

Arterial Stiffness Differences between Aerobically and Resistance Trained Turkish Elite Athletes

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ABSTRACT The purpose of this study was to evaluate whether arterial stiffness was different in aerobically trained elite athletes than in anaerobically or resistance trained elite athletes. The cohort comprised 36 healthy male volunteers, aged between 17 and 32 years. All subjects were basketball players (n=10), weightlifters (n=11) or sedentary controls (n=15). The Pulse Trace System (Micro Medical Ltd., Rochester, UK) was employed to record central and peripheral arterial stiffness. Echocardiographic images were taken by the use of a commercially available machine (Vivid 7 GE-Vingmed, Horten, Norway) with a 2.5 MHz transducer. Aortic elastic properties derived from echocardiographic measurements did not differ between the groups ($p < 0.05$). Pulse wave velocity measurements reflected significantly lower values in both the basketball players and weightlifters compared to controls ($p < 0.001-0.05$). No significant difference was seen between the basketball players and weightlifters ($p < 0.05$). Contrary to existing knowledge, arterial stiffness of athletes that perform more resistance exercise such as weightlifters improved significantly and did not get worse. This result implies that in the long-term arterial stiffness improves with sports activities that are predominantly comprised of resistance exercises despite increased arterial stiffness in the acute phase.

INTRODUCTION

Arterial pulse wave velocity (PWV) is widely used for the indirect assessment of arterial stiffness. PWV is measured as the transit time of the pressure wave between two remote parts of the arterial tree (Eugene et al. 1982). If the vessel is

stiffer, the pulse pressure wave moves faster along the vessel.

Regular physical training has a protective effect against cardiovascular diseases such as hypertension, coronary heart diseases, and peripheral arterial occlusive diseases. Although the mechanisms underlying this protective effect probably include favorable changes in blood pressure, plasma lipids and glucose-insulin metabolism (Shephard et al. 1999), an additional possibility is that regular aerobic exercise is associated with enhanced arterial compliance. Accordingly, several studies provide evidence of reductions in central arterial stiffness with en-

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duration training in young (Cameron et al. 1994; Kakiyama et al. 2005) and older populations (Collier et al. 2008a; Tanaka et al. 2000), and in competitive endurance athletes (Edwards et al. 2005). Such results were noted from both cross-sectional (Edwards et al. 2005; Vaitkevicius et al. 1993; Kingwell et al. 1995; Monahan et al. 2001) and mostly longitudinal studies (Tanaka et al. 2000; Cameron et al. 1994; Edwards et al. 2004).

However, in longitudinal studies exercising durations were between 8 to 12 weeks in general (Ciolac et al. 2010; Ferrier et al. 2001; Madden et al. 2009; Yoshizawa et al. 2009). In few studies the duration of exercise was 26 weeks long (Madden et al. 2013). The effects of the exercise training on arterial stiffness have never been conclusive (Ciolac et al. 2010; Ferrier et al. 2001; Madden et al. 2009; Yoshizawa et al. 2009; Madden et al. 2013). Tanaka et al. (1998) showed that arterial stiffness of the upper and lower limbs in well-trained postmenopausal female runners was not different from that in sedentary counterparts. Hayashi et al. (2005) evaluated the effects of aerobic exercise training on the stiffness of central and peripheral arteries. Following the aerobic exercise training, they found reductions in central arterial stiffness, but no difference in peripheral arterial stiffness. The researchers stated that the stiffness of the peripheral artery was difficult to change in the short-term and moderate-intensity aerobic exercise training. In contrast, Schmidt-Trucksass et al. (2000) indicated that femoral arterial compliance and diameter in endurance-trained males were significantly larger than those in age-matched sedentary counterparts.

Today strength training takes up more space in exercise programs. National and international healthcare institutions continue to recommend resistance to be part of physical activity programs designed for both recreational and health purposes (Diabetes Mellitus and Exercise 2000; ACSM Position Stand 1998; Pollock 2000).

