Effect of ingested sodium bicarbonate on muscle force, fatigue, and recovery

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Verbitsky, O., J. Mizrahi, M. Levin, and E. Isakov. Effect of ingested sodium bicarbonate on muscle force, fatigue, and recovery. J. Appl. Physiol. 83(2): 333-337, 1997.-The influence of acute ingestion of NaHCO₃ on fatigue and recovery of the quadriceps femoris muscle after exercise was studied in six healthy male subjects. A bicycle ergometer was used for exercising under three loading conditions: test A, load corresponding to maximal oxygen consumption; test B, load in test A + 17%; test C, load in test B but performed 1 h after acute ingestion of NaHCO₃. Functional electrical stimulation (FES) was applied to provoke isometric contraction of the quadriceps femoris. The resulting knee torque was monitored during fatigue (2-min chronic FES) and recovery (10-s FES every 10 min, for 40 min). Quadriceps torques were higher in the presence of NaHCO₃ (P < 0.05): with NaHCO₃ the peak, residual, and recovery (after 40 min) normalized torques were, respectively, 0.68 \pm 0.05 (SD), 0.58 \pm 0.05, and 0.73 \pm 0.05; without NaHCO3 the values were 0.45 \pm 0.04, 0.30 \pm 0.06, and 0.63 \pm 0.06. The increased torques obtained after acute ingestion of NaHCO₃ indicate the possible existence of improved nonoxidative glycolysis in isometric contraction, resulting in reduced fatigue and enhanced recovery.

functional electrical stimulation; knee torque; isometric contraction; quadriceps femoris; peripheral muscle fatigue

DURING SUSTAINED ISOMETRIC CONTRACTION, the tension developed by the muscle declines, a phenomenon referred to as fatigue. A sustained contraction can be obtained in able-bodied subjects either voluntarily, with the subjects actively contracting their muscles (4), or by means of functional electrical stimulation (FES), with the subjects remaining passive (8). One advantage of the latter method is that it provides a means to standardize the contraction, enabling one to reproducibly detect the progressively developing fatigue.

At least two components of fatigue have been reported (13). One is due to a direct effect of intracellular acidosis, acting directly on the myofibrils and accounting for a part of the suppression of the contractile force. The second is due to changes of the state of the excitation-contraction coupling process. Recovery of the excitation-contraction process is sensitive to external pH. Thus a drop of pH due to an increase of lactic acid within the muscle plays a significant role, both directly and indirectly, in muscle contraction (13). This situation takes place in high-intensity exercise of short duration (2, 9, 22). Studies on able-bodied (3, 8, 9, 16, 23) subjects have shown that also after intensive electrical stimulation of either isolated or in situ muscle there is a fast accumulation of H⁺. A mechanism acting to decrease H⁺ concentration is the evacuation of lactic acid from the cell to the surrounding plasma. This takes place by means of pH-dependent transport across

the cell membrane while lactic acid is in the undissociated condition (20, 21). Lactic acid efflux across the muscle cell membrane is an important regulator of intracellular pH during lactic acidosis. An increase of extracellular NaHCO₃ buffer capacity by NaHCO₃ ingestion is a way to facilitate the efflux of H⁺ and lactate from the muscle cell and thereby delay the critical increase in intracellular pH, which negatively affects muscle glycolysis (12).

It has been reported that NaCHO₃ ingestion of at least 300 mg/kg of body mass prolongs high-intensity exercise with a duration ranging between 60 s and 7 min (5, 12, 14, 15, 28). Most studies examining the ergogenic effect of NaHCO₃ have used overall performance criteria as measures of comparison, such as performance time or maximum power. The effect of NaHCO₃ injection on the process of progressive muscle fatiguing and recovery was not studied.

