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# ACUTE EFFECTS OF SELF-MYOFASCIAL RELEASE USING A FOAM ROLLER ON ARTERIAL FUNCTION

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## ABSTRACT

Okamoto, T, Masuhara, M, and Ikuta, K. Acute effects of self-myofascial release using a foam roller on arterial function. *J Strength Cond Res* 28(1): 69–73, 2014—Flexibility is associated with arterial distensibility. Many individuals involved in sport, exercise, and/or fitness perform self-myofascial release (SMR) using a foam roller, which restores muscles, tendons, ligaments, fascia, and/or soft-tissue extensibility. However, the effect of SMR on arterial stiffness and vascular endothelial function using a foam roller is unknown. This study investigates the acute effect of SMR using a foam roller on arterial stiffness and vascular endothelial function. Ten healthy young adults performed SMR and control (CON) trials on separate days in a randomized controlled crossover fashion. Brachial-ankle pulse wave velocity (baPWV), blood pressure, heart rate, and plasma nitric oxide (NO) concentration were measured before and 30 minutes after both SMR and CON trials. The participants performed SMR of the adductor, hamstrings, quadriceps, iliotibial band, and trapezius. Pressure was self-adjusted during myofascial release by applying body weight to the roller and using the hands and feet to offset weight as required. The roller was placed under the target tissue area, and the body was moved back and forth across the roller. In the CON trial, SMR was not performed. The baPWV significantly decreased (from  $1,202 \pm 105$  to  $1,074 \pm 110$   $\text{cm} \cdot \text{s}^{-1}$ ) and the plasma NO concentration significantly increased (from  $20.4 \pm 6.9$  to  $34.4 \pm 17.2$   $\mu\text{mol} \cdot \text{L}^{-1}$ ) after SMR using a foam roller (both  $p < 0.05$ ), but neither significantly differed after CON trials. These results indicate that SMR using a foam roller reduces arterial stiffness and improves vascular endothelial function.

**KEY WORDS** fascia, arterial stiffness, pulse wave velocity, vascular endothelial function, nitric oxide

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## INTRODUCTION

Stiffer arteries determined by pulse wave velocity (PWV) are associated with increased risk for a first cardiovascular event (18). Increases in arterial stiffness impair arterial buffering function and contribute to elevation of systolic blood pressure and/or left ventricular hypertrophy (1,11). Therefore, an increase in arterial stiffness should be prevented.

The myofascial system is a protective 3-dimensional web matrix of connective tissue that envelops all muscles, organs, glands, and cells in the body and surrounds the circulatory, nervous, and musculoskeletal systems and the digestive tract (24). Each of the 12 fascia or connective tissues comprises various concentrations of collagen and/or elastin (12). Collagen provides support, shape, and stability, and elastin allows for flexibility. Myofascial release is used to treat myofascial restrictions and restore muscles, tendons, ligaments, fascia, and/or soft-tissue extensibility (23). Such release not only stretches muscles and tendons but can also relax soft-tissue adhesions and scar tissue, which might confer benefits similar to those of stretching or massage. Self-myofascial release (SMR) using a foam roller is a relatively simple technique that can be easily applied to release tension in muscles, tendons, fascia, and/or soft tissues and acutely enhance the range of motion of the knee joint without a concomitant deficit in muscle performance (15). Therefore, this technique has become popular among athletes.

A study has suggested that flexibility might predict arterial stiffening that is independent of other fitness components (32). Both arterial stiffness and flexibility might be determined by a similar structural composition to that of muscles or connective tissues (i.e., elastin-collagen) (19). Moreover, individuals who practice yoga have significantly less arterial stiffness than those who are sedentary (7). Thus, flexibility exercises such as stretching or yoga might reduce arterial stiffness and thus that SMR might serve as an alternative method of reducing arterial stiffness.

The stiffness of large elastic muscular arteries is influenced by vascular endothelial function (30). Vascular endothelial cells play an important role in the regulation of vascular activity by producing vasoactive substances such as nitric oxide (NO) (25). However, the effect of SMR using a foam

roller on arterial stiffness and vascular endothelial function remains unknown.

This study investigates the acute effect of SMR using a foam roller on arterial stiffness and vascular endothelial function. We hypothesized that SMR using a foam roller would decrease PWV and increase the plasma NO concentration.

