Original Article

Age-Related Reduction of Systemic Arterial Compliance Relates to Decreased Aerobic Capacity during Sub-Maximal Exercise

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A decrease in systemic arterial compliance (SAC) increases left ventricular load along with the demand for excessive myocardial oxygen consumption when the age-related reduction of SAC reaches a marked level, and consequently may depress left ventricular pump function. Reduced left ventricular pump function decreases aerobic capacity, and some study groups have shown that SAC and/or central arterial distensibility is correlated with maximal aerobic capacity in humans. We thus hypothesize that, once the age-related reduction of SAC reaches a marked level, the participation of SAC in aerobic capacity will be significant even during sub-maximal exercise. Thirty young humans and 46 elderly humans participated in this study. SAC, oxygen uptake at the ventilatory threshold ($\dot{V}O_{2\,VT}$), and the ratio of increase in oxygen uptake, in cardiac output, and in effective arterial elastance to increase in work rate ($\Delta\dot{V}O_2/\Delta$WR, ΔCO/ΔWR and $\Delta E_a/\Delta$WR) were measured. SAC was significantly higher in young subjects compared with elderly subjects, and was significantly related to $\dot{V}O_{2\,VT}$ in elderly subjects. SAC also significantly correlated with $\Delta\dot{V}O_2/\Delta$WR, ΔCO/ΔWR and $\Delta E_a/\Delta$WR in elderly subjects. When total subjects were divided by the value of SAC into 6 groups, the $\dot{V}O_{2\,VT}$ values in the 3 groups with lower SAC were significantly lower than those in the 3 groups with higher SAC, and gradually decreased with the reduction of SAC. There were no changes in $\dot{V}O_{2\,VT}$ among the 3 groups with higher SAC. These results suggest that the participation of SAC in aerobic capacity is significant even during sub-maximal exercise in individuals who show a pronounced age-related reduction of SAC. (*Hypertens Res* 2006; 29: 759–765)

***Key Words*: arterial distensibility, effective arterial elastance, left ventricular load, oxygen delivery function, ventilatory threshold**

Introduction

Large arteries, particularly central arteries (*e.g.*, the aorta and carotid artery), are distensible. Consequently, they are able to buffer the pulsatile systolic output of the ventricle (*1*), and their buffering function is usually described in terms of compliance (*1*), *e.g.*, total arterial compliance or systemic arterial compliance (SAC) (*2*, *3*). It has been well established that arterial compliance decreases with aging (*4–6*). A reduced

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Table 1. Risk Factors for Coronary Heart Disease

Risk factors	*n*	Mean±SD
Total cholesterol≥220 mg dl^{-1}	18	250±24
Triglycerides≥150 mg dl^{-1}	5	272±124
Glucose≥110 mg dl^{-1}	6	117±7
Systolic blood pressure≥140 mmHg	7	155±11
Diastolic blood pressure≥90 mmHg	1	91

Table 2. Subject Characteristics

	Young (n=30)	Elderly (n=46)
Age (years)	24.3±1.2	64.0±4.6*
Height (cm)	168±8	155±7*
Weight (kg)	63.0±9.0	55.5±7.5*
Body mass index (kg m^{-2})	22.3±1.9	23.1±2.7
Systolic blood pressure (mmHg)	110±10	128±16*
Diastolic blood pressure (mmHg)	63±9	74±9*
Heart rate (bpm)	68±10	71±10
Peak oxygen uptake (ml kg^{-1} min^{-1})	39.0±5.0	23.2±4.0*
Peak work rate (W)	243±52	106±21*

Values are means±SD. *p<0.05 *vs.* young subjects.

arterial compliance increases systolic blood pressure (SBP) (*7*), and consequently may enhance left ventricular (LV) afterload and may lead to increases in myocardial energy cost (*8*). Recently, we showed that an age-associated reduction of arterial compliance was significantly related to the increases in SBP and LV work load (*i.e.*, double product: SBP × heart rate) during sub-maximal aerobic exercise (*5*). However, the relationship of arterial compliance and double product remains not significant until the reduction of arterial compliance reaches a marked level (*5*).