The aim of the present work, therefore, was to evaluate the effect of ingestion of NaHCO₃ on muscle progressive fatigue as induced by FES and on muscle recovery after intensive cycling exercise. The relationship between short-term physical effort performed during cycling and the resulting decrease in blood pH level due to lactic acid accumulation is well documented (7, 11, 12, 14). End-tidal PCO₂ (PET_{CO2}) was used in this study to indirectly reflect humoral acid-base changes due to NaHCO₃ decrease (19, 27). Isometric contraction of the quadriceps femoris was used as a procedure to monitor the muscle torque during the progressively developing fatigue and recovery. This procedure provided standardized testing protocols and ensured reproducibility of the testing conditions.

MATERIALS AND METHODS

Subjects

Six volunteer male subjects took part in this study. Their mean age, height, and mass were 36.5 ± 6.0 (SD) yr, 172 ± 2.3 cm, and 68.8 ± 5.8 kg, respectively. All subjects were in an excellent state of health and practiced calisthenics at least twice a week. No previous history of muscle weakness, neurological disease, or drug therapy was recorded.

Methodology

Determination of load at maximum oxygen consumption $(\dot{V}O_{2max})$. Before the actual tests, $\dot{V}O_{2max}$ was determined for each subject as follows: the subjects were asked to cycle continuously at the constant speed of 50 revolutions/min on a bicycle ergometer (Monark Ergometric 818E) while the load was increased every 3 min from an initial load of 50 W by increments of 50 W, until physical exhaustion. The load obtained at $\dot{V}O_{2max}$ was denoted by load $\dot{V}O_{2max}$. The mean values for all subjects for load $\dot{V}O_{2max}$ and for the actual

 $\dot{V}_{0_{2}max}$ were 200 \pm 44 W and 44.5 \pm 4.0 ml·kg⁻¹·min⁻¹, respectively.

Respiratory parameters. Values of oxygen consumption ($\dot{V}O_2$), minute ventilation ($\dot{V}E$), and PET_{CO_2} were monitored continuously by means of a respiratory diagnostics system (model MTS 4400 metabolic and respiratory diagnosis system, Alpha Technologies). These quantities were measured continuously, and their average values were calculated every 5 s, corresponding to a sampling rate of 12 samples/min.

Testing procedure. Each subject performed three different tests, which were made on separate days. At least a 1-wk interval of time was taken between the tests to ensure fatigue-free initial conditions. Loading conditions differed from one test to another as follows: *test A* was at load \dot{Vo}_{2max} ; *test B* was at supramaximal loading, determined as load $\dot{Vo}_{2max} + 17\%$; and *test C* was at the supramaximal loading as defined in *test B* but was performed 1 h after acute ingestion of NaHCO₃ with a dosage of 400 mg/kg body mass.

Preload isometric test by FES. Each test was started with 2 min of surface FES of the right quadriceps femoris muscle, during which this muscle was fatigued (preload fatigue). This stage served for reference for the subsequent stages. The subject was instructed to remain relaxed during FES so that voluntary contraction of the muscle would be avoided.

The knee torque was continuously monitored during the preload isometric test, by using a specially constructed testing apparatus. The subject was seated on an adjustable testing chair, and his right thigh was belted to the seat to ensure that the hip angle remained constant at 90° during the tests. The lower leg was hinged at the level of the ankle to a pendulum, which was smoothly hinged at the level of the knee joint. The pendulum could be locked at any desired angle for isometric activation. The knee angle chosen in this study was 60°, around which isometric torque was reported to be maximal (24). The torque at the knee, which was a result of activation of the quadriceps femoris and corresponding to the external force at the ankle level, was measured by means of an instrumented horizontal cantilever at the lower part of the pendulum arm.

Transcutaneous electrical stimulation was applied through a pair of rectangular rubber electrodes (4 \times 5 cm) with Karaya gum as an interface. One electrode was placed on the quadriceps' motor point, which corresponds approximately to the center of the muscle belly area, and the second was placed more distally, near the patella. The stimulator (17) provided a train of rectangular monophasic positive pulses at 20 Hz. Pulse width was kept constant at 0.25 ms, and the current intensity was adjusted for each subject in accordance with his pain limit. This corresponded to ~75% of maximum voluntary contraction of each subject. The mean current intensity for all subjects was 62.5 ± 6.9 (SD) mA.

