. An AOFAS study of 356 healthy women found that 88% were wearing shoes that were too small. Of the group, 80% exhibited foot deformities.

In November, Tosi testified before the National Institutes of Health Office of Research on Women's Health that funding for research and prevention efforts must be increased to avoid problems in coming generations.

"Education of parents as well as children about shoes is necessary. We need to investigate more vigorously early preventive measures. We also need further studies about changing shoe trends and the effects on the feet in the long term," Tosi told NIH officials.

Book Review

Kreighbaum, E.F. and Smith, M.A. (eds.) 1996. *Sports and Fitness Equipment Design.* Champaign, IL: Human Kinetics.

What makes running shoes different from other footwear? How do I estimate my grip size when buying a new tennis racquet? What is a derailleur? These are just a few questions easily answered with a new book from Human Kinetics titled *Sport and Fitness Equipment Design.* Dr. Ellen Kreighbaum and Mark Smith have teamed up to edit this unique, 218-page reference that investigates foot-ground interfaces, striking implements, and personal fitness equipment. The authors successfully describe the biomechanical aspects of more than a dozen pieces of exercise and sport equipment including aerobic exercise and resistance training tools. The descriptions are accompanied by black and white illustrations and photographs that help the reader understand the anatomy of modern exercise equipment.

One outstanding strength of this book lies in the careful organization of components relating to each piece of equipment. Readers who know nothing about a bicycle's suspension for example, can see its relationship to bicycle design and then thoughtfully consider its relative importance when choosing a new bike. This organization is also helpful to enthusiasts who already have their hearts set on a formation of tubing but would like to know more about how that material will handle under a certain design.

Another way that this book successfully enlightens the reader is through the use of bolded terms within the text that are defined in the margins. This design allows the reader to make quick use of new vocabulary as the topic develops.

The only weakness that I could find with this book is probably better described as a strength instead. Since there are many manufacturers of sports equipment, it is tempting to look for brand names recommended by the experts and base purchases accordingly. Although there is no doubt in my mind that the authors are experts indeed, readers would do better to compare brands with a periodical such as Consumer Reports. The authors of this book have done an excellent job of maintaining objectivity by keeping brand names and logos to a minimum and have produced an engaging reference for both beginning and advanced exercise participants.

Christopher Berger

Steroid Use in Adolescents

By September Nelson

Introduction. Steroid use often has been associated with high profile athletes. Unfortunately, these athletes make up a small percentage of the total users. Many competitive athletes, recreational athletes, and non-athletes also use these illegal chemicals. Just what do anabolic steroids do for users, and how many are using them?

Anabolic and androgenic steroids mimic the action of hormones normally present in all humans. Anabolic compounds stimulate the building of muscle, while androgenic compounds stimulate the development of male characteristics. The hormone testosterone is produced by the testes and adrenal glands in males and in the adrenal gland in females.

Testosterone is generally thought of as a male hormone because it is found in higher levels in men than in women, but it is necessary for both genders. Heightened levels of this hormone promote development of secondary male characteristics such as increased muscle mass and strength, growth of body hair, and deepening of the voice. Accepted clinical applications of steroids involve the treatment of disorders related to growth and development (1). Steroids also are used to prevent and reverse the muscle wasting associated with infection with human immunodeficiency virus (HIV). Unfortunately, these drugs also accelerate the closure of the growth plates in bones, which can result in decreased stature of immature skeletons (2).

Many studies have examined the demographics of who actually takes steroids. The surveys reported motives for using steroids, frequency of use, age of onset, knowledge about steroids, and attitudes toward use. They focused on anabolic steroids and did not include information about other performance enhancing drugs.

Buckley et. al. and Scott et. al. reported that 6.6% of male high school seniors use steroids or have used them in the past (3, 4). The incidence of steroid use among females, 2.5%, is much lower. Of these users, a large majority initiated use before they were 17 years old. Buckley reported that more than one third of steroid users began taking the drugs before age 15. These surveys report that the primary reason high school males take steroids is to improve athletic performance.

The second most popular reason for turning to steroids is a desire to improve appearance (3,5). Junior high and high school students are faced with dilemmas regarding both physical performance and appearance. At a time where peer acceptance is so important, young people may try anything to increase their popularity.

High school students' motives for participating in athletics influence their decision to use steroids. In 22.4% of anabolic steroid users and 9.1% of nonusers, the primary reason for participating in school-sponsored sport was to earn a college scholarship (5). Those who participate in sport in order to get a college scholarship or get into professional sports may be more willing to compromise themselves and try these illegal drugs. The pressure to win placed on athletes by parents, coaches, and friends may also influence the decision to use steroids.

In addition, a strong physique is promoted by the media and Western culture. The use of steroids as a cosmetic agent by non-athletes needs to be addressed by the media, schools, and parents. Genetics plays a large factor in physical size and stature, and there are limits to how much the body can adapt. It is unlikely that a child of parents who are small-framed and of average height

will develop large musculature. Nonetheless, the body can continue to get stronger even with minimal gains in muscle size. It is important to recognize and emphasize individual differences.

