Arterial wall elasticity and various physical activities

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Abstract

Most physical activities contribute to improvement of age-associated arterial stiffening, such as reductions in arterial compliance or increases in arterial stiffness. From the viewpoint of exercise physiology, exercise can be divided into aerobic and resistance exercises. Although it is widely accepted that arterial compliance or stiffness is improved by habitual aerobic exercise, it has been reported that resistance training decreases arterial compliance and increases arterial stiffness. Interestingly, combined aerobic and resistance exercise did not affect arterial compliance, which suggests that simultaneously performed aerobic training may negate and prevent the reduction in arterial compliance induced by resistance exercise. It seems that not only moderate-high intensity physical activity but also daily light physical activity, such as cooking, cleaning, washing clothes, or deskwork, decreases arterial stiffness. Arterial wall viscosity reflects dissipation of energy during conversion of cardiac pulsatile energy into arterial elastic energy, which is related to sympathoexcitation and intima-media thickening in the carotid artery. In addition, arterial wall viscosity increases with advancing age, and this age-associated increase in wall viscosity is attenuated in men with high cardiorespiratory fitness. Therefore, high volume physical activity or habitual aerobic exercise negates and improves arterial stiffening with age or resistance training.

Introduction

People usually perform various kinds of physical activity, such as standing, walking, running, housework, or leisure activity, etc. "Exercise and Physical Activity Reference for Health Promotion 2006" and "Physical Activity Reference for Health Promotion 2013" were reported by the Ministry of Health, Labour and Welfare of Japan. These references define "Physical activity is as any bodily movement produced by skeletal muscles results in energy expenditure above resting energy expenditure" and "physical activity can be

divided into two; one is nonexercise physical activity (e.g. daily work, housework, commuting to office/school) and the other is exercise that is practiced intentionally for maintaining and improving one's physical fitness." Many studies have indicated favorable and unfavorable effects of various types of exercise on arterial compliance or stiffness. Recently, both exercise and physical activity have been the focus of attention, and the effects of various physical activities on arterial mechanical characteristics have been reported. This review presents findings regarding the effects of type and intensity of exercise and physical activity on arterial stiffness or compliance, and proposes effective physical activity for preventing and improving age-associated arterial stiffening.

Arterial stiffening with advancing age

Osler said that "man is as old as his arteries." Many cross-sectional studies have demonstrated that large artery stiffness becomes progressively greater (compliance is lower) with aging even in the healthy population regardless of sex [1-3]. There are 40%-50% differences in stiffness and compliance in large elastic arteries between 25 and 75 years in healthy adults without clinical disease or major coronary risk factors [4, 5]. Moreover, lumen diameter and intima-media thickness in large elastic arteries increase with advancing age [6] (Fig. 1). This morphological adaptation in large elastic arteries is thought to contribute to age-associated arterial stiffening. In contrast to large elastic arteries, peripheral arteries do not obviously stiffen with aging in healthy humans, because peripheral arteries have less elastin and more collagen than central arteries, leading to difficulty in changing the elastin and collagen ratio in peripheral arteries with advancing age.

It is believed that age-associated arterial stiffening is mainly affected by changes in the composition of the arterial wall, including fragmentation of elastin and increases in collagen deposition, collagen cross-linking as well as vascular smooth muscle cell hypertrophy [7, 8]. Functional changes that result in increased vascular smooth muscle tone, such as increased sympathetic nervous activity and bioactivity of locally synthesized vasoconstrictor molecules (e.g. endothelin-1) and reduced endothelial dilator production [9], also likely contribute to arterial stiffening. It has been reported that the rural Chinese population has a low prevalence of atherosclerotic disease [1], which suggests that arterial stiffening with aging does not depend on atherosclerosis.

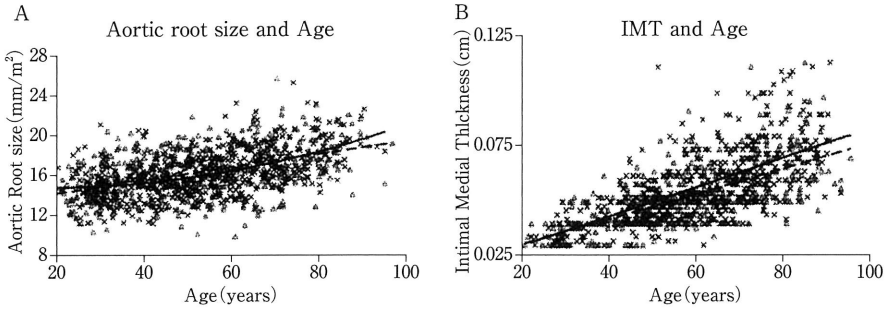

Fig. 1

Age-associated changes in vascular structure in men (×) and women (△). A, Aortic root size measured by M-mode ultrasonography. B, Common carotid intima-media thickness.

This age-associated arterial stiffening is called "arteriosclerosis."

Assessments of arterial function

The large elastic arteries are viscoelastic tubes, the principal functions of which are to act as conduits and to buffer flow pulsatile energy imposed by cardiac contractions. The buffering function permits disappearance of pulsation in peripheral arteries (e.g. capillaries). Developed buffering dysfunction may induce coronary artery diseases or stroke through increased stress and blood pressure in peripheral arteries. Methods have been established and used for assessment of arterial stiffening.

