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The Cause of Heat Stroke During Practice

On August 1, 2001, a football player died of a heat stroke that could have been prevented. All that was needed was professionalism on the part of the coach.

Professional coaches—those who have professional habits instilled in them in the course of their education—know signs of various degrees of fatigue and of heat exhaustion. (Heat exhaustion precedes heat stroke.) Recognizing these signs is part of their training in exercise physiology and the methodology of sports training. They know too that an athlete who shows signs of heavy fatigue or of heat exhaustion must be stopped from exercising. The cause of heat stroke during practice is not heat or humidity—its cause is a coach oblivious to the warning signs of heavy fatigue and heat exhaustion.

On the day he died, the player (Korey Stringer) vomited three times during the morning conditioning drills. After the first incident he should have been removed from the field and sent to a team doctor, regardless of the weather. Actually, he showed signs of ill health two days before he died, so the whole affair is even more damning. Photos of Korey Stringer taken on the first day of training camp (two days before he died) show that he was not coping with exercising in the heat. He was carted off the field on that first day of practice. Allowing him to practice on the day after was unprofessional.

Signs of heavy fatigue: head and arms hang down, the rib cage caves in, the frequency of breathing suddenly increases, athlete leans forward at rest, the upper body sweats heavily, cold sweat on the face, face pale or very red, eyes dull, voice muffled and interrupted, and movements lose precision, rhythm, and amplitude. At these signs the exercises should be stopped.

A coach who does not recognize and react to signs of unhealthy fatigue—and even worse, of heat exhaustion—is dangerously unprofessional. It is the coach's job to know these signs, to know his or her athletes, to notice small signs of impending injury and to stop athletes from hurting themselves. What was this coach doing during the practice? What was he watching if not his players?

As beach volleyball coach Charles Richardson said, "The best athletes are the ones with the most drive and motivation and even obsession," so it is up to the coach to coldly notice signs (a wince, a limp, wrong skin color, and other signs given in *Science of Sports Training*) that some athletes, with their high threshold of pain, or just ignorance, may disregard.

A coach does not have to like the athletes to watch them carefully—this is not a matter of liking or of fuzzy "caring," but of drilled-in professional habit.

It is well illustrated by an anecdote from the time of the Bolshevik revolution. Once a White artillery unit was captured by the Reds. The Reds killed all prisoners except one White officer who was in charge of directing and calculating artillery fire (a job that requires excellent mathematical skills). The Reds needed his skills so they spared him and, at gunpoint, put him in charge of their artillery.

In the next battle the Reds were routed

Signs of heat exhaustion: heavy sweating, thirst, cold and pale skin, breathlessness, muscle cramps, weakness, dizziness, nausea, vomiting, headache, fainting. There is no excuse for exercising past these signs. Actually, in rationally conducted sports training heat exhaustion does not happen. The next step after heat exhaustion is heat stroke.

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by a White unit even though the Reds' artillery inflicted lots of casualties on the Whites. The recaptured White officer was debriefed by his liberators and asked who was in charge of the Reds surprisingly effective artillery fire. He said, "I was." They asked, "Why did you do it so well and inflict so many losses on your own people?" He answered, "A professional habit."

Signs of heat stroke: red, hot and dry skin, rapid breathing, dizziness, headache, nausea, confusion, loss of consciousness.

Without proper medical attention heat stroke progresses to coma, permanent central nervous system damage, and death.

Muscle Fatigue

by [Piotr Drabik](#)

Coaches and athletes need to know what fatigue is to understand all issues of the training process.

The whole training process is predicated on fatigue and on recovery from it—the changes of training load, means of recovery, frequency and sequence of exercises and workouts, periodization, and nutrition. Without understanding fatigue it is not possible to understand all these issues as well as the upsides and downsides of different supplements.

Fatigue is a decreased effort capacity of a body, or part of it, resulting from exertion or excessive stimulation. There are several types of fatigue: mental (boredom), sensory (a result of intense activity of one or more of the senses), emotional (a consequence of intense emotions, observed after performance at important sports competitions, or after executing movements that demand overcoming fear), and physical (caused by muscle work). While workouts cause all types of fatigue (in various degrees) the most obvious is physical fatigue. Physical fatigue directly affects the function and structure of the musculoskeletal system and muscle fatigue is its main component.

