Dynamic Chiropractic

SPORTS / EXERCISE / FITNESS

Exercise Physiology Issues, Part 2

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Exercise Stiffness

Many rehabilitation patients inevitably have stiff, painful, slow-functioning leg muscles after an exercise. Myoglobinemia has been reported in many endurance activities. Hansen et al. determined the degree of leakage of myoglobin and other muscle proteins into the circulation during various types of muscular activity.[1] Sixteen highly-trained marathon runners aged 19-44 years were examined, along with nine nonrunners of similar age and six rowers aged 15-24 years. All subjects were men.

The runners trained 100-350 km per week and had maximal oxygen uptake values of 72-90 ml oxygen per minute per kilogram of body weight. The nonrunners had maximal oxygen uptake values of about 40 ml, and the rowers' values were at 60-75 ml. Four runners were examined after running 17-27 km on a hilly course at a mean rate of 17.4 km per hour. Six runners and six nonrunners were compared during and after a noncompetitive test run of 27 km or 12 km, respectively. Runners also participated in 15 km and 30 km events. The rowers were examined after hard rowing for 1 1/2 hours.

Runners returned from the 17-27 km race with plasma-free hemoglobin values of 2.4-10 mole/L. High transferrin iron-binding capacity and low haptoglobin values also were noted. The runners received iron daily and had normal serum iron levels. All the runners had abnormally high serum myoglobin values immediately after the race. Minimal myoglobinuria was observed for 24 hours. No runner had significant proteinuria. Serum lactic dehydrogenase, creatinine kinase and alkaline phosphatase values were increased. Nonrunners had a greater increase in serum myoglobin levels than did the runners. Most runners had increased values of serum creatinine kinase B after the competitive run, in contrast to those values after noncompetitive runs or rowing. The rowers had elevations of serum enzymes and serum myoglobin similar to those seen in the runners. Free plasma hemoglobin was present in all the rowers.

Both distance running and rowing damage skeletal muscle cells. The myopathic condition resolves within 24 hours except for soreness in leg muscles. The changes after competitive running are consistent with some leakage of muscle proteins from myocardial cells. Neither myoglobinemia nor hemoglobinemia appears to explain adequately the iron deficiency that occurs in athletes.

This study makes a careful comparison of the biochemical damage sustained by muscle in athletes and nonathletes after running, and in rowers after rowing. The fact that protein leaks from the muscles of rowers makes a traumatic explanation unlikely. This phenomenon is possible after endurance exercise within the clinic. Nutritional intervention is helpful.

Exercise-Induced Soreness

Acute soreness develops during exercise and is related by Abraham[2] to stress-induced ischemia. Pain occurs when exercise is intense enough to produce muscle ischemia and the muscle cannot remove metabolic waste products such as lactic acid and potassium quickly enough. Critical

concentrations of these substances can stimulate pain, which continues until exercise intensity is reduced or work ceases, both of which result in increased blood flow.

Delayed muscle soreness develops 24-48 hours after exercise. It has been attributed to torn tissues, spasm and the strain placed on the elastic component of muscle by eccentric work. Increases in urinary myoglobin excretion have been associated with exercise-induced muscle soreness, but they may also follow exercise when soreness does not develop. The relationship between exercise-induced muscle soreness and urinary hydroxyproline excretion can be compared. Significant correlation is apparent between the time of maximum hydroxyproline excretion and the day when the most soreness is reported. Delayed soreness appears to be linked to an irritation of the connective tissue in muscles.

Exercise-Delayed Soreness

Exercise that uses untrained muscles results in delayed muscle soreness and increased muscle enzyme activities. Tiidus[3] examined the time course of these effects and the importance of the intensity and duration of exercise in untrained individuals of both sexes aged 20-45 years. All exercised less than once a week. A dynamic leg-extension apparatus was used. Six persons performed sets of up to 10 contractions to establish the time course of post-exercise serum enzyme changes and muscle soreness. Twenty-one subjects then performed exercise at various intensities and durations at 10-day intervals.

The most severe muscle soreness and the highest serum creatine phosphokinase (CPK), lactic dehydrogenase and glutamic oxaloacetic transaminase activities occurred 48 hours and 8-24 hours after exercise, respectively, in the pilot study. Increasing intensities and durations of exercise resulted in corresponding increases in enzyme activities and muscle soreness. High-intensity, brief exercise led to greater enzyme activities and more muscle soreness than low-intensity exercise of long duration. The serum CPK activity correlated significantly with the change of degree of muscle soreness.

The findings suggest that exercise-induced muscle damage leads to leakage of muscle enzyme into the blood and physiologic reactions to the muscle damage that result in a sensation of muscle soreness. Studies in rate have shown myofibrillar disruption, macrophage and fibroblast accumulation, and necrosis in exercised muscles in association with serum enzyme elevations. The exercise-induced changes may be casually related to delayed muscle soreness. What is important is that the enzyme elevation and subjective symptoms of soreness are signals of profound cellular change, which includes cellular disruption, fibroblastic change and necrosis.

Exercise and Sleep

Slow-wave sleep (SWS) has been suggested to be a restorative phase of sleep. Exercise has been hypothesized to create a demand for SWS proportional to energy expenditure. Bunnell[4] used a quantified submaximal exercise procedure, carried out to volitional exhaustion, to test the exercise-SWS hypothesis. Measures of total caloric expenditure permitted comparisons on individual energy costs and changes in SWS.

Five women and four men, aged 21-30 years, participated in the study. All were moderately active, but none engaged in vigorous physical training. Four patients typically exercised regularly. Recordings were made on adaptation and baseline nights and for two nights after an afternoon exercise bout in which the subjects walked on a treadmill for 50 minutes at 50-70% of maximal oxygen uptake to the point of volitional exhaustion.

The duration of SWS before the onset of rapid eye movement (REM) sleep increased markedly on the exercise night. Moderate increases in stage four sleep and total SWS were observed. An increased latency to first REM onset and decreased duration of the first REM period were also found. Initial REM cycle length decreased. The increases in SWS before the onset of REM sleep averaged 24 minutes in women and six minutes in men. In women, this increase correlated with total caloric expenditure during exercise with a coefficient of 0.85. Heart rate and cardiac output during sleep were significantly increased on the exercise night. A significant fall in nocturnal urinary cortisol excretion followed exercise.

The findings suggest that exhaustive exercise affects sleep primarily in the early part of the night. An increase in SWS pressure is observed at the expense of REM sleep. The increase in SWS in the first non-REM period is consistently greater in women than in men. Any explanation of the increase in SWS after exhaustive exercise must take into account the apparently greater effect of exercise intensity, compared with caloric expenditure itself. An explanation that holds that SWS reflects primarily neural functioning seems to account for the changes in both SWS and REM sleep.

The metabolic and biochemical alterations induced by vigorous exercise have not been identified precisely. It is of some interest that in rats that have swum to exhaustion, there is a 15% increase in whole brain serotonin and a 20% drop in norepinephrine levels. Whether this pattern is reduplicated or paralleled in the higher vertebrates remains to be seen. The main points are well documented: slow wave sleep is increased early on; REM sleep is decreased; and the effect is greater in women than in men.

References

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