Pulse wave velocity (PWV) was devised by Hill and Bramwell, for which they received the Nobel Prize [10], as an index of arterial stiffness. High distensibility in the artery can absorb amplified energy induced from each pulsation, and then decrease the energy propagating to the periphery, which results in a delayed pulse wave. In contrast to high distensibility in artery, PWV of decreased distensibility in arteries with advancing age is higher, because the arteries cannot buffer pulsation energy. This method is applied by applanation tonometry or ultrasound Doppler technique. Generally, central arterial PWV is assessed between the common carotid and common femoral arteries (cfPWV), which independently predicts all-cause mortality, coronary artery diseases, and diabetes mellitus [11-13]. However, the cfPWV is limited to use in clinical sites, because technicians must have a high level of skill to measure it by applanation tonometry or ultrasound. On the other hand, brachial-ankle PWV (baPWV) was devised in Japan as an index of systemic arterial stiffness. The baPWV measurement, which can be performed more

easily than cfPWV measurement, has become available as a means of measuring PWV [14-17]. The baPWV can be obtained simply by wrapping the four extremities with blood pressure cuffs, and it serves as a simple marker of the severity of vascular damage [17]. This baPWV is strongly correlated with chPW V ($r = 0.76$) [18].

Dynamic arterial compliance and beta-stiffness index are measured by a combination of ultrasound imaging of the pulsatile common carotid artery with simultaneous applanation of tonometrically obtained arterial pressure from the contralateral carotid artery [5, 19]. The carotid artery diameter is measured from images obtained from an ultrasound machine equipped with a high-resolution linear-array transducer. A longitudinal image of the cephalic portion of the common carotid artery is acquired 1-2 cm distal to the carotid bulb. Pressure waveforms and amplitudes are obtained from the common carotid artery with a pencil-type probe incorporating a high-fidelity strain-gauge transducer [5, 20]. As baseline levels of blood pressure are subjected to hold-down force, the pressure signal obtained by tonometry is calibrated by equating the carotid mean arterial and diastolic blood pressure to the brachial artery value [5, 21]. Dynamic arterial compliance and the beta-stiffness index are calculated using the following equations [22] :

$$\text{dynamic arterial compliance} = \frac{(D_1 - D_0)/D_0/}{2 \cdot (P_1 - P_0)} \cdot \pi \cdot D_0^2$$

and

$$\text{beta-stiffness index} = \frac{\ln(P_1/P_0)/}{(D_1 - D_0)}/D_0$$

where D_1 and D_0 are maximal and minimal diameters, and P1 and P0 are the highest and lowest blood pressures, respectively. Although, these parameters have accurately been measured as optical arterial function [5, 21-25], it is difficult to measure accurately for inexperienced technicians. This paper will present findings obtained by PWV and arterial compliance or beta-stiffness index.

Effects of different modes of exercise on arterial stiffness or compliance

From the viewpoint of exercise physiology, exercise is divided into aerobic

and resistance exercises. Many studies have indicated that habitual aerobic exercise improves arterial stiffening. In a cross-sectional study, Tanaka et al. [5] showed that endurance-trained individuals had the lowest value of beta-stiffness index and highest value of dynamic arterial compliance compared to sedentary and recreationally active groups (Fig. 2). In addition, the study also tried to verify the findings in a cross-sectional study by 3-month intervention of aerobic exercise training (e.g. walking and jogging) in middle-aged and older women. The results indicated that dynamic arterial compliance and beta-stiffness index of middle-aged and older women were increased and decreased by 25% and 18% for three months, respectively, comparable to the values of the recreationally active group (Fig. 3). In another study, it was reported that 16-week aerobic exercise (e.g. walking or jogging) improved cfPWV in middle-aged men [18]. Thus, many studies have demonstrated the favorable effects of regular aerobic exercise on arterial compliance and arterial stiffness.

In contrast to aerobic exercise, it has been reported that resistance exercise may reduce dynamic arterial compliance and increase arterial stiffness. Miyachi et al. [25] reported that resistance-trained men who performed weight training at heavy intensity for two years or more showed lower dynamic arterial compliance and greater beta-stiffness index compared with age-matched sedentary healthy men (Fig. 4). We confirmed this result in a cross-sectional study, and showed that arterial compliance was reduced (beta-stiffness index was increased) by 4 months of resistance training in sedentary healthy young men [21] (Fig. 5). Moreover, other randomized control trials have also supported this finding that resistance training induces arterial stiffening [23, 26-29]. On the other hand, some studies indicated no changes in arterial compliance and stiffness with chronic resistance training in young, middle-aged, and older populations [30-33]. Recently, Miyachi [34] examined the negative relationship between arterial compliance and habitual resistance training by meta-analysis for randomized control trial studies, and suggested that high-intensity resistance training is associated with increased arterial stiffness or decreased arterial compliance in young subjects with low baseline levels of arterial stiffness, but no changes in older or low intensity resistance training groups (Fig. 6).

