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Viscoelastic characteristics of muscle: passive stretching versus muscular contractions

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ABSTRACT

TAYLOR, D. C., D. E. BROOKS, and J. B. RYAN. Viscoelastic characteristics of muscle: passive stretching versus muscular contractions. Med. Sci. Sports Exerc., Vol. 29, No. 12, pp. 1619-1624, 1997. This study compared the effects of repeated contractions and repeated passive stretches on the viscoelastic properties of muscle. The tibialis anterior (TA) muscles of eight anesthetized male New Zealand white rabbits were studied. In each rabbit, one hindlimb was randomly assigned to the repeated muscular contraction group (CON) and the contralateral hindlimb to the repeated passive stretch group (STRETCH). The passive tension at neutral length was measured in all muscles before and after both repeated muscular contractions or repeated passive stretches. In the CON hindlimb, the peroneal nerve was stimulated with a nerve stimulator for 1 s, and the resulting contractile force was measured. Stimulations were repeated every 10 s for a total of 10 contractions. In the STRETCH hindlimb, the TA was stretched from its shortest in vivo length to its maximum in vivo length 10 times at 20 cm·min⁻¹. The maximum force generated during the first contraction in the CON group averaged 21.74 ± 1.41 N, with a subsequent reduction with each muscle contraction to 12 65 + 43 N by the tenth contraction. The average peak tensile force in the STRETCH group was 17.39 \pm 2.61 N for the first passive stretch, decreasing to 13.57 \pm 1.84 by the tenth stretch. After repeated muscular contractions in the CON hindlimbs, the passive tension at neutral length decreased from 0.88 ± 0.22 N to 0.42 ± 0.08 N. After repeated passive stretches in the STRETCH hindlimbs, the passive tension at neutral length decreased from 1.16 \pm 0.17 N to 0.67 \pm 0.09 N. The percentage decreases in passive tension between the CON and STRETCH groups were not statistically significant (P = 0.24). The results show that stretching and contracting both result in tissue relaxation of the muscle-tendon unit. This finding may be a result of changes in the viscous elements of the connective tissue secondary to the forces generated by either stretches or contractions. This study suggests that well controlled isometric muscular contractions may result in decreased passive tension in a muscle at neutral length, a finding that one normally associates with passive stretching.

ISOMETRIC, FLEXIBILITY, CONTRACTILE FORCE, VISCOELASTICITY

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Submitted for publication April 1996. Accepted for publication July 1997. Passive stretching exercises are ubiquitous in athletics and rehabilitation today. Stretching is almost universally emphasized in preparticipation warmup, postinjury and postoperative rehabilitation, and for performance enhancement. Stretching is emphasized because it elongates soft tissues, which may result in greater joint range of motion. Greater range of motion implies improved flexibility. Some have suggested that greater flexibility may improve performance (2,6,10,13,24,25,30) and reduce the risk of injury (4,5,7–9), while others have been unable to demonstrate these stretching benefits (11,28).

Exercises using repeated muscular contractions are also inherent to preparticipation warm-ups, rehabilitation, and performance enhancement. Examples include preparticipation exercises conducted at slower speeds than the actual competition, contract-relax stretching and various strengthening techniques in rehabilitation, and weight lifting and drill repetition to enhance performance. The primary focus of these exercises is on improving strength and function, with less emphasis on increasing flexibility.

Biologic soft tissues react viscoelastically to stretching, meaning that the tissue response to stretching is time and history dependent. Characterizing the biomechanical properties of muscle is more complex because a muscletendon unit is comprised of passive (connective tissue) and contractile (muscle fibers) elements. However, passive stretching does cause a viscoelastic response in the muscle-tendon unit (1,3,18,27), perhaps because of the changes in the connective tissues (3,22,23).

After stretching, the passive tension of the muscletendon unit is reduced for any given length of the tissue (tissue relaxation) (27). This viscoelastic response provides the basis for passive stretching and has been demonstrated in humans and animals (16,18,27).

Some authors have suggested that exercises using repeated muscle contractions may also result in greater flexibility (12,19,21,26,29), and there is some basic science work to support this idea (14,22). However, a muscle contraction involves shortening of the contractile

element of muscle and increased tension, so greater flexibility following muscle contractions may seem contradictory.

