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Determinants of Brachial-Ankle Pulse Wave Velocity and Vascular Aging in Healthy Older Subjects

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Background: Vascular aging is characterized by increasing arterial stiffness as measured by pulse wave velocity. The present study evaluated the factors influencing vascular aging in Chinese healthy older subjects.





Material/Methods: Disease- and treatment-free aged (≥ 60 years) participants were recruited from 2014 to 2019. Cardiometabolic risk factors and brachial-ankle pulse wave velocity (baPWV) were assessed. We defined healthy vascular aging (HVA) as the lowest 10% and early vascular aging (EVA) as the highest 10% of the baPWV distribution, after adjustment for age and blood pressure (BP). We fitted linear and logistic regression models to assess the determinants.

Results: In all, 794 subjects (mean age 66.5 ± 6.8 years, 71.0% male) were recruited; the 10th and 90th percentiles of baPWV were 1278 cm/s and 1955 cm/s, respectively. Age, BP, heart rate, and triglycerides were all positively associated with baPWV, whereas male subjects and body mass index (BMI) were negatively associated with baPWV. The number of participants diagnosed with either HVA or EVA was 80. Logistic regression models showed that sex, BMI, heart rate, and triglycerides were associated with HVA and EVA after adjustment for age, BP, and other confounding factors.

Conclusions: Male, high BMI, low heart rate, and low triglycerides are protective factors for vascular aging in the healthy aged population. Management of BMI, heart rate, triglycerides in a reasonable range may help to alleviate the vascular aging process.

MeSH Keywords: **Aging • Pulse Wave Analysis • Vascular Stiffness**

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Background

Cardiovascular disease (CVD) has become the leading cause of death in China, and the increasing age of patients has further aggravated the burden of CVD [1]. Aging of the vasculature, resulting in high elastic artery stiffness and endothelial dysfunction, plays a central role in CVD in older people [2]. Clinically, arterial stiffness contributes to the pathophysiological process of hypertension, atherosclerosis, and stroke. Moreover, increased arterial stiffness has been regarded as the fundamental and vital link in the Vascular Aging Continuum [3]. The degree of arterial stiffness reflects the cumulative damaging effects of cardiovascular risk factors on the arterial wall with aging and has been used as an established hallmark of vascular aging [4–9].

Not all individuals undergo the vascular aging process in a uniform way. Like other clinical features, vascular aging also has a polygenic inheritance pattern and a multi-factorial etiology [10,11]. Vascular aging, evaluated by pulse wave velocity (PWV), progresses at different rates. Although there is no standard method for quantifying vascular aging [5–9], the core concept is that healthy vascular aging (HVA) means the lack of the age-related increase in arterial stiffness and can be diagnosed in subjects who present a lower PWV than the expected level for their age and blood pressure (BP). Conversely, early vascular aging (EVA), the opposite of HVA, can be diagnosed in subjects who present a higher PWV for their age and BP.

There is significant heterogeneity in the health trajectories among the elderly population, resulting in varying aging patterns, such as disease-free aging, or aging with increased risk for CVD or diabetes [12]. However, older participants enrolled in epidemiological studies are often affected by a variety of chronic diseases. To reduce the potential bias from disease and maximize the chance to capture the age-related differences, it is reasonable to select healthy older individuals according to strict criteria. Healthy aged subjects with HVA remain protected for a long period of time. We aimed to identify the factors protecting against vascular aging, owing to the high predictive value of vascular aging for cardiovascular events [4–6]. Another goal of our study was to assess the distribution of baPWV in the Chinese healthy aged population.

Material and Methods

This study was conducted at the physical examination center of the Geriatrics Department of Tongji Hospital and was approved by the Medical Ethics Committee. Healthy older subjects (aged ≥ 60 years), who underwent arterial stiffness assessment between January 2014 and October 2019 were consecutively recruited. We excluded individuals with hypertension (systolic

BP ≥ 140 mmHg or diastolic BP ≥ 90 mmHg, or use of antihypertensive drugs), diabetes [fasting glucose level of ≥ 7.0 mmol/L or glycated hemoglobin A1c (HbA1c) $\geq 6.5\%$, or use of hypoglycemic medications] [13], previous CVD, stroke, obvious arrhythmia (persistent atrial fibrillation, frequent premature beats, or wearing a pacemaker), cardiomyopathy, valvular heart disease, cancer, lung disease, severe chronic liver/kidney disease, and ankle-brachial index (ABI) less than 0.9 [14]. We also excluded patients receiving any medical treatment (e.g., lipid-lowering drugs, aspirin, antibiotics). Consequently, 794 participants were included in the analysis.