Since an increased LV afterload deteriorates LV pump function (*9*) and this pump function is related to aerobic capacity (*10–12*), the reduced arterial compliance may decrease aerobic capacity. Indeed, central arterial compliance and/or distensibility has been shown to correlate with maximal aerobic capacity in young (*2*), middle-aged (*13*), and elderly humans (*14*, *15*), as well as in populations with a wide age range (*4*, *16*). We have previously demonstrated that arterial compliance was related to aerobic capacity at sub-maximal exercise levels (*i.e.*, oxygen uptake [$\dot{V}O_2$] at ventilatory threshold [VT], $\dot{V}O_{2\,VT}$) in elderly women (*17*). However, it is unclear whether arterial compliance would be related to aerobic capacity at sub-maximal exercise levels in a group with a wider age range. We thus hypothesize that, once the age-related reduction of SAC reaches a marked level, the participation of SAC in aerobic capacity will be significant even during sub-maximal exercise. In the present study, we measured SAC, aerobic capacity during sub-maximal exercise ($\dot{V}O_{2\,VT}$), and the ratio of increases in $\dot{V}O_2$ and in effective arterial elastance (E_a) to increase in work rate below the VT ($\Delta\dot{V}O_2/\Delta$WR and $\Delta E_a/\Delta$WR) as indices of oxygen delivery function (*10*, *18*, *19*) and LV afterload. Although $\Delta\dot{V}O_2/\Delta$WR is considered as an index of the function of oxygen delivery to the exercising muscles (*18*), the ability of the muscles to extract oxygen could also affect $\Delta\dot{V}O_2/\Delta$WR. Therefore, we also measured the ratio of the increase in cardiac output to the increase in work rate (ΔCO/ΔWR). Then, the relationships of SAC to $\dot{V}O_{2\,VT}$, $\Delta\dot{V}O_2/\Delta$WR, ΔCO/ΔWR, and $\Delta E_a/\Delta$WR were compared between elderly and young humans.

Methods

Subjects

Thirty young subjects (18 men and 12 women) and 46 elderly subjects (11 men and 35 women) volunteered to participate in this study. Elderly subjects without risk factors for coronary heart disease (n=17; serum total cholesterol 195±17 mg dl^{-1}, serum triglycerides 84±30 mg dl^{-1}, plasma glucose 95±5 mg dl^{-1}, blood pressure 125±11/71±9 mmHg) and with risk factors (n=29; 233±30 mg dl^{-1}, 125±89 mg dl^{-1}, 99±13 mg dl^{-1} and 129±19/75±8 mmHg, respectively; Table 1) were included in this study. Subjects were either sedentary or recreationally active, and were not involved in strenuous exercise training. All subjects were free from overt cardiovascular disease, and none of the participants smoked or took medication for hypertension, hyperlipidemia or hyperglycemia.

This study was approved by the Ethical Committee of the Institute of Health and Sport Sciences of the University of Tsukuba. The present study conformed to the principles outlined in the Helsinki Declaration and all subjects gave their written informed consent before participating.

Exercise Tolerance Test and Measurements

Subjects were examined after a rest in a quiet, temperature-controlled room during experimental sessions. After a more than 15-min rest period in a seated position, the subjects moved onto an electromagnetically braked cycle ergometer (232 CXL; COMBI WELLNESS, Tokyo, Japan) and underwent a symptom-limited (heart rate>[220 − age] bpm, subjects felt exhausted, or SBP>250 mmHg) exercise test. The cycle ergometer work rate protocol consisted of three phases: 2 min of rest then 4 min at 20 W followed by a ramp work rate (young men, 30 W min^{-1}; young women, 20 W min^{-1}; elderly men and women, 10 W min^{-1}). A beat-to-beat analogue waveform of finger arterial pressure was recorded using the volume clamp method (Portapres; BMEYE, Amsterdam, Netherlands) (*20*, *21*). The antialiasing filter in the Portapres has its −3 dB point at 100 Hz. After the antialiasing process, the analogue waveform was converted to a 200 Hz digital file (*22*). We further converted this file to a 100 Hz digital file using analysis software (Beatscope; BMEYE). Breath-by-