During pilot studies examining techniques for evaluating skeletal muscle contractile strength, we found that in our set-up the maximum contractile force generated by the rabbit tibialis anterior (TA) at its neutral length was of similar magnitude to the passive tension of the TA stretched to maximum in vivo length. In other words, the loads were similar when generated either centrally, with a contraction, or peripherally, with a stretch. Based on these findings, we felt that we had a model that could examine how these two processes, contracting and stretching, altered the biomechanical characteristics of the muscle. With so much current clinical emphasis on stretching and contracting in rehabilitation, performance enhancement, and injury prevention, we elected to evaluate how this relationship between similar forces generated by contractions and stretches altered the viscoelastic characteristics of skeletal muscle.

MATERIALS AND METHODS

The experimental studies described in this report were reviewed and approved by the Institutional Review Committee/Animal Care and Use Committee at Letterman Army Institute of Research. The manuscript was peer reviewed for compliance before submission for publication. In conducting the research described here, the author adhered to the *Guide for the Care and Use of Laboratory Animals*, DHEW Publication (NIH) 85–23.

The tibialis anterior (TA) muscles of eight New Zealand white rabbits (3.5 to 3.7 kg) were studied. The rabbits were anesthetized with a 1.0-cc intramuscular injection of the following: 10 cc of ketamine Hcl at a concentration of 100 mg·cc⁻¹ (Vetalar, Fort Dodge Laboratories, Fort Dodge, IA); 0.5 cc of acepromazine maleate, 10 mg·cc⁻¹ (PromAce, Aveco Co., Fort Dodge, IA); and 1.0 cc of xylazine, 20 mg·cc⁻¹ (Anased, Lloyd Laboratories, Shenandoah, IA).

An intravenous line was placed in the rabbit forelimb. All animals were maintained under effective general anesthesia throughout the exposure and evaluation phases using 0.1 cc to 0.3 cc intravenous injections of the following: 1.0 cc of ketamine Hcl, 100 mg·cc⁻¹; 0.5 cc of acepromazine maleate, 10 mg·cc⁻¹; and 1.0 cc of xylazine, 20 mg·cc⁻¹. Past experience in our laboratory has demonstrated the effectiveness of these anesthetic combinations in achieving adequate levels of anesthesia while minimizing cardiorespiratory side effects.

With the rabbits under general anesthesia, we shaved and exposed the TAs of both hindlimbs through an anterior longitudinal incision over the leg. We measured the length of the TA by placing a 2–0 silk suture from the muscle origin to the cruciate ligament on the dorsum of the foot. The suture followed the contour of the muscle,

including passage deep to the crural ligament (hock joint extensor retinaculum). We measured the origin-cruciate ligament suture length with precision calipers accurate to 0.1 mm. Three length measurements were made: 1) L-MIN: TA length with the hock in maximal dorsiflexion, 2) L-NEU: TA length with the hock at 90° and 3) L-MAX: TA length with the hock maximally plantarflexed.

The neurovascular pedicle to the TA was maintained throughout testing. Exposed muscle was kept moist at 37°C with a continuous drip of 0.9% NaCl solution maintained at that temperature using a prototype microwave fluid warmer (Microwave Medical Systems, Littleton, MA). The hindlimb was immobilized in a frame and the TA tendon of insertion was clamped within the crosshead of a United SFM 1 Testing System (United Calibration Corporation, Huntington Beach, CA). The crosshead clamp was linked to a load cell, and loads and deformations were recorded using United Calibration software. With this software, measurements of change in load were sensitive to 0.027 N. This testing setup is similar to that previously described (27).

In each rabbit, one hindlimb was randomly assigned to the repeated muscular contraction group (CON) and the contralateral hindlimb to the repeated passive stretch group (STRETCH).

The passive tensions at length L-MIN and L-NEU were measured in muscles from both groups before and after repeated contractons (CON) or repeated stretching (STRETCH). After clamping the TA tendon of insertion, the muscle-tendon U was elongated from length L-MIN to L-NEU and immediately returned to L-MIN. The tensile forces at these lengths were obtained from the computer reaching. The tensile forces were performed, followed by a second measurement of tensile forces of L-MIN and L-NEU.

In the hindlimbs assigned to the CON group, the TA was maintained at length L-NEU. An isometric contraction of the TA was generated by stimulating the peroneal nerve for 1 s with a Grass S9B nerve stimulator (Grass

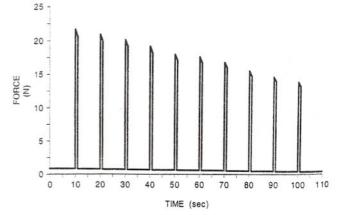


Figure 1—The average contractile forces generated by the muscles in the CON group. The peak force generated during the first contraction averaged 21.74 N. declining to 13.66 N by the tenth contraction.