Resistance exercise is known to strengthen the connective tissue and increase the muscle bone mass (Pollock et al. 2000). Some studies have shown that resistance training benefitted the cardiovascular system although it has not been clearly elucidated in literature. In marked contrast to regular aerobic exercise, it has been shown in longitudinal studies that high-intensity resistance training may increase central arterial stiffness in young (Cortez-Cooper et al. 2005;

Miyachi et al. 2004; Okamoto et al. 2006) and middle-aged adults (Collier et al. 2008a). Some cross-sectional comparisons between sedentary and resistance-trained individuals have shown also reduced central arterial stiffening (Bertovic et al. 1999; Miyachi et al. 2003). The possible increased stiffness with both acute (Heffernan et al. 2007a) and chronic resistance exercise (Cortez-Cooper et al. 2005; Miyachi et al. 2004) was restricted to central elastic arteries and not found in peripheral muscular arteries. In contrast, no such changes were observed in several other studies (Casey et al. 2007a; Cortez-Cooper et al. 2008; Rakobowchuk et al. 2005; Yoshizawa et al. 2009). These studies reported contradictory results, that arterial stiffness was not significantly increased after resistance exercise (Rakobowchuk et al. 2005). Rakobowchuk et al. (2005) found that central arterial compliance was unaltered after three months of resistance training in young men. As can be seen, there is no unified view on the effects of resistance training on arterial stiffness and further studies are needed to evaluate the effects of longer duration resistance exercise on arterial stiffness.

Therefore, the purpose of this study was to evaluate whether arterial stiffness was different in aerobically trained elite athletes than in anaerobically or resistance trained elite athletes.

MATERIAL AND METHODS

Subjects

The cohort comprised 36 healthy male volunteers, aged 17 to 32 years (Table 1). All subjects were basketball players (n=10), weightlifters (n=11) or sedentary controls (n=15). All of them were non-obese (BMI: 30 kg/m²), non-smoking, normolipidemic, normotensive (140/80 mmHg), and free of cardiovascular and other chronic diseases such as diabetes mellitus, chronic renal disease, peripheral arterial disease, alcohol addiction, history of vascular surgery, arrhythmia, cardiac valvulopathy or myocardial ischemia according to medical history, and clinical and electrocardiographic evaluation. None of the subjects were taking anti-diabetic or cardiovascular-acting medications and antioxidants or lipid-lowering supplements. Physical activity and nutrition habit was documented using a questionnaire.

Subjects in group 1 (basketball players, $n=10$) were athletes undergoing training 15 hours per week for the last 12 months. The training comprised 6 hours of specific basketball training, 2 hour of strength training, 5 hours of endurance activities, 1 hour of balance and coordination exercises and 1 hour of anaerobic training.

Subjects in group 2 (weightlifters, $n=11$) were athletes training 18 hours per week (3 hours/day, 6 time/week) for the last 12 months. Most of the training comprised specific weightlifting with a duration of 15 hours per week. The athletes performed aerobic warm-up training before starting specific weightlifting training with a duration of 3 hours per week. The sport specific training comprised general types of weightlifting training like squats, clean and jerk and snatch activities and lifting weight as much as they could. The amount that was targeted for the forthcoming competition was determined as the training amount of the weight that the athletes should aim to lift. The athletes are capable to lift 300 kg weight on average.

Subjects in group 3 were sedentary (less than 1-hour physical activity per week, $n=15$) and were matched with the other groups for age.

The study was approved by the ethics board of the university. An informed consent form was filled in and signed by all subjects.

Study Design

All laboratory procedures were performed at supine rest in a quiet and temperature controlled room at 22°C. Subjects had fasted for at least 6 hours and had abstained from caffeine and alcohol for at least 12 hours before the measurements. Additionally, to avoid the effects of acute exercise, the subjects were directed to perform neither training nor other vigorous physical activity other than walking on the day before measurements. All tests were conducted between the hours of 07:00 and 10:00.

Anthropometric Measures

Upon arrival at the laboratory, measurements for body mass and body fat were taken from each subject. Body fat was calculated by measuring sub-dermal fat content from 5 body areas (abdomen, sub-scapulae, triceps, lateral side of the thigh and lateral side of the abdomen) using a

skinfold caliper and body mass was recorded using standard laboratory scales.