Isotonic load. After the 2-min FES fatigue a 3-min cycling exercise at load $\dot{V}_{0_{2max}}$, supramaximal load, and supramaximal load after 1 h of acute ingestion of NaHCO₃ was performed for *tests A, B,* and *C*, respectively, as described in *Testing procedure.*

The respiratory parameters were measured continuously during the exercise and averaged every 5 s. Monitoring of the respiratory parameters was extended to an additional period of 3 min after termination of the exercise. This was done because, as has been previously reported, after heavy exercise, the change in blood lactate (consequently also the change in PET_{CO₂}) continues for a few minutes (2, 6).

Postload isometric test by FES. After the 3-min cycling load and the subsequent 3-min rest, surface FES was given to the right quadriceps for 2 min under isometric testing conditions similar to those of the previously described preload FES (see *Preload isometric test by FES*). The knee torque was monitored during the isometric postload test (postload fatigue).

Recovery. After the postload isometric test by means of FES, there was a 40-min recovery period for the loads of *tests B* and *C*. During this period the knee torque resulting from a 10-s stimulation train was monitored every 10 min. The FES conditions were similar to those used during the previously described pre- and postload isometric test protocols (see *Preload isometric test by FES* and *Postload isometric test by FES*).

Intrasubject variability. On one of the subjects the above procedure was repeated five times to verify repeatability of the results.

Statistics

To identify statistically meaningful variations, the Student's *t*-test was used, with P < 0.05 as level of significance. Differences between intrasubject variability and variability within the whole group were verified by using analysis of variance (ANOVA).

RESULTS

Figure 1 presents the variations of VE during the 3-min isotonic exercise and the subsequent 3-min rest. The mean values (\pm SD) for all six subjects are plotted for each of the three tests. Statistical comparisons between the tests were made on the means every minute on the minute. It was found that VE was significantly higher (P < 0.05) in *test B* (supramaximal load) compared with *test A* (load Vo_{2max}). There was a statistically significant difference between *tests B* and *A*. However, when *test C* (supramaximal plus NaHCO₃) is compared with *test A* (load Vo_{2max}), the differences were not statistically significant.

The mean variation $(\pm SD)$ of Vo_2 during isotonic exercise and rest for all tested subjects is shown in Fig. 2. The results of all three tests did not differ significantly from one another except after the first minute of



Fig. 1. Minute ventilation during 3-min exercise load and subsequent 3-min rest. Mean values for all 6 subjects are plotted for each of the 3 tests. *Test A* (A): load [maximal oxygen consumption ($\dot{V}O_{2max}$)]; *test B* (B): load in *test A* + 17%; *test C* (C): load in *test B* + NaHCO₃. Bars, SD. Statistical comparisons between tests were made on mean values every minute on the minute. *Significant difference between *tests A* and *B*, *P* < 0.05.



Fig. 2. Oxygen consumption during 3-min exercise load and subsequent 3-min rest. Mean values for all 6 subjects are plotted for each of the 3 tests. Bars, SD. Statistical comparisons between tests were made on mean values every minute on the minute. *Significant difference between *test A* and *test B*, P < 0.05. ***Significant difference between *test A* and *test C*, P < 0.05.

exercise, when the value in *test* A was smaller than those of *tests* B and C(P < 0.05).

Mean (\pm SD) levels of Pet_{CO_2} for all subjects are plotted in Fig. 3. Significantly lower Pet_{CO_2} values were found in *test B* compared with both *tests A* and *C*. The peak value of Pet_{CO_2} (after 1 min of exercise) in *test C* was significantly higher than those of *tests A* and *B*. Similar results were found at the end of the rest period (6th min).