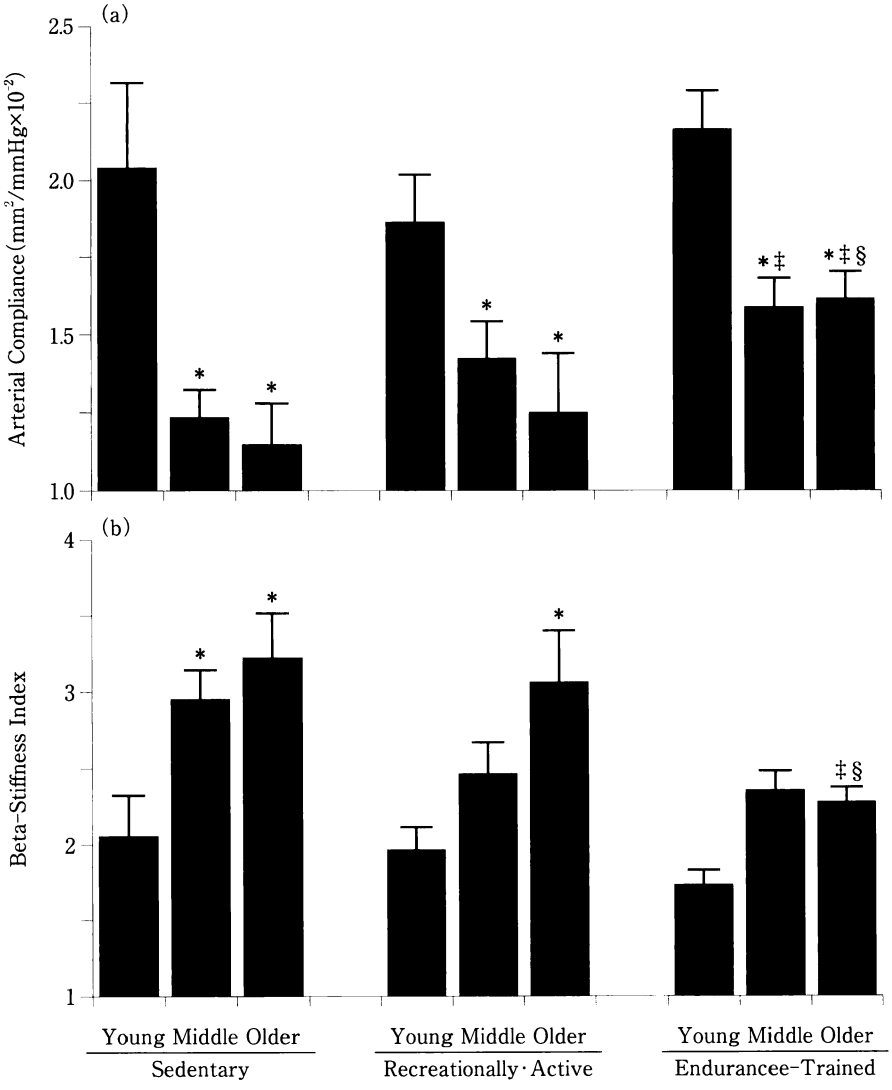

Fig. 2

Dynamic arterial compliance (a) and beta-stiffness index (b) of subjects in cross-sectional study. *P<0.05 vs. young within the same activity group; ‡ P<0.05 vs. sedentary of same age group; and § P<0.05 vs. recreationally active of same age group. Data are means ± SEM.

Effects of combination of aerobic and resistance training on arterial stiffness or compliance

As described above, arterial stiffness is decreased with habitual aerobic exercise and increased with resistance exercise. Both types of exercise have become popular modalities of exercise performed by most populations, and

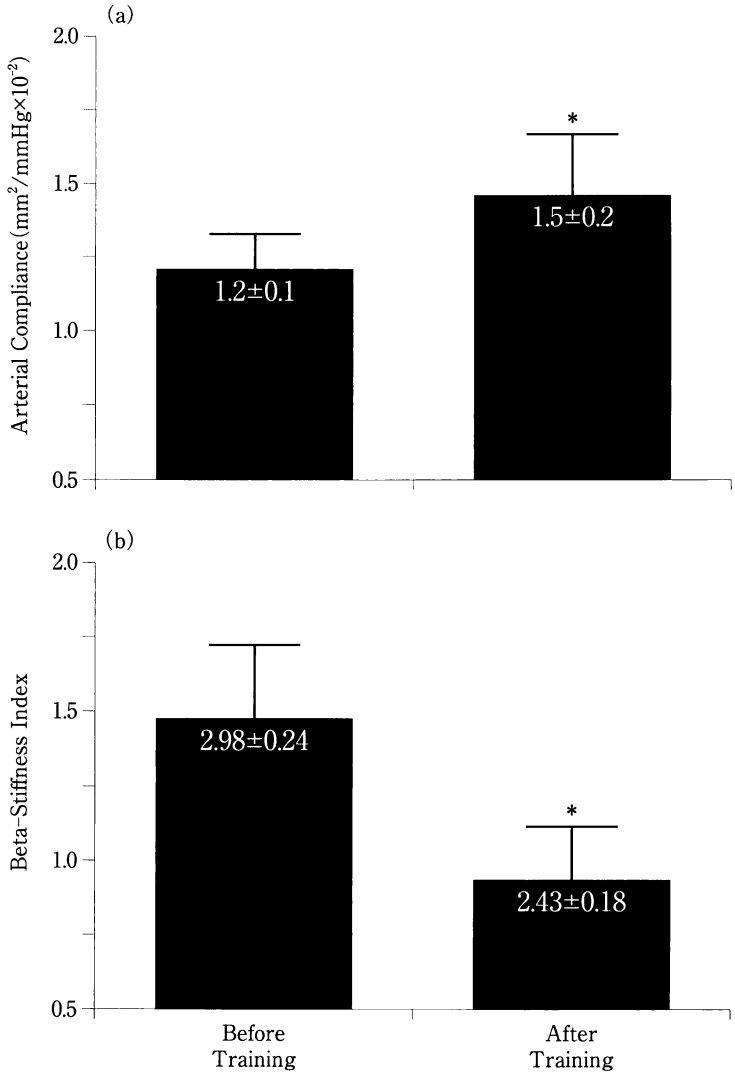

Fig. 3

Dynamic arterial compliance (a) and beta-stiffness index (b) before and after aerobic exercise intervention. *P<0.05 vs. before training. Data are means ± SEM.

have become integral components of exercise recommendations endorsed by a number of national health organizations [35-37]. Generally, many people would perform a combination of aerobic and resistance exercises to maintain cardiorespiratory fitness and muscular strength and to prevent cardiovascular diseases. Therefore, we examined the effects of combined habitual aerobic and resistance exercise on arterial compliance and beta-stiffness index [23]. The