## METHODS

### Experimental Approach to the Problem

This study examined the acute effects of SMR on arterial function. We tested the hypothesis that SMR using a foam roller reduces arterial stiffness and improves vascular endothelial function. Individuals were randomly assigned to either SMR or control (CON) group and participated in each trial in pairs. Self-myofascial release and CON trials proceeded on separate days at an interval of 3 days in random order. Brachial-ankle PWV (baPWV), an index of arterial stiffness, and plasma NO concentration were measured before and 30 minutes after both trials.

### Subjects

The 10 healthy individuals who participated in this study comprised 7 men and 3 women (age,  $19.9 \pm 0.3$  y; height,  $162.7 \pm 8.1$  cm; weight,  $60.6 \pm 11.2$  kg, mean  $\pm$  SD). All of them were normotensive (140/90 mm Hg), with no signs, symptoms, or history of overt chronic diseases. Although PWV is not affected by the menstrual cycle (31), all female participants were studied during the early follicular phase of the cycle to avoid any hormonal influences. None of the female participants were taking oral contraceptives. Although some of the participants had regularly exercised at some time, most had not exercised for over 1 year, and their activity levels were thus considered essentially identical. Moreover, none of them had previously applied any form of SMR or associated exercise. All participants were fully informed about the experimental procedures and the purpose of the study, and all provided written informed consent before participating. This study was approved by the Ethical Committee of Kinki Welfare University and proceeded in accordance with the guidelines for human experimentation published by our Institutional Review Board.

### Brachial-Ankle Pulse Wave Velocity

The participants abstained from caffeine and intense physical activity, including exercise, for 24 hours, and they fasted for at least 4 hours before being tested. Moderate water intake was permitted. All participants slept for 7–8 hours on the night before measurements and arose at least 6 hours before starting the study. Brachial-ankle PWV was measured between 1:00 and 4:00 PM. After resting supine for at least 30 minutes in a quiet and temperature-controlled room ( $25^\circ$  C), baPWV was measured using an automatic oscillometric device (form PWV/ABI; Omron-Colin Co., Ltd., Komaki, Japan) (26). Briefly, baPWV was measured using sensory cuffs wrapped around both cubital fossae and ankles. The cuffs were connected to a plethysmographic sensor to

determine volume pulse form and to an oscillometric sensor to measure blood pressure. The pulse volume waveforms were recorded using a semiconductor pressure sensor, with the sample acquisition frequency for PWV set at 1,200 Hz. Volume waveforms for the brachial and ankle pulses were stored for 10 seconds with automatic gain analysis and quality adjustment.

The interval between the wave front of the brachial waveform and that of the ankle waveform was defined as the time between the brachial region (cubital fossa) and ankle (Tba) (27). The distance between the sampling points of the baPWV was calculated according to the height of each participant. The length of the path from the suprasternal notch to the measuring point in the brachial region (Lb) was determined and is expressed as:  $Lb = 0.2195 \times \text{height of participant (cm)} - 2.0734$ . The path length from the suprasternal notch to the ankle (La) was determined from superficial measurements and is expressed as:  $La = (0.8129 \times \text{height of the subject (cm)}) + 12.328$ . The distance between the 2 recording sites for baPWV was calculated based on the height of the individual and anthropomorphic data for the Japanese population (27). We calculated baPWV as:  $baPWV = (La - Lb / Tba)$ .