Recognizing Steroid Abuse. The introduction of steroids has many effects on the body. The desirable and undesirable effects provide signals that an individual may be using steroids. Unexpected rapid increase in muscle size and accompanied weight gain is one sign. This increased muscle development places extra force on the tendons and sites of attachment to the bone, resulting in tendinitis and pain (6). Secretion by the skin's oil, or sebaceous glands, is increased, which results in severe acne on the face, chest, and back. The apocrine sweat glands also increase secretions. Decreased liver function can cause the individual to appear jaundiced (7, 8).

The presence of just one of these factors should not be a cause for concern. In combination, however, steroid use may be suspected. An adolescent who becomes significantly more muscular within a relatively short amount of time and surpasses the growth and development of peers should be observed for the presence of additional indicators. Rapid growth in conjunction with worsening acne, and perhaps a yellowing of the skin from jaundice may suggest steroid use. Complaints of musculoskeletal pain (tendinitis and insertion pain) that cannot be attributed to a specific injury or event should be addressed by an athletic trainer or physician in order to rule out training errors or overuse injuries.

In women, anabolic steroids induce a marked masculinization accompanied by an irreversible lowering of the voice, increased body and facial hair, loss of menstruation and fertility, and enlargement of external genitalia (6, 7). Other symptoms include breast shrinkage and a recession in the hair line. As the woman's body becomes more masculine, the breasts decrease in size and become more dense. At a time when secondary characteristics are developing, steroid use poses problems. An adolescent girl's reproductive health may be jeopardized. Steroid use in this population can delay the onset of menstruation or cause amenorrhea, the absence of menstruation.

In males, normal production of testosterone decreases when high levels of synthetic steroids are present. This suppression of normal testicular and adrenal function can result in decreased testicular size, sexual dysfunction, loss of fertility, and breast development (2,7,8). The presence of any of these symptoms could indicate steroid use.

Steroid use can be recognized by psychological changes, too. Steroids have been associated with increased hostility and aggression. Other emotional disturbances such as mood swings, anxiety, depression, and an increased or depressed libido may accompany steroid use in both males and females. Of course, there are many causes for mood disturbances, and they should never be treated lightly. A sudden onset of any of these psychological symptoms, particularly in conjunction with physical signs, may indicate that steroids are being used. Fortunately, these mood-related symptoms usually subside when steroid use is discontinued.

Parents, coaches, and others who works with adolescents need to be aware of the signs of steroid use. Because the age of initial use can be quite young (junior high), education of youth and the adults who work with them must begin while they are young. All young people, not just athletes, need to be taught about the serious side effects of steroid use. Shifting the emphasis of sport from winning to enjoyment and personal mastery, avoiding unrealistic performance goals, and emphasizing personal characteristics other than appearance may help by reducing the pressures placed on youth.

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Book Review

Fleck, S.J. & Kraemer, W.J. 1997. *Designing Resistance Training Programs (2nd ed.). Champaign, IL: Human Kinetics.*

Although beginning resistance trainers can call upon several excellent guidebooks for learning training basics, advanced participants and instructors can't always easily find sources appropriate for their needs too. Steven J. Fleck and William J. Kraemer have improved those odds with the second edition of their book *Designing Resistance Training Programs.*

This book is an excellent reference for resistance trainers of every ability, but is especially useful to advanced participants who want to know more about the scientific foundations of resistance training. In twelve chapters, Fleck and Kraemer tightly integrate the field of resistance training with concepts from biomechanics, motor control, and exercise physiology. In the section on bioenergetics, the authors obviously realize the importance of energy production as it relates to human movement; the section is so well written that it is difficult to determine whether they are resistance trainers who really know their physiology or physiologists who really know their resistance training. In any case, some important points are made in an engaging, practical way.

Four chapters of this book are devoted to resistance training and special populations. Topics pertaining to the training of women, children, and seniors are discussed. In addition, a chapter discussing Olympic weightlifting and powerlifting is included. Principles of plyometrics are presented early in the book. Much of the book would be useful to clinicians prescribing exercise and to exercise test technologists who need a current review of the health benefits of resistance training.

In the end, two suggestions come to mind that could benefit the reader. First, because one of the outstanding strengths of this text is its multidisciplinary approach to the foundations of resistance training, it may require the reader to review concepts from other areas of exercise science. It would have been helpful to have included a glossary of terms at the end of the book to assist all readers with unfamiliar vocabulary and concepts. Also, little mention is made of the importance of sound nutritional practices for exercise participants. In light of athletes' determination to change body composition and gain perfect physique, such a section would have complemented those on bioenergetics, body composition, and detraining. Sound nutritional practices should be the basis for designing any training program.