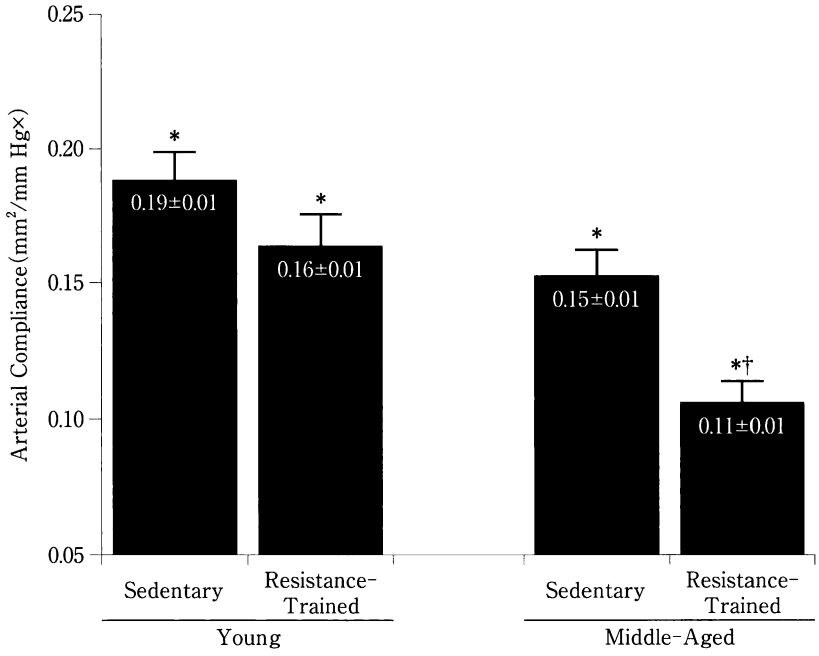

Fig. 4

Dynamic arterial compliance of sedentary and resistance-trained men. *P<0.05 vs. young of same activity group; † P<0.05 vs. sedentary of same age group. Data are means ± SEM.

combined aerobic and resistance training group showed no change in arterial compliance and beta-stiffness index over a 4-month training period (Fig. 7), which suggests that simultaneously performed aerobic training could negate and prevent the stiffening of carotid arteries caused by resistance training. Moreover, Okamoto et al. [38] investigated the training effects of the influence of timing of aerobic exercise with respect to resistance exercise on baPWV in healthy young adults. The study demonstrated that although baPWV was not improved by aerobic exercise before resistance training, performing aerobic exercise afterwards can prevent the deterioration of baPWV (Fig. 8). These findings may have important clinical implications for exercise prescription.

Exercise modes and arterial stiffness or compliance

There are many modes of exercise involving combinations of various physical motions. Thus, it is difficult for exercise to be completely divided into aerobic and resistance exercise. Therefore, table 1 shows the associations between arterial stiffness and mode of exercise or sport reported by original

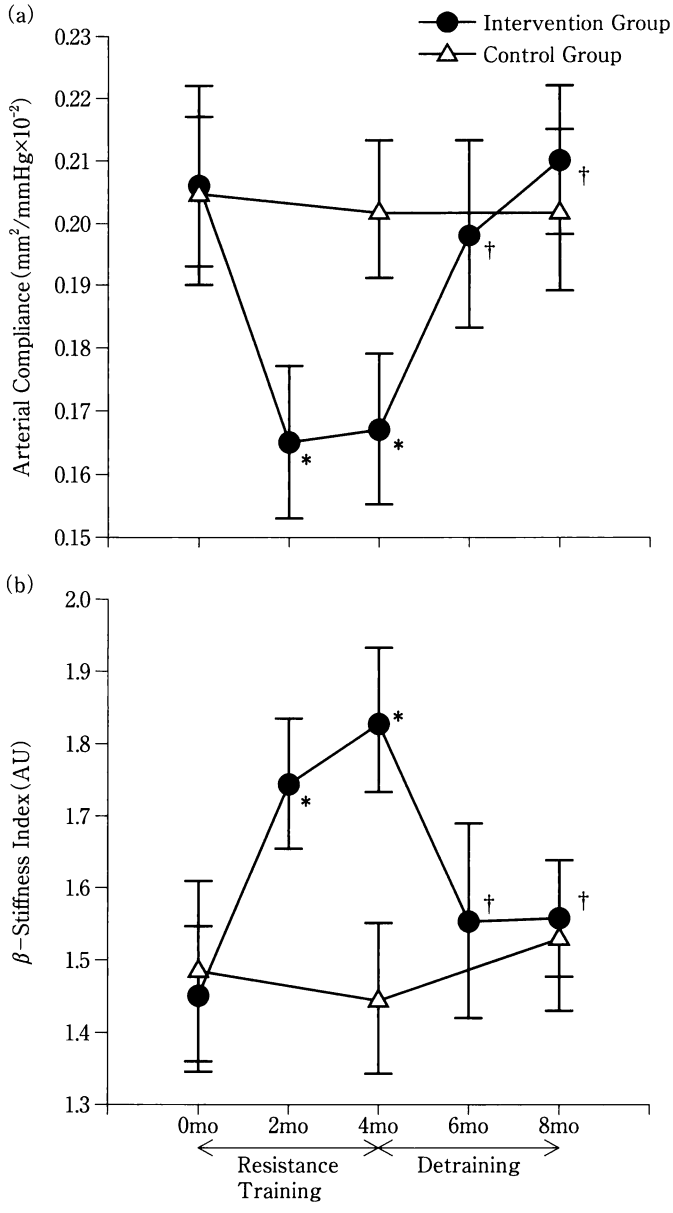

Fig. 5

Changes in dynamic arterial compliance (top) and beta-stiffness index (bottom) in the intervention group (black circles) and control group (white triangles). *P<0.05 vs. baseline; † P<0.05 vs. resistance training period (2- and 4-month values). Data are means ± SEM.