### Plasma Nitric Oxide Concentration

Plasma was collected from the ulnar vein of each participant and converted to nitrite using nitrate reductase. Levels of plasma NO were then measured in triplicate using the Griess reaction (8). Briefly, 80  $\mu$ l of each sample was incubated for 60 minutes at  $25^\circ$  C in 270  $\mu$ l containing 140  $\mu$ l of 125  $\text{mmol} \cdot \text{L}^{-1}$  KPi (pH, 7.5), 10  $\mu$ l of 87.5  $\mu\text{mol} \cdot \text{L}^{-1}$  of flavin adenine dinucleotide (Sigma, St. Louis, MO, USA), 10  $\mu$ l of 3.5  $\text{mmol} \cdot \text{L}^{-1}$  NADPH, 90  $\mu$ l of demineralized distilled water, and 20  $\mu$ l of nitrate reductase (1.75 U  $\cdot$  mL $^{-1}$ ; Sigma). Plasma samples were diluted fourfold with distilled water and deproteinized with 5% (v/v) zinc sulfate (300  $\text{g} \cdot \text{L}^{-1}$ ) to yield a final concentration of 15  $\text{g} \cdot \text{L}^{-1}$ . After centrifugation at 10,000g for 5 minutes at room temperature (or 1,000g for 15 minutes), 100  $\mu$ l of supernatant was applied to microtiter plate wells, followed by 100  $\mu$ l of the Griess reagent (1  $\text{g} \cdot \text{L}^{-1}$  sulfanilamide, 25  $\text{g} \cdot \text{L}^{-1}$  phosphoric acid, and 0.1  $\text{g} \cdot \text{L}^{-1}$  N-1-naphthyl-ethylenediamine). After observing 10 mm of color development at room temperature, absorbance was measured using an  $E_{\text{max}}$  (Molecular Devices, Sunnyvale, CA, USA) at a wavelength of 550 nm.

### Self-Myofascial Release

The participants received instruction about using the SMR technique with a 15  $\times$  91-cm (diameter  $\times$  length) uniform polystyrene roller (LPN Co., Ltd., Nagoya, Japan). The upper and lower extremities and the trunk were moved across the roller when pressure (direct force) was directed at the lower sacrum, mid thoracic spine, and posterior head. The participant dynamically stretched the upper and lower extremities and trunk over a turning roller as a warm-up. We examined the effects of this technique on the adductors, hamstrings,

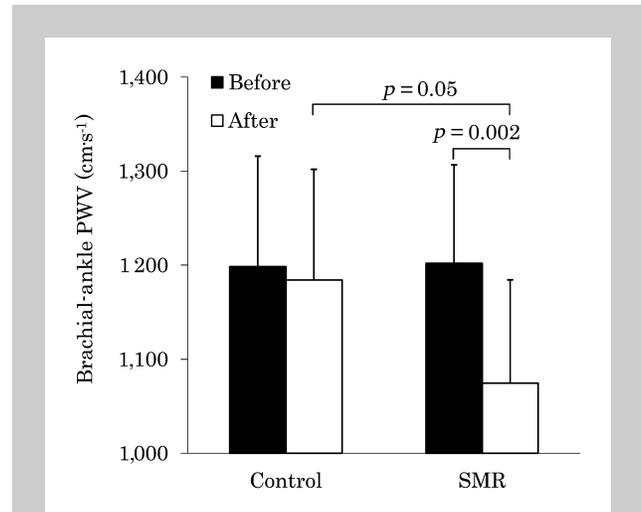
quadriceps, iliotibial band, and upper back including trapezius (23). Pressure was adjusted by applying body weight to the roller and using the hands and feet to offset weight as required. The roller was placed under the target tissue area, and the body was moved back and forth across the roller. Briefly, to accomplish SMR of the adductor, the thigh is extended and the roller is placed in the groin region with body prone on the floor. For SMR of the hamstrings, the lower extremities are extended, and the roller is placed on the hamstrings with the hips unsupported. For SMR of the quadriceps, the thigh is extended and the roller is placed on the quadriceps with the body prone on the floor. For SMR of the iliotibial band, the roller is placed on the iliotibial band with the body lateral on the floor. For SMR of the upper back, the hands are placed behind the head and the roller is positioned on the trapezius with the hips unsupported. The head is maintained in a neutral position with the ears and shoulders aligned. The bottom leg is raised slightly off floor. The hips are raised until they are unsupported and the head is stabilized in the neutral position. The SMR proceeded in the order of adductors, hamstrings, quadriceps, iliotibial band, and trapezius. Each participant practiced 2 or 3 times to learn the correct foam rolling technique with the guidance of a trainer and performed 20 SMR repetitions on each muscle group at 1-minute intervals. In the CON trial, participants rested supine in a quiet temperature-controlled room. Both trials began around the same time of day to minimize possible diurnal changes in the dependent variables. All sessions were completed within about 15 minutes. A trainer supervised the SMR trials and provided feedback to the participants to ensure that correct SMR technique was applied.