Don't let the title fool you: this book is a great source of information beyond designing resistance training programs. It is both an outstanding reference for veterans wishing to know more about the science of resistance training and an approachable text for others anxious to expand their knowledge of resistance training beyond that of the experienced beginner.

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Book Review

Clark, N. 1997 *Nancy Clark's Sports Nutrition Guidebook (2nd ed.) Champaign, IL: Human Kinetics, 1997.*

If you are like most people, you are bound to be confused by the explosion of contradictory information out there about eating right for peak performance. Is fat good or bad? Exactly how important are anti-oxidants? How should I shop for food? These are just a few questions answered in the second edition of Nancy Clark's popular *Sports Nutrition Guidebook.*

As a registered dietitian and athlete, Nancy Clark, MS, RD practices what she preaches. Her expertise and easy-to-read writing style combine to form an approachable resource for anybody wanting to know more about eating for peak performance. The book contains 16 chapters organized under four major themes: The Training Table, Sports Nutrition for Success, Weight Management, and Recipes for Health and Fitness. In addition, there are three helpful appendices that direct the reader to books, organizations, and articles for additional sport nutrition information. Appendix B even provides information for the budding sport nutritionist hoping to break into the field. The 455-page book is illustrated throughout with black and white images.

The major strength of this book lies in the breadth of information presented. Clark also skillfully integrates important principles of exercise physiology throughout the text without intimidating the reader. She does this through the careful use of facts and figures, as well as with case studies that help the reader put this wealth of information to use. Even the casual sport enthusiast can pick up this book and learn some important tips for better eating and performance. Not to be missed are pages 5 (Three Basic Keys to Healthful Eating) and pages 231-234 (Body Fat Facts). Pages 204 and 205 also provide a handy table of vitamins along with easy to remember illustrations of good sources for each. Anyone who has ever wondered about eating and performance will come away from this book with new ideas about how to maximize both. Interested readers will be happy to see that chapter 16 is devoted completely to those needing information about gaining weight safely and effectively.

Part IV of the book is a 134-page "cookbook" for readers wanting to put sound nutrition and training practices into their next meal. Because this section comprises such a large portion of this edition, it significantly increases the size and (presumably) the cost of the book. Some readers may view the addition of these recipes as a strength while others may consider it a weakness. Since there are so few reliable exercise nutrition textbooks out there though, it would be hard to rule out this publication for use in class as the primary reference. In any case, even though some of the recipes are unique (to say the least), it might be more appropriate to advance this book into two editions with one emphasizing the science of sport nutrition and leave the recipes for a book presenting the creative art of healthy eating.

Readers of Nancy Clark's Sports Nutrition Guidebook are in for a real treat whether they try some of the featured recipes or not. This sensible book will certainly make a positive difference in the training programs of athletes of every ability.

Christopher Berger

Muscular Conditions that May Limit Activity in Persons Diagnosed Positive for the Human Immunodeficiency Virus

By Carolyn Petersen

Introduction. Research into the human immunodeficiency virus (HIV) during the past decade has expanded the current understanding of how this virus affects people. Individuals diagnosed HIV+ are receiving more effective medical treatment, and as a result, are living longer. With this lengthened lifespan comes a desire to return to usual daily activities, including and fitness programs.

HIV+ persons, however, face health issues not common to those testing negative for the virus. In addition to the challenges other immunocompromised persons experience (e.g. fatigue, increased susceptibility to infections), HIV+ persons frequently undergo a number of health problems specific to HIV infection. Muscular and joint dysfunctions are among the most widespread maladies experienced by persons infected with HIV over several months and/or years. Such conditions are never pleasant, but their presence poses special problems for active HIV+ persons. When exercise or physical labor is part of the daily routine, the potential for development of these activity-impairing conditions must be taken into account. Activity may provide the HIV+ individual with numerous benefits, but activity must be planned to avoid hastening the onset of acquired immune deficiency syndrome (AIDS).

Safe, beneficial exercise prescription requires an understanding of potential disease states affecting muscle. This paper will identify and describe muscle dysfunctions commonly experienced by HIV + persons. It will also address the prevention and treatment of these conditions when such information is known.

Among the conditions affecting skeletal muscle in HIV+ persons, nontropical pyomyositis is acknowledged as the most common. Polymyositis-dermatomyositis, too, has been associated with HIV infection. HIV+ persons also experience drug-induced myopathies, with zidovudine (AZT)-induced myopathy occurring in significant numbers of persons remaining on the drug for more than a few months. The clinical presentations of these conditions in HIV+ populations will be explored individually.

Myositis in HIV+ Persons. Of all the forms of chronic inflammation associated with HIV+ status that are described in the literature, pyomyositis appears to be most prevalent. Pyomyositis involves the formation of abscesses in striated muscle, and the usual signs of infection such as fever typically accompany it. Prior to its presentation in the HIV+ population, it was confined primarily to tropical regions. Several clinicians who regularly treat large numbers of AIDS patients have reported emergence of a non-tropical strain in HIV+ persons.