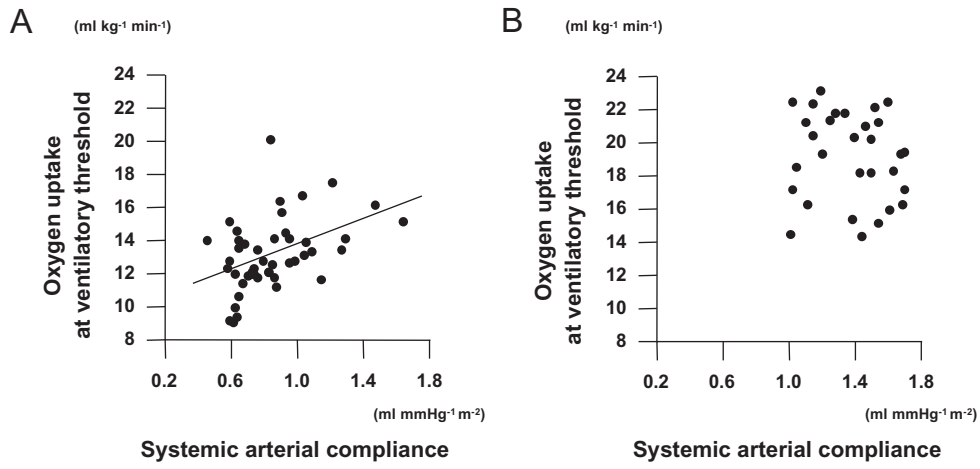

Fig. 1. *Relationships between systemic arterial compliance (SAC) and oxygen uptake at the ventilatory threshold ($\dot{V}O_{2\,VT}$). SAC was significantly related to $\dot{V}O_{2\,VT}$ in total subjects (*n = *76,* r = *0.65) and elderly subjects (A:* n = *46,* r = *0.44) but not in young subjects (B:* n = *30,* r = *−0.14).*

breath $\dot{V}O_2$ and carbon dioxide output were also measured (AE 300S; Minato Medical Science, Osaka, Japan).

Data Analysis

SAC was estimated as previously described by our laboratory (*5*). Briefly, the finger arterial waveform was transformed to a brachial arterial waveform using analysis software (Beatscope; BMEYE) (*20*, *21*), and the transformed waveform was used to calculate SAC (*23*, *24*). The stroke volume (SV) was estimated by means of the Modelflow method using Beatscope (BMEYE) (*25*, *26*). SAC was calculated using the area method (*3*) as follows:

$$SAC = SV \times \{(A_s + A_d) \times A_d^{-1}\}^{-1} \times (P_s - P_d)^{-1},$$

where A_s is the area under the arterial waveform during systole, A_d is that during diastole, P_s is the end-systolic pressure, and P_d is the end-diastolic pressure. The arterial waveform immediately before the start of ramp-fashion exercise (*i.e.*, at the end of the 20-W warm-up exercise) was used for the SAC calculation. Since physical size may affect SV, SAC was normalized for body surface area.

Mean values of $\dot{V}O_2$ and carbon dioxide output were calculated for every 15 s, and the VT was determined using the V-slope method (*27*). $\Delta\dot{V}O_2/\Delta WR$ was calculated by a linear regression of $\dot{V}O_2$ plots from 1 min after the start of ramping up to the VT point according to the method described in previous studies with minor modification (*10*, *19*). E_a can be calculated as follows (*28*):

$$E_a = 0.9 \times SBP \times SV^{-1}.$$

Brachial arterial SBP was used for calculation of E_a during exercise according to the previous studies (*29*, *30*). $\Delta E_a/\Delta WR$ and $\Delta CO/\Delta WR$ were also determined by a linear regression of mean values for every 15 s.

Statistics

Data are expressed as the means±SD. To evaluate differences between two groups, statistical analysis was carried out by an unpaired Student's *t*-test. Univariate correlation analyses were used to determine the relations between variables of interest. Total subjects were divided into 6 groups by the value of SAC, and the effects of SAC on age and $\dot{V}O_{2\,VT}$ were analyzed by one way ANOVA followed by Fisher's PLSD test for multiple comparisons. Values of $p<0.05$ were accepted as significant.

Results

Table 2 shows the physiological parameters in young and elderly subjects. Blood pressure was significantly higher in elderly subjects compared with young subjects. $\dot{V}O_2$ and work rate at peak exercise were markedly lower in elderly subjects than in young subjects.

SAC (elderly: 0.84±0.25 ml mmHg^{-1} m^{-2}; young: 1.36±0.23 ml mmHg^{-1} m^{-2}) and $\dot{V}O_{2\,VT}$ (13.2±2.2 *vs.* 19.2±2.7 ml kg^{-1} min^{-1}) were significantly lower in elderly subjects than in young subjects. Blood pressure at the measurement of SAC was higher in the elderly group than in the young group, and SBP was inversely correlated with SAC in total subjects ($r=-0.74$) and in the elderly ($r=-0.52$) and young groups ($r=-0.44$). SAC was significantly related to $\dot{V}O_{2\,VT}$ in total subjects and the elderly group, but not in young subjects (Fig. 1). Thus, aging reduced SAC and aerobic capacity, and the relation of SAC to aerobic capacity during sub-maximal exercise became more pronounced with aging.