Fig. 6

Relative changes in arterial stiffness in individual studies of resistance training vs. no intervention. Relative changes in arterial stiffness index (%) of individual studies included in the meta-analysis of resistance exercise training vs. no intervention. Studies with young (< 40 years old) or middle-aged (≥ 40 years old) participants were evaluated as separate observations. Weights are from random-effects analysis.

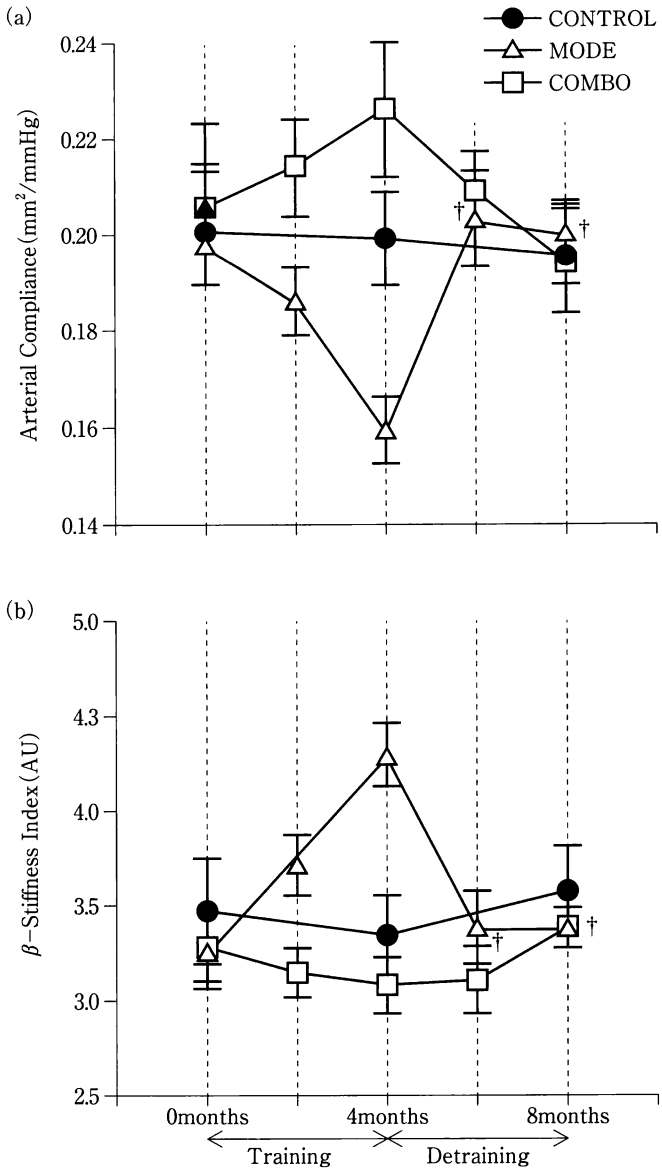

Fig. 7

Changes in (a) dynamic arterial compliance and (b) beta-stiffness index for the sedentary control group (CONTROL), the moderate-intensity strength training group (MODE), and the combined aerobic and strength training group (COMBO). *P<0.05 vs. baseline; † P<0.05 vs. 4 months. Data are means ± SEM.

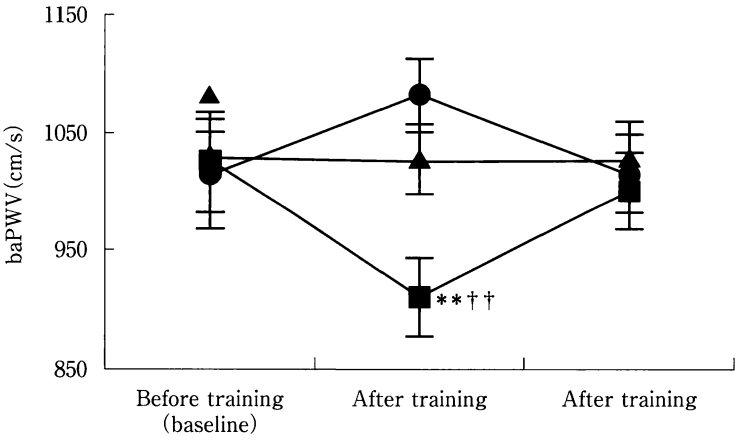

Fig. 8

Changes in baPWV in groups that ran before resistance training (BRT; circles), ran after resistance training (ART; squares), or remained sedentary (SED; triangles). *P<0.05; **P<0.01 vs. baseline; † P<0.05; † † P<0.01 vs. BRT group. Data are means ± SEM.

papers except for basic resistance and aerobic exercise (e.g. weight lifting, walking, or jogging) [39-44]. The association between arterial stiffness or compliance and rowing training was reported, followed by swimming and yoga. Most of these studies indicated favorable effects of these exercise modes on arterial compliance and stiffness. Future studies should determine the effects of sports with many players on arterial compliance and stiffness.

