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Effects of Low-Load Resistance Training With Vascular Occlusion on the Mechanical Properties of Muscle and Tendon

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The present study aimed to investigate the effects of low-load resistance training with vascular occlusion on the specific tension and tendon properties by comparing with those of high-load training. Nine participants completed 12 weeks (3 days/week) of a unilateral isotonic training program on knee extensors. One leg was trained using low load (20% of 1 RM) with vascular occlusion (LLO) and other leg using high load (80% of 1 RM) without vascular occlusion (HL). Before and after training, maximal isometric knee extension torque (MVC) and muscle volume were measured. Specific tension of vastus lateralis muscle (VL) was calculated from MVC, muscle volume, and muscle architecture measurements. Stiffness of tendon-aponeurosis complex in VL was measured using ultrasonography during isometric knee extension. Both protocols significantly increased MVC and muscle volume of quadriceps femoris muscle. Specific tension of VL increased significantly 5.5% for HL, but not for LLO. The LLO protocol did not alter the stiffness of tendon-aponeurosis complex in knee extensors, while the HL protocol increased it significantly. The present study demonstrated that the specific tension and tendon properties were found to remain following low-load resistance training with vascular occlusion, whereas they increased significantly after high-load training.

***Key Words*:** knee extension, tendon stiffness, physiological cross-sectional area

Recent studies demonstrated that low-load resistance training combined with vascular occlusion induced remarkable increases in muscle strength and hypertrophy (Takarada et al., 2000; Takarada, Sato, & Ishii, 2002). According to the size principle of neuromotor control (Hammarsten, Bylund-Fellenius, Holmm, Schersten, & Krotkiewski, 1980), larger motor units for type II fibers are gradually recruited with an increase in force production level. Therefore, higher load resistance training has been considered to recruit type II fibers during the exercises (e.g., MacDougall, Elder, Sale, Moroz, & Sutton, 1980).

In fact, some previous studies have shown that high-load resistance training enhances the ability to activate the motor units of muscles, and consequently to increase the muscle force per physiological cross-sectional area (specific tension; Narici, Roi, Landoni, Minetti, & Cerretelli, 1992; Reeves, Narici, & Maganaris, 2004). However, no studies have investigated the effects of low-load training with vascular occlusion on the activation of motor unit and/or the specific tension.

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Tendons play a very important part in the transmission of tension from muscle fiber to bone. Previous findings obtained from animal and human experiments have shown that the adaptations of tendon to training vary with the mode of exercise performed (e.g., Kubo, Kanehisa, & Fukunaga, 2001; Simonsen, Klitgaard, & Bojsen-Moller, 1995). Recent studies using ultrasonography demonstrated that the stiffness of human tendon increased after higher load resistance training (Kubo et al., 2001; Reeves, Maganaris, & Narici, 2003). Furthermore, we have reported that the isometric training regimen using higher internal muscle force increased tendon stiffness whereas that using a lower force level did not (Kubo et al., in press). Accordingly, it seems reasonable to suppose that the effect of low-load training with vascular occlusion on the tendon was different from that of normal high-load resistance training, even if the possible change in muscle strength and/or size are nearly the same between both protocols.

Low-load resistance training with vascular occlusion would be potentially useful for accelerating the recovery of muscle strength and size without large mechanical stress for patients and aged populations. Therefore, information on the effects of this training regimen on the nervous system and tendon properties is essential for an understanding of the increment of physical performances and/or preventing sports related injuries. In the present study, we aimed to examine the effects of the low-load resistance training with vascular occlusion on the specific tension and tendon properties through the comparison with those of high-load resistance training.

Methods

Nine healthy young men (age 25 ± 2 yrs, height 172 ± 4 cm, body mass 73 ± 13 kg, mean ± *SD*) voluntarily participated in this study. They were fully informed of the procedures to be utilized as well as the purpose of this study. Written informed consent was obtained from all participants. This study was approved by the Department of Sports Sciences, University of Tokyo, and complied with their requirements for human experimentation.