In the comparison between elderly subjects without and

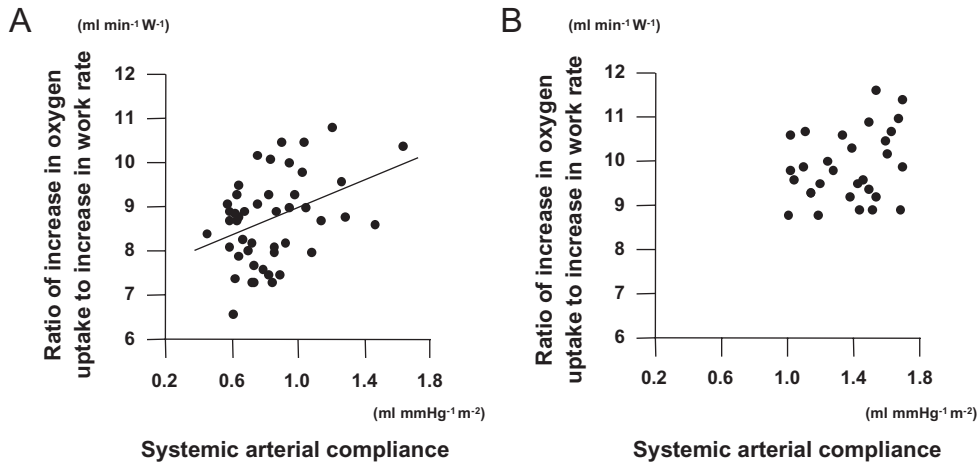

Fig. 2. *Relationships between systemic arterial compliance (SAC) and the ratio of increase in oxygen uptake to increase in work rate ($\Delta\dot{V}O_2/\Delta WR$). SAC was significantly related to $\Delta\dot{V}O_2/\Delta WR$ in total subjects (*n = *76,* r = *0.60) and elderly subjects (A:* n = *46,* r = *0.39) but not in young subjects (B:* n = *30,* r = *0.28).*

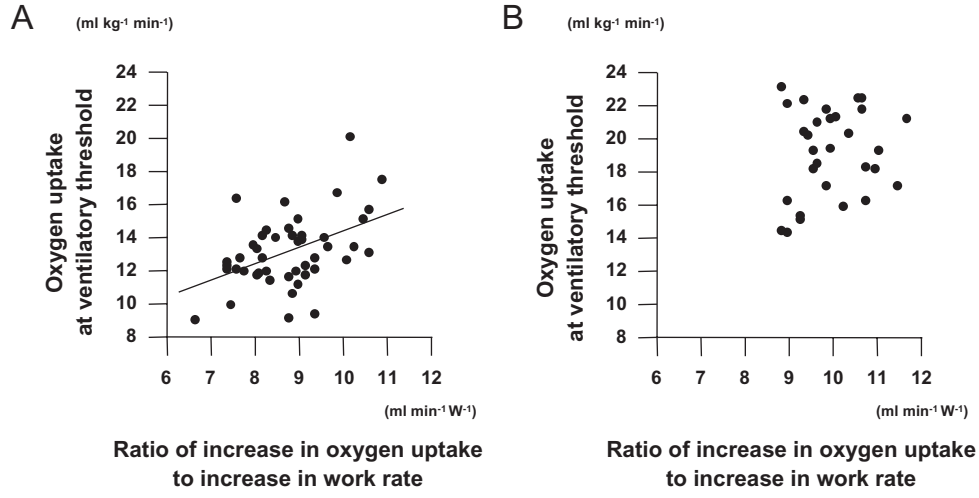

Fig. 3. *Relationships between the ratio of increase in oxygen uptake to increase in work rate ($\Delta\dot{V}O_2/\Delta WR$) and oxygen uptake at the ventilatory threshold ($\dot{V}O_{2\,VT}$). $\Delta\dot{V}O_2/\Delta WR$ was significantly related to $\dot{V}O_{2\,VT}$ in total subjects (*n = *76,* r = *0.60) and elderly subjects (A:* n = *46,* r = *0.46) but not in young subjects (B:* n = *30,* r = *0.17).*