Intensity of physical activity and arterial stiffness

Many studies have shown that high physical activity contributes to improvement of arterial stiffness. However, it is difficult to correctly determine the duration and intensity of physical activity, because most studies used questionnaires. Therefore, it remained unclear what kind of physical activity (e.g. duration and intensity) affects arterial stiffness or compliance. Recently, use of accelerometers has permitted evaluation of physical activity volume from duration and intensity. Sugawara et al. [45] examined the associations between beta-stiffness index of carotid artery and physical activity durations of each intensity (low < 4 METs; 4 METs ≤ moderate < 6 METs; 6 METs ≤ vigorous) in a day for 103 postmenopausal women. The beta-stiffness index was inversely related to the duration of physical activity at all intensities. Beta-stiffness index was significantly correlated with the duration of physical

Table 1 The associations between arterial stiffness/compliance and modes of exercise/sport

Study design	Subjects	n	Sex	Age	Results	Paper
Cross-sectional	Yoga-trained	8	Male and Female	48	Arterial stiffness in yoga-trained and endurance-trained groups was lower than controls.	Duren et al. Dyn Med. 2008.
	Endruance-trained	10	Male and Female	52		
	Control	8	Male and Female	51		
Cross-sectional	Swimmer	25	Male and Female	56	Arterial stiffness in swimmers and runners was lower than controls.	Nualnim et al. Am J Cardiol. 2011.
	Runner	25	Male and Female	52		
	Control	25	Male and Female	54		
Cross-sectional	Rowing-trained	15	Male end Female	50	Arterial stiffness in rowing-trained group was lower than controls.	Cook et al. J Appl Physiol. 2005.
	Contoro	15	Male end Female	52		
Cross-sectional	Rowing-trained	28	Male and Female	24	Arterial compliances of aorta, carotid and brachial arteries in rowing-trained group were similar to controls.	Petersen et al. J Am Coll Cardiol. 2006
	Control	21	Male and Female	28		
Cross-sectional	Young rowing-trained	26	Male	20	baPWV in middle-aged rowing-trained men was lower than age-matched controls.	Sanada et al. J Sports Sci. 2006.
	Young control	23	Male	25		
	Middle-aged rowing-trained	24	Male	65		
	Middle-aged control	22	Male	65		
Cross-sectional	Rowing-trained	11	Male	68	Carotid arterial compliance and beta-stiffness index in rowing-trained men were similar to tocontrols.	Kawano et al. J Sports Sci. 2012.
	Control	11	Male	65		

activity at moderate and vigorous intensity, but not low intensity, even adjusting for confounders (e.g. age, body mass index, and blood pressure, etc.). In addition, the investigation also reported findings of exercise intervention for 17 menopausal women as follows: beta-stiffness indexes of both moderate (< 4 METs) and vigorous (< 9 METs) intensity groups showed the same reduction (30%-33%) in beta-stiffness for 12 weeks. These cross-sectional and interventional results suggest that physical activity at moderate to vigorous intensity needs to be included in daily life to improve arterial stiffening.

Light physical activity and arterial stiffness

Generally, we perform physical activity at various intensities for daily life, ranging from 1 MET (e.g. sleeping) to 15 METs (e.g. climbing stairs). Although previous studies have applied uniaxial accelerometry to assess

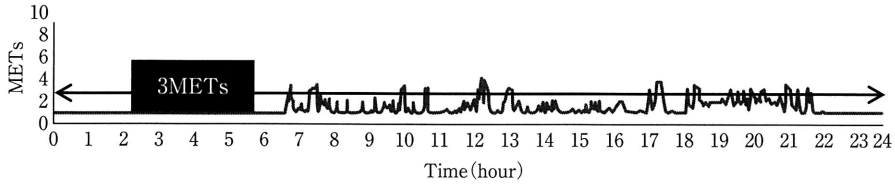

Fig. 9

Typical physical activity of one reference subject for one day.

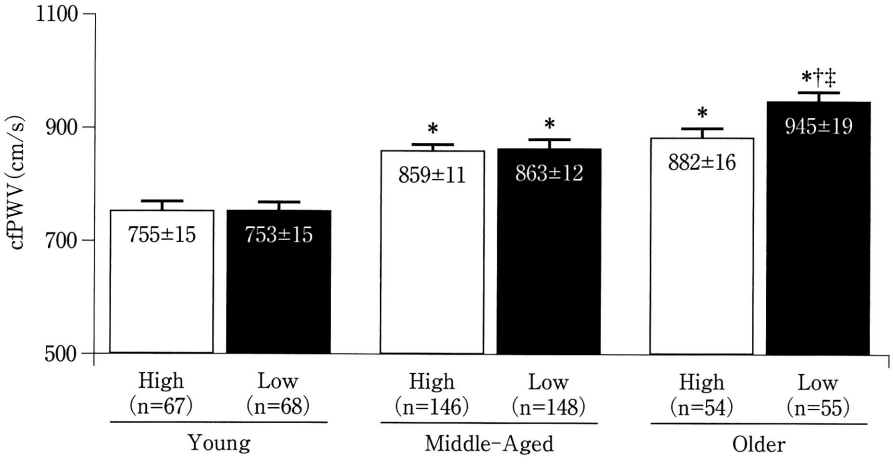

Fig. 10

cfPWV in high-light physical activity and low-light physical activity groups. *P<0.05 vs. young; † P<0.05 vs. middle-aged; ‡ P<0.05 vs. high in the same age group. Data are means ± SEM.

physical activity volume through duration and intensity, uniaxial accelerometry may be unsuitable for light (< 3 METs) physical activity. However, light physical activity (e.g. cooking, cleaning, washing clothes, desk work etc.) generally account for most of one day (Fig. 9). This raises the question of whether light physical activity affects arterial stiffness. In recent years, there have been advances in triaxial accelerometry, which permitted adequate assessment of light physical activity of less than 3 METS. Gando et al. [46] tested the effects of durations of light (≤ 2.9 METs), moderate (3.0-5.9 METs) and high (≥ 6.0 METs) physical activities on cfPWV for 538 healthy men and women. The results indicated that durations of not only moderate and high but also light physical activity were negatively correlated with cfPWV. Furthermore, in the older subjects, cfPWV was greater in the high duration of light physical activity group than in the low duration of light physical activity

group, and the differences remained significant after normalizing cfPWV for sex and durations of moderate and high physical activities (Fig. 10). These results suggest that performing light physical activity may prevent age-associated arterial stiffening. Moreover, older adults in the study showed a correlation between cfPWV and duration of light physical activity ($r = -0.39$) similar to their relationship between cfPWV and durations of moderate physical activity ($r = -0.31$), suggesting that moderate as well as light physical activity plays an important role in preventing and improving arterial stiffening. In modern society, decreased light physical activity may need to return to the previous level for prevention and improvement of arterial stiffening.