The specific causes of pyomyositis vary by patient. Muscle injury resulting from exercise or trauma (both direct and indirect) was observed in some patients (1), while others experienced bacterial, viral, and fungal infections as the cause of onset. In trauma-related cases, the trauma is believed to have rendered the muscle vulnerable to the subsequent infection. Among causative disease agents, Staphylococcus aureus was found in the muscle of 90% of HIV+ patients (2). Streptococci and gram-negative bacilli also have been implicated. Staphylococcus often is introduced via in-dwelling catheters used in treatment of other conditions; because HIV+ persons typically experience many opportunistic infections, they may be at especially high risk for pyomyositis through this mechanism of transmission. Of 12 patient profiles reviewed, four had histories of excessive exercise or trauma to the affected muscles (2), so exercise program design is

clearly a concern for HIV+ persons.

The condition affects primarily the large muscles including the hamstrings, quadriceps, gluteal group, and back, though a small number of patients experienced involvement of smaller muscles located in the upper extremities. A correlation between cause of onset and site(s) of involvement has not been reported. Accompanying symptoms include localized and systemic fevers, muscle stiffness and/or inflammation, general myalgia, and moderate to extreme pain. The disease is characterized by three stages: an invasive stage during which the organism enters the muscle; a purulent stage in which an abscess develops and fluid collects; and a late stage manifesting as a general systemic infection (3). Patients usually seek medical care during the second stage.

Certain pathologic phenomena also predominate. Schwartzman, Lambertus, Kennedy, and Goetz reported that the neutrophils of HIV+ persons often show a decreased ability to fight Staphylococcal infections, as well as chemotactic and oxidative dysfunctions (4). Leukocytosis may or may not be present. The condition is sometimes misdiagnosed as osteochondritis, thrombophlebitis, or various sarcomas. Ultrasound may be helpful in diagnosing nontropical pyomyositis (5).

Treatment in HIV+ persons typically includes removal of pus via needle aspiration or open surgical drainage. One or more courses of such antibiotics as nafcillin, cephalexin, vancomycin, cefotaxime, oxacillin, and gentamicin given over several weeks invariably are needed to eliminate the causative infection. Treatment protocols involving only intravenous antibiotics, as described by Hoyle and Goldman (6), may reduce the physiological stress experienced by immunocompromised individuals with pyomyositis. However, such protocols may not be sufficient to resolve widespread or well-established infections. Immunosuppressive compounds such as cyclosporine and azathioprine have been used with some success in persons not infected with HIV (7), but their use may be counterproductive in HIV+ persons.

Polymyositis-dermatomyositis (PM-DM) is another type of muscle damage that has been observed in HIV+ persons. Because (PM-DM) manifests in a number of ways, diagnosing it poses a challenge for clinicians. Bunch (8) has identified five criteria that can be used to establish the diagnosis, including symmetric proximal muscle weakness, increased blood levels of muscle enzymes, inflammatory and myopathic changes observable through muscle biopsy, and rash on the hands, elbows, and knees. Electromyographic examinations reveal normal nerve conduction velocities, insertional activity, fibrillation potentials, and motor unit potentials with greater frequency and lesser duration.

Persons presenting with PM-DM typically list general weakness and moderate to severe muscle pain as their primary concerns. Heightened levels of creatine kinase and decreased erythrocyte sedimentation rates, as well as the EMG irregularities noted above, support a diagnosis of PM-DM. Muscle biopsies usually reveal muscle fiber degeneration and regeneration, inflammatory mononuclear cell infiltration, and central nuclei (8). Dalakas, Pezeshkpour, Gravell, and Sever (9) report that polymyositis may be the first sign of HIV infection. The mechanism by which HIV infection facilitates PM-DM development has not been identified.

Treatment for PM-DM typically includes moderate to high doses (5 to 60 mg/day) of corticosteroids such as prednisone. Espinoza et al. (10) reported that HIV+ persons respond to corticosteroid therapy more slowly than PM-DM-diagnosed persons without HIV infection. Oral candidiasis may occur in HIV+ persons treated with prednisone.

Intravenous gamma globulin infusions may offer another possible treatment for PM-DM. In a study of 11 persons diagnosed with PM-DM, Cherin et al. (11) noted serum profile improvements (e.g. reduced CK levels) in 8 patients. HIV infection was not reported for any of these subjects, however, so no conclusion can be drawn about the therapy's effectiveness for HIV+ populations.

Zidovudine-Induced Myopathy Progression of HIV+ status to AIDS-related complex (ARC) and full-blown AIDS may occur within several months or several years. Once individuals reach this degree of immune function failure, however, their quality of life usually deteriorates fairly rapidly. As a result, large numbers of HIV+ persons take pharmaceutical substances in an effort to slow the development and progression of ARC and AIDS. Zidovudine (AZT), the medication most commonly prescribed to inhibit disease development, has been associated with myopathy among long-term users.