with risk factors for coronary heart disease, we did not find statistically significant differences in SAC (without risk factors: 0.91±0.31 ml mmHg^{-1} m^{-2}; with risk factors: 0.80±0.20 ml mmHg^{-1} m^{-2}) and $\dot{V}O_{2\,VT}$ (13.3±1.8 *vs*. 13.2±2.2 ml kg^{-1} min^{-1}). In both groups, SAC tended to correlate with $\dot{V}O_{2\,VT}$ (r=0.66, p<0.05; r=0.35, p=0.06). Thus, the effects of risk factors were not statistically significant in this study.

$\Delta\dot{V}O_2/\Delta$WR was significantly lower in elderly subjects compared with young subjects (elderly: 8.7±1.0 ml min^{-1} W^{-1}; young: 9.9±0.8 ml min^{-1} W^{-1}). The relationships between SAC and $\Delta\dot{V}O_2/\Delta$WR are shown in Fig. 2. SAC was significantly related to $\Delta\dot{V}O_2/\Delta$WR in total subjects and in elderly subjects, but not in young subjects. Significant correlations also existed between $\Delta\dot{V}O_2/\Delta$WR and $\dot{V}O_{2\,VT}$ in total subjects and in elderly subjects (Fig. 3). ΔCO/ΔWR was lower in the elderly group than in the young group (40.1±12.6 *vs*. 53.4±16.0 ml min^{-1} W^{-1}). ΔCO/ΔWR was also related to SAC and $\dot{V}O_{2\,VT}$ in total subjects (r=0.54 and r=0.43) and in the elderly group (r=0.39 and r=0.36), but not in the young group (r=0.33 and r=−0.04). There was a significant difference in $\Delta E_a/\Delta$WR between elderly and young subjects (1.09±0.81 *vs*. 0.11±0.22 × 10^{-3} mmHg ml^{-1} W^{-1}). SAC correlated with $\Delta E_a/\Delta$WR only in total subjects and elderly subjects (Fig. 4). $\Delta E_a/\Delta$WR was also

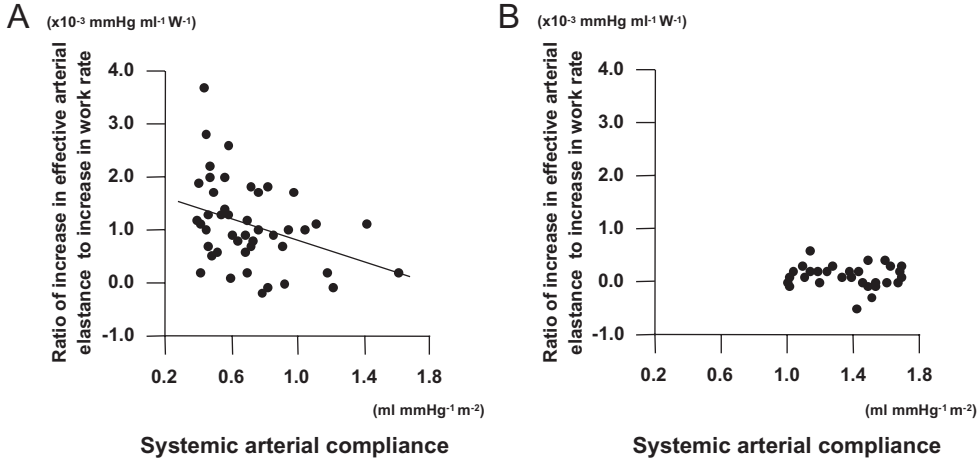

Fig. 4. *Relationships between systemic arterial compliance (SAC) and the ratio of increase in effective arterial elastance to increase in work rate ($\Delta E_a/\Delta WR$). SAC was significantly related to $\Delta E_a/\Delta WR$ in total subjects (*$n = 76$, $r = -0.60$*) and elderly subjects (A:* $n = 46$, $r = -0.36$*) but not in young subjects (B:* $n = 30$, $r = -0.08$*).*

significantly related to $\Delta\dot{V}O_2/\Delta WR$ and $\dot{V}O_{2\,VT}$ in total ($r = -0.61$ and $r = -0.60$) and elderly ($r = -0.53$ and $r = -0.43$) subjects but not in young subjects ($r = 0.04$ and $r = 0.24$).