Pulse Wave Velocity (PWV) Measurement

In the laboratory, subjects were required to rest in a seated position for approximately 15 minutes at a constant temperature of 22°C. After that their examinations were made in the supine position. The Pulse Trace System (Micro Medical Ltd., Rochester, UK) was employed to record central and peripheral arterial stiffness. Pulse Trace PWV uses the standard Doppler method to detect the onset of flow in the artery. This equipment has a 4 MHz Doppler probe for identifying the arrival of the arterial pulse. The waveform was measured sequentially in two arterial locations, from right carotid to right brachial artery (cb) and from right carotid to right femoral artery (cf). The transit time ($\dot{A}T$) was computed from the foot-to-foot time difference in milliseconds between the two sequentially measured Doppler waveforms from the above mentioned locations using the 'R' wave of the ECG as a timing reference. The Pulse Trace PWV automatically detects the foot-to-foot of the waveform. The average of ten waveforms from each location was used and the SD was calculated and displayed. At each time point, the measurements were made three times for each individual subject and the mean value was recorded. For the measurements of arterial stiffness, the distance (D) between the carotid artery and brachial artery, and the distance between the carotid artery and femoral artery was measured using a measuring tape, respectively. PWV in m/sec was automatically calculated by dividing the time delay by the externally measured distance between locations ($PWV=D/\dot{A}T$). All waveforms were displayed and the detected onset of the pulse was marked.

Echocardiography (Arterial Compliance)

Images were taken using a commercially available machine (Vivid 7 GE-Vingmed, Horten, Norway) with a 2.5 MHz transducer. All patients were examined at rest while in the left lateral recumbent position following the recommendations of the American Society of Echocardiography (Lang et al. 2005). Images were obtained in the parasternal long and short axis and in the apical four and five chamber views using two dimen-

sion and M-Mode Doppler. All of the measurements were digitized at the peak of the R wave by the average of three cardiac cycles. The ascending aorta was recorded in the two-dimensional guided M-Mode tracings. Aortic diameters were recorded 3cm above the aortic valve. Aortic systolic diameter was determined at the time of the full opening of the aortic valve, and aortic diastolic diameter was determined at the peak of QRS. Blood pressure was measured simultaneously from the right brachial artery with an aneroid sphygmomanometer. Korotkoff phases I and V of the measurements were used to determine the systolic and diastolic blood pressure.

Calculations

1. The following formula was used to measure aortic strain (AS):

$$AS = (AoS - AoD) / AoD$$
(AoS=systolic aortic diameter and AoD=diastolic aortic diameter).
2. The following formula was used to measure aortic distensibility (AD):

$$AD = 2 \times (AoS - AoD) / [AoD \times (SBP - DBP)]$$
(SBP=systolic blood pressure, DBP= diastolic blood pressure, AoS= systolic aortic diameter and AoD= diastolic aortic diameter).

3. The following formula was used to measure aortic stiffness index (beta stiffness index, BSI):

$$BSI = \ln(SBP/DBP) / [(AoS - AoD) / AoD]$$

(SBP=systolic blood pressure and DBP= diastolic blood pressure).

Data Analysis

Statistical analysis was performed using SPSS version 16.0 (SPSS, SPSS Inc, Chicago, Illinois, USA) software. For comparison among the groups, one-way analysis of variance (ANOVA) was used. Scheffe's post hoc test was performed to evaluate a significant *F*-value. All measurements were expressed as mean±SD. All tests were two-tailed and the level of significance was set at $p < 0.05$.

RESULTS

Demographic and physical characteristics of the study groups are shown in Table 1. Basketball players had a significantly higher height and body weight as compared to both weightlifters and controls ($p < 0.001-0.01$). Heart rate was significantly reduced in basketball players than only the control group ($p < 0.05$). As indicated in Table 2, aortic elastic properties derived from echocardiographic measurements did not differ between

Table 1: Physical characteristics of athletes and controls. (Data are mean ± SD)