The torque curves during the pre- and postload isometric FES tests are shown in Fig. 4. It should be



Fig. 3. End-tidal Pco_2 during 3-min exercise load and subsequent 3-min rest. Mean values for all 6 subjects are plotted for each of the 3 tests. Bars, SD. Statistical comparisons between tests were made on mean values every minute on the minute. *Significantly lower end-tidal Pco_2 values in *test B* compared with *test A*, P < 0.05. **Significantly lower end-tidal Pco_2 values in *test B* compared with *test B* compared with *test C*, P < 0.05. **Significantly higher end-tidal Pco_2 values in *test C* compared with *test B*, P < 0.05.



Fig. 4. Torque curves during pre- and postload functional electrical stimulation fatigue. Postload functional electrical stimulation fatigue was carried out after *tests A*, *B*, and *C*, which correspond to load $\dot{V}O_{2max}$, supramaximal load, and supramaximal load 1 h after acute ingestion of NaHCO₃, respectively. Mean values for all 6 subjects are plotted in each of the curves. Bars, SD. Statistical comparisons between tests were made on mean values every 20 s. *Significant difference between preload and postload in *test A*, *P* < 0.05. ***Significant difference between preload and postload in *test C*, *P* < 0.05.

remembered that the postload FES test was carried out after each of the three loads in *tests A, B,* and *C*, which correspond to load Vo_{2max}, supramaximal load, and supramaximal load 1 h after acute ingestion of NaHCO₃, respectively. All knee torque values were normalized to the peak values of the preload test. Comparison of the postload torque traces between 20 and 60 s of FES reveals that in *tests A* and *B* the torques decline after having reached their respective early peaks. In test C, however, the torque curve has an elevated and delayed peak, taking place at \sim 60 s of FES. At 60 s, the mean normalized magnitudes are 0.46 \pm 0.05 (SD), 0.34 \pm 0.08, and 0.67 \pm 0.05 for *tests A*, *B*, and *C*, respectively, compared with 0.71 \pm 0.06 for the preload test. The preload peak was significantly higher than all the postload peaks (P < 0.05). In a comparison of the postload protocols to each other, it is seen that after NaHCO₃ administration (*test C*), both the peak and residual torques are higher than those of *tests A* and *B*. It is also seen that while the preload residual torque was higher (P < 0.05) than the postload residual torque in *tests* A and B, this was not the case with *test* C, in which NaHCO₃ was ingested.

The peak torques obtained during the 40-min recovery post-FES are presented in Fig. 5. As described in *Recovery*, the torques were measured by stimulation trains of short durations, 10 s each, delivered to the quadriceps femoris every 10 min of the recovery time. The mean normalized torque magnitudes for *tests B* and *C* were, respectively, 0.40 ± 0.07 and 0.55 ± 0.05 after 10 min and 0.63 ± 0.06 and 0.73 ± 0.05 after 40 min of recovery. In all the four measurements, values of



Fig. 5. Peak torques obtained during 40 min post-functional electrical stimulation recovery after *tests B* and *C*, which correspond to supramaximal load without and with acute ingestion of NaHCO₃, respectively. Mean values of all 6 subjects are plotted. Bars, SD. * Significant difference between values of *tests B* and *C*, P < 0.05.

the peak torque in *test C* significantly exceeded those of *test B* (P < 0.05).

The results obtained for the subject on whom the whole experimental procedure was repeated five times indicate that there was no significant difference between intra- and intersubject variability, as revealed by performance of ANOVA (P > 0.05).