The participants performed unilateral knee extension exercises in a seated position with an isotonic knee extension machine. The range of motion of the knee joint was 90° to 0° (0° = full extension). Participants were instructed to lift and lower the load at an approximately constant velocity, taking about 1 second for concentric action and 3 s for eccentric action. They trained three times per week for 12 weeks. One leg was trained using the low load with vascular occlusion (LLO) while the other leg was trained using the high load without vascular occlusion (HL). For each participant, the right and left legs were randomly allocated to either training protocol. That is, in a given training session the participant would train the LLO protocol leg first, then the HL protocol leg, and in the next session the order would be reversed.

In LLO, the proximal portion was compressed by a specially designed elastic belt. The belt contained a small pneumatic bag along its inner surface which could be connected to an electronic pressure gauge so as to monitor the occlusion pressure (model MPS-700 developed by Y. Sato and manufactured by VINE Medical Instruments, Tokyo). During the training period, the occlusion pressure increased gradually every 4 weeks. The participants performed 4 sets of exercise with an inter-set interval of 30 s. The intensity of exercise was 20% of the weight that could just be lifted once throughout the complete range of movement (1 RM), which was determined in the initial stage of exercise training and remained unchanged throughout the training period. Each set included 25, 18, 15, and 12 repetitions (means of repetition / one set; 17.5 reps). The load and repetition for LLO were decided according to the previous report (Takarada et al., 2000).

In HL the participants performed 4 sets of exercise with an inter-set interval of 1 min, which consisted of unilateral knee extension at 80% of 1 RM with 10 repetitions per set. The 1 RM was measured every 4 weeks to adjust the training load.

Muscle Strength and Neural Activation

Maximal voluntary isometric strength (MVC) of the knee extensor muscles was determined using an electrical dynamometer (Myoret, Asics, Japan). The participant sat in an adjustable chair with support for the back and the hip joint flexed at an angle of 80° (full extension = 0°) to standardize the measurements and localize the action to the appropriate muscle group. The ankle was firmly attached to the

lever arm of the dynamometer with a strap and fixed with the knee joint flexed at an angle of 90°.

When the voluntary torque peaked, evoked twitch contractions were imposed by supramaximal electrical stimulations. The stimulating electrodes were placed on the skin over the femoral nerve at the inguinal region (cathode) and the midbelly of the quadriceps muscle (anode). Rectangular pulses (triple stimuli with a 500-μs duration for one stimulus and an interstimulus interval of 10 ms) were delivered using a high-voltage stimulator. The difference between peak twitch force and MVC force (twitch force) was measured. Afterward the twitch imposed on the resting muscle after MVC (control twitch force) was measured. The voluntary activation (%) of the knee extensor muscles was calculated as: {1 – (twitch force during MVC / control twitch force)} * 100 as previously reported (Becker & Awiszus, 2001).

Stiffness of Tendon-Aponeurosis Complex and Patella Tendon

The participants exerted isometric knee extension torque from zero (relax) to MVC within 5 s. An ultrasonic apparatus (SSD-2000, Aloka, Tokyo) with an electronic linear array probe was used to obtain longitudinal ultrasonic images of each muscle and tendon. To evaluate the elongation (L) of tendon and aponeurosis, we recorded the movements of the following two points (P1 and P2; see below) by ultrasonography (see Figure 1 of Kubo et al., 2005). The longitudinal images of the vastus lateralis muscle (VL) and patella tendon were obtained at the level of 50% of thigh length (P1) and at the apex of the patella (P2), respectively (see Figure 1 of Kubo et al., 2005). The displacement of P1 was considered as the tendon-aponeurosis complex elongation, and the displacement of P2 was considered as the patella tendon elongation.

According to Kubo, Kanehisa, and Fukunaga (2005) and Reeves et al. (2003), angular joint rotation needs to be accounted for in order to avoid an overestimation of tendon displacement during contraction. To monitor joint angular rotation, we placed an electrical goniometer on the lateral aspect of the knee joint. To correct the measurements taken for the tendon and aponeurosis elongation, we took additional measurements under passive conditions. Thus, for each participant the displacement of P1 obtained from the ultrasound images could be corrected for that attributed to joint rotation alone (Kubo et al., 2005; Reeves et al., 2003). However, we did not correct for the measurement of patella tendon elongation (displacement of P2) in the present study (Kubo et al., 2005; Reeves et al., 2003).

