

Stretch and Contraction Specific Changes in Passive Torque in Human M. Rectus Femoris

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In 43 subjects stretch procedures of the rectus femoris muscle (rf) were produced before and after 15-min treatments: SG ($n = 11$), 3×5 stretches (st1–st15) of rf with 3-min rest after 5 reps; CG ($n = 11$), resistance training of rf (5×12); RG ($n = 10$), 15-min rest without any activity; AG ($n = 11$), 15-min submaximal activity.

During the pretest, subjects underwent 3 and, in retest, 2 stretch procedures. Throughout every stretch procedure resting tension (TS) at 9° below the maximal range of motion of pretest-PS was quantified and calculated as percent of TS.

In pretest, TS of the 4 treatment groups declined to 89.3%. During treatment of SG, TS decreased until st2 (the fifth stretch altogether; 77.8%; $p < .01$). In the retest, TS of SG (82.3%) was significantly lower than in the other 3 treatment groups (96.8%, $p < .01$). No significant differences exist between the increases of these 3 treatment groups.

Low TS during the retest of the SG group has to be attributed to the stretching load of the titin filaments in the course of the treatment. In contrast to this, titin filaments don't seem to be under any strain during the treatment in the other groups and thus recover to the full extent after the few stretch procedures between pre- and retest.

Key Words: flexibility, stretching, resting tension, resistance training, titin

Key Points:

1. Stretching of rectus femoris muscle resulted in a decrease in resting tension of 22.2%.
2. Resistance training did not affect resting tension in an other way than a rest without any activity and than a rest with submaximal activity (walking, stairs climbing, or squats).
3. The significantly lower resting tension in retest of the stretch group has to be attributed to the stretching load of titin filaments in the course of the treatment.
4. In contrast to this, titin filaments do not seem to be under any strain during the treatment in the other groups and thus recover after the few stretch procedures between pre- and retest.

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Introduction

When stretch- and contraction-specific changes in range of motion or in passive torque are investigated in humans, the most frequently used muscles are the hamstrings, and the most frequently used test is the straight leg raise (Figure 1). Whenever the hamstrings are stretched twice in a row, the comparison between the two hip-angle stretching tension curves shows three stretch specific changes: First, an increase in range of motion and, second, an increase in peak torque. It could be demonstrated that the enlargement of range of motion after short-term stretching programs, but also after long-term stretching programs, has to be attributed to an increase in the subject’s tolerance to stretching strain (26).

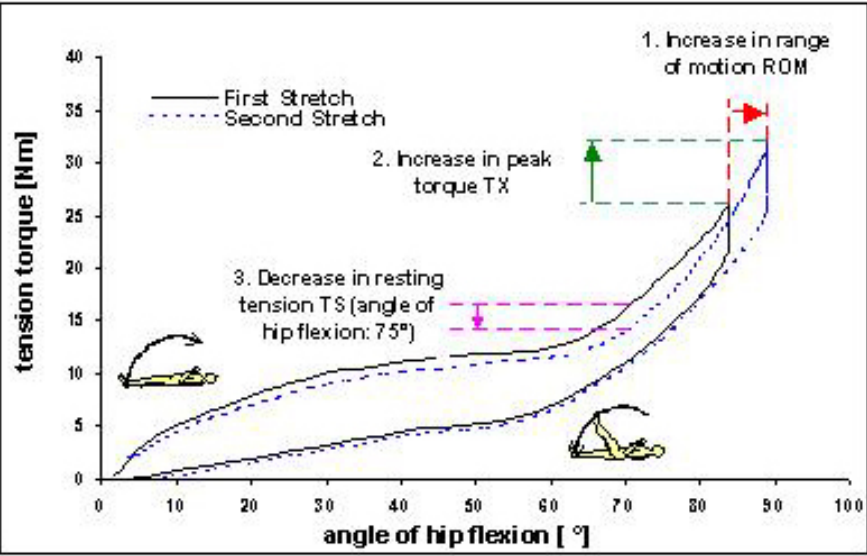


Figure 1 — The hip-angle stretching tension curves of two successive stretches of the hamstrings.