Clinicians report varying rates of myopathy associated with AZT. Chalmers, Greco, and Miller (12) reported AZT-induced myopathy in 17 of 50 patients studied, while Dalakas and Illa (13) suggest that 15 to 20% of those taking AZT develop this condition. Myopathies may become apparent within a few weeks of the start of AZT therapy, but more typically present after 200 days of continued use at standard dosage levels (14). Persons with AZT-induced myopathies complain of fatigue, myalgia, and proximal muscle weakness. Muscle fibers typically display a classic "ragged red" appearance connoting disorganization and significant macrostructural disturbance.

Inflammatory necrosis, Type II fiber and neurogenic atrophy, and nemaline rod formation were associated with development of AIDS even before AZT was available. Histopathological studies of muscle tissue from long-term AZT users reveal dysfunction at the level of muscle mitochondria (15; 16). Under an electron microscope, Pezeshkpour et al. (16) observed increased numbers of subsarcolemmal mitochondria and large, irregular cytoplasmic bodies in the muscle tissue of all 13 AZT users in the study. Immunocytochemical testing revealed a marked decrease in stainable mitochondrial DNA as compared to levels in non-AZT-treated HIV+ persons. Other muscle fiber morphology was consistent with fiber structure of HIV+ persons who had not taken AZT. They concluded that AZT disrupts normal muscle mitochondrial reproduction by preventing accurate DNA replication.

In another study of energy metabolism within muscle, Gherardi et al. (17) reported increased muscle production of interleukin-1a and significant accumulation of the compound within mitochondria, but did not identify the cause of this phenomenon.

Sinnwell et al. (15) evaluated the effects of AZT use on active HIV+ persons through study of metabolites following a graded steady state exercise protocol. They compared changes in phosphocreatine, adenosine triphosphate (ATP), and intracellular pH levels and post-exercise phosphocreatine recovery in the gastrocnemius muscle of 9 HIV+ AZT users, 6 HIV+ AZT non-users, and 19 HIV- persons. Five HIV+ AZT users were able to complete two levels of the four-level exercise test, and four completed three levels, while all but one of the HIV+ AZT non-users completed four levels. Magnetic resonance spectroscopy supported the apparent differences in muscle capability in that persons who had used AZT exhibited greater phosphocreatine depletion than HIV+ AZT non-users and HIV- subjects. Although some HIV+ persons experience muscle atrophy, the leg muscle cross-sectional areas of the AZT users did not differ significantly ($p = 0.05$) from those of the AZT non-users or the HIV- control group.

Although a connection between AZT use and muscle dysfunction clearly exists, the nature of that correlation and the circumstances precipitating myopathy development have not been established conclusively. The fact that a substantial number of AZT users -- at least two-thirds of those on the drug, if the highest estimates of AZT-induced myopathy are accepted -- do not develop myopathies suggests that other factors may be involved.

Lane, McLean, Moss, and Woodrow (18) examined muscle tissue from 23 HIV+ persons experiencing myopathy, 18 of whom had taken AZT for 8 to 20 months. Based on the presence (or lack) of tubuloreticular inclusions in capillary endothelial cells, they concluded that AZT causes myopathies only when other HIV-associated inflammatory myopathy(s) have already been

established. They hypothesized that TRI activity is the initial sign of HIV activity within muscle.

Treatment for AZT-induced myopathies takes two primary forms, cessation of AZT use and corticosteroid therapy. Chalmers et al. (12) reported decreases in myalgia and CK levels, increases in strength, and trends toward normal EMG test results in all 17 persons who discontinued AZT use following diagnosis of AZT-induced myopathy.

The results of that study, however, have been challenged by a more recent initiative. Manji et al. (19) noted improvement in the condition of 5 of 11 AZT users with myopathies. In persons with moderate myalgia, they found increases in quadriceps strength 8 weeks after cessation of AZT. Persons with more severe impairment required treatment with corticosteroids (e.g. prednisolone) or thalidomide to regain muscle function. Four patients who later began taking AZT experienced return of myalgia and other symptoms. The mechanism of action for thalidomide was unknown at the time of the study, although its use demonstrated actions in muscle tissue similar to those of the steroids.

Cessation of AZT use may permit and/or facilitate improvements in myopathies, but the drug's ability to inhibit progression of ARC and AIDS makes discontinuation an unacceptable option for large numbers of HIV+ persons. Corticosteriods, too, pose an unpalatable form of treatment due to their immunosuppressive actions. Despite the potential problems AZT may create for active HIV+ persons, many if not most users will be reluctant to stop taking this medication until other equally effective antiviral drugs become available. Thus, exercise professionals must take into account AZT's deleterious effects and monitor active AZT users for possible health problems.