The knee joint torque (TQ) measured by the dynamometer was converted to muscle force (Fm) by the following equations:

$$Ft = TQ \cdot MA^{-1}$$

$$Fm = k \cdot Ft$$

where Ft and k represent tendon force and relative contribution of VL to the quadriceps femoris muscles in terms of physiological cross-sectional area, and MA is the moment arm length of quadriceps femoris muscles at 90° of knee flexion, which is estimated from the thigh length of each participant (Kubo et al., 2001). The Fm-L and Ft-L curves above 50% MVC were fitted to a linear regression equation, the slope of which was adopted as an index of the stiffness of tendon-aponeurosis complex and patella tendon, respectively (Kubo et al., 2001).

Electromyographic Activity

The electromyographic activity (EMG) was recorded during the measurement of maximal voluntary isometric strength and tendon properties. Bipolar surface electrodes (5 mm in diameter) were placed over the bellies of VL, the rectus femoris muscle (RF), vastus medialis muscle (VM), and biceps femoris muscles (BF) with a constant interelectrode distance of 25 mm. The electrodes were connected to a preamplifier and differential amplifier with a bandwidth of 5 Hz to 500 Hz (Model 1253A, NEC Medical Systems, Tokyo). The EMG signals were transmitted to a computer at a sampling rate of 1 kHz. The EMG was full-wave rectified and integrated for a 1.0-s period of steady-force output for the measurement of MVC to give integrated EMG. In addition, the mean of integrated EMG in the knee extensors (VL, RF, VM) was defined as mEMG.

To investigate the antagonist muscle activity of the BF (coactivation level), we measured the integrated EMG of the BF during knee extension contraction. To determine the maximal activation of the BF, we had the participant perform a maximal knee flexion isometric contraction at the same angle (90° of knee joint). We normalized the integrated

EMG value of BF with respect to the integrated EMG value of BF at the same angle when acting as agonist at maximal effort.

Measurements

The pennation angle and fascicle length of VL were measured during MVC using an ultrasonic apparatus. The pennation angle was defined as the angle between the fascicle and deep aponeurosis. The fascicle length was defined as the distance between the insertions of the fascicle into the superficial and deep aponeurosis.

The cross-sectional area (CSA) of the quadriceps femoris muscle was measured by magnetic resonance imaging scans (Resona, 0.5 Tesla System, GE). T1-wighted spin-echo, axial-plane imaging was performed with the following variables: TR 450 ms, TE 20 ms, matrix 256 × 172, field of view 300 mm, slice thickness 10 mm, and interslice gap 0 mm. The participants were imaged in a prone position with the knee kept at 0°. Consecutive axial images were obtained from spina illiaca anterior superior to extremitas distal of tibia. The muscles examined were RF, VL, vastus intermedius (VI), and VM. From the series axial images, outlines of each muscle were traced, and the traced images were transferred to a computer for calculation of the anatomical CSA using digitizing software. Muscle volume was determined by summing the anatomical CSA of each image times the thickness (10 mm). Furthermore, the physiological CSA of VL was calculated by dividing muscle volume by fascicle length (e.g., Reeves et al., 2004).

In addition, the measurement of patella tendon CSA was taken at a knee joint angle of 90° from axial plane ultrasound images taken at 25, 50, and 75% of patella tendon length according to Reeves et al. (2003). The average of CSA at the three positions was calculated as representative of tendon CSA (Reeves et al., 2003).

Specific tension of VL was calculated by dividing fascicle force by physiological CSA (e.g., Reeves et al., 2004). The fascicle force (Ff) of VL was calculated by the following equation:

$$Ff = Fm \cdot \cos P^{-1}$$

where cos P is the cosine of the angle of pennation of VL.

Descriptive data included means ± *SD*. A two-way ANOVA with repeated measures (2 groups × 2 test times) was used to analyze the data. The *F* ratios for main effects and interactions were considered significant at $p < 0.05$. Significant difference among means at $p < 0.05$ were detected using a Tukey post hoc test.