—Thomas Kurz

Muscle fatigue is any reduction in force-generating capacity of the involved parts of the neuromuscular system.

For the last 150 years the nature and mechanism of fatigue were the objects of a great number of studies. Fatigue in a voluntary muscular effort is a complex phenomenon influenced by central nervous system factors and peripheral factors,

such as damage to muscle cells. According to the latest scientific data, the two main factors causing muscle fatigue are mechanical damage and chemical damage to muscle cells. There is strong evidence to suggest a relationship between physico-chemical damages and exercise-induced muscle soreness.

Mechanical damage

Mechanical damage is caused by mechanical stress.

Mechanical damage is most likely to occur when the vector of force acting on a muscle fiber (muscle cell) is not parallel to the long axis of this muscle fiber. This is usually the case because of the way muscle fibers are arranged in most muscles. (And indeed, hardly anyone has gotten strained or sore muscles of one's arms, for example, by merely holding heavy weights with the arms hanging straight down. This is because in such a position the majority of muscle fibers lie parallel to the line of force and the angles of those that are not parallel are very acute.)

Mechanical stress destroys partially or completely three types of structures in the muscle cell: membranes, myofibrils, and the cytoskeleton.

Membranes (cellular membrane, mitochondrial membranes, nuclear membrane, sarcoplasmic reticulum membrane, lysosomal membrane, and more) separate various processes going on within the cell, and when the membranes are damaged the molecules mix and all cell functions are impaired.

Myofibrils are the contractile elements of muscle cells, responsible for muscle

action, whether concentric, static, or eccentric. They are built of long, thin proteins that are easy to tear. When its myofibrils are destroyed, a muscle cell cannot contract.

The cytoskeleton is the internal scaffolding of the cell. Among its many functions, the cytoskeleton gives shape to the cell, and in the case of muscle cells transmits forces generated by the myofibrils. When the cytoskeleton is damaged the end effect is the same as destroying myofibrils—the cell cannot contract.

The more intense the effort, the bigger is the number of destroyed muscle cells.

Chemical damage

Chemical damage is caused by oxidative stress—the overproduction of oxygen-derived free radicals. Oxidative stress damages all the structures of the cell as well as enzymes and nucleic acids that store and pass genetic information.

Free radicals are oxygen metabolites with unpaired electrons that are highly reactive. Examples of free radicals are superoxide (O_2^-) and hydroxyl (HO^\cdot). Their unpaired electrons make the free radicals extremely reactive, being capable of interacting with various organic molecules—building blocks of cells—(proteins, lipids, and nucleic acids that comprise the genetic material of living cells and regulate all cell activity) and interfering with their structure and metabolism.

Though most of these radicals are inactivated by naturally occurring products of the body, a certain fraction escapes the cellular defense systems and may react with cellular components.

Self-Defense Tip

Being proficient with the method of an attack is necessary for realistic practice, and thus for acquiring effective self-defense skills. People who do not know what makes attacks work can be persuaded to practice useless “defenses” that won't work against the real thing.

Phony self-defense experts show “impressive” defenses that work only against unrealistic attacks. (By *realistic attack* I do not mean professional or very well-practiced, just done with real intent.)

Take a simple bear hug from behind. Phonies show various “clever” releases, but all these releases work only against a bear hug that is done

wrong in every respect. The attacker in such a self-defense demo attacks like a complete moron—grabs the defender just below the shoulders (where the cross section of the defender's upper body is largest, and the defender's arms are at their strongest position). In addition, the attacker does not tuck his or her head into the defender's back (to protect it from defender's head butts), does not keep his hips low for stability, and does not continue the attack that started with the bear hug (the bear hug is just a grab that leads to a takedown or a slam on the ground). On top of that, the grab itself is usually done at less than full strength.