Total subjects were divided by the value of SAC (ml $mmHg^{-1}$ m^{-2}) into group 1 (–0.70, $n = 16$), group 2 (0.71–0.90, $n = 16$), group 3 (0.91–1.10, $n = 14$), group 4 (1.11–1.30, $n = 10$), group 5 (1.31–1.50, $n = 9$) and group 6 (1.51–, $n = 11$), and then age and $\dot{V}O_{2\,VT}$ were compared among these groups. ANOVA revealed the significant effects of SAC on age and $\dot{V}O_{2\,VT}$. Age was significantly higher in the groups 1–2 than in the groups 3–6, and in the groups 3–4 than in the groups 5–6. $\dot{V}O_{2\,VT}$ were markedly lower in the groups 1–3 with lower SAC (12.2±2.0, 13.3±2.3 and 15.9±3.1 ml kg^{-1} min^{-2}) than in the groups 4–6 with higher SAC (18.6±4.1, 18.5±2.7 and 18.5±2.7 ml kg^{-1} min^{-2}). We also found significant differences in $\dot{V}O_{2\,VT}$ between the groups 1–2 and group 3.

Discussion

In the present study, we measured SAC and $\dot{V}O_{2\,VT}$ in young and elderly subjects. SAC was significantly lower in elderly subjects compared with young subjects, and was significantly related to $\dot{V}O_{2\,VT}$ in elderly subjects, but not in young subjects. Since it is well known that LV pump function is one of the important contributing factors to aerobic capacity (*11*, *12*), and since increased LV afterload deteriorates LV pump function (*9*), we also measured the oxygen delivery function to the working muscles (*i.e.*, $\Delta\dot{V}O_2/\Delta WR$) as an index of LV pump function and the increase rate of LV afterload (*i.e.*, $\Delta E_a/\Delta WR$) during sub-maximal exercise. SAC correlated with $\Delta\dot{V}O_2/\Delta WR$ and $\Delta E_a/\Delta WR$ in elderly subjects, but not in young subjects. The relationships among $\dot{V}O_{2\,VT}$, $\Delta\dot{V}O_2/\Delta WR$ and $\Delta E_a/\Delta WR$ were also significant only in elderly subjects. In addition, subjects were divided into 6 groups by the value of SAC, and age and $\dot{V}O_{2\,VT}$ were compared among these groups. With the decrease in SAC, age was significantly increased and $\dot{V}O_{2\,VT}$ was significantly decreased. However, we did not find statistically significant differences in $\dot{V}O_{2\,VT}$ among the groups 4–6 with higher SAC. On the other hand, $\dot{V}O_{2\,VT}$ in the groups 1–3 with lower SAC gradually decreased with reduction of SAC. These results suggest that the age-related reduction of SAC may significantly deteriorate LV oxygen delivery function with increasing LV afterload even during sub-maximal exercise, and consequently may decrease $\dot{V}O_{2\,VT}$ when the reduction of SAC reaches a marked level.

Previous studies that focused on maximal exercise level have demonstrated that SAC, central arterial compliance, and/or central arterial distensibility are related to maximal aerobic capacity. These studies either employed a population of subjects with a wide range of ages (*4*, *16*) or limited themselves to young (*2*), middle-aged (*13*) or elderly (*14*, *15*) individuals. In contrast, the present study focused on the sub-maximal exercise level and compared the relationship between SAC and aerobic capacity in elderly subjects to that in young subjects. As a result, we were able to show that SAC was significantly related to $\dot{V}O_{2\,VT}$ in elderly but not in young subjects. No subjects were involved in high performance athletic activities in the present study. Therefore, in young humans generally, it is possible that SAC may not significantly influence aerobic capacity during sub-maximal exercise, even though SAC might correlate with aerobic capacity during maximal exercise. The present study demonstrated that SAC correlated with $\dot{V}O_{2\,VT}$ in elderly humans. Thus, we considered that decreased SAC in elderly humans participates in age-related loss of aerobic capacity not only at maximal exercise intensity but also at sub-maximal exercise intensity.