Summary. HIV+ individuals face numerous challenges to regular exercise completely separate from progression of AIDS and AIDS-related complex. Pyomyositis, polymyositis-dermatomyositis, and AZT-induced myopathies may not only reduce physical performance, but also curtail it entirely. In persons capable of exercising without noticeable pain or fatigue, trauma or excessive exertion may trigger the development of myositis and thereby limit future mobility. Use of AZT to extend survival may, in addition, reduce the quality of life through physical impairment.

Despite the obstacles posed by muscle dysfunctions related to HIV infection and its therapy, exercise prescription for HIV+ persons is possible. HIV+ status introduces additional variables into the activity equation, but the science of exercise management has always focused on individuals. With the appropriate monitoring of health status and a willingness to adjust activity as necessary based on day to day circumstances, regular exercise can be both manageable and beneficial.

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Adaptation of Skeletal Muscle to Resistance Training

By Christopher Berger

Introduction. All too often success in the weight room is measured by the number of reps performed or the amount of weight lifted. Certainly bench pressing your own weight for the first time can be a gratifying and motivating experience. Unfortunately, many exercise participants experience difficulty seeing beyond such markers of performance, and their workouts become stale, predictable periods in the gym with little or no improvement over time. The ultimate goal of a safe and effective resistance training program is to elicit and maintain an adaptive response from the skeletal musculature. Although this concept may seem simple, the body possesses a number of complex mechanisms by which it adapts to the stress of resistance training. Not all of these mechanisms are well understood, but participants who appreciate the complexity of the body's response are in the best position to maximize their time in the weight room.

General Adaptation Syndrome (GAS)..A good place to begin to understand the adaptation of skeletal muscle to resistance training is with the work of Hans Selye. After a failed attempt 40 years ago to design a new hormone, Selye theorized that the human body possesses a "non-specific response to stress" (1). This theory incorporates three stages: the alarm reaction, the stage of resistance, and the stage of exhaustion.

The alarm reaction is an acute stage often eliciting a "fight or flight" response from the central nervous system. For resistance trainers, this stage may be characterized by a decrease in performance shortly after beginning a program. Common problems involving muscle soreness and fatigue may be disappointing hindrances to eager participants, but they are two of the body's many important protective mechanisms and should not be ignored.

The stage of resistance is also commonly referred to as the "stage of adaptation." For the resistance trainer, performance increases during this stage as the body adapts to the program. Most of the physical or mental exertions, infections, and other stresses which act upon us produce changes corresponding only to the alarm reaction and stage of resistance.

Finally, the stage of exhaustion is reached when the body's ability to resist or adapt to a stress fails. The exhaustion stage is marked by one of two possible events in the resistance trainer (2). In one case, despite strict adherence to a tried and true program, the participant notices that performance neither decreases nor increases but plateaus. In the second case, performance decreases as the resistance trainer reaches a state of overtraining characterized by the over-stressed body's inability to adapt to the program. Both cases are excellent examples of the peril of paying too much attention to the absolute amount of weight lifted. Whether or not the weight can be lifted becomes irrelevant because the body has already failed to adapt.

The Overload Principle and Overtraining. Both cases of exhaustion described above present problems to resistance trainers even though it is tempting to think of exhaustion only in terms of being overtrained. Exhaustion also occurs when the overload principle is abused. The overload principle states that the strength, endurance, and cross-sectional area (CSA) of a muscle fiber will increase only when the muscle performs for a given period of time at near its maximal strength and endurance capacity (3). In other words, muscle will not demonstrate improved performance unless called upon to do so. If the muscle fiber is loaded beyond its adaptive capacity, it will be destroyed.

On the other hand, persistent and unexplained poor performance, difficulty in recovering from exercise bouts, and overuse injuries are all characteristics of overtraining. These problems are

highly individualized systemic responses and do not subside without adequate rest. Typically, overtraining represents a level of training that exceeds the whole body's capacity to adapt to multiple stresses rather than a specific muscle or muscle fiber's ability to adapt to a load, as is seen in the abuses of the overload principle.

Muscle Damage. In the context of Selye's GAS, muscle damage occurs because the muscle fails to adapt to loading. In essence, the muscle has reached its exhaustion stage. Muscle damage can vary from imperceptible molecular changes to gross, whole-muscle changes (4). Evidence of muscle damage (following repetitive eccentric muscle actions) includes morphological changes, delayed-onset muscle soreness (DOMS), decrements in markers of performance, and increases in muscle proteins (especially creatine kinase) (5). These events generally are not considered to be good things for the resistance trainer, but participants who do not appreciate the adaptive response of muscle tissue often pursue doomed programs under the impression that these symptoms are indicative of a normal progression toward strength or endurance. It is worth noting, however, that muscle damage following these actions is temporary and repairable in muscles unaccustomed to strenuous activity, but muscle that has adapted to the stress of repeated exercise results in little or no damage. Moreover, Brown et al. (6) found that skeletal muscle adaptation can be brought about by a single bout of relatively few eccentric muscle contractions.