Light intensity exercise and arterial stiffness

Generally, because it is often defined as moving the body vigorously, we would imagine exercise as entailing physical activity of 3 or more METs, such as walking or running. However, there is also light intensity exercise, such as stretching, which is categorized as a form of exercise of less than 3 METs. Yamamoto et al. [47] examined the relationship between flexibility and cfPWV in a cross-sectional study, because flexibility is increased by performing stretching exercise. In addition, stiffness in both arteries and flexibility are determined by elastin-collagen composition of smooth muscle and/or connective tissue and skeletal muscle and/or tendons, respectively, and then age-related alterations in the muscles or connective tissues in the arteries may correspond to similar age-related alterations in the whole body [48]. The investigation revealed negative relationships between flexibility and cfPWV or baPWV in middle-aged and older populations (Fig. 11). Stepwise multiple-regression analysis revealed that flexibility, age, and cardiorespiratory fitness were independent correlates of baPWV. Another study confirmed these findings by yoga exercise intervention, which indicated that 8 weeks of Bikram yoga improved beta-stiffness index in young but not older adults [49]. Therefore, light intensity exercise may also prevent and improve arterial stiffening, leading to prevention and improvement of arterial stiffening for older and low-fitness populations.

Effects of age and cardiorespiratory fitness on arterial wall viscosity and elasticity

Arterial mechanical properties are assessed by various methods (e.g.

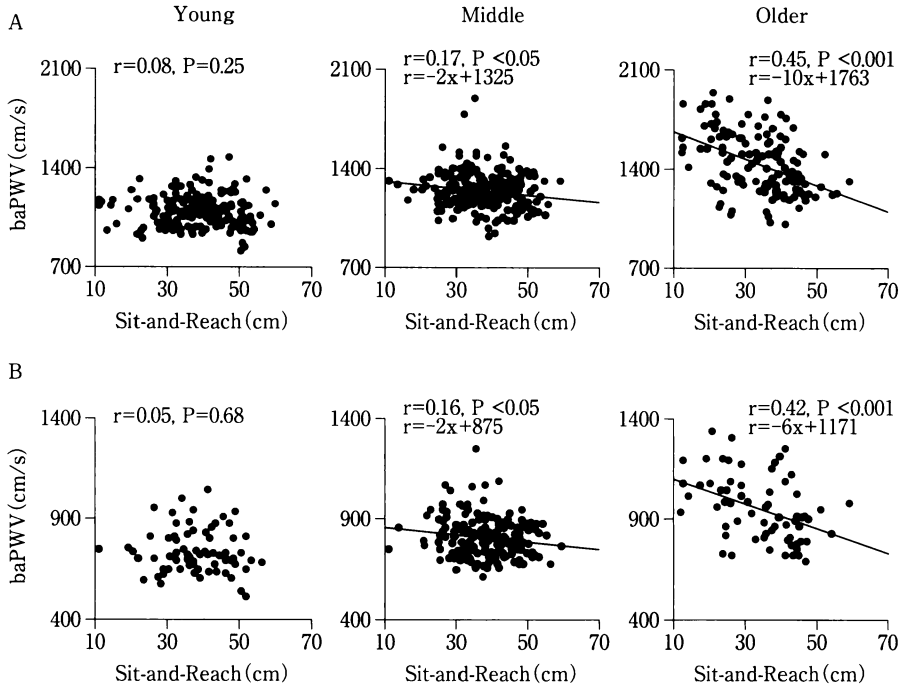

Fig. 11

Relationships between sit-and-reach and baPWV (A) or cfPWV (B) in each age category. The baPWV and cfPWV correlated with sit-and-reach in middle-aged (middle) and older (right) subjects. In both baPWV and cfPWV, the slope of the relationship was steeper in older subjects than in middle-aged subjects ($P < 0.001$).

dynamic arterial compliance, beta-stiffness index, or PWV). This chapter focuses on viscosity and elasticity in central arteries. The central elastic arteries are viscoelastic tubes, which function to buffer flow pulsatile energy imposed by the left ventricle. Although purely elastic materials permit all of the stored energy to be restored during the unloading phase, arteries are not purely elastic and exhibit marked viscous behavior. Arterial wall viscosity is a source of energy dissipation, considering viscosity as an energy dissipating phenomenon during mechanical transduction (conversion of cardiac pulsatile energy into arterial elastic energy) [50-53]. Therefore, the arterial mechanical characteristics include both elastic and viscous properties. Although dynamic arterial compliance or beta-stiffness index is assessed as arterial elastic properties, viscous properties have been considered less important in assessment of vascular function. Arterial wall viscosity may be affected by neural and physical factors. It has been reported that wall viscosity is related

to intima-media thickening in the carotid artery [54] (Fig 12). On the other hand, hypertensive patients showed greater wall viscosity in the carotid artery than normotensive patients, and greater wall viscosity in hypertensive patients was attenuated by treatment with an antihypertensive drug [55]. Interestingly, Armentano et al. reported that the isolated carotid artery showed lower wall viscosity and greater wall strain in vitro [55], suggesting that reduction of wall viscosity caused by denervation is associated with greater wall strain. Therefore, wall thickness, blood pressure, and sympathetic nerve activation may account for wall viscosity in central elastic arteries.