Trained personnel conducted standardized in-person interviews with the patients to collect information regarding their lifestyle, medical history, and medication use. Based on smoking status, the subjects were categorized as non-current smokers or current smokers. Anthropometric measurement (weight and height) and resting heart rate were also recorded. Body mass index (BMI) was computed as the weight in kilograms divided by the square of the height in meters. Fasting venous blood samples were collected and sent to the hospital's clinical chemistry laboratory to measure total cholesterol, triglycerides, low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), glucose, HbA1c, creatinine, and uric acid.

We evaluated arterial stiffness with brachial-ankle pulse wave velocity (baPWV). BaPWV is widely used to screen vascular damages in East Asian populations. BaPWV, BP, and ABI were measured using the Vascular Profiler BP-203RPEIII (Omron, Kyoto, Japan). The examination room was maintained at a standardized temperature. Trained technicians placed 4 pressure cuffs on the subjects (one on the upper part of each arm and one on each ankle). Then, the subjects were examined after 10 minutes of rest in the supine position. The device simultaneously recorded the bilateral systolic and diastolic BP, ABI, and baPWV. BaPWV was calculated as the ratio of the traveled distance (which was automatically estimated from the body height) divided by the transit time of the pulse wave between the brachial and posterior tibial arteries. The higher-side baPWV values were used for analysis [14]. Pulse pressure was calculated as the systolic BP minus the diastolic BP, and the mean arterial pressure was calculated as the diastolic BP plus the pulse pressure divided by 3. We adjusted baPWV by age and mean arterial pressure using a general linear model to calculate the adjusted values with the residual method. For this healthy older sample with normal BP, we defined HVA as the lowest 10% and EVA as the highest 10% of the adjusted baPWV distribution, and the rest of the subjects were categorized as the non-HVA group.

Data were analyzed using R 3.6.1. Continuous variables are presented as means \pm standard deviation (SD) or

Table 1. Characteristics of the study population by vascular aging status.

Variables	All (n=794)	HVA (n=80)	Non-HVA (n=634)	EVA (n=80)	p overall	p for interaction
Age, years	66.5±6.8	68.6±7.6	66.0±6.5*	68.0±7.7#	<0.001	0.76
Male, %	564 (71.0%)	61 (76.2%)	454 (71.6%)	49 (61.2%)	0.09	–
Smoker, %	154 (19.4%)	14 (17.5%)	131 (20.7%)	9 (11.2%)	0.12	–
BMI, kg/m ²	23.6±2.8	24.9±3.2	23.7±3.6*	23.1±3.9*	<0.001	0.20
Systolic BP, mmHg	123±10	124±9	123±10	126±10#	0.03	0.15
Diastolic BP, mmHg	72±8	74±7	72±8	73±8	0.04	0.55
Pulse pressure, mmHg	51±8	50±8	51±8	53±9	0.06	0.04
Mean arterial pressure, mmHg	89±8	91±7	89±8	91±8	0.03	0.57
Heart rate, beats/min	67±10	63±9	67±10*	75±10*.#	<0.001	0.08
Total cholesterol, mmol/L	4.59±0.87	4.54±0.92	4.59±0.86	4.60±0.90	0.86	0.15
Triglycerides, mmol/L	1.35±0.77	1.17±0.58	1.34±0.75	1.57±1.04*.#	0.004	0.93
HDL-C, mmol/L	1.25±0.32	1.23±0.30	1.26±0.31	1.26±0.42	0.82	0.86
LDL-C, mmol/L	2.85±0.75	2.87±0.84	2.86±0.74	2.72±0.74	0.26	0.06
Glucose, mmol/L	5.06±0.51	5.00±0.54	5.07±0.52	5.06±0.45	0.44	0.97
HbA1c, %	5.68±0.30	5.66±0.31	5.67±0.30	5.71±0.33	0.45	0.40
Uric acid, umol/L	347±83	355±80	346±83	342±86	0.56	0.43
Creatinine, umol/L	78.0±15.5	80.2±15.7	78.0±15.3	75.5±16.0	0.16	0.37
ABI	1.13±0.07	1.11±0.07	1.13±0.07*	1.13±0.09*	0.02	0.52
BaPWV, cm/s	1570±281	1320±155	1540±215*	2060±290*.#	<0.001	0.07

* p<0.05 vs. HVA; # p<0.05 vs. Non-HVA.