	Basketball players (n=10)	Weight lifters (n=11)	Controls (n=15)
Age (years)	22.2 ± 4.3	19.5 ± 3.7	22.6 ± 2.4
Height (cm)	191 ± 7	169 ± 7***	174 ± 5***
Weight (kg)	93.1 ± 10.6	76 ± 15.8**	76.5 ± 6.6**
Heart rate (beat.m ⁻¹)	65 ± 13	73 ± 14	79 ± 11*
Systolic blood pressure (mm.Hg)	119 ± 7	120 ± 4	118 ± 8
Diastolic blood pressure (mm.Hg)	80 ± 3	80 ± 3	78 ± 8
Body fat percentage (%)	8.2 ± 3.5	17.8 ± 6.9	21.2 ± 4

* $p < 0.05$, ** $p < 0.01$ and *** $p < 0.001$ compared with basketball players.

Table 2: Aortic elastic properties in athletes and controls. (Data are mean ± SD)

	Basketball players (n=10)	Weight lifters (n=11)	Controls (n=15)
Systolic aortic diameter (cm)	2.8 ± 0.2	2.6 ± 0.4	2.8 ± 0.3
Diastolic aortic diameter (cm)	2.6 ± 0.2	2.4 ± 0.4	2.6 ± 0.3
Aortic distensibility (mm.Hg ⁻¹ .10 ⁻³)	5.21 ± 2.05	5 ± 4.19	4.45 ± 3.05
Aortic stiffness index	4.35 ± 1.26	6.91 ± 4.31	6.63 ± 3.63
Aortic strain	0.10 ± 0.04	0.12 ± 0.10	0.08 ± 0.05

$P < 0.05$.

the groups ($p < 0.05$). On the other hand, pulse wave velocity measurements reflected significantly lower values both in basketball players and weightlifters compared to controls ($p < 0.001-0.05$) (Table 3). No significant difference was seen between basketball players and weightlifters ($p < 0.05$).

DISCUSSION

Ageing, hypertension, increased insulin resistance, and diabetes causes are reported to increase arterial stiffness by inducing collagen and calcium accumulation in the arterial walls, which is associated with arterial dilatation, elevated systolic blood pressure and increased cardiac disease risk (Henry et al. 2003; Mitchell et al. 2010; Vlachopoulos et al. 2010; Ben-Shlomo et al. 2014). Carotid-femoral pulse wave velocity above 12 m/sec (or increased arterial stiffness) is a cardiac risk factor according to guidelines (Giuseppe et al. 2007).

Many studies have been conducted to elucidate the association between increased arterial stiffness and physical activity and exercise. Some of these studies investigated the acute effects of aerobic exercise (Kingwell et al. 1997; Heffernan et al. 2007 a; Heffernan et al. 2007 b; Currie et al. 2009; Tordi et al. 2010; McClean et al. 2011; Tomoto et al. 2015; Liu et al. 2015) whereas others studied the chronic effects of aerobic exercise (Tanaka et al. 2000; Edwards et al. 2004; Edwards et al. 2005; Na et al. 2009; Koshiha et al. 2015). In addition to studies investigating the acute (DeVan et al. 2005; Yoon et al. 2010; Hoonjan et al. 2013) and chronic (MacDougall et al. 1986; Miyachi et al. 2004; Cortez-Cooper et al. 2005; Kawano et al. 2006) effects of resistance exercise there are studies that investigate the combined effects of aerobic and resistance exercise (Cook et al. 2006; Nualhim et al. 2011). However, these studies report the outcome after 8-12 weeks of exercise training (Ciolac et al. 2010; Ferrier et al.

2001; Madden et al. 2009; Yoshizawa et al. 2009). Unlike these studies, the present study compares basketball players who had been performing long-term aerobic training and weightlifters who had been resistance training for a long time with a sedentary group and aims to study the effect of aerobic exercise and resistance training on arterial stiffness.