**Statistical Analyses**

All data are expressed as mean ± SD. Statistical analyses were performed using Statistica software (SPSS ver.19, Chicago, IL, USA). Data were analyzed by 2-way analysis of variance (ANOVA) (time × intervention) with repeated measures. When a significant interaction was observed, group differences were assessed by student's *t*-tests for paired values. Measures were considered statistically significant if  $p \leq 0.05$ . Relative effect sizes for performance data were calculated using Cohen's *d* and are defined as small ( $d = 0.2$ ), medium ( $d = 0.5$ ), or large ( $d = 0.8$ ).

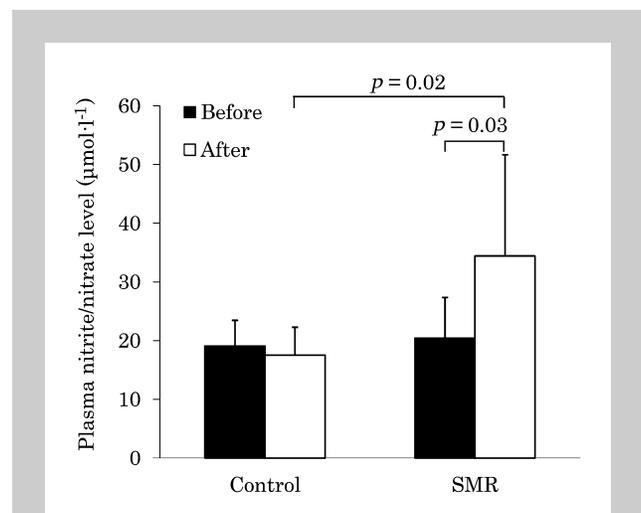
**RESULTS**

Figure 1 shows changes in baPWV before and after both trials. The 2-way ANOVA repeated measures test revealed a significant interaction effect of 2 trials ( $F = 4.12, p = 0.05$ ). Brachial-ankle pulse wave velocity significantly decreased after SMR (from  $1202 \pm 105$  to  $1073 \pm 106 \text{ cm} \cdot \text{s}^{-1}$ ,  $T = 4.48, p = 0.002$ , Cohen's  $d = 1.01$ ). The baPWV did not significantly differ before and after the CON trial (from  $1198 \pm 118$  to  $1184 \pm 105 \text{ cm} \cdot \text{s}^{-1}$ ,  $T = 0.83, p = 0.43$ , Cohen's  $d = 0.12$ ). The baPWV significantly decreased after SMR compared with the CON trial ( $1073 \pm 106$  vs.  $1184 \pm 105 \text{ cm} \cdot \text{s}^{-1}$ ,  $T = 2.28, p = 0.05$ , Cohen's  $d = 0.95$ ).



**Figure 1.** Changes in brachial-ankle pulse wave velocity (PWV) before and after control and self-myofascial release (SMR) trials. Values are mean ± SD. *N* = 10.

Figure 2 shows changes in plasma NO concentration before and after both trials. The 2-way ANOVA repeated measures test revealed a significant interaction effect of 2 trials ( $F = 6.25, p = 0.017$ ). Plasma NO concentration significantly increased after SMR (from  $20.4 \pm 6.9$  to  $34.4 \pm 17.2 \mu\text{mol} \cdot \text{L}^{-1}$ ,  $T = 2.56, p = 0.03$ , Cohen's  $d = 0.95$ ). Plasma NO concentrations did not significantly differ before and after the CON trial (from  $19.1 \pm 4.3$  to  $17.5 \pm 4.7 \mu\text{mol} \cdot \text{L}^{-1}$ ,  $T = 1.14, p = 0.29$ , Cohen's  $d = 0.35$ ). Plasma NO concentration significantly increased after SMR in the SMR compared with the CON trial ( $34.4 \pm 17.2$  vs.  $17.5 \pm 4.7 \mu\text{mol} \cdot \text{L}^{-1}$ ,  $T = 2.77, p = 0.02$ , Cohen's  $d = 1.05$ ).



**Figure 2.** Changes in plasma nitrite/nitrate level before and after control and self-myofascial release (SMR) using a foam roller trials. Values are mean ± SD. *N* = 10.

## DISCUSSION

This first study on the effects of SMR using a foam roller on arterial stiffness discovered that baPWV acutely decreased and that the plasma NO concentration significantly increased. These findings suggest that SMR using a foam roller exerts a favorable effect on arterial function.