$\Delta\dot{V}O_2/\Delta WR$ is relatively constant in normal humans, but the slope should be shallower than normal if the muscle is unable

to obtain oxygen due to inadequate oxygen delivery (*18*). Although there may be several theoretical reasons why this slope can be reduced, a reduction is most likely to be evident under the condition of impaired oxygen delivery to the exercising muscles (*18*). Oxygen delivery by the LV pump function plays an important role in aerobic exercise. The reserve in LV pump function is greater in young than in elderly humans (*31*), and the age-related decrease in this function is associated with a decreased aerobic capacity in elderly humans (*11*). In this study, SAC correlated with $\Delta\dot{V}O_2/\Delta$WR and $\dot{V}O_{2\,VT}$, and $\dot{V}O_{2\,VT}$ correlated with $\Delta\dot{V}O_2/\Delta$WR in elderly subjects with reduced SAC. In addition, we found that ΔCO/ΔWR was also related to SAC and $\dot{V}O_{2\,VT}$ in elderly humans. In elderly humans with a reduced cardiac reserve, the decreased SAC may be one of the factors contributing to deteriorating LV pump function, which in turn would cause a reduction in aerobic capacity even during sub-maximal exercise.

The interaction between the left ventricle and systemic arterial tree participates in the regulation of aortic blood flow. Elzinga and Westerhof (*9*) have demonstrated that an increased LV afterload reduces SV by using isolated hearts connected to a hydraulic model simulating the input impedance. In general, SAC may make a relatively small contribution to the LV afterload, because it has been reported that the ratio of pulsatile to total (steady + pulsatile) LV external work is 10% on average (*32*). Nevertheless, the ratio of the pulsatile work could be increased up to 50% with the decrease in distensibility (*32*), and a reduction of central arterial compliance increases SBP and depresses LV pump function (*33*). It has been shown that arterial distensibility decreases with age (*15*, *16*, *34–36*), and thus the contribution of SAC to LV afterload should not be ignored, particularly in elderly humans with markedly reduced arterial distensibility. It has also been reported that E_a during sub-maximal exercise (50% maximal exercise level) markedly increased in elderly humans although the increase in young humans was slight (*30*). In the present study, we found that the increase in E_a was significantly related to SAC and the oxygen delivery function during sub-maximal exercise in elderly subjects. The reduced SAC in elderly humans may increase LV afterload and thereby reduce LV pump function and aerobic capacity during sub-maximal exercise.

Most physical activities of daily living are done at the sub-maximal exercise level in the general population. It is considered that the decreased VT produces adverse effects when doing sub-maximal physical activity *via* the changes in safety and comfort (*27*, *37*). The prevention of age-related reduction of SAC would thus have important significance for quality of life, especially in elderly humans. In the present study, most elderly subjects belonged to the groups with lower SAC and $\dot{V}O_{2\,VT}$ (groups 1–3), but 6 elderly subjects belonged to the groups with higher SAC and $\dot{V}O_{2\,VT}$ (groups 4–6). Thus, it is considered that elderly humans can maintain or improve SAC and aerobic capacity under some physiological conditions. On the other hand, 6 young subjects belonged to the group with lower SAC and $\dot{V}O_{2\,VT}$ (group 3), suggesting that the reduction of SAC would be related to sub-maximal aerobic capacity even in young subjects when it becomes pronounced. It has previously been shown that aerobic exercise training increased arterial compliance and/or distensibility in elderly humans (*4*, *17*) and young humans (*2*, *38*). In addition, we have previously reported that the amount of daily physical activity was positively associated with arterial compliance and/or distensibility (*35*, *39*, *40*). Thus, we propose that increasing SAC by aerobic exercise training and/or increasing daily physical activity would improve aerobic capacity, and consequently would improve the quality of life in elderly and even in some young humans.

In conclusion, we showed that SAC was significantly lower in elderly subjects than young subjects, and that SAC positively correlated with $\dot{V}O_{2\,VT}$ in elderly subjects but not in young subjects. There were no differences in $\dot{V}O_{2\,VT}$ among the groups with higher SAC (groups 4–6), but $\dot{V}O_{2\,VT}$ gradually decreased with the reduction of SAC in the groups with lower SAC (groups 1–3). SAC significantly correlated with $\Delta\dot{V}O_2/\Delta$WR and $\Delta E_a/\Delta$WR only in elderly subjects. These results suggest that the age-related reduction of SAC is related to aerobic capacity even during sub-maximal aerobic exercise, but the effect is not significant until the reduction of SAC becomes pronounced. The deterioration in LV pump function by reduced SAC may be one of the factors decreasing the aerobic capacity during sub-maximal exercise, especially in elderly humans.

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