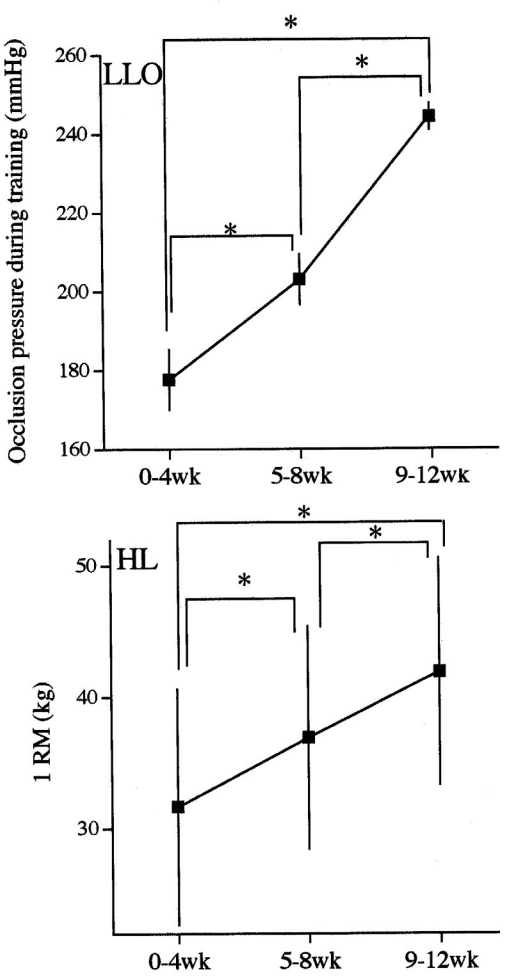

Figure 1 – Changes in occlusion pressure for LLO and the 1 RM for HL. Both measured variables increased significantly. *Significantly different at $p < 0.05$

Results

For LLO, the occlusion pressure increased significantly, 37.7% (Figure 1). Similarly, 1-RM value for HL increased significantly, 37.8% (Figure 1). The volume of quadriceps femoris muscles increased significantly, 5.9% for LLO ($p = 0.022$) and 7.4% for HL ($p = 0.016$) (Figure 2). No significant difference was found in the relative increase in muscle volume between LLO and HL ($p = 0.195$). The PCSA of VL increased significantly, 6.1% for LLO

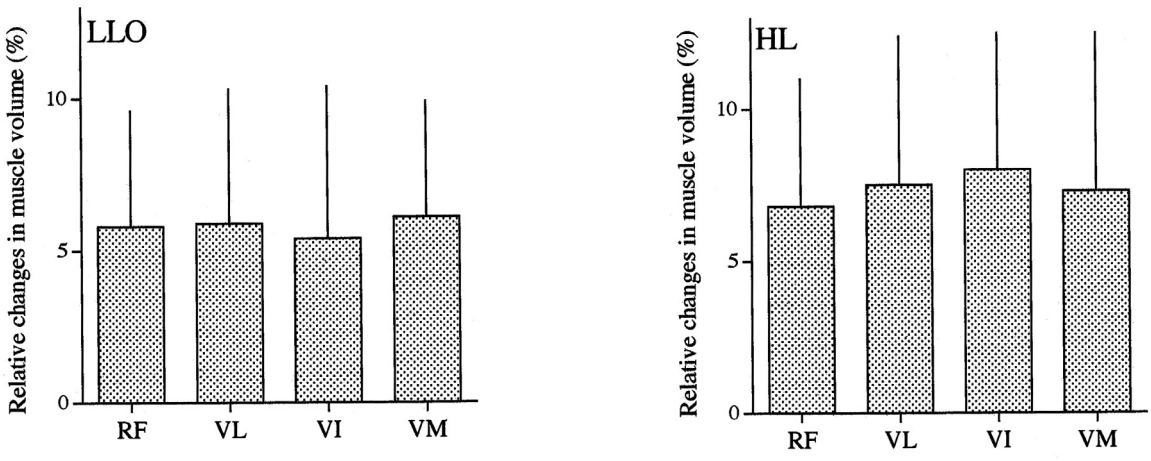

Figure 2 – Relative changes in muscle volumes of rectus femoris (RF), vastus lateralis (VL), vastus intermedius (VI), and vastus medialis (VM) before and after training for 12 weeks. All muscle volumes of knee extensors (RF, VL, VI, VM) increased significantly for both protocols.