Some studies have shown that myofascial release can improve the flexibility of muscles, tendons, ligaments, and fascia by releasing tension in tight muscles or fascia (9,10) while increasing blood flow and circulation to the soft tissues, which in turn improve flexibility and range of motion (15,24). Mechanical stress caused by flexibility training can affect hemodynamic responses (21). Stretched muscle fibers activate mechanoreceptors, which elicit cardiovascular adjustments through parasympathetic withdrawal and sympathetic activation (6). Therefore, because SMR using a foam roller improves flexibility by releasing tension in muscles or fascia, it might also help modify arterial stiffening.

The compressed and isolated area in contact with the roller suggests a potential benefit of SMR (5). Changes in arterial stiffness might be because of mechanical/load-bearing properties of vessel walls such as elastin/collagen recruitment (2). Smooth muscle in the arterial wall is in series with collagen and both are in parallel with elastin (24). Muscular fascia is connective tissue comprising collagen and elastin. When stress is released from collagen and transferred to the more distensible elastin, strain on the smooth muscle is released. In addition, with a reduction in smooth muscle tension, stress is transposed from collagen to the elastic lamellae rendering the vessel wall more flexible (20). Reduced arterial stiffness might be almost exclusively attributable to collagen and elastin in the arterial wall. Under very low pressure or stress, the elastic modulus of the arterial wall is equal to the elastic modulus of elastin because little or no collagen bears stress under this condition (22). Therefore, we speculate that the release of myofascial strain by SMR using a foam roller reduces arterial stiffness.

The physiological implication of reduced arterial stiffness is important. However, the mechanisms responsible for the reduction in arterial stiffness after myofascial release are unclear. Stiffness in large elastic and muscular arteries is influenced by vascular endothelial function (29). Therefore, 1 potential mechanism for reduced arterial stiffness might be enhanced endothelial function. Vascular endothelial cells play an important role in the regulation of vascular activity by producing vasoactive substances such as NO. We found a significantly increased plasma NO concentration after SMR and others have suggested that NO participates in the regulation of arterial stiffness (25,30). Changes that occur in endothelial function during SMR might therefore provide a stimulus for both acute and chronic changes in vascular function.

Mechanical stimuli, such as compression of the arterial muscle, induce arterial vasodilation, the magnitude of which is not affected by increasing the duration of compression, but

it is enhanced by increasing the number of compressions (4). Compression might distort the vascular endothelium, which could trigger the release of vasodilator substances such as NO (28). External leg compression causes elevated shear stress in the walls of the underlying vasculature through increasing flow velocity in the deep veins of the extremities (17). Shear stress on endothelial cells is a potent stimulus for NO production. Rapid cuff inflation might increase shear stress on the vascular wall, which stimulates the endothelial release of NO (13,14). The participants repeatedly performed SMR using a foam roller in addition to external compression in this study. Therefore, external compression might be a major pathway of vasodilation induced by the increased release of NO. Furthermore, consequential changes in vasodilator function persist for several weeks (16), which might decrease baseline levels of arterial stiffness. These results support the notion that this mechanism contributes to reducing arterial stiffness in relaxed skeletal muscle. However, the precise mechanisms responsible for the changes in arterial function induced by SMR using a foam roller remain unknown, and further studies are required.

Several important limitations to this study should be emphasized. Because the participants were healthy young adults, the findings may not be generalized to older adults or athletes. Further studies are warranted to determine the effects of SMR using foam roller on arterial function in older adults and/or athletes. Moreover, the sample size was small but similar to those used in the previous studies of post-exercise PWV or NO (25,30). Furthermore, we measured baPWV, which reflects changes in the stiffness of both elastic and peripheral muscular arteries. Carotid-femoral PWV (cfPWV) is an established method for measuring PWV (3), but measurements involving the femoral artery require attaching a transducer to the inguinal region. This can have a powerful psychological impact on patients, which was considered to be an issue, particularly because the PWV investigator in this study was a male. As PWV is closely determined by blood pressure level per se, the psychological pressor effect might increase cfPWV. In contrast, measurement of baPWV minimizes psychological stress by simply using exposed extremities. Recently, baPWV, a noninvasive measurement of PWV, has been closely associated with aortic PWV and cfPWV (26).