The third effect is a decrease in resting tension at a low angle of hip flexion—in this case, at 75° (Figure 1). Formerly, resting tension was attributed to elastic resistance of connective tissue and of sarcolemma (17). Later, it was demonstrated that in intact muscles up to a stretching rate of 160%, resting tension arises from the elastic resistance of the myofibrils (13). Recently, the titin filaments, elastic molecular springs within the sarcomeres (12, 16, 22), have been identified as the source of resting tension. If the difference between the resting tension of the two stretches is transferred to a graph (see x-axis: series of stretch procedures), the decrease in resting tension as a result of repeated stretches can be illustrated (Figure 2). In this way, Magnusson et al. (15) demonstrated that resting tension can be reduced by about 20% by 4 stretches and that resting tension recovers after an interval rest of 60 min. But, it is still unknown so far how dynamically the already-decreased resting tension recovers.

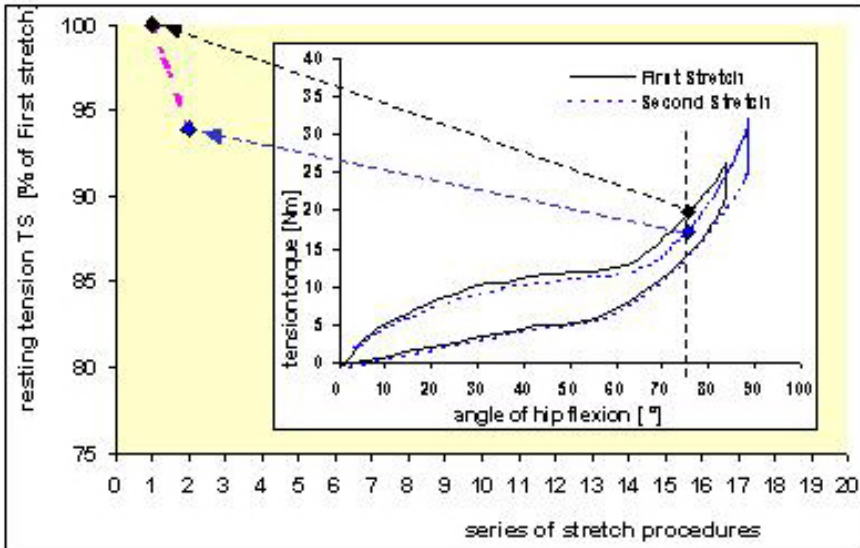


Figure 2— Transfer of absolute values of resting tension to a graph, with relative values displaying a decrease of resting tension from the first to the second stretch procedure.

Moreover the specific characteristics of hamstrings—for example, muscle fiber type distribution, arrangement of fibers—require a verification of scientific results before generalizing them by means of an adequate comparison with other groups of muscles. Therefore, the purpose of the present study was to investigate the stretch specific changes in resting tension of rectus femoris muscle.

In addition to investigations demonstrating a decrease in resting tension after short-term stretching (15), other examinations fail to reveal any lowering of stretching tension in the course of several stretch procedures (26). The reason for this may be that during these examinations, there was a break interval of 5 to 10 min between treatment and retest. Besides that, subjects had muscular contractions while climbing up and being fixed on the experimental station. This may have diminished the effects of stretching on resting tension and thus is probably responsible for the fact that a decrease in resting tension could not be demonstrated in that investigation. Due to these results, we intended to investigate effects of a rest with submaximal activity.

Besides this, there was another open question to be investigated: In vitro experiments show that stretching tension of muscles can be lowered by means of contractions as well as by stretches (19). Comparable in vivo investigations lead to inconsistent results. Thus stretching tension increased after eccentric resistance training (25) but, after auxotonic resistance training, remained unaltered (26). Magnusson et al. (14) demonstrated that submaximal stretching tension did not change after 40 eccentric contractions whereas, after 40 concentric contractions, it had decreased significantly by 22%. Investigations with finger muscles, which demonstrated contradictory results, show obviously (4, 9) that there is great need for clarification concerning effects of different types of contractions on different stretch-

ing parameters. It was therefore a main purpose of the investigation to determine the effects of short-term resistance training.