Table 1 Measured Variables (mean ± *SD*) for LLO and HL Protocols

	LLO		HL	
	Before	**After**	**Before**	**After**
Muscle volume (cm^3)	1892 (205)	2032 (214) *	1878 (230)	2011 (242) *
Physiologocal CSA (cm^2)	75.6 (9.2)	81.3 (9.3) *	74.5 (9.8)	79.0 (8.9) *
Fascicle length (mm)	83.1 (10.1)	85.4 (9.2)	84.2 (10.8)	86.1 (8.6)
Pennation angle (deg)	14.8 (1.2)	15.6 (1.1) *	15.1 (1.4)	16.0 (0.9) *
MVC (Nm)	224 (62)	241 (69) *	225 (66)	262 (73) *
Activation level (%)	94.2 (4.7)	93.7 (5.6)	93.5 (5.1)	96.5 (4.5) *
mENG (mV)	0.58 (0.13)	0.60 (0.11)	0.62 (0.09)	0.75 (0.12) *
Coactivation level (%)	16.9 (7.6)	17.4 (8.2)	17.7 (6.8)	17.1 (7.7)
Specific tension (N/cm^2)	23.1 (5.2)	23.9 (4.7)	23.8 (4.3)	25.1 (4.6) *
Stiffness of tendon-aponeurosis complex (N/mm)	45.1 (14.6)	49.3 (21.8)	45.6 (19.1)	59.3 (23.0) *
Stiffness of patella tendon (N/mm)	1694 (692)	1722 (704)	1676 (662)	1819 (710)
CSA of patella tendon (cm^2)	78.9 (18.7)	78.4 (19.2)	78.1 (19.3)	77.6 (19.9)

* Significantly different from before.

($p = 0.024$) and 7.5% for HL ($p = 0.021$) (Table 1). Furthermore, no significant changes were found in the patella tendon CSA in either the LLO or HL. The fascicle length of VL did not change significantly in either the LLO or HL. Pennation angle of VL increased significantly, 5.2% for LLO ($p = 0.038$) and 5.8% for HL ($p = 0.029$).

The MVC value increased significantly, 7.8% for LLO ($p = 0.019$) and 16.8% for HL ($p = 0.008$) (Table 1). The relative increase in MVC tended to be lower for LLO than for HL ($p = 0.107$). Activation level of the quadriceps femoris muscle assessed by superimposing electrical stimuli increased significantly, 3.2% for HL ($p = 0.031$), but not for LLO ($p = 0.457$). Similarly, the mEMG of the quadriceps femoris muscle increased significantly, 20.5% for HL ($p < 0.001$), but not for LLO ($p = 0.654$). For both LLO and HL protocols, the coactivation level

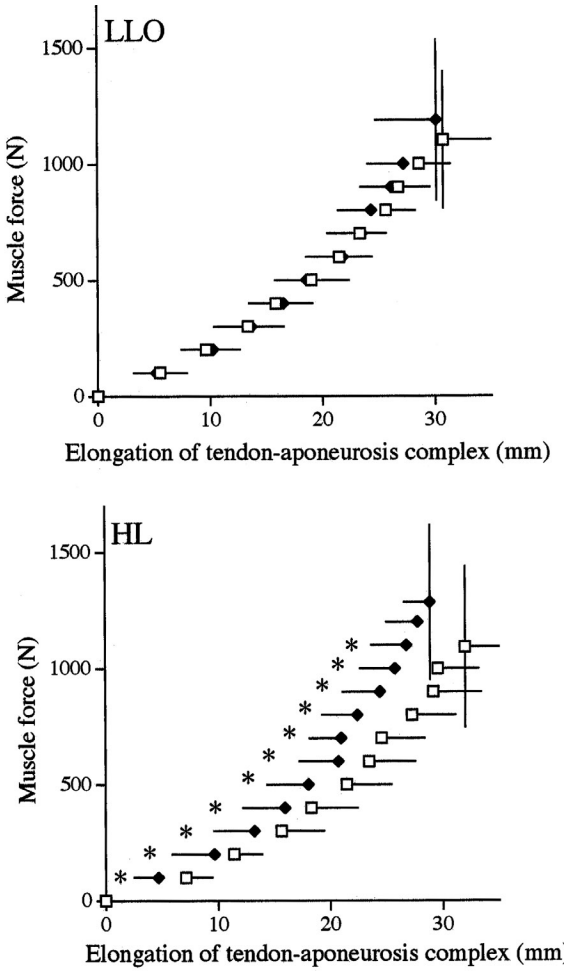

Figure 3 – Muscle force–elongation of tendon-aponeurosis complex before (open symbols) and after (closed) training for 12 weeks. The L values of tendon-aponeurosis complex at all force production levels decreased significantly after training for HL (lower), although that did not change for LLO (upper). *Significantly greater than before at $p < 0.05$