In conclusion, the present findings indicated that SMR using a foam roller reduces arterial stiffness and improves vascular endothelial function. These results imply that this technique exerts a favorable effect on arterial function. We believe that SMR using a foam roller can promote the cardiovascular health of the general population. The present results require prospective confirmation in an intervention study.

## PRACTICAL APPLICATIONS

This is the first study to examine the effects of SMR using a foam roller on arterial function. We found that PWV

decreases and plasma NO concentration increases after SMR in healthy young adults. The present findings extend the beneficial influence of SMR to arterial function in this population. The present findings suggest that 1 bout of SMR confers many favorable cardiovascular benefits. Moreover, because 1 bout of SMR reduces PWV and increases plasma NO concentration, repeated long-term SMR might decrease baseline arterial stiffness. Therefore, SMR could be included in exercise programs to promote health.

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#### REFERENCES

1. Abhayaratna, WP, Barnes, ME, O'Rourke, MF, Gersh, BJ, Seward, JB, Miyasaka, Y, Bailey, KR, and Tsang, TS. Arterial stiffness to left ventricular diastolic function and cardiovascular risk prediction in patients  $\geq 65$  years of age. *Am J Cardiol* 98: 1387–1392, 2006.
2. Bank, AJ, Wilson, RF, Kubo, SH, Holte, JE, Dresing, TJ, and Wang, H. Direct effects of smooth muscle relaxation and contraction on in vivo human brachial artery elastic properties. *Circ Res* 77: 1008–1016, 1995.
3. Blacher, J, Guerin, AP, Pannier, B, Marchais, SJ, Safar, ME, and London, GM. Impact of aortic stiffness on survival in end-stage renal disease. *Circulation* 99: 2434–2439, 1999.
4. Clifford, PS, Kluess, HA, Hamann, JJ, Buckwalter, JB, and Jasperse, JL. Mechanical compression elicits vasodilatation in rat skeletal muscle feed arteries. *J Physiol* 572: 561–567, 2006.
5. Curran, PF, Fiore, RD, and Crisco, JJ. A comparison of the pressure exerted on soft tissue by 2 myofascial rollers. *J Sport Rehabil* 17: 432–442, 2008.
6. Drew, RC, Bell, MP, and White, MJ. Modulation of spontaneous baroreflex control of heart rate and indexes of vagal tone by passive calf muscle stretch during graded metaboreflex activation in humans. *J Appl Physiol* 104: 716–723, 2008.
7. Duren, CM, Cress, ME, and McCully, KK. The influence of physical activity and yoga on central arterial stiffness. *Dyn Med* 7: 2, 2008.
8. Green, LC, Wagner, DA, Glogowski, J, Skipper, PL, Wishnok, JS, and Tannenbaum, SR. Analysis of nitrate, nitrite, and [15N]nitrate in biological fluids. *Anal Biochem* 126: 131–138, 1982.
9. Hanten, WP, Olson, SL, Butts, NL, and Nowicki, AL. Effectiveness of a home program of ischemic pressure followed by sustained stretch for treatment of myofascial trigger points. *Phys Ther* 80: 997–1003, 2000.
10. Hou, CR, Tsai, LC, Cheng, KF, Chung, KC, and Hong, CZ. Immediate effects of various physical therapeutic modalities on cervical myofascial pain and trigger-point sensitivity. *Arch Phys Med Rehabil* 83: 1406–1414, 2002.
11. Kingwell, BA and Gatzka, CD. Arterial stiffness and prediction of cardiovascular risk. *J Hypertens* 20: 2337–2340, 2002.
12. Langevin, HM and Huijing, PA. Communicating about fascia: History, pitfalls, and recommendations. *Int J Ther Massage Bodywork* 2: 3–8, 2009.
13. Liu, K, Chen, LE, Seaber, AV, Johnson, GW, and Urbaniak, JR. Intermittent pneumatic compression of legs increases microcirculation in distant skeletal muscle. *J Orthop Res* 17: 88–95, 1999.
