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Original Article

# Influence of detraining on temporal changes in arterial stiffness in endurance athletes: a prospective study

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**Abstract.** [Purpose] We examined the effects of detraining on temporal changes in arterial stiffness in endurance athletes. [Subjects] Eighteen female university athletes requiring high endurance exercise capabilities were classified into 2 groups: 10 retired players (detraining group) and 8 active players (training group). [Methods] Brachial-ankle pulse wave velocity, an index of arterial stiffness, was measured a total of 6 times: immediately before retirement of the detraining group and at 1, 2, 3, 6, and 12 months after retirement. [Results] Brachial-ankle pulse wave velocity was measured in the training group at the same 6 points to allow comparison with the detraining group. The brachial-ankle pulse wave velocity in the detraining group increased significantly at 3 and 12 months as compared with that at 0 months and showed a significant increase at 12 months compared with that at 1 month. Moreover, the brachial-ankle pulse wave velocity in the detraining group was significantly higher at 3, 6, and 12 months than in the training group. [Conclusion] These results revealed that detraining may result in increased arterial stiffness from 3 months onward in endurance athletes.

**Key words:** Branchial-ankle pulse wave velocity, Blood pressure

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## INTRODUCTION

It is well known that heart and cerebrovascular diseases rank high among causes of death associated with arteriosclerosis progression in the Japanese population. Therefore, preventing the onset of arteriosclerosis or inhibiting its progression is key to reducing mortality rates. Pulse wave velocity (PWV) is a widely used index of arteriosclerosis. PWV represents the speed of an arterial wave caused by cardiac contraction and circulation of blood from the center to the periphery, reflecting arterial stiffness, a functional characteristic of arteriosclerosis[1)]. This noninvasive method was established by Bramwell and Hill in 1922, over 90 years ago[2)]. While carotid-femoral PWV (cfPWV) is the current gold standard, the development of devices that can easily measure brachial-ankle PWV (baPWV) and cardio-ankle vascular index (CAVI) with high reproducibility has resulted in renewed interest concerning PWV measurement in clinical research.

Although genetic factors are involved in arteriosclerosis, it is also known to be greatly affected by lifestyle habits such as lack of exercise. Thus, there are many reports on the relationship between arterial stiffness and exercise. For example, aerobic exercise reduces arterial stiffness, and the PWV is low in individuals who habitually engage in aerobic exercise as compared with those who do not[3)]. Furthermore, arterial stiffness is reportedly low in endurance athletes who continue to perform advanced aerobic exercise as compared with healthy people in general[4)]. Arterial stiffness improvement is observed after an aerobic exercise intervention with a relatively short period of just 8 weeks[5)], and high efficacy is observed at an exercise frequency of 2 to 5 days a week[5–9)]. Meanwhile, the effect of muscle training on arterial stiffness has also been studied. According to these studies, arterial stiffness was higher in muscle strength-trained athletes than in untrained subjects[10)]; high-intensity muscle training increased arterial stiffness, medium-intensity muscle training did not[11, 12)], and low-intensity muscle training reduced arterial stiffness[13)].

Thus, the types of sports, as well as their optimal durations and frequencies, needed to reduce arterial stiffness have been clarified. However, the duration of persistence of such arterial stiffness improvement has not been elucidated. In this study, we examined the effects of detraining on temporal changes in arterial stiffness in endurance athletes.

## SUBJECTS AND METHODS

### *Subjects*

Eighteen female university athletes requiring high-endurance exercise capabilities were included in this study. We examined these 18 subjects by classifying them into 2

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groups: 10 retired players (detraining group; DeTr group) and 8 active players (training group; Tr group). The subjects exercised for 2.5 hours a day 4 to 5 times a week. In previous studies, female university field hockey players had $O_2$ max values of 42–52 mL·kg$^{-1}$·min$^{-1}$, which were markedly higher than the average $O_2$ max in young women[14–16]. Past research demonstrates that field hockey requires a substantial amount of cardiovascular fitness[17]. None of the subjects in the present study smoked or habitually drank alcohol, and none had a family history of coronary heart disease or its risk factors. None of the females were taking oral contraceptives or undergoing hormone replacement therapy. In addition, no subjects in the DeTr group began new exercise habits after retirement. The objective, methods, and safeness of the study were explained to the subjects in writing or verbally, and each participant submitted an informed consent form after obtaining a full understanding of the study contents. The study was approved by the ethics review board of Osaka University of Health and Sport Sciences. Table 1 presents the physical profiles of the subjects at the start of the experiment. Owing to the presence of a detraining period of several months before entering the university from high school, the sports history after university entrance is indicated.