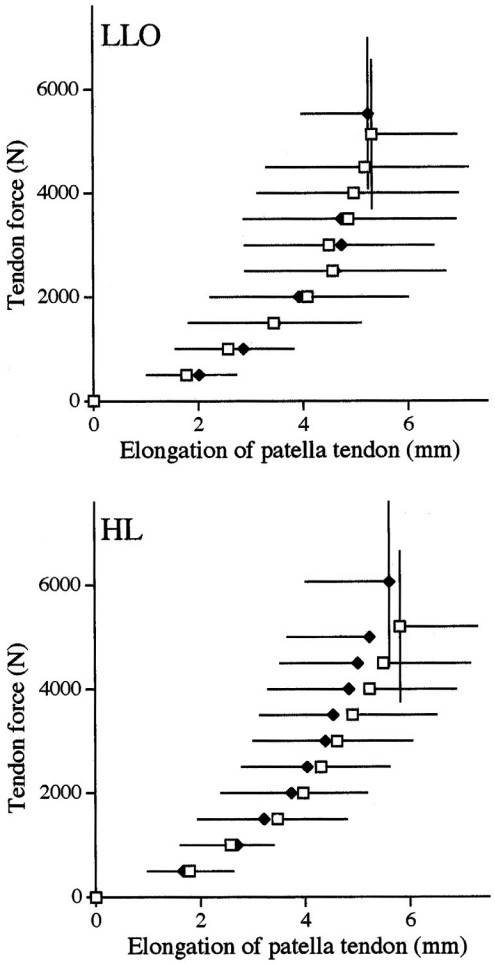

Figure 4 – Tendon force–elongation of patella tendon before (open symbols) and after (closed) training for 12 weeks. The L values of patella tendon at all force production levels did not change after training for both protocols.

of BF did not change after training. The specific tension of VL increased significantly, 5.5% for HL ($p = 0.018$), but not for LLO ($p = 0.217$).

The LLO protocol produced no significant differences in the L values of the tendon-aponeurosis complex at all force production levels between before and after (Figure 3). In the case of the HL protocol, the L values at all force levels were significantly shorter after training. The stiffness of the tendon-aponeurosis complex increased significantly for HL ($p = 0.038$), but not for LLO ($p = 0.468$) (Table 1). The relative increase in stiffness of the tendon-aponeurosis complex tended to be greater for HL than for LLO ($p = 0.131$).

For both LLO and HL protocols, there were no significant differences in the L values of the patella tendon at all force production levels between before and after (Figure 4). In addition, the stiffness values of the patella tendon did not change after training for both protocols (Table 1).

Discussion

Recent studies showed that the low-load training regimen with vascular occlusion caused a marked muscular hypertrophy, even if the load of exercise was much lower than expected to increase in muscle size (Takarada et al., 2000; 2002). In the present study, there was no significant difference in the relative increase of muscle volume between LLO and

HL. This result was consistent with the findings of Takarada et al. (2000; 2002). However, the relative increase in MVC tended to be lower for LLO than for HL ($p = 0.107$). One possible reason for the difference in the increment of MVC between two protocols might be a change in the activation level of the quadriceps femoris muscles.

In the present study, the activation levels of the quadriceps femoris muscles assessed by surface electromyogram and twitch-interpolation technique increased significantly for HL, but not for LLO (Table 1). For HL, the activation levels of assessed by surface electromyogram and twitch-interpolation technique increased significantly, 20% and 3.2%, respectively (Table 1). These results were similar to previous findings concerning resistance training using high load (e.g., Narici et al., 1996). As far as we know, only Moore et al. (2004) have assessed the contribution of neuromuscular adaptations to the increment of muscle strength after low-intensity occluded resistance training. They also reported that the motor unit activation assessed by the twitch-interpolation technique did not change after the low-load (50% of 1 RM) resistance training with vascular occlusion.