Instruments, Quincy, MA) set at a frequency of 50Hz, pulse duration of 0.05 s, and voltage of 5T (where T is the minimum voltage that leads to a measurable contractile response). A 5T voltage was selected because we found in our pilot studies that this was the minimum voltage that could assure consistent maximal neuromuscular activation. The peroneal nerve stimulation was repeated every 10 s for ten contractions. This sequence simulates repeated isometric muscle contractions over a 90-s interval (Fig. 1).

In the STRETCH hindlimbs, the TA was continuously stretched from length L-MIN to L-MAX 10 times at 20 cm·min⁻¹ (Fig. 2). This sequence simulates a gentle, low velocity, repetitive stretching regimen from the shortest to the longest in vivo musculotendinous lengths.

We initially attempted to compare EDL muscles in the same rabbits. We abandoned these efforts when we found that unlike the TA, the EDL STRETCH passive tensions did not correlate well with the CON contractile forces.

While under general anesthesia, all animals were killed with a lethal intravenous injection of pentobarbital sodium (Beuthanasia-D, Steris Laboratories, Phoenix, AZ).

The primary measure in this study was the percentage change in passive tension at L-NEU. Statistical analysis was carried out using a paired Student's t-test comparing the percentage change in passive tension at L-NEU and L-MIN for CON muscles versus STRETCH muscles. Comparisons used percentage changes instead of absolute changes to account for differences in initial values that resulted from measurement and biologic variability. Statistical significance was defined as P < 0.05.

RESULTS

The average TA excursion (L-MAX minus L-MIN) was 17.0 ± 0.6 mm (15.0% strain) for the CON group and 17.5 ± 0.7 mm (15.6%) for the STRETCH group.

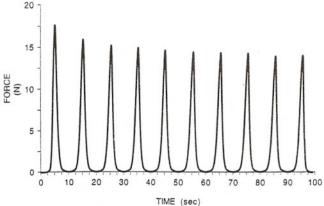


Figure 2-The average tensile forces resulting from passive stretching in the STRETCH group. The average tensile force at L-MAX was 17.39 N for the first stretch, declining to 13.57 N by the tenth stretch.

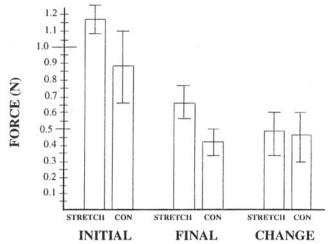


Figure 3-The initial, final, and change in force at length L-NEU for STRETCH and CON muscles.

Excursion to L-NEU (L-NEU minus L-MIN) was 5.8 ± 0.7 mm (5.0%) for the CON group and 6.2 \pm 0.5 mm (5.5%) for the STRETCH group. L-MIN averaged 113.1 ± 0.8 mm and 112.3 ± 0.7 mm, L-NEU 118.9 ± 0.7 0.8 mm and 118.4 \pm 1.0 mm, and L-MAX 130.1 \pm 0.9 mm and 129.7 \pm 0.8 mm for the CON and STRETCH groups, respectively.

The force generated for the first contraction in the CON group averaged 21.74 ± 1.41 N. The force gradually declined with each contraction to 13.66 \pm 0.81 N by the tenth contraction (Fig. 1). In the stretch group, the tensile force at L-MAX was 17.39 ± 2.61 N for the first stretch. The tensile force at L-MAX decreased with each stretch to 13.57 ± 1.84 N by the tenth stretch (Fig. 2).

Figures 3 and 4 show the initial, final, and change in force at L-NEU and L-MIN for the STRETCH and CON muscles. In the CON muscles, the passive tension at L-NEU decreased from 0.88 ± 0.22 N before the 10 isometric contractions to 0.42 ± 0.08 N afterward (change in L-NEU passive tension of $0.46 \pm 0.15 \text{ N}$). The passive tension at L-MIN decreased from $0.16 \pm$ 0.02 N to $0.09 \pm 0.01 \text{ N}$ (change in L-MIN passive tension of $0.07 \pm 0.02 \text{ N}$).

In the STRETCH muscles, the passive tension at L-NEU decreased from 1.16 \pm 00.17 N before the 10 passive stretches to 0.67 ± 0.09 N afterward (change in L-NEU passive tension of 0.49 \pm 0.11 N). The passive tension at L-MIN decreased from 0.19 ± 0.02 N to 0.12 ± 0.02 N (change in L-MIN passive tension of 0.07 ± 0.02).