14. Liu, K, Chen, LE, Seaber, AV, and Urbaniak, JR. Influences of inflation rate and duration on vasodilatory effect by intermittent pneumatic compression in distant skeletal muscle. *J Orthop Res* 17: 415–420, 1999.
15. Macdonald, G, Penney, M, Mullaley, M, Cuconato, A, Drake, C, Behm, DG, and Button, DC. An acute bout of self myofascial release increases range of motion without a subsequent decrease in muscle activation or force. *J Strength Cond Res* 27: 812–821, 2013.
16. Maiorana, A, O'Driscoll, G, Taylor, R, and Green, D. Exercise and the nitric oxide vasodilator system. *Sports Med* 33: 1013–1035, 2003.
17. Mayrovitz, HN and Larsen, PB. Effects of compression bandaging on leg pulsatile blood flow. *Clin Physiol* 17: 105–117, 1997.
18. Mitchell, GF, Hwang, SJ, Vasani, RS, Larson, MG, Pencina, MJ, Hamburg, NM, Vita, JA, Levy, D, and Benjamin, EJ. Arterial stiffness and cardiovascular events: The Framingham Heart Study. *Circulation* 121: 505–511, 2010.
19. Nichols, W and O'Rourke, M. *McDonald's Blood Flow in Arteries* (5th ed.). London, United Kingdom: Hodder Arnold, 2005.
20. Nichols, WW and Edwards, DG. Arterial elastance and wave reflection augmentation of systolic blood pressure: deleterious effects and implications for therapy. *J Cardiovasc Pharmacol Ther* 6: 5–21, 2001.
21. Rassier, DE, MacIntosh, BR, and Herzog, W. Length dependence of active force production in skeletal muscle. *J Appl Physiol* 86: 1445–1457, 1999.
22. Roach, MR and Burton, AC. The reason for the shape of the distensibility curves of arteries. *Can J Biochem Physiol* 35: 681–690, 1957.
23. Schlei, R. Fascial plasticity—a new neurobiological explanation: Part 1. *J Bodyw Mov Ther* 7: 11–19, 2003.
24. Schlei, R. Fascial plasticity—a new neurobiological explanation: Part 2. *J Bodyw Mov Ther* 7: 104–116, 2003.
25. Sugawara, J, Maeda, S, Otsuki, T, Tanabe, T, Ajisaka, R, and Matsuda, M. Effects of nitric oxide synthase inhibitor on decrease in peripheral arterial stiffness with acute low-intensity aerobic exercise. *Am J Physiol Heart Circ Physiol* 287: H2666–H2669, 2004.
26. Tanaka, H, Munakata, M, Kawano, Y, Ohishi, M, Shoji, T, Sugawara, J, Tomiyama, H, Yamashina, A, Yasuda, H, Sawayama, T, and Ozawa, T. Comparison between carotid-femoral and brachial-ankle pulse wave velocity as measures of arterial stiffness. *J Hypertens* 27: 2022–2027, 2009.
27. Tomiyama, H, Yamashina, A, Arai, T, Hirose, K, Koji, Y, Chikamori, T, Hori, S, Yamamoto, Y, Doba, N, and Hinojara, S. Influences of age and gender on results of noninvasive brachial-ankle pulse wave velocity measurement—a survey of 12517 subjects. *Atherosclerosis* 166: 303–309, 2003.
28. Tschakovsky, ME and Sheriff, DD. Immediate exercise hyperemia: contributions of the muscle pump vs. rapid vasodilation. *J Appl Physiol* 97: 739–747, 2004.
29. Wilkinson, IB, Franklin, SS, and Cockcroft, JR. Nitric oxide and the regulation of large artery stiffness: from physiology to pharmacology. *Hypertension* 44: 112–116, 2004.
30. Wilkinson, IB, MacCallum, H, Cockcroft, JR, and Webb, DJ. Inhibition of basal nitric oxide synthesis increases aortic augmentation index and pulse wave velocity in vivo. *Br J Clin Pharmacol* 53: 189–192, 2002.
31. Williams, MR, Westerman, RA, Kingwell, BA, Paige, J, Blombery, PA, Sudhir, K, and Komesaroff, PA. Variations in endothelial function and arterial compliance during the menstrual cycle. *J Clin Endocrinol Metab* 86: 5389–5395, 2001.
32. Yamamoto, K, Kawano, H, Gando, Y, Iemitsu, M, Murakami, H, Sanada, K, Tanimoto, M, Ohmori, Y, Higuchi, M, Tabata, I, and Miyachi, M. Poor trunk flexibility is associated with arterial stiffening. *Am J Physiol Heart Circ Physiol* 297: H1314–H1318, 2009.