The present result was consistent with the finding of Moore et al. (2004). Thus it seems reasonable to suppose that the low-load with occlusion training induces hypertrophy without the increment of activation level, whereas muscle size and activation level increase after high-load resistance training.

Furthermore, the specific tension increased significantly for HL, but not for LLO (Table 1). Previous studies concerning low-load training with vascular occlusion investigated the change in one slice of the anatomical CSA of the trained muscle (Takarada et al., 2000; 2002). Therefore, the effect of low-load training with occlusion on the specific tension were unclear, since muscle volume and muscle fiber or fascicle length were required for accurate determination of specific tension (e.g., Reeves et al., 2004). According to previous findings (Narici et al., 1992; Reeves et al., 2004), the increment of muscle strength after high-load resistance training was caused by the hypertrophy and the improvement of activation level of muscles. On the contrary, we may say that the hypertrophy produced by LLO does not accompany the increment of neural activation level of muscle; consequently, the specific tension remained after low-load training with occlusion.

To calculate the accurate specific tension, the "optimal" fascicle length should be used. In the present study, however, we aimed to examine the influences of LLO and HL protocols on neuromuscular and tendon properties rather than the "accurate" specific tension. The present result showed that the fascicle length during MVC did not change after training (Table 1). In addition, as far as we know, no studies have ever demonstrated that the optimal joint angle changed after training (due to changes in the tendon properties). In light of this, we considered that the calculation of the specific tension was valid for studying the changes of specific tension after training.

Muscle contractions are accompanied by simultaneous changes in many variables, including increased mechanical stress and metabolic alteration. In the present study, the stiffness of both the tendon-aponeurosis complex and patella tendon did not change after low-load training with vascular occlusion. When resistance exercise was performed with occlusion, an elevated energy consumption and repeated muscle contractions with the increase in intramascular pressure would have complex effects on blood circulation and accumulation of lactate (Takarada et al., 2000). The lactate is produced in response to tissue hypoxia and is a strong stimulator of collagen production by resident tissue macrophages (Klein, Pham, Yalamanchi, & Chang, 2001; Yalamanchi, Klein, Pham, Longaker, & Chang, 2004). Therefore, it was hypothesized that tendon stiffness increased for LLO protocol by the accumulation of lactate due to vascular occlusion.

However, this hypothesis was not borne out in the present study. Our recent observation showed that the isometric training regimen using higher internal muscle force led to an increase in tendon stiffness, whereas that using lower force level did not (Kubo et al., in press). This finding suggests that the mechanical stress was important for changes in tendon stiffness. Taking the present result into account along with the previous finding (Kubo et al., in press), we may say that only "mechanical" stress contributed to adaptation in tendon stiffness whereas metabolic and mechanical stresses were related to muscle hypertrophy.

The present study demonstrated that low-load training with vascular occlusion increased muscle strength and size without the increment of tendon stiffness. Our previous study showed that tendon

stiffness was negatively correlated to performance during stretch-shortening cycle exercises (Kubo et al., 1999). Therefore it might be assumed that, compared to the HL protocol, the LLO protocol is more effective at improving performance during stretch-shortening cycle exercises. In the future it will be necessary to investigate the effect of low-load training with vascular occlusion on performance during various exercises as well as the morphology of muscle and tendon.

We must draw attention to the negative effects of the LLO protocol contrary to the above mentioned positive effects. The ultimate strength of tendons after low-load training with vascular occlusion would not provide the strength corresponding to the increment of muscle strength after training. Furthermore, it may safely be assumed that this leads to tendon injuries. Similarly, actual cases of spontaneous rupture of tendons have been reported in athletes who have ingested large doses of anabolic steroids (Freeman & Rooker, 1995). Therefore, we should notice whether tendon strength and property correspond to muscle strength after training. Regardless, further studies using animal models are needed to clarify this point.

In conclusion, the present study demonstrated that low-load resistance training with vascular occlusion did not alter the specific tension and stiffness of the tendon-aponeurosis complex, while high-load training increased it significantly. These results suggested that low-load resistance training with vascular occlusion did not affect the motor unit activation of muscle and tendon properties.

Acknowledgments

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