In a realistic bear hug the attacker grabs at the level of the elbows and floating ribs—this is the narrowest part of the defender's trunk, it is easy to squeeze breath out of the defender, and the defender's arms are at their weakest position.

All sorts of grabs and chokes are applied in similarly unrealistic fashion in phony demos. The same lack of realism can be seen in popularly taught defenses against strikes and kicks. It all arises from the demonstrator's lack of fighting experience and of common sense or from disregard for the viewers. To learn realistic unarmed attacks and defenses see the video [Basic Instincts of Self-Defense](#).

Muscle Fatigue (continued from page 2)

Exercise leads to an increase in the free radicals produced, and the following lipid peroxidation* provokes changes in all biological membranes. Their fluidity changes, thus reducing the ability to communicate with other cells. So does their integrity, which causes the dissolution of the cell.

Proteins are the most important components of all living species. They are the constituents of enzymes, receptors, and structural building blocks. All cellular proteins are endangered by free radicals, which cause cascades of changes that severely impair cellular metabolism.

It has been shown that exercising to exhaustion increases DNA damage in white blood cells of untrained subjects. Moderate exercise of intensities below the anaerobic threshold shows no such effects. Supplementation with the antioxidant vitamin E prevents exercise-induced DNA damage. Adaptation to exercise (being in good shape) seems to reduce free radical-associated effects, such as DNA damage.

Many indicators of muscular damage caused by oxygen radicals are restored to normal in the after-effort recovery period. Even well-trained individuals after a bout of hard exercise display indicators of both damage and repair.

Other causes of fatigue

Apart from mechanical and chemical damages, which are the two major factors causing fatigue, there are some additional changes that contribute to muscle fatigue. They are the consequences of or a background for physicochemical damages.

Muscle performance declines during prolonged and intense activity—the force and velocity of contractions are reduced and time needed for relaxation is increased (meaning that muscles do not relax as rapidly after a contraction as they do when not fatigued). The changes in metabolites (particularly H^+ , Ca^{2+} , and ATP^{**}) lead to the observed changes in force, contraction, and relaxation.

It is generally stated that muscle H^+ accumulation may contribute to fatigue during intense exercise. According to the latest research results, however, the changes in pH (an indicator of H^+ concentration in a solution) may have little or no role in the loss of force production associated with muscle fatigue.

During fatigue, the sarcoplasmic reticulum (SR), an organelle which is a cellular calcium reservoir, undergoes basic changes. The Ca^{2+} uptake and Ca^{2+} -ATPase activity of the SR are depressed in the fatigued muscles, thus reducing calcium reuptake by the SR and increasing the binding of calcium by calcium-binding proteins (and altering, in that way, the functions of these proteins, which impairs the cellular metabolism). These changes likely result in altered force production and energy consumption by the intact muscle.

The cellular processes contributing to fatigue may also lead to decreased force or power output due to an insufficient rate of neurotransmitter (acetylcholine) synthesis in the synapses and a reduced rate of ionic pumps function in cellular membranes of both muscle cells and nerve cells.

The information about molecular changes induced by mechanical damage, chemical damage, and the other changes is sent to the brain via pain receptors and makes a person feel muscle soreness or pain.

Pain receptors are free nerve endings within the tissue that are excited by chemical, mechanical, and thermal stimuli. In health, pain informs of damage and so is a part of the body's defense against injury. The stimuli that excite pain receptors are signals to the brain indicating damage. Substances released from damaged muscle cells include bradykinin, histamines, prostaglandins, excess potassium ions, serotonin, and proteolytic enzymes.

Apart from the cellular changes characteristic to muscle cells, fatigue seems to be connected with other important type of

cells, namely neurons.

Relatively little attention has been placed on the role of the central nervous system (CNS) in fatigue during exercise. Several biological mechanisms have been proposed to explain CNS fatigue. Hypotheses have been developed related to several neurotransmitters including serotonin (5-HT—5-hydroxytryptamine), dopamine, and acetylcholine. The most prominent one involves an increase in 5-HT activity in various brain regions. Good evidence suggests that increases and decreases in brain 5-HT activity during prolonged exercise hasten and delay fatigue, respectively, and nutritional manipulations designed to reduce brain 5-HT synthesis during prolonged exercise improve endurance performance.