The differences between the CON and STRETCH values for percentage decrease in passive tension at L-NEU were not statistically significant (P = 0.24). The differences between the CON and STRETCH values for percentage decrease in passive tension at L-MIN were also not statistically significant (P = 0.99).

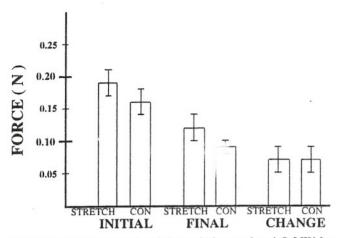


Figure 4—The initial, final, and change in force at length L-MIN for STRETCH and CON muscles.

DISCUSSION

In this study we measured the tensile load changes in muscle after repeated passive stretches or repeated isometric contractions. However, both stretches and contractions resulted in passive tension reductions, indicating stress relaxation of the muscle-tendon unit. The similar magnitudes of stress relaxation effects with both sequences suggest that the sequences result in similar viscoelastic changes.

The methods we used to stretch and contract the muscles provided similar force patterns for comparison. Each routine generated 10 peak forces of similar magnitude, each approximately 10 s apart (Figs. 1 and 2). The peak forces of the CON group were similar to those of the STRETCH group because the combination of passive and contractile forces at L-NEU (CON peak force) was similar to the magnitude of the passive force at L-MAX (STRETCH peak force). Figure 5 demonstrates this relationship. We were able to directly control the separation time between peak forces in the CON group by stimulating the peroneal nerve every 10 s. In the STRETCH group, the time between peak forces varied based on the TA excursion (L-MAX minus L-MIN). The TA excursion averaged 17.5 mm in the STRETCH group so that stretching at 20 cm·min-1 resulted in an average of 10.5 s between peak forces. By generating contractile and stretching forces in this way, we were able to create a system that allowed for a fair comparison of the two routines.

One limitation of this study was that the viscoelastic changes were measured only to length L-NEU. Passive tension changes at L-MAX were not compared because measurement of L-MAX before repeated contractions (CON) would have represented a significant stretch and not allowed a valid comparison between the CON and STRETCH groups.

The magnitude that the peak contractile force decreased from the first contraction to the tenth (Fig. 2) may

seem surprising. This large decrease may have been secondary to a combination of factors. One may expect neuromuscular fatigue with consecutive, relatively rapidly stimulated, maximal isometric contractions. Additionally, with each contraction, the resting tension at L-NEU decreased. The decreased resting tension and associated relative lengthening of the muscle-tendon unit may result in a shift to the left on the length-force curve. A lower passive tension should result in a lower contractile force. The combination of neuromuscular fatigue and a change in the length-force relationship could explain the large decrease in maximum contractile force. Other possible explanations include decreased neuromuscular response associated with the recurrent mechanical manipulation of the nerve during stimulation and slippage of the fixation points during testing. We saw no evidence of either of these phenomena during the study.

The passive tension reductions in both groups were of similar magnitude, a finding that was somewhat unexpected. Why do stretches, a lengthening process, and muscular contractions, a process which involves shortening of the contractile element of muscle, both result in stress relaxation effects and a relative lengthening of the muscle-tendon unit?

In an isometric contraction, force is created by the shortening of the contractile element, the muscle fibers. For the entire muscle-tendon unit to remain a fixed length (isometric), a compensatory lengthening must occur. Because the tendons of origin and insertion are fixed, the connective tissues (tendon, muscle-tendon junction, perimysium, epimysium, and endomysium) must lengthen as the muscle fibers shorten.

Stretching is parely anothering process. The muscle fibers and connective tissues elongate because of a pe-

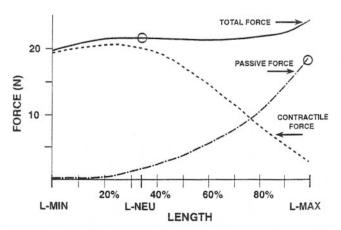


Figure 5—Length-force curves for the TA muscle. The CON peak forces result from the total force of passive stretch and contraction at length L-NEU. The STRETCH peak forces result from the passive force at length L-MAX. The peak forces shown (circles) are the average values for the first contraction at L-NEU in the CON group (21.749) and the peak passive tension at L-MAX in the STRETCH group (17.39N). The actual curves are approximations for load-elongation curves for the rabbit TA muscle, based on the average values for passive and contractile forces at L-MIN, L-NEU, and L-MAX.