Aerobic Exercise

A study investigating the effect of acute arterial exercise on arterial stiffness revealed that a 30-minute exercise performed once at an intensity of sixty-five percent VO_2 max raised arterial compliance by thirty to forty percent, and values returned to baseline in 60 minutes due to increased vasodilatation (Kingwell et al. 1997). Heffernan et al. (2007a) purported that 20 minutes after acute aerobic cycling (exercise) arterial stiffness was reduced, whereas after resistance exercise central arterial stiffness increased but peripheral arterial stiffness was not affected. Moreover, the same study showed that central arterial stiffness did not change after acute aerobic exercises but peripheral arterial stiffness was reduced in athletes who performed resistance training (Heffernan et al. 2007b). Currie et al. (2009) reported a decrease in peripheral and central PWV versus baseline after a 6-day endurance exercise, but there was no significant difference in the augmentation index ($Aix@75$ bpm), HR and VO_2 max and reduced arterial stiffness was not found to be associated with pulse count and vascular permeability. Tordi et al. (2010) measured peripheral stiffness 30 minutes after intermittent and constant exercise and found that HR and PWV were lower after intermittent exercise. Intermittent exercise was associated with a stronger effect on vascular endothelial and vasoactive factors. McClean et al. (2011) looked at central PWV one hour after moderate aerobic exercise, however did not find any significant difference compared to the non-exercising group.

Table 3: Pulse wave velocity in athletes and controls. (Data are mean \pm SD)

	<i>Basketball players (n=10)</i>	<i>Weight lifters (n=11)</i>	<i>Controls (n=15)</i>
cb PWV (m/s)	5.12 \pm 0.61***	5.95 \pm 0.93*	6.83 \pm 0.86
cf PWV (m/s)	5.66 \pm 0.50***	5.89 \pm 0.77***	7.85 \pm 0.56

* $p < 0.05$ and *** $p < 0.001$ compared with controls.

PWV=Pulse wave velocity. cb: carotid-brachial cf: carotid-femoral

Many studies pointed out that chronic aerobic exercise reduced cardiovascular risk factors and mortality risk (Tanaka et al. 2000; Edwards et al. 2004; Edwards et al. 2005; Na et al. 2009). The present study found that carotid-brachial and carotid-femoral arterial stiffness were reduced in basketball players commonly performing aerobic training compared to sedentary controls. In parallel, the aortic stiffness index of basketball players was lower than that of weightlifters and sedentary subjects but not significant. Although aortic distensibility of basketball players and weightlifters was higher compared to sedentary controls it was not significantly different. Edwards and Lang (2005) purported that central arterial stiffness and augmentation index (Ai) of endurance athletes was lower compared to controls that exercised recreationally and was associated with decreased sympathetic tonus and endothelial improvement. Hayashi et al. (2005) observed a reduction in central PWV after 16 weeks of walking and jogging exercises performed by middle-aged men but did not see any significant difference in peripheral PWV. They reported that aerobic exercise might have this effect due to the amount of elastin in the arterial wall structure because central arteries had more elastin than peripheral arteries. Contrary to Hayashi (2005), Ashor et al. (2014) purported that peripheral arterial PWV was more affected than central PWV and the effect intensified following exercise of longer duration and intensity in a meta-analysis. Tanaka et al. (2000) reported that regular aerobic endurance exercise prevented reduction of central arterial compliance associated with age, reversed previous deterioration and decreased the risk for cardiovascular disease in middle and advanced aged men. Kakiyama et al. (2005) found that cycling for an hour 3-4 times per week at seventy percent VO₂ max intensity for 8 weeks reduced arterial stiffness in 8 weeks, however arterial stiffness returned to baseline after 8 weeks of absence of training. Some researchers suggested that aerobic exercise caused reduction of arterial stiffness by modifying the structural and functional factors in the walls, increasing expression of genes that act on factors stimulating vasodilation and reducing oxidative stress (Maeda 2005; Moreau 2005).

Tanaka et al. (2000) suggested that aerobic exercise reduced arterial stiffness independently regardless of conventional cardiovascular risk factors. In postmenopausal women exercise re-

portedly prevents increased central arterial stiffness, which is independent from total cholesterol and LDL cholesterol and also reduces cardiovascular morbidity risk (Tanaka et al. 1998). Again in postmenopausal women aerobic exercise was found to decrease the arterial stiffness index (Matsubara et al. 2014). Aerobic exercise reportedly decreases arterial stiffness in people with metabolic syndrome and reduces cardiovascular morbidity related risk factors (Donley et al. 1985).