Other neuromodulators that may influence fatigue during exercise include cytokines and ammonia. Increases in several cytokines have been associated with reduced exercise tolerance associated with acute viral or bacterial infection. Accumulation of ammonia in the blood and brain during exercise could also negatively affect the CNS function and thus contribute to fatigue because impaired CNS function worsens coordination.

Conclusion

In conclusion, muscular fatigue is caused by many factors. Mechanical damage and chemical damage are the major factors, however. The role of each factor depends on the kind of exercise, individual characteristics, and external conditions.

Let us know what you think about our newsletter. Have you learned something that improved your or your athletes' performance or health? What would you like to learn more about? Write to us at our address:
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* Lipid peroxidation may be defined as oxidative deterioration of lipids by free radical reactions

** ATP by its very presence (in addition to its role as an energy carrier) is needed for relaxation of muscles. Without ATP, myosin crossbridges are stuck to actin and cannot move. This lack of ATP is the cause of rigor mortis.

Q and A on STRETCHING and TRAINING

(continued from previous issue)

Study these typical questions on stretching and training carefully. You may find information that relates to questions of yours. Questions are in *italic boldface*.

■ **In the book *Science of Sports Training* you touched on the subject of sunshine being beneficial to the body, with the amount of exposure being between 30–60 minutes a day and gradually increasing. Do you recommend that athletes use sunscreen or just expose the body to get any benefits from this exposure?**

I admit that this information on exposure to sunshine is not very specific. Here are the specifics:

The duration of exposure should be such as to cause no reddening of the skin 24 hours after the exposure (slight reddening without other sensations after 8-12 hours is permissible). Therefore, duration of the exposure will differ depending on the following factors:

- geographical latitude—the lower the latitude, the shorter the exposure;
- position of the sun—the higher the sun, the shorter the exposure because of the greater amount of ultraviolet radiation;
- altitude—the higher the altitude, the shorter the exposure; and
- reflectivity of the surroundings—the more reflective, the shorter the exposure because, for example, snow, water, or sand reflect more of the sun's radiation than grass.

If one follows the “no reddening after 24 hours” rule, sunscreen should not be needed.

Children must have their heads protected with hats or caps and eyes with sunglasses.

The durations of exposure given in the book are typical for latitudes of Central Europe.

■ **I just finished reading *Stretching Scientifically* by Thomas Kurz, and I really enjoyed it! I just have a question—if doing dynamic stretches every morning resets the nervous control of muscular length, can't static stretches be done in the morn-**

ing to achieve the same effect, except for static flexibility rather than dynamic? So then if every morning I did static stretching, I wouldn't need a warm-up to achieve full static flexibility, would I?

Perhaps. I do not know. Athletes who use the method of stretching explained in the book *Stretching Scientifically* for a few months can display their maximal static flexibility at any time anyway, so how would one evaluate the effect of doing a static stretch in the morning?

To find this out it would take an experiment that is not likely to be ever conducted. Why? Because from the viewpoint of practice-oriented coaches, not every question is worthy of research.

You see, doing dynamic stretches in the morning gives an observable increase in athletes' dynamic flexibility later in the day regardless of whether the athletes do any full ROM (range of motion) static stretches in their training or not. But athletes who do full ROM static stretches invariably do dynamic stretches too. So finding out what helps them show a large ROM without a warm-up in static stretches—whether it is due to dynamic stretches done in the morning, or to static stretches done in the morning, or just an effect of static stretches done during their regular workouts—requires conducting an experiment that could interfere with their training.

After a few months of continuing isometric stretching, those who achieve full ROM, can display it (for example, do a split) anytime—even though initially such a split did require warming up. This is much like lifting weights—establishing a new personal record may require carefully warming up, but with continuous training, after a few months, the same weight may be lifted cold.

A question on the average time frame from being able to do a side split after several isometric stretches to doing the split instantly was answered in the *Winter 2000 issue of Stadion News*.

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