median (interquartile range), as appropriate for distribution. Categorical variables are shown as count (percentage). The normal distribution was tested using the probability density function. The difference in baPWV between males and females was assessed by *t* tests. Correlations between age, mean arterial pressure, and baPWV were assessed by Pearson's correlation. A linear regression model was fitted to identify the independent determinants of baPWV; total cholesterol was excluded to avoid the effect of multicollinearity. We compared the variables between vascular aging status using ANOVA or chi-squared test and also did post hoc pairwise comparisons. We performed multivariable logistic regression analysis to determine the independent influencing factors for HVA and EVA, resistively. Since the residuals which were used to define vascular aging represented the fraction of baPWV that could not be explained by age and mean arterial pressure, we did not add those 2 variables in the models, and total cholesterol was also excluded. Only variables staying in the final model were presented. Give the sex imbalance, we also tested for

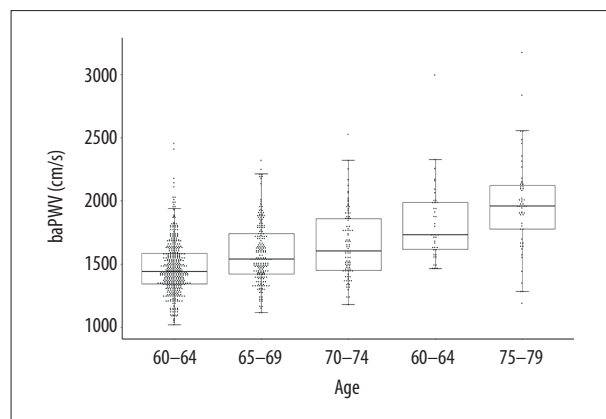


Figure 1. The distribution of baPWV by different age groups in healthy older subjects.

Table 2. Reference values of baPWV (cm/s) by different age groups of the healthy older sample.

Age groups	Min-max	Means±SD	Percentiles		
			10%	50%	90%
60-64 (n=412)	1020-2460	1470±210	1244	1441	1731
65-69 (n=193)	1120-2320	1590±242	1311	1538	1903
70-79 (n=134)	1180-3000	1710±285	1387	1662	2052
≥80 (n=55)	1190-3180	1970±367	1562	1960	2417
All (n=794)	1020-3180	1570±281	1278	1518	1955

Table 3. Linear regression analysis of determinants of baPWV.

Variables	β	SE	p
Age, years	18.2	1.2	<0.001
Male, %	-48.9	15.2	0.001
BMI, kg/m ²	-15.6	3.0	<0.001
Mean arterial pressure, mmHg	13.0	1.0	<0.001
Heart rate, beats/min	6.7	0.8	<0.001
Triglycerides, mmol/L	26.6	10.7	0.01
Adjusted R ²	0.48		

interactions with sex by entering corresponding cross-product terms into the models. Two-tailed P values less than 0.05 were considered significant.

Results

Study population and reference values for baPWV

In all, 794 healthy older subjects (mean age 66.5±6.8 years, 71.0% male) were recruited and the characteristics are presented in Table 1. The mean value of baPWV was 1570±281 cm/s, and there was no significant difference between males and females (1570±280 cm/s vs. 1580±284 cm/s, p=0.82). The baPWV value was positively associated with age (r=0.52, p<0.001) and mean arterial pressure (r=0.38, p<0.001). The baPWV distribution stratified for the age of this healthy older sample is shown in Figure 1 and the exact reference values according to age categories are presented in Table 2. The 10th and 90th percentiles of baPWV of the sample were 1278 cm/s and 1955 cm/s, respectively. This could help doctors judge the vascular aging status in the aged population.

Table 4. Determinants of HVA and EVA as compared with the control group by multivariable logistic regression analysis.

Variables	OR	95% CI	p
HVA			
Male	2.36	1.24-4.71	0.01
BMI, per kg/m ²	1.22	1.05-1.42	0.01
Heart rate, per beats/min	0.85	0.80-0.90	<0.001
Triglycerides, per mmol/L	0.38	0.17-0.71	0.01
EVA			
Male	0.65	0.44-0.96	0.03
BMI, per kg/m ²	0.91	0.82-0.99	0.04
Heart rate, per beats/min	1.08	1.06-1.11	<0.001
Triglycerides, per mmol/L	1.41	1.07-1.84	0.01

Linear regression analysis of determinants of baPWV

Using baPWV as the dependent continuous variable in multiple linear regression, age, BP, heart rate, and triglycerides were all positively associated with baPWV, whereas male subjects and BMI were both negatively associated with baPWV after adjusting for confounding factors, as illustrated in Table 3.