Materials and Methods

The subjects ($n = 43$) were placed on the experimental station (Figure 3) on their left side to perform the stretch procedure in the horizontal plane in order to eliminate the effects of gravity—that is, displacement of blood (5). Stretch procedures of the rectus femoris muscle (rf) lasting 24 s were produced before (pretest) and after (retest) 15-min treatments. The subjects were randomly assigned to one of the four treatment groups (Figure 4):

- SG ($n = 11$): 3×5 stretches (st1–st15) of rf with 3-min rest after 5 reps
- AG ($n = 11$): 15-min rest with submaximal activity (walking, stairs climbing, and squats)
- CG ($n = 11$): auxotonic resistance training of rf (5 sets of 12 repetitions each, resistance weight $\sim 70\%$ of one repetition maximum, with 3-min resting periods between the sets)
- RG ($n = 10$): 15-min rest with no activity—this group served as the control group.

During the pretest, subjects underwent 2 maximal (PX1, PX2) and 1 submaximal (PS) stretch procedures of rf. In retest, 1 maximal (RX) stretch procedure and 1 submaximal (RS) stretch procedure were performed. Throughout every stretch procedure of pretest, SG-treatment (st1–st15) or retest maximal range of motion (ROM) and maximal tension torque (TX) were measured. Furthermore, resting

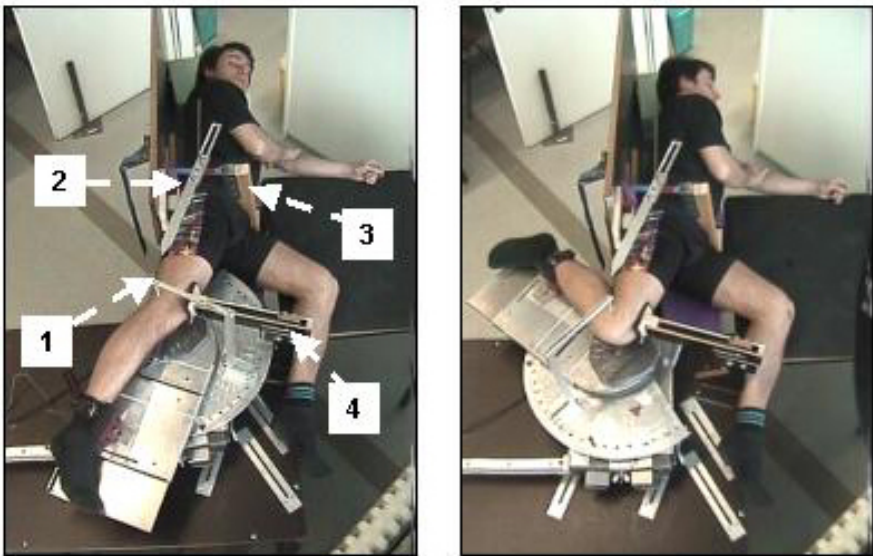


Figure 3 — Experimental conditions: (1) pointer to align the axis of knee joint with the axis of rotation disc; (2) hip joint extension: 20° ; (3) pelvic strap; (4) thigh strap.