DISCUSSION

The central issue in this study was to investigate the influence of acute ingestion of NaHCO₃ on fatigue and recovery. The methodology adopted was to use FES to provoke an isometric contraction of the quadriceps femoris as a means of testing the muscle before and after the isotonic cycling exercise. The rationale of this methodology was as follows. 1) Because the subject was instructed to remain relaxed, voluntary contractions of the muscle were avoided when FES was applied, thus isolating the quadriceps muscle and minimizing problems associated with performance-based testing criteria. We found that, in the preload fatigue, the torque decreased to $\sim 75\%$ of the initial value after 50 s of activation (Fig. 4). Similar results were obtained by other investigators using similar testing conditions (8), confirming that this test can serve as a standardized and reliable procedure for monitoring the quadriceps muscle developing fatigue, before and after load, with and without NaHCO₃. 2) Although, in the cycling exercise, several muscle groups are being activated, involving both peripheral and central mechanisms, it has been reported that in high-intensity bicycle exercise the dominant subjective sensation limiting further exercise was fatigue of the quadriceps (10), which was the basis for the previously described isometric tests (see Preload isometric test by FES and Postload isometric test by FES). It should also be mentioned that a similar approach of combining isotonic exercise with isometric FES as a means of testing was previously

used to assess the effect of exercise training on resistance to fatigue of the quadriceps femoris (25).

The isotonic active load was given in three different modes, applied in separate tests, during which the ventilation variables were monitored. Because the dynamics of PET_{CO_2} were reported to possibly reflect the arterial PCO_2 in men (19), the measured PET_{CO_2} served as an indicator of the humeral acid-base changes due to metabolic acidosis (27). Decreased levels of CO_2 in the blood create unfavorable metabolic conditions for the extracellular buffering capacity, necessary to counterbalance the acidosis that results in reduced physical work capacity (1, 26).

The ergogenic properties of NaHCO₃ ingestion have in the past been the focus of numerous investigations (see, e.g., Refs. 12, 14). It has been shown that during short-term, high-intensity physical activity progressive metabolic acidosis due to lactic acid accumulation and a drop of pH takes place, in both the blood and the working muscle (2). The resultant accumulation of H^+ within the muscle directly inhibits the contractile process by inhibiting the release of Ca^{2+} from the sarcoplasmic reticulum, as well as by reducing the activity of glycolytic enzymes, thus impairing the propagation of neural impulses (8, 9). As exercise progresses, various buffering mechanisms function to neutralize this effect. Eventually, when the intracellular buffering capacity is exceeded, H⁺ diffuse into the blood, causing a drop in extracellular pH. This, in turn, stimulates extracellular buffering mechanisms of which HCO₃⁻ is one of the most effective constituents (18). It is thus expected that acute ingestion of NaHCO₃ should at this stage enhance the buffering capacity in the blood and hence delay exhaustion and improve performance (12). The presence of NaHCO₃ induces an increased efflux of H⁺ and lactate ions from the cell across the membrane, reported to take place via pH-sensitive transport of the lactic acid in the undissociated condition (20, 21).

The results obtained indicate that during supramaximal load (*test B*) a significant decrease in PET_{CO_2} resulted compared with load $\dot{V}O_{2max}$ (*test A*), reflecting a decrease in NaHCO₃ buffer formation (27). However, when the supramaximal load was combined with acute ingestion of NaHCO₃ (*test C*), a significantly higher PET_{CO_2} level resulted, indicating an increased CO_2 accumulation in the blood and a more effective NaHCO₃ buffer activity (26). It should be remembered, however, that PET_{CO_2} served as an indirect measure of extracellular NaHCO₃ buffer capacity (27), limiting its role as a major indicator of performance (22).

A more direct evidence for the beneficial effect of NaHCO₃ came from the biomechanical measurements. The fatigue curves obtained during FES indicated that both the peak and the residual torques decreased significantly as a result of isotonic load increase (comparison between *tests B* and *A*). However, when the supramaximal load was combined with acute ingestion of NaHCO₃ (*test C*), higher peak and residual torques resulted, compared with both the maximal (*test A*) and supramaximal (*test B*) loads. Of special interest was

comparison of the torque traces between 20 and 60 s of FES (Fig. 4). Whereas in *tests A* and *B* the torques were declining after having reached their respective peaks, in *test C* the torque continued increasing toward its elevated and delayed peak at \sim 60 s of FES. Higher torques in the presence of NaHCO₃ were also obtained in the postfatigue measurements, indicating that the buffer capacity in the blood was enhanced in the recovery process as well.