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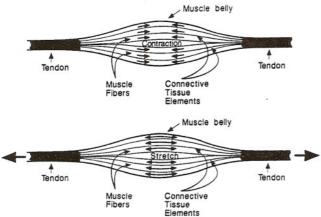


Figure 6-Muscle-tendon unit undergoing an isometric contraction (top) and a passive stretch (bottom). During an isometric contraction, muscle fibers shorten, resulting in an elongation of the connective tissue elements and tendons. During a passive stretch, both the connective tissue elements, tendons, and muscle fibers elongate (modeled after Safron et al. (22)).

ripherally applied force. Figure 6 demonstrates that in both an isometric contraction and a passive stretch there s connective tissue elongation.

Apparently the passive tension changes are related only to the magnitude of the load, independent of how that load is applied, either centrally with a contraction, or peripherally with a stretch. These findings suggest that the stress relaxation effects that result from the two routines occur primarily in the connective tissues because lengthening of the connective tissue is the only result common to both routines. Thus, muscle stretching, which has received such great emphasis, may be more appropriately thought of as connective tissue stretching, as others have suggested (3,22,23).

Applied clinically, these results suggest that either passive stretches or repeated contractions can lead to relaxation of skeletal muscle. When used alone, each method has been shown to improve joint motion (12,19). Medeiros et al. (19), in a clinical study similar to our study in design, compared 20 isometric hip extension contractions with 20 passive stretches. Like our study, they found that both techniques similarly improved the range of hip flexion.

The combination of stretches and contractions may be most effective because, with the application of greater forces to the muscle-tendon unit, the muscle-tendon unit undergoes more tissue relaxation and therefore could result in greater joint motion. This concept provides a better explanation than crediting reflex effects for the findings of several clinical studies that have suggested that the combination of muscle contractions and passive stretches is the most effective means of improving joint range of motion (20,21,26,29).

There are other considerations when examining the viscoelastic changes associated with repeated contractions. The type of contraction may be important. In this study, we examined repeated isometric contractions. Similar to our study, Magnusson et al. (15) found that passive stretch combined with concentric contractions decreased the passive tension in human hamstrings. However, they also found that the addition of eccentric contractions had no effect on passive tension.

Another consideration is the effect of fatigue. A recent study by Mair et al. (17) suggests that care should be used not to cause muscle fatigue if a repeated contraction regimen is being used to improve feasibility as a means of injury prevention. They found in a rabbit model similar to ours that fatigued muscles absorbed less energy when stretched to complete failure. Even though we have shown similar viscoelastic responses associated with comparable stretching and contracting regimens, other factors, such as fatigue and the types of contractions, should also be considered when applying these results clinically.

CONCLUSIONS

This study provides information on the viscoelastic characteristics of muscle that has been repeatedly stretched or contracted. The forces generated by the stretching and contracting techniques used in this study were of similar magnitude. Both repeated stretching and contracting resulted in similar decreases in passive tension of the TA muscle at its neutral length. The data suggest that an exercise regimen consisting of controlled muscular contractions may lead to connective tissue alterations similar to those seen with passive stretches. More clinical studies evaluating the functional and prophylactic effects of regimens using stretches and/or contractions are necessary to provide the information required for sound recommendations in this area.

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The opinions and assertions contained herein are the private views of the authors and are not to be construed as official nor do they reflect the views of the Department of the Army or the Department of Defense. (AR360-5)