Schmitz et al. (2001) found that habitual physical activity did not affect arterial distensibility, however regular physical activity might increase arterial distensibility. Vaitkevicius et al (1993) found that age related arterial stiffness increased to a lesser extent in those with higher VO₂ max. Laurent et al. (2011) compared central systolic blood pressure and aortic stiffness of aerobically trained athletes and sedentary controls and found that central arterial blood pressure of athletes was higher and their stiffness lower (Laurent 2011). Terenzi et al. (2000) identified a positive correlation between aerobic fitness and arterial compliance. In a recent study, Liu et al. (2015) compared the acute effects of a cycling exercise on carotid arterial hemodynamics between basketball players and sedentary controls. They found that carotid arterial stiffness was lower in the basketball group compared to sedentary controls at rest. Immediately following the cycling intervention, carotid arterial stiffness showed no obvious changes in the basketball group but significantly changed in the sedentary group. They concluded that long-term basketball exercise had a significant impact on common carotid arterial hemodynamics variables not only at rest but also after cycling interventions. As can be seen, studies have revealed that aerobic exercise reduced PWV and improved arterial compliance both in men and women. This study found a significant reduction of arterial stiffness in basketball players who predominantly exercised aerobically compared to sedentary controls, which is in parallel with previous study results.

Resistance Exercise

A study investigating the effect of acute resistance (75% RM) training on arterial stiffness found that 30 minutes after the exercise central arterial stiffness increased, arterial compliance

decreased and systolic arterial tension increased, however after 60 minutes these values returned to baseline (DeVan et al. 2005). Yoon et al. (2010) reported that 20 minutes after resistance exercise (60% RM) arterial stiffness, carotid femoral PWV and augmentation index increased but central and brachial blood pressure did not change. Central arterial stiffness has been found to increase due to blood pressure rising up to 310 to 250 mmHG during acute resistance exercise (MacDougall et al. 1986).

Many studies found that contrary to acute aerobic exercise, chronic resistance exercise increased central arterial stiffness (Miyachi et al. 2004; Miyachi et al. 2003; Cortez-Cooper et al. 2005; Kawano et al. 2006). Chronic resistance exercise is suggested to increase stiffness associated with increased smooth muscle content in the vessel walls and modified elastin and collagen load-bearing properties (MacDougall et al. 1986). Fahs et al. (2010) found a negative correlation independent from VO₂ max between upper extremity relative muscle strength and central PWV, whereas no correlation was shown between fPWV, AIx75, SBP vs DBP, MAP and BP. Miyachi et al. (2004) reported in a study including young men that resistance exercise performed 3 days a week for 4 months reduced aortic compliance, increased β stiffness index and in the absence of training these values returned to baseline, however brachial and carotid systolic tension and femoral arterial compliance and carotid intima media thickness did not change. Miyachi et al. (2003) found that resistance exercise reduced central arterial compliance of middle-aged men in a study including young and middle aged men. This value was even lower in resistance-trained men compared to sedentary controls and was concomitant with left ventricular hypertrophy, however peripheral arterial compliance did not change. Maeda et al. (2006) found that knee flexion and extension exercise performed by advanced age men in three sets twice a week for 12 weeks increased muscle strength however did not affect aortic PWV, increased plasma nitric oxide, however did not alter endothelin-1. Cortez-Cooper et al. (2005) found that high-intensity resistance exercise performed by young women increased cfPWV, did not change faPWV and increased carotid augmentation index, central arterial stiffness and arterial wave reflex. Rakobowchuk et al. (2005) showed that whole body resistance exercise performed by young men for

6 and 12 weeks increased brachial and carotid arterial pressures but did not alter carotid compliance, β stiffness index and intima media thickness and cardiac diameter.

