Original Research

Title: Acute Effects of Self-Myofascial Release Using a Foam Roller on Arterial Function

Brief Running Head: Self-Myofascial Release and Arterial Function

Authors: Takanobu Okamoto¹, Mitsuhiko Masuhara², Komei Ikuta³

Affiliation
1 Institute of Exercise and Sport Physiology, Nippon Sport Science University, 7-1-1, Fukasawa, Setagaya-ku, Tokyo, 158-8508, Japan.
2 Institute of Exercise Physiology and Biochemistry, Osaka University of Health and Sport Sciences, 1-1 Asashirodai, Kumatori-cho, Sennan-gun, Osaka 590-0496, Japan.
3 Institute of Health and Child Sciences, Osaka Aoyama University, 2-11-1 Niina, Minoh, Osaka, 562-8580, Japan.

Corresponding author
Takanobu Okamoto
Institute of Exercise and Sport Physiology, Nippon Sport Science University, 7-1-1, Fukasawa, Setagaya-ku, Tokyo, 158-8508, Japan.
tel: +81-3-5706-0966 fax: +81-3-5706-0966 e-mail: tokamoto@nittai.ac.jp

Funding
This work was supported by Research Grants from the Japan Core Conditioning Associate, Japan.
Abstract

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. The present 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, blood pressure, heart rate and plasma nitric oxide concentration were measured before and 30 min after both SMR and CON trials. The participants performed SMR of the adductor, hamstrings, quadriceps, iliobial 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 brachial-ankle pulse wave velocity significantly decreased (from 1202 ± 105 to 1074 ± 110 cm/s) and the plasma nitric oxide concentration significantly increased (from 20.4 ± 6.9 to 34.4 ± 17.2 µmol/L) 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
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 three-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 as well as the digestive tract (23). 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 (24). 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 recent 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 (that is, elastin-collagen) (19). Moreover, individuals who practice yoga have significantly less arterial stiffness that 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.

The present 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) groups and participated in each trial in pairs. SMR and control (CON) trials proceeded on separate days at an interval of three days) in random order. Brachial-ankle PWV (baPWV), an index of arterial stiffness, and plasma NO concentration were measured before and 30 min after both trials.

Participants

The 10 healthy individuals who participated in this study comprised seven males and three females (age, 19.9 ± 0.3 y; height, 162.7 ± 8.1 cm; weight, 60.6 ± 11.2 kg, means ± 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 one 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 as well as 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.

baPWV

The participants abstained from caffeine and intense physical activity, including exercise, for 24 h, and they fasted for at least 4 h before being tested. Moderate water intake was permitted. All participants slept for seven to eight hours on the night before measurements and arose at least six hours before starting the study. Brachial-ankle PWV was measured between 1:00 and 4:00 PM. After resting supine for at least 30 min in a quiet and temperature-controlled room (25°C), baPWV was measured using an automatic oscillometric device (form PWV/ABI, Omron-Colin Co. Ltd., Komaki, Aichi, 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 s 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 x 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 two 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 NO 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 µL of each sample was incubated for 60 min at 25°C in 270 µL containing 140 µL of 125 mmol/L KPi (pH 7.5), 10 µL of 87.5 µmol/L FAD (Sigma, St. Louis, MO, USA), 10 µL of 3.5 mmol/L NADPH, 90 µL of DDW and 20 µL nitrate reductase (1.75 U/mL; Sigma). Plasma samples were diluted 4-fold with distilled water and deproteinized with 5% (v/v) zinc sulfate (300 g/L) to yield a final concentration of 15 g/L. After centrifugation at 10,000 × g for 5 min at room temperature (or 1,000 × g for 15 mm), 100 µL of supernatant was applied to microtiter plate wells, followed by 100 µL of the Griess reagent (1 g/L sulfanilamide, 25 g/L phosphoric acid, and 0.1 g/L N-1-naphthyl-ethylenediamine). After observing 10 mm of color development at room temperature, absorbance was measured using an Emax (Molecular Devices, Sunnyvale, California, USA) at a wavelength of 550 nm.

SMR

The participants received instruction about using the SMR technique with a 15 × 91-cm (diameter × length) uniform polystyrene roller (LPN Co. Ltd., Nagoya, Aichi, 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 (24). 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 two or three 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-min intervals. In the control 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 min. A trainer supervised the SMR trials and provided
feedback to the participants to ensure that correct SMR technique was applied.

Statistical analysis

All data are expressed as means ± SD. Statistical analyses were performed using Statistica software (SPSS ver.19, Chicago, IL, USA). Data were analyzed by two-way 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 ≤ 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 two-way ANOVA repeated measures test revealed a significant interaction effect of two trials (F = 4.12, P = 0.05). baPWV significantly decreased after SMR (from 1202 ± 105 to 1073 ± 106 cm/s, T = 4.48, P = 0.002, Cohen’s d = 1.01). The baPWV did not significantly differ before and after the control trial (from 1198 ± 118 to 1184 ± 105 cm/s, T = 0.83, P = 0.43, Cohen’s d = 0.12). The baPWV significantly decreased after SMR compared with the CON trial (1073 ± 106 vs. 1184 ± 105 cm/s, T = 2.28, P = 0.05, Cohen’s d = 0.95).

Figure 2 shows changes in plasma NO concentration before and after both trials. The two-way ANOVA repeated measures test revealed a significant interaction effect of two trials (F = 6.25, P = 0.017). Plasma NO concentration significantly increased after SMR (from 20.4 ± 6.9 to 34.4 ± 17.2 µmol/L, T = 2.56, P = 0.03, Cohen’s d = 0.95). Plasma NO concentrations did not significantly differ before and after the control trial (from 19.1 ± 4.3 to 17.5 ± 4.7 µmol/L, 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 ± 17.2 vs. 17.5 ± 4.7 µmol/L, T = 2.77, P = 0.02, Cohen’s d = 1.05).
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 upon 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 improves flexibility and range of motion (15, 23). Mechanical stress caused by flexibility training can affect hemodynamic responses (21). Stretched muscle fibres 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 due to 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 (23). 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, since 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, one 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 the present
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 the present 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 a foam roller on arterial function in older adults and/or athletes. Moreover, the sample size was small, but similar to those used in 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 favourable 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 one bout of SMR confers many favorable cardiovascular benefits. Moreover, because one 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.
References


8. Green LC, Wagner DA, Glogowski J, Skipper PL, Wishnok JS, and Tannenbaum


Acknowledgment

The funding source for this research was Japan Core Conditioning Associate. Foam roller was provided by LPN Co. Ltd, Nagoya, Aichi, Japan. There was no conflict of interest between LPN Co. Ltd and the researchers.

Figure Legends

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

Figure 2. Changes in plasma nitrite/nitrate level before and after control and self-myofascial release (SMR) using a foam roller trials. Values are means (SD). N=10.
Figure 1

Brachial anlkle PWV (cm/sec)

- Black: Before
- White: After

Control

SMR

P=0.05

P=0.002
Figure 2

Plasma nitrite/nitrate level (µmol/l)

- **Before**
- **After**

Control

SMR

P = 0.02

P = 0.03