A recent study confirmed the effects of age and cardiorespiratory fitness on arterial wall viscosity. The study indicated that carotid arterial wall viscosity was increased with advancing age, and the age-associated increase in wall viscosity was attenuated in cardiorespiratory fit men [56] (Fig. 13). These findings may have clinical implications for prevention and improvement of age-associated arterial stiffening by maintaining cardiorespiratory fitness. Central elastic artery flexibly receives the pulsatile energy imposed by cardiac contractions to store energy, and the stored energy is effectively used for flowing blood into peripheral tissues. In this successive progression, wall viscosity reflects energy loss during conversion of mechanical energy from cardiac pulsation into elastic energy in the arterial wall. Thus, the results of

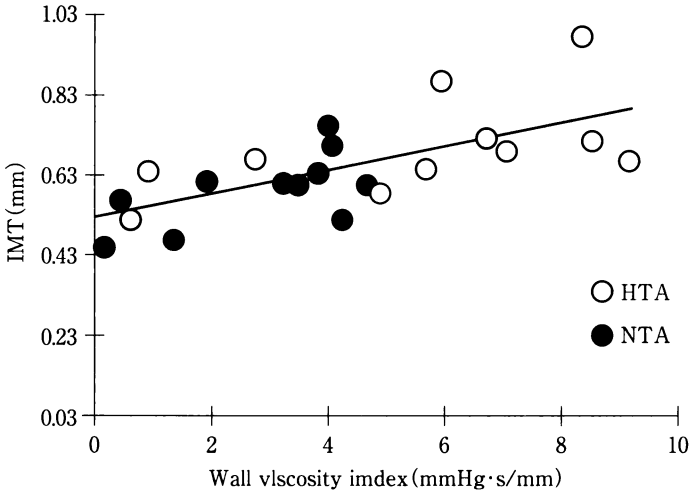

Fig. 12

Linear regression analysis between intima-media thickness (IMT) and arterial wall viscosity index in the normotensive (NTA: black circles) and hypertensive (HTA: white circles) groups.

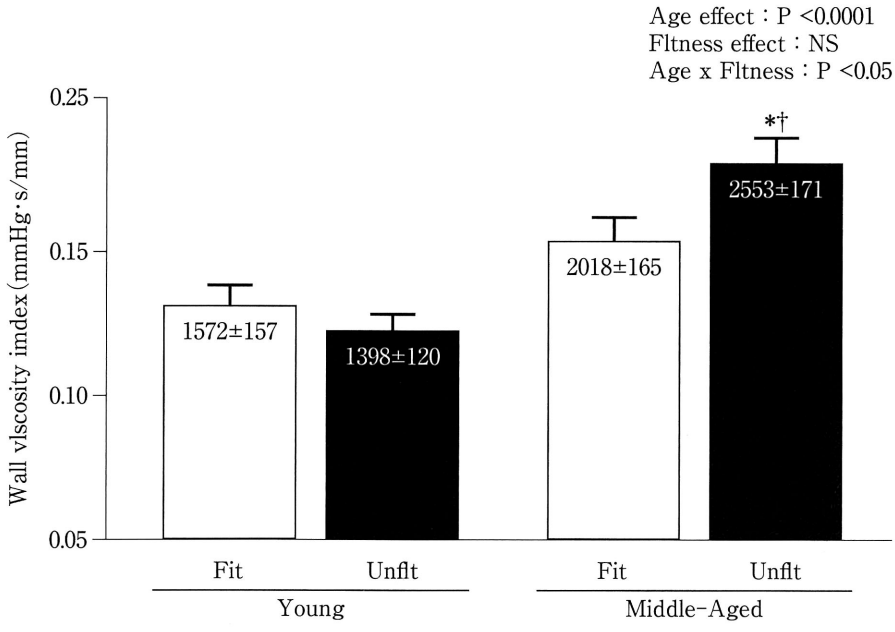

Fig. 13

Bar graph showing arterial wall viscosity index in each group. *P<0.05 vs. young in the same cardiorespiratory fitness level; † P<0.05 vs. men of the same age in the cardiorespiratory fit group. Data are means ± SEM.

the present study indicating greater wall viscosity of middle-aged men than young men suggest that advancing age results in inefficient mechanical transduction, and consequently the efficiency of blood flow in elastic arteries may be impaired with advancing age. In addition, our results indicated that the age-related increase in wall viscosity was smaller in cardiorespiratory fit men compared with unfit men, and there were no significant differences in wall viscosity or elasticity between young and middle-aged cardiorespiratory fit men. These findings suggest that the attenuation of age-associated reduction in cardiorespiratory fitness level prevents energy loss during mechanical transduction, leading to maintenance of effective blood flow in elastic arteries.

These results may be expanded to understand the mechanisms underlying changes in autonomic regulation with advancing age. Arterial wall viscosity is mainly related to vascular smooth muscle cells [50, 51, 53, 57].Thereby, age-associated sympathoexcitation [58] may induce an increase in wall viscosity through smooth muscle contraction. On the other hand, increased wall viscosity leads to a reduction in wall strain [55]. Decreased wall strain may contribute

to attenuation of baroreceptor sensitivity in response to blood pressure fluctuation. Therefore, age-related reductions in baroreflex sensitivity [59] may be associated with age-related increases in wall viscosity. Accordingly, age-associated arterial stiffening and its prevention can be clarified by physical methods.

Perspectives

In 2013, "Physical Activity Reference for Health Promotion 2013" and "Active Guide 2013" were released by the Ministry of Health, Labour and Welfare of Japan, which recommends appending physical activity for 10 min per a day (Plus 10). In the near future, the effects of Plus 10 will be verified for various outcomes, and dynamic arterial compliance, beta-stiffness index, PWV, and arterial wall viscosity are not exceptions, either. Nevertheless, the underlying physiological mechanisms and clinical implications of adaptations of vascular function to age and various physical activities or exercise warrant further investigation.

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