Factors associated with HVA and EVA

Eighty participants were diagnosed with HVA and the other 80 participants were diagnosed with EVA. Characteristics of the study population by vascular aging status are also presented in Table 1. The unadjusted baPWV values were significantly different among the HVA, the non-HVA, and the EVA groups. In contrast with those in the HVA group, individuals without

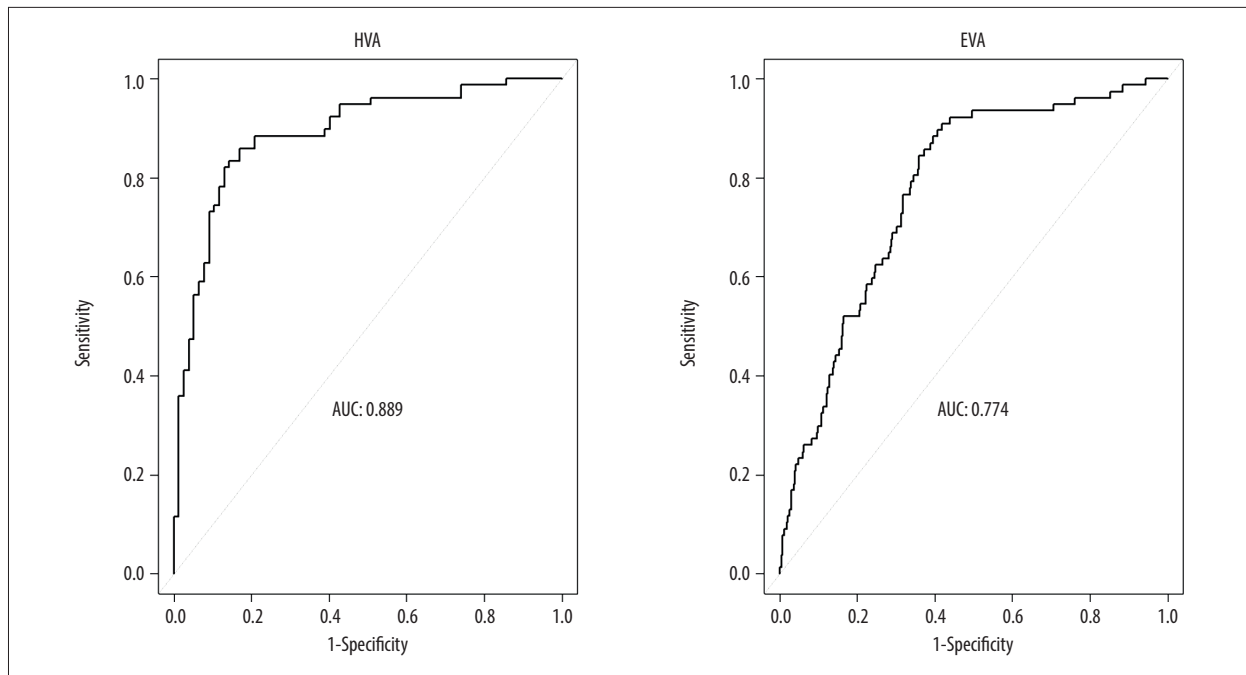


Figure 2. Receiver operating characteristic curves for the multivariable logistic models in predicting the presence of HVA or EVA. The independent variables of the 2 models included sex, BMI, heart rate, and triglycerides.

HVA had significantly lower BMI and younger age, and higher heart rate and ABI; while subjects with EVA had lower BMI and higher heart rate, triglycerides, and ABI. No statistically significant interactions were observed between sex and any of the variables except for blood pressure.

In the logistic regression models (Table 4), we observed that male [OR (95% CI) 2.36 (1.24–4.71), $p=0.01$], higher BMI [OR (95% CI) 1.22 (1.05–1.42), $p=0.01$], lower heart rate [OR (95% CI) 0.85 (0.80–0.90), $p<0.001$], and lower triglycerides [OR (95% CI) 0.38 (0.17–0.71), $p=0.01$] were all associated with a higher likelihood of having HVA. The area under the receiver operating characteristic curve (AUC) for the multivariable logistic models in predicting the presence of HVA was 0.89 is illustrated in Figure 2. Female sex [male OR (95% CI) 0.65 (0.44–0.96), $p=0.03$], lower BMI [OR (95% CI) 0.91 (0.82–0.99), $p=0.04$], higher heart rate [OR (95% CI) 1.08 (1.06–1.11), $p<0.001$], and higher triglycerides [OR (95% CI) 1.41 (1.07–1.84), $p=0.01$] added excessive risk to EVA; and the AUC for the model was 0.77 in predicting the presence of EVA. Similarly, there was no significant interaction between sex and any of the significant correlates of HVA and EVA in the multivariable models.