**Table 1.** Subjects' characteristics

| | DeTr | Tr |
|---|---|---|
| Age (years) | 21.7±0.2 * | 20.0±0.3 |
| Sport career (months) | 42.7±0.3 * | 19.0±3.2 |
| Height (cm) | 156.8±1.5 | 158.9±1.5 |
| Body mass (kg) | 53.3±2.2 | 54.0±1.1 |
| Body mass index (kg/m$^2$) | 21.6±0.7 | 21.5±0.3 |
| Body fat (%) | 22.7±1.5 | 24.4±1.2 |

Values are shown as the mean ± SE.
*p<0.05 for the difference between DeTr and Tr
DeTr: detraining group; Tr: training group

*Methods*

Arterial stiffness was measured at 6 time points: immediately before retirement of the DeTr group (0 M) and at 1 (1 M), 2 (2 M), 3 (3 M), 6 (6 M), and 12 months (12 M) after retirement. In order to examine the effects of detraining, arterial stiffness was measured at the same 6 time points in the Tr group, which served as the control and continued training as usual. Arterial stiffness was measured approximately 3 hours after breakfast; subjects were prohibited from performing strenuous exercises within 12 hours before measurement and from consuming caffeine after waking up. Arterial stiffness was measured in a room with the temperature controlled at approximately 24 °C after the subjects had rested for at least 5 minutes[18, 19].

Systolic blood pressure (SBP) and diastolic blood pressure (DBP) levels were measured at the left and right brachial arteries by the oscillometric method using form PWV/ABI® (Omron Colin Co., Ltd., Komaki, Japan), and the mean left and right brachial artery blood pressures were adopted as the representative level.

Arterial stiffness was measured by baPWV, which was determined using form PWV/ABI® (Komaki, Japan). With baPWV, artery stiffness can be measured noninvasively; a cuff for blood pressure measurement is used to detect pulse waves at both forearms and ankle joints, and the PWV is obtained based on the time difference in the pulse wave rise point, from pulse waves at the forearms and ankle joints, and the blood vessel length (the distance obtained by subtracting the blood vessel length from the valvular ostium to the ankle joints and forearms). Because baPWV corresponds to the PWV between the brachial and peroneal arteries, a muscular artery is measured instead of elastic arteries such as the aorta; however, as the value obtained for the muscular artery shows a high correlation with the PWV of the aorta by the catheter method and has favorable reproducibility, it is widely applied clinically as the index of arteriosclerosis in Japan[1]. The mean of the left and right baPWVs was used as the representative value[20]. In previous research, the within- and between-observer coefficients of brachial ankle PWV variation were 6.5% ± 4.1% and 3.6% ± 3.9%, respectively[21].

All results of this study are presented as means with standard errors. The baPWV and blood pressure levels were compared using two-factor analysis of variance (group × time). For statistical calculations, IBM SPSS Statistics (Version 21.0, IBM Japan, Tokyo, Japan) was used, and a p value of less than 5% was considered to indicate a statistically significant difference.

## RESULTS

No interaction between groups and time was observed for SBP. Table 2 presents the changes in DBP in both the DeTr and Tr groups and at the 6 time points from 0 M to 12 M. An interaction between group and time was observed for DBP ($p < 0.05$), which showed a significant increase at 12 M as compared with that at 0 M in the DeTr group ($p < 0.05$). In the intergroup comparison, the diastolic pressures at 6 M and 12 M were significantly higher in the DeTr group than in the Tr group ($p < 0.05$).

Table 3 presents the changes in arterial stiffness in both the DeTr and Tr groups and at the 6 time points from 0 M to 12 M. An interaction was observed in baPWV between group and time ($p < 0.05$), showing significant increases at 3 M and 12 M compared with 0 M in the DeTr group ($p < 0.05$). Moreover, in the intergroup comparison, the DeTr group had significantly higher levels at 3 M, 6 M, and 12 M than in the Tr group ($p < 0.05$).