Muscle Fiber Hypertrophy. Muscle fiber hypertrophy is simply an increase in the size of the muscle fiber (3). This increase is usually measured at the cross-sectional diameter of the muscle fiber and is a function of the type of fiber in question. Five changes have been linked to muscle fiber hypertrophy (7): increased number of myofibrils (proportionate to the degree of hypertrophy), up to 120% increase in mitochondrial enzymes, up to 60-80% increase in the components of the phosphagen metabolic system (including both ATP and phosphocreatine), up to 50% increase in stored glycogen, and up to 75-100% increase in stored triglyceride. Capillary changes proportional to muscle fiber growth have also been observed in college men following resistance training (8).

Genetics and testosterone determine the basic size of a person's muscles but, with training, muscles can increase in size by 30-60% (7). Despite the importance of testosterone, relative changes in muscle hypertrophy (and strength of the muscle due to resistance training) have been found to be similar in men and women (9).

So what accounts for this increase in size? One plausible explanation is that muscle hypertrophy may involve a repeated injury of the muscle fiber followed by an overcompensation of protein synthesis resulting in a net anabolic effect (10). The physical condition of the participant is believed to play a role in the timing of these events. Ruthner et al. (11) observed that in sedentary individuals engaged in a resistance training program, inhibition of activation during voluntary effort early in the program was a limitation to hypertrophy. The neuromuscular system is believed to possess a conservative strategy to enhance performance. Enhanced performance early in resistance training occurs while sparing the costly metabolic event of net contractile protein accumulation. The authors caution that these results are likely to be limited to sedentary individuals, however, since athletes and other physically active participants already possess the requisite neural adaptations to resistance exercise and can impose significant mechanical stress on skeletal muscle.

Muscle Fiber Hyperplasia. Muscle fiber hyperplasia represents an increase in the net number of muscle fibers within the whole muscle. The role of muscle fiber hyperplasia in increasing human muscle size has been a source of controversy. It seems likely that in several animal species, there is an increase in the number of muscle fiber numbers developed in response to mechanical overload (12) but this has been suggested to be a compensatory adjustment to overload since massive cellular hypertrophy (as observed in humans) does not occur in many animal species (13). Even if hyperplasia is not the primary adaptational response of most muscle fibers, it might represent an

adaptation to resistance training occurring when certain muscle fibers reach a theoretical upper limit in cell size (2). Mikesky et al. (14) have suggested that in cats increases in muscle fiber CSA do not account for all of the increase in muscle mass resulting from resistance training. Factors in addition to hyperplasia were believed to include disproportionate increases in water content, protein content, connective tissue, and/or muscle fiber length.

Muscle Fiber Transformation and Changes in Connective Tissue. There are two main types of muscle fibers, slow-twitch (ST) and fast-twitch (FT). Each type of muscle fiber possesses a subtype of fibers classified according to how each appears when stained and viewed in cross-section. It is now believed that transformation within a particular muscle fiber subtype is a common adaptation to resistance training. Changes may occur only within fiber type and are related to the function of the training program and sequential changes in contractile proteins (2).

In addition, as muscle becomes stronger, ligament, tendon, and bone also adapt in order to support greater force and weight. This increase in connective tissue mass is related to a net increase in the size of a muscle because connective tissue surrounds the entire muscle (epimysium), groups of muscle fibers (perimysium), and individual muscle fibers (endomysium).

Neuromuscular Adaptations. The ability of a muscle fiber to generate increased force is related to the extent to which the motor unit can activate the muscle. This concept is referred to as "neural adaptation" (15) and is less understood than factors discussed previously. A motor unit consists of a motor neuron and all of the muscle fibers it innervates. As discussed above, muscle fibers vary in type and how they function, but it has been observed that muscle hypertrophy generally involves structural changes in FT fibers and that motor units are preferentially recruited during exercise (small ones first and large ones last) (3). These adaptations to strength and endurance are advantageous just as the same is true for the adaptations that follow periods of reduced activity (16). It simply does not make sense functionally and bioenergetically to recruit larger, slower fibers when smaller, faster fibers are able to perform the same task.

Conclusion. The human body possesses a remarkable capacity to adapt to the stress of life. Resistance trainers who understand the adaptive capacity of the musculature are more able to develop safe and effective training programs that provide a continuous challenge. Appreciating the adaptive processes of skeletal muscle also serves to shift a participant's thought processes from, "How much should I lift in the weight room today?" to "What response am I trying to evoke?" This change in thinking is likely to stimulate a new understanding of the principles underlying success in the weight room.

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Book Review

Thomas, J.R. and Nelson, J.K. *Research Methods in Physical Activity (3rd ed.)* Champaign, IL: Human Kinetics, 1997.