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REFERENCES

- ABBOTT, B. C. and J. LOWLY. Stress relaxation in muscle. Proc. R. Soc. Lond. 146B:281–288, 1956.
- Anderson, B. and J. Anderson. Stretching. Bolinas, CA: Shelter Publications, 1980, pp. 8–11.
- BANUS, M. G. and A. M. ZETLIN. The relation of isometric tension to length in skeletal muscle. J. Comp. Physiol. 12:403–420, 1938.
- BEAULIEU, J. E. Developing a stretching program. Phys. Sportsmed. 9:59-66, 1981.
- DAVIS, V. P. Flexibility conditioning for running. In: Prevention and Treatment of Running Injuries, R. D'Ambrosia and D. Drez (Eds.). Thorofare, NJ: Slack, 1982, pp. 147–153.
- DINTIMAN, G. B. Effects of various training programs on running speed. Res. Q. 35:456, 1964.
- DORMAN, P. A report on 140 hamstring injuries. Aust. J. Sports Med. 4:30-36, 1971.
- GLICK, J. M. Muscle strains: prevention and treatment. Phys. Sportsmed. 8:73-77, 1980.
- HOLT, L. E. Scientific Stretching for Sport. Halifax, Nova Scotia: Sports Research Limited, 1973, p. 42.
- HORTOBAGYI, T., J. FALUDI, J. THANYI, and B. MERKELY. Effects of intense "stretching" flexibility training on the mechanical profile of the knee extensors and on the range of motion of the hip joint. *Int. J. Sports Med.* 6:317–321, 1985.
- Hubley-Kozey, C. L. and W. D. Stanish. Can stretching prevent athletic injuries? J. Musculoskel. Med. 7:21–31, 1990.
- Hubley, C. L., J. W. Kozey, and W. D. Stanish. The effects of static stretching exercises and stationary cycling on range of motion at the hip joint. J. Orthop. Sports Phys. Ther. 6:104–109, 1984.
- KREJCI, V. and P. KOCH. Muscle and Tendon Injuries in Athletes. Chicago: Year book, Medical Publishers, 1979, pp. 61–86.
- LEVIN, A. and J. WYMAN. The viscous elastic properties of muscle. Proc. R. Soc. B. 101:218–243, 1927.
- MAGNUSSON, S. P., E. B. SIMONSEN, P. AAGAARD, U. MORITZ, and M. KJAER. Contraction specific changes in passive torque in human skeletal muscle. *Acta Physiol. Scand.* 155:377–386, 1995.
- Magnusson, S. P., E. B. Simonsen, P. Aagaard, and M. Kjaer. Biomechanical responses to repeated stretches in human hamstring muscle in vivo. Am. J. Sports Med. 5:622-628, 1996.
- 17. Mark, S. D., A. V. Seaber, R. R. Glisson, and W. F. Gerrett, Jr. The role of fatigue in susceptibility to acuse mesers in anjury. Am. J. Sports Med. 2:137–143, 1996.

- McHugh, M. P., S. P. Magnusson, G. W. Gleim, and J. A. Nicholas. Viscoelastic stress relaxation in human skeletal muscle. *Med. Sci. Sports Exerc.* 12:1375–1382, 1992.
- Medeiros, J. M., G. L. Smidt, L. F. Burmeister, and G. L. Soderberg. The influence of isometric exercise and passive stretch on hip joint motion. *Physiol. Ther.* 57:518–523, 1977.
- MOORE, M. A. and R. S. HUTTON. Electromyographic investigation of muscle stretching techniques. *Med. Sci. Sports Exerc.* 12:322– 329, 1980.
- SADY, S. P., M. WORTMAN, and D. BLANKE. Flexibility training: ballistic, static or proprioceptive neuromuscular facilitation. Arch. Physiol. Med. Rehabil. 63:261–263, 1982.
- SAFRAN, M. R., W. E. GARRETT, A. V. SEABER, R. R. GLISSON, and B. M. RIBBECK. The role of warm-up in muscular injury prevention. Am. J. Sports Med. 16:123–129, 1988.
- SAPEGA, A. A., T. C. QUEDENFELD, and R. A. MOYER. Biophysical factors in range of motion exercise. *Phys. Sportsmed.* 9:57–65, 1981.
- SOLVEBORN, S. A. The Book About Stretching. New York: Japan Publications, 1985, p. 116.
- STANISH, W. D. Neurophysiology of stretching. In: *Prevention and Treatment of Running Injuries*, R. D'Ambrosia and D. Drez (Eds.). Thorofare, NJ: Slack, 1982, pp. 135–145.
- TANIGAWA, M. C. Comparison of the hold-relax procedure and passive mobilization on increasing muscle length. *Physiol. Ther*, 52:725–735, 1972.
- TAYLOR, D. C., J. D. DALTON, A. V. SEABER, and W. E. GARRETT. Viscoelastic properties of muscle-tendon units: the biomechanical effects of stretching. *Am. J. Sports Med.* 18:300–309, 1990.
- VAN MECHELIN, W., H. HLOBIL, H. C. G. KEMPER, W. M. VORN, and H. R. DEJONGH. Prevention of running injuries by warm-up, cooldown, and stretching exercises. Am. J. Sports Med. 21:711–719, 1993
- Wallin, D. B., R. Ekblom, R. Grahn, and T. Nordenborg. Improvement of muscle flexibility: a comparison between two techniques. *Am. J. Sports Med.* 13:263–268, 1985.
- WILSON, G. J., B. C. ELLIOTT, and G. A. WOOD. Stretch shorten cycle performance enhancement through flexibility training. *Med. Sci. Sports Exerc.* 24:116–123, 1992.