Otsuki et al. (2007) reported increased central PWV, reduced compliance in subjects performing strength and endurance sports exercises versus sedentary controls. They showed that endothelin-1 concentration was directly proportional to arterial PWV and might contribute to increased stiffness in those that performed strength (endurance) exercise. There was no difference in nitric oxide values. Collier et al. (2008a) reported in a study that investigated the effects of resistance and aerobic exercise on pre-hypertensive and stage 1 hypertensive men and women that resistance exercise increased central and peripheral arterial stiffness, whereas aerobic exercise reduced stiffness and both types of exercise had the same effect on blood pressure. In addition, Collier (2008b) purported that resistance and endurance exercise affected gender differently in terms of arterial stiffness. Some studies show that resistance exercise increases central arterial stiffness although it has not been clearly elucidated yet (Ashor et al. 2014). Recently it was suggested that resistance exercise should be part of physical activity (Pollock 2000; Haskell 2007). Casey et al. (2007b) purported that moderate whole body (twice a week for 18 weeks) resistance exercise performed by postmenopausal healthy women did not alter central aortic wave reflex. Okamoto et al. (2006) said that 8-week long concentric exercises performed by young women increased PWV, however eccentric exercise did not have such an effect.

In middle and advanced age female and male rowers that performed resistance and endurance exercise, carotid arterial compliance was higher, beta stiffness index was lower compared to sedentary controls, however there was no significant difference in peripheral stiffness. The same study did not detect any significant difference in blood pressure, but carotid baroreceptor sensitivity increased parallel to compliance (Cook et al. 2006). Nualnim et al. (2011) found reduced arterial stiffness in swimmers that performed both aerobic and resistance training. Kawano et al. (2006) found reduced central arterial compliance especially in resistance trained subjects in a study including healthy men that performed resistance, aerobic and combined exercises 3 days a week for 4 months, however no significant difference

was found in aerobic and combined training. These results suggest that resistance exercise reduces central arterial compliance, which may be prevented by aerobic exercise and no significant difference may occur in peripheral arterial stiffness (Kawano et al. 2006).

Figueroa et al. (2011) reported that a moderate combined exercise program for 12 weeks for postmenopausal women positively affected arterial stiffness. In a recent study, Hoonjan et al. (2013) measured central and peripheral pulse wave velocity before and at 3, 15 and 30-minute intervals post-exercise (moderate intensity) in resistance trained athletes, endurance trained athletes and sedentary controls. They found no differences in resting pulse wave velocity values between the groups. Peripheral pulse wave velocity did not change significantly from respective baseline values in any group. However, central pulse wave velocity increased at 3 minutes post-exercise, followed by a decline to baseline within 15 minutes of exercise cessation. Hoonjan et al. (2013) concluded that the endurance trained and control group exhibited differences in their central arterial response to exercise compared to the resistance trained group, although peripheral arterial response was similar between the groups. They stated that for more pronounced differences, higher intensity and/or prolonged exercise was required.

This study did not show any increased arterial stiffness in weightlifters who performed strengthening exercises. To the contrary, the researchers found reduced stiffness. Nevertheless, the reduction was not statistically significant and was lower compared to basketball players. Unlike basketball players the aortic stiffness index of weightlifters was the same as sedentary controls. These results show that arterial stiffness may be affected independently from cardiac parameters as reported in literature. Moreover, decreased arterial stiffness may be caused by aerobic exercise that is part of the training program for weightlifters.

CONCLUSION

In conclusion, arterial stiffness improves in athletes that perform more aerobic training such as basketball players compared to sedentary controls. Contrary to existing knowledge arterial stiffness of athletes that perform more resistance exercises such as weightlifters did improve sig-

nificantly and did not get worse. This result implies that in the long-term arterial stiffness improves although arterial stiffness increases in the acute phase in sports activities that predominantly comprise of resistance exercises. Arterial stiffness improvement may be expected in both athlete groups. More comprehensive studies that investigate both the acute and the chronic effects of both exercise types on arterial stiffness need to be conducted.

RECOMMENDATIONS

Endurance and resistance training can be used to decrease arterial stiffness values effectively. The researchers' opinion is that regular exercise is mostly important for the protection of vessel health.

LIMITATIONS

The number of subjects reduces the power of this study. Another limitation was lack of VO₂ max and global muscle strength data of subjects. If this data was available, their aerobic and strength characteristics could have been elaborated. Another limitation was the lack of investigation of the acute effects of aerobic and resistance exercise on stiffness.

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