Jerry Thomas and Jack Nelson have teamed up again to produce a third edition of their popular book *Research Methods in Physical Activity*. Readers familiar with previous editions will be pleased (or maybe annoyed) to see that the authors have retained the humorous writing style and corny examples that made older editions so engaging. (Be sure to read, "Top 10 Problems That Have Not Been Resolved by Humankind" on page 26 - it will inspire you to whole new levels of research!) The book consists of 485 pages with an ample supply of photos, tables, and illustrations. Although the expertise and writing style of the authors is present throughout the text, there are two additional contributing authors. Nancy L. Struna shares her knowledge of Historical Research in chapter 12 and, R. Scott Kretchmar does the same for chapter 13 on Philosophic Research.

One of the major strengths of this book is that it is comprehensive enough to serve the needs of both beginning and advanced researchers. The text is not only thorough in its 20-chapter treatment of research methods topics, but also a helpful reference throughout the process of research. For example, chapter two contains a list of some periodicals in exercise science, as well as a table describing some databases available for computer searches. These would be excellent resources for the first-time researcher in the field. The four appendices at the end of the book contain statistical tables and sample consent forms that would be especially helpful to those with additional experience.

Another strength of this book lies in the end-of-chapter features called "Check Your Understanding." These pages follow each chapter and serve as opportunities for the reader to practice the concepts just explained. Since research is about doing, it only makes sense to get involved in activities that do such things as consider case studies in ethical issues (pp. 87-88) or calculate effect sizes (p. 304). Most exercises are short and clearly explained, but they would have been more helpful had the authors included the answers when appropriate. The authors refer readers to the instructor for the answers.

There are few weaknesses to this edition, but there is one feature that the authors may wish to consider prior to the next revision. Because those who do physical activity research likely will be using statistical methods throughout the process, it is helpful to find a research methods book that links the two areas so effectively. Readers of Research Methods can be assured that the authors provide an effective treatment of statistical methods in this edition, but it almost appears as though this book is starting to become two books in one. Statistical concepts are woven throughout the text (even in chapter 17 on Qualitative Research), but even elementary statistics these days requires material and coursework running beyond the scope of research methods. Perhaps the size and cost of the book could be reduced if topics such as multivariate techniques and nonparametric techniques (chapters 9 and 10 respectively) were eliminated and covered more effectively in a separate statistical methods text.

Overall, Research Methods is clearly a book to buy and keep. It won't be happy stored on some bookshelf either. With its engaging readability and complete treatment of research methods in all areas of physical activity research, Research Methods is an important resource for such a growing discipline.

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Book Review

Foss, M.L. and Keteyian, S.J. *Fox's Physiological Basis for Exercise and Sport (6th ed.)* Boston, MA: WCB:McGraw-Hill, 1998.

If you are looking for an exercise physiology book that will bridge the gap between your undergraduate and graduate years, look no further than the latest edition of *Fox's Physiological Basis for Exercise and Sport*. Readers of previous editions of this textbook will recognize it as "The physiological basis of physical education and athletics" or more recently as, "The physiological basis for exercise and sport". Fortunately, many of the positive qualities of previous editions have been retained. This new edition consists of 620 pages including eight appendices and a glossary.

An immediately apparent strength of this book is the expansive use of color and illustrations that clarify points described in the text. For instance, Figure 6.9, along with corresponding Table 6.2, succinctly describes skeletal muscle contraction events more effectively than could be achieved through a tedious narrative on the subject. Physiology is about action and the authors underscore this fact. The book is an excellent example of how exercise physiology books should be written.

There are seven sections to this text including bioenergetics, neuromuscular concepts, cardiorespiratory considerations, physical training, nutrition and body weight control, humoral responses and performance aids, and the environment. These sections are representative of the current state of exercise physiology research. The authors organized topics in logical sequences that contribute to the reader's understanding of the bigger picture. For example, section four discusses methods for anaerobic training in Chapter 11 and then proceeds to methods for aerobic training in Chapter 12. Having been exposed to the bioenergetic spectrum of physical activities, the reader is prepared to tackle the development of strength, endurance, and flexibility in Chapter 13 and physical activity and health in Chapter 14. Yet, it is not absolutely essential to follow the chapters in sequence. Depending upon the background and expertise of the reader, each chapter can stand on its own.

A few issues could have benefited from clearer, simpler explanations. For example, in pages 340-344 the definition and types of muscular contractions (isotonic, isometric, eccentric, and isokinetic) are discussed. Understanding these muscle actions is very important. Foss and Keteyian could have tapped into their effective use of tables and figures to help the reader visualize the meaning and interrelationships of muscle actions to physical activity.

Overall, Foss and Keteyian have written an outstanding book that will serve a diverse audience during a rapid period of change in exercise physiology. *Fox's Physiological Basis for Exercise and Sport* will be a helpful addition to any student's bookshelf.

Christopher Berger