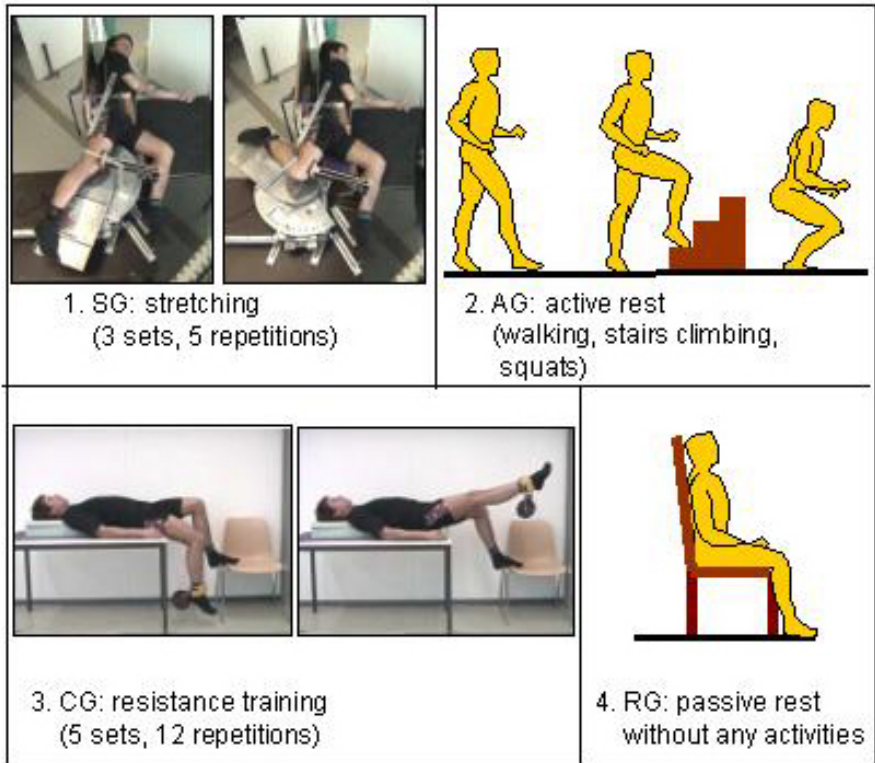


Figure 4 — The four treatments.

tension (TS) at 9° below the maximal range of motion of pretest-PS was quantified and calculated as percent of TS_{PX1} . The function of the first maximal stretch procedure PX1 was to familiarize the subjects with the experimental station. For evaluation of changes in ROM and TX, PX2 and RX were compared. For evaluation of changes in TS, all 20 measurements (PX1, PX2, PS, st1–st15, RX, RS) from the SG group were compared and in groups CG, RG, AG all 5 measurements (PX1, PX2, PS, RX, RS) were compared. Because the values were normally distributed, a paired t test was used to examine the differences between all single stretch procedures within the treatment groups, and t test for independent samples was employed to examine the differences between the treatment groups. All statistical calculations were computed using *Excel 97*.

Results

Regarding ROM, this study could only demonstrate a significant increase of 4.3° ($p < .01$) in SG. This increase of ROM in retest did not go along with any rise of TX. Neither short-term auxotonic resistance training nor the two break intervals had any influence on ROM and TX, respectively.

to be attributed to the stretching load of titin filaments. In contrast to this, titin filaments do not seem to be under any strain during the treatment in the other groups and thus recover after the few stretch procedures between pre- and retest.

The titin filaments, making up ~ 10% of the combined muscle protein, link the myosin filaments with the Z-discs and bear most of the resting tension during physiological extension in skeletal muscles. Preventing sarcomeric overstretch beyond acto-myosin overlap and restoring slack sarcomere length after stretching seem to be the most important functions of titin. This physiological function may be the reason for the discovery that resting tension of human muscles could not be lowered by stretching exercises in the course of long-term stretching programs (7, 26). There is just one segment of the titin filaments within the I-band, which generates the resting tension, the PEVK-segment (70% of its residues are P-proline, E-glutamate, V-valine, and K-lysine).

The stretching of a myofibril in vitro from slack sarcomere length of $2.1\ \mu\text{m}$ (100%) generates exponential rise in tension (Figure 6). At a sarcomere length of $3.5\ \mu\text{m}$ (167%), the passive length tension curve flattens. At this point, the so-called *yield point* (22), titin is released from the distal ends of the myosin. A study by Goulding et al. (3) showed that this point is prevented in vivo by skeletal, tendinous, and ligamentous structures. Measurements of the elastic forces generated during stretching of single titin molecules indicate that the sources of passive tension are within the titin molecules (1). Both entropic and enthalpic origins are supposed (12).

The theory that titin bears most of the resting tension is supported by two other studies. Tskhovrebova et al. (20) discovered in their investigation of isolated titin molecules the same nonlinear visco-elastic properties (stress-relaxation and hysteresis) as Taylor et al. (18) in their examination of entire muscle-tendon units. Moreover, Kellermayer et al. (8) found out in their investigation of isolated titin molecules, that several stretching procedures lead to a “wearing out” of titin. “By pre-unfolding just as much titin as necessary, the maximum of the range of efficient elastic response may be adjusted. Regulating the range of the efficient elastic response in muscle