## DISCUSSION

We examined temporal changes in arterial stiffness with 12 months of detraining in endurance athletes. Although several past reports have examined the effects of detraining after relatively short exercise interventions[5, 22–24], no report has examined changes in arterial stiffness caused by detraining following continuous long-term exercise as in this study. A prior study, which included an 8- to 12-week exercise intervention, showed that the arterial stiffness value returned to the pre-exercise level after approximately 4 weeks of detraining. Although the value before continuous exercise was

**Table 2.** Changes in DBP

| | 0 M | 1 M | 2 M | 3 M | 6 M | 12 M |
|---|---|---|---|---|---|---|
| DeTr | 56.1±1.6 | 58.3±1.6 | 63.2±1.9 | 58.9±1.5 | 61.1±2.1 † | 65.4±1.5 *§† |
| Tr | 58.8±1.2 | 57.1±1.4 | 58.6±1.9 | 57.2±1.4 | 55.8±1.0 | 60.0±2.1 |

Values are shown as the mean ± SE.
*significantly higher than 0 M ($p<0.05$). §significantly higher than 1 M ($p<0.05$). †significantly higher than the Tr group ($p<0.05$).
DBP: diastolic blood pressure (mmHg); DeTr: detraining group; Tr: training group

**Table 3.** Changes in baPWV

| | 0 M | 1 M | 2 M | 3 M | 6 M | 12 M |
|---|---|---|---|---|---|---|
| DeTr | 1002.3±23.1 | 1028.3±29.3 | 1081.2±32.1 | 1085.0±24.1 *† | 1059.5±30.7 † | 1107.7±29.2 *§† |
| Tr | 966.0±25.8 | 964.4±21.7 | 993.6±34.3 | 965.1±12.6 | 913.8±38.5 | 1007.2±27.4 |

Values are shown as the mean ± SE.
*significantly higher than 0 M ($p<0.05$). §significantly higher than 1 M ($p<0.05$). †significantly higher than the Tr group ($p<0.05$).
baPWV: brachial-ankle pulse wave velocity (cm/sec); DeTr: detraining group; Tr: training group

not measured in the study subjects, baPWV increased after 3 months (12 weeks) of detraining as compared with the value after continuous exercise, suggesting that the effects of exercise on arterial stiffness might be more sustained as the period of continued exercise is increased. This concept is supported by a previous study conducted by Otsuki et al. that compared arterial stiffness in endurance athletes based on years of exercise continuation and showed that arterial stiffness levels were lower in athletes who continued exercise for a longer duration[4]. It was assumed that the effects of exercise on arterial stiffness would be sustained longer as the duration of exercise increased. The factors most affecting baPWV, an index of arterial stiffness, were reported to be age, gender, and blood pressure[25]. Based on its measurement principles, baPWV is considered highly dependent on blood pressure. In the study subjects, DBP increased significantly after detraining. However, a significant increase in DBP was not observed until 6 months of detraining, whereas a significant increase in baPWV was observed at 3 months of detraining.

Next, we attempted to determine the mechanism by which baPWV increased in the DeTr group. Increased baPWV, i.e., an increase in arterial stiffness, is mainly attributable to organic factors associated with changes in the elastin and calcium contents of the arterial wall and functional factors associated with autonomic nerves and vascular endothelial function. Considering that baPWV increased in the relatively short period of just 3 months, it is highly likely that increased arterial stiffness is a change associated with functional factors. It is assumed that arterial stiffness would be particularly affected by the presence of vasoactive substances such as the vasorelaxant nitric oxide (NO) and the vasoconstrictor endothelin-1 (ET-1), both of which have key vascular endothelial functions. These vasoactive substances are reportedly increased and decreased (NO and ET-1, respectively) by exercise; furthermore, blood NO levels increased and blood ET-1 levels decreased after an 8-week aerobic exercise intervention, returning to the pre-exercise levels after detraining for 8 weeks[26]. It has also been reported that blood NO levels are high in young atheletes[27, 28]. Animal studies have shown that administration of NO synthetic inhibitors increases PWV in the common iliac artery without accompanying changes in mean blood pressure (MBP)[29], while administration of ET-1 receptor antagonists decreases PWV in the common iliac artery without accompanying changes in MBP[30]. Presumably, an increase in baPWV due to detraining may be the result of reduced NO production and increased ET-1 production.

An increase in baPWV was observed earlier than an increase in DBP, suggesting that baPWV is a useful vascular index for understanding the effects of detraining at an early stage.

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