Discussion

Arterial stiffness, as measured by baPWV, is one of the major age-related arterial phenotypes, and is an independent predictor of future CVD in older adults [15]. While baPWV has been

widely applied in clinical work, investigations of the reference values of baPWV are limited, especially for healthy aged individuals, and the absence of reference values hampers the understanding of its implications. At present, individuals with a baPWV level greater than 1400 cm/s are considered to have arterial stiffness [16–18]; however, this is unreasonable, since the baPWV values are greatly influenced by age and BP. Just like carotid-femoral PWV [19], practical reference values of baPWV should be established according to age, BP, and other cardiovascular risk factors. Previous similar studies have been carried out in other areas of China [20–22], but the numbers of the healthy aged people included were small. Our study only focused on healthy older subjects with normal BP, and the sample size was relatively large. We provided reliable reference values for the healthy aged population of central China.

There are still no generally accepted methods for quantifying vascular aging. While PWV was used in most studies, other structural and functional parameters of arteries also provide information about vascular aging, such as augmentation index, artery intima-media thickness, or coronary artery calcium score [23,24]. Understanding of vascular aging is far from sufficient. A variety of factors (e.g., arterial hypertension, cardiometabolic factors, adiposity, unhealthy lifestyle) have all been linked to vascular aging in previous studies [25,26]. We tried to determine the modifiable determinants of vascular aging for the healthy aged population, especially those with HVA. After adjustment for age and BP, we found that sex, BMI, heart rate, and triglycerides were all independently associated with both HVA and EVA.

In the healthy aged subjects, high BMI was found to be a protective factor for vascular aging, even when adjusted for age, BP, and other confounding factors. Our findings are quite similar to the research by Yang et al., which also found a negative correlation between BMI and baPWV in 414 elderly subjects [27]. Moreover, a similar relationship also existed in other demographic groups, such as youth [28] and individuals with hypertension [29]. Increased blood volume and larger vessel inner diameter of the subjects with higher weight might be a potential mechanism. Numerous studies reported that obese individuals often had a lower CVD and mortality risk than normal-weight individuals, a phenomenon termed the “obesity paradox”; and this was also elucidated in the elderly population [30–32]. The specific mechanisms are unclear, and it may be related to the greater muscle mass and muscular strength, earlier presentation of heavier patients, greater likelihood of receiving optimal medical therapy, and so on [30,31]. We demonstrated that those with higher BMI had lower baPWV (i.e., younger arteries with greater elasticity) than those with normal BMI, and this might be a vascular mechanism that contributes to the “obesity paradox”.

The sex differences in the time course of aging-related arterial stiffness have been shown in previous studies, and the rate of change in PWV was higher in females [33,34]. We further confirmed that aged males were more likely to have HVA and less likely to have EVA compared with aged females. Low heart rate and triglycerides also played a protective role in vascular aging of the healthy aged population. The value of triglycerides in predicting arterial stiffness was also found in other studies [35–37]. However, ApoA5-1131T>C polymorphism was not associated with increased baPWV, and this suggests that the association between circulating triglyceride level and baPWV may reflect confounding and reverse causation [38]. Longitudinal studies are needed to confirm the causal effect. The independent influence of resting heart rate on arterial stiffness remained controversial since results were confounded by

BP and other confounding factors [39,40]. In our study, one beat per minute increase in heart rate led to a 15% decrease in the likelihood of having HVA and an 8% increase in the likelihood of having EVA independently. Moreover, long-term resting heart rate pattern was also proven to predict the risk of having arterial stiffness [17].

We have some limitations to consider. First, all the findings were based on analyses of healthy aged people and may not apply to younger individuals or to those at high risk for CVD. Second, this study was conducted at a single center, resulting in a sample that was comprised of more males than females. Third, subjects with untreated dyslipidemia were not excluded since they accounted for 54% of the sample [41]. In addition, a study of patients with heart failure demonstrated that patients with the lowest baPWV values had the highest frequency of heart failure-related events [42]. However, its study population was quite different from the healthy elderly. The follow-up of the individuals with very low baPWV value in our study has important value.

Conclusions

In conclusion, in this study, we describe the distribution of baPWV in a Chinese healthy older sample. We also show that after adjustment for age and BP, vascular aging is associated with several traditional cardiovascular risk factors. Male sex, high BMI, low heart rate, and low triglycerides are the most significant protective factors. Management of BMI, heart rate, and triglycerides in a reasonable range may help to alleviate the vascular aging process of healthy older adults.

Conflict of interest

None.

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