The relationship between performance and levels of HCO_3^- and CO_2 in the blood was demonstrated (26); hence the effect of H^+ is obvious: H^+ accumulation is a limiting factor in the usage of nonoxidative sources of energy (12). NaHCO₃ ingestion clearly results in a more alkaline extracellular environment.

Published work on the effect of NaHCO₃ on anaerobic performance shows variable results (14). The inconsistency in results has been attributed to differences in the methodologies used, including dosage ingested, time between completion of ingestion and initiation of exercise, exercise protocols and methodology of controls. However, there is agreement that NaHCO₃ ingestion of at least 300 mg/kg body mass prolongs high-intensity exercise with a duration ranging between 60 s and 7 min (12). The protocols selected in the present study matched those of reported enhanced performance after NaHCO₃ administration: dosage of 400 mg/kg body mass (5, 26) and time of 1 h between completion of ingestion and initiation of exercise. As previously mentioned in the introductory section, the exercise protocols were designed to objectively monitor the progressively developing fatigue and the subsequent recovery of the quadriceps femoris with and without acute digestion of NaHCO₃. On the basis of the results obtained, it may thus be concluded that acute ingestion of NaHCO₃ is an effective means for increasing the torques in isometric contraction, thus reducing muscle fatigue and enhancing recovery.

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REFERENCES

- Coggan, A. R., D. L. Habash, L. A. Mendenhall, S. C. Swanson, and C. L. Kien. Isotopic estimation of CO₂ production during exercise before and after endurance training. *J. Appl. Physiol.* 75: 70–75, 1993.
- Costill, D. L., F. Verstappen, H. Kuipers, E. Janssen, and W. Fink. Acid-base balance during repeated bouts of exercise: influence of HCO₃. *Int. J. Sports Med.* 5: 228–231, 1984.
- Edman, K. A. P., and A. R. Mattiazzi. Effects of fatigue and altered pH on isometric force and velocity of shortening at zero load in frog muscle fibers. *J. Muscle Res. Cell Motil.* 2: 321–334, 1981.
- Fulco, C. S., S. F. Lewis, P. N. Frykman, R. Boushel, S. Smith, E. A. Harman, A. Cymerman, and K. B. Pandolf. Quantitation of progressive muscle fatigue during dynamic leg exercise in humans. *J. Appl. Physiol.* 79: 2154–2162, 1995.
- Goldfinch, J., L. McNaughton, and P. Davies. Induced metabolic alkalosis and its effects on 400-m racing time. *Eur. J. Appl. Physiol.* 57: 45–48, 1988.
- Gollnick, P. D., and L. Hermansen. Biochemical adaptions to exercise: anaerobic metabolism. *Exerc. Sport Sci. Rev.* 1: 1–43, 1973.