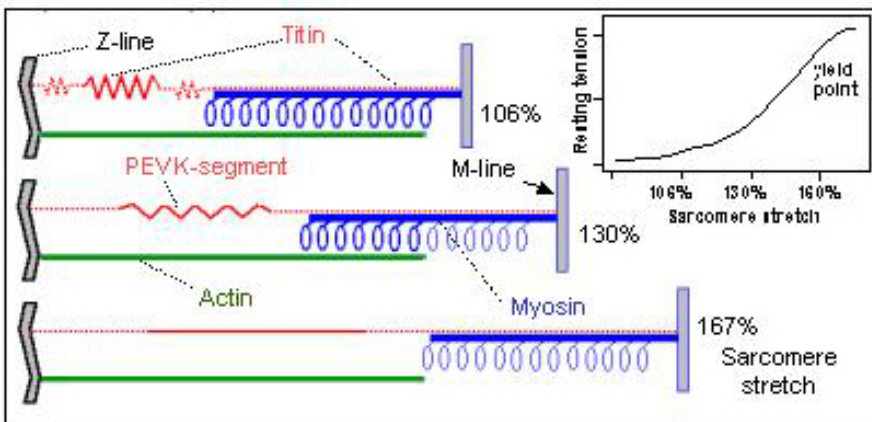


Figure 6 — Model of titin extension with sarcomere stretch. Inset: increase in resting tension (see 11, 22).

through titin unfolding and refolding may serve as an adaptive mechanism during the repetitive mechanical loading of skeletal or cardiac muscle.” (8)

If one complements the demonstrated decrease of TS after 3 min (by 4%) and after 15 min (by 13%, the increase of resting tension of RG and AG in a ratio of the 22.2% decrease of SG caused by 4 stretch procedures) with the findings of Magnusson et al. (15), who established a complete recovery after 60 min, one is able to depict recovery of resting tension as shown in Figure 7.

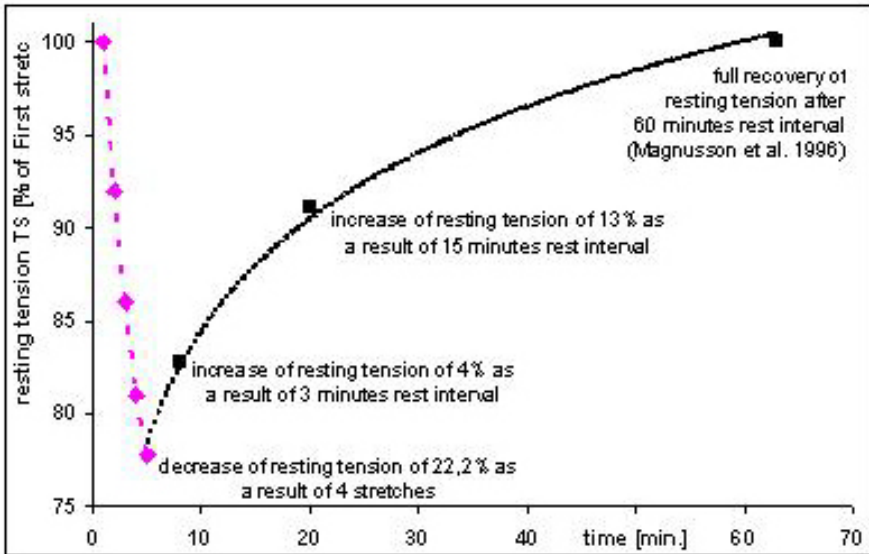


Figure 7 — The decrease of resting tension as a result of 4 stretches and recovery after 3 min, 15 min, and 1 hour.

Like the decrease in resting tension, its recovery has a biological significance, too. If the “wearing out” were not reversible, titin could not fulfill its function to restore slack sarcomere length after stretching (27). This assumption is supported by the result that long-term stretching does not lead to a decrease of resting tension, but contrary to hypertrophy and thus to an increase in resting tension. This has been demonstrated in experiments with animals (2) as well as human subjects (10, 23).

As resting tension already decreases by 6.7% after one stretch procedure and reduces by a further 4.1% after a second one, stretches in order to familiarize subjects with the test procedure have to be performed very carefully. To avoid this problem, stretch procedures before the test should not be carried out at all. As resting tension starts to increase already after a 3-min break, retest should be performed as quickly as possible after treatment.

These findings are relevant and applicable in the practice of sports because they show that 4 stretch procedures are sufficient to reduce resting tension in the run-up to top performance in a competition. Furthermore, there should not be a long interval between stretching and sport activity.

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