- Hermansen, L., and J. Osnes. Blood and muscle pH after maximal exercise in man. J. Appl. Physiol. 32: 304–308, 1972.
- 8. Hultman, E., and H. Sjoholm. Energy metabolism and contraction force of human skeletal muscle in situ during electrical stimulation. *J. Physiol. (Lond.)* 345: 525–532, 1983.
- Hultman, E., and H. Sjoholm. Biochemical causes of fatigue. In: *Human Muscle Power*, edited by N. L. Jones, N. McCartney, and A. J. McComas. Champaign, IL: Human Kinetics, 1986, p. 215–238.
- Jones, N. L., J. R. Sutton, R. Taylor, and C. J. Toews. Effect of pH on cardiorespiratory and metabolic responses to exercise. *J. Appl. Physiol.* 43: 959–964, 1977.
- Lambert, C. P., P. L. Greenhaff, D. Ball, and R. J. Maughan. Influence of sodium bicarbonate ingestion on plasma ammonia accumulation during incremental exercise in man. *Eur. J. Appl. Physiol.* 66: 49–54, 1993.
- Linderman, J., and T. D. Fahey. Sodium bicarbonate ingestion and exercise performance (an update). *Sports Med.* 11: 71–77, 1991.
- Mainwood, G. W., and J. M. Renaud. The effect of acid-base balance on fatigue of skeletal muscle. *Can. J. Physiol. Pharma*col. 63: 403–416, 1985.
- Matson, L. G., and Z. V. Tran. Effects of sodium bicarbonate ingestion on anaerobic performance: a meta-analytic review. *Int. J. Sport Nutr.* 3: 2–28, 1993.
- McNaughton, L. R. Sodium bicarbonate ingestion and its effects on anaerobic exercise of various durations. *J. Sports Sci.* 10: 425–535, 1992.
- Merton, P. A., D. K. Hill, and H. B. Morton. Indirect and direct stimulation of fatigued human muscle. In: *Human Muscle Fatigue: Physiological Mechanisms*, edited by R. Porter and J. Whelan. London: Pitman Medical, 1981, p. 120–126. (Ciba Found. Symp. 82)
- Minzly, J., J. Mizrahi, E. Isakov, Z. Susak, and M. Verbeke. Computer controlled portable stimulator for paraplegic patients. *J. Biomed. Eng.* 15: 333–338, 1993.
- Parkhouse, W. S., and D. C. McKenzie. Possible contribution of skeletal muscle buffers to enhance anaerobic performance: a brief review. *Med. Sci. Sports Exerc.* 16: 328–338, 1984.
- Robbins, P. A., J. Conway, D. A. Cunningham, S. Khamnei, and D. J. Paterson. A comparison of indirect methods for continuous estimation of arterial Pco₂ in men. *J. Appl. Physiol.* 68: 1727–1731, 1990.
- Roth, D. A., and G. A. Brooks. Lactate and pyruvate transport is dominated by a pH gradient-sensitive in rat skeletal muscle sarcolemmal vesicles. *Arch. Biochem. Biophys.* 279: 368–374, 1990.
- Roth, D. A., and G. A. Brooks. Lactate transport is mediated by a membrane-bound carrier in rat skeletal muscle sarcolemmal vesicles. *Arch. Biochem. Biophys.* 279: 377–385, 1990.
- Sahlin, K. Intracellular pH and energy metabolism in skeletal muscle of man, with special reference to exercise. *Acta Physiol. Scand. Suppl.* 455: 1–56, 1978.
- Sahlin, K., L. Edstrom, and H. Sjoholm. Fatigue and phosphocreatine depletion during carbon dioxide-induced acidosis in rat muscle. Am. J. Physiol. 245 (Cell Physiol. 14): C15–C20, 1983.
- Selkowitz, D. M. Improvement in isometric strength of the quadriceps femoris muscle after training with electrical stimulation. *Phys. Ther.* 65: 186–196, 1985.
- Sinacore, D. R., R. B. Jacobson, and A. Delitto. Quadriceps femoris muscle resistance to fatigue using an electrically elicited fatigue test following intense endurance exercise training. *Phys. Ther.* 74: 930–939, 1994.
- Verbitskii, O. N., L. V. Skorik, E. V. Melnichenko, and V. A. Gaevoi. Administration of hydrocarbonate-containing salt mixture for correction of deadaptation processes in men. *Fiziol. Cheloveka* 22: 116–122, 1996.
- Wasserman, K., B. J. Whipp, S. N. Koyal, and W. L. Beaver. Anaerobic threshold and respiratory gas exchange during exercise. J. Appl. Physiol. 35: 236–243, 1973.
- Wilkes, D., N. Gledhill, and R. Smyth. Effect of acute induced metabolic alkalosis on 800-m racing time. *Med. Sci. Sports Exerc.* 15: 277–280, 1983.