Original Article

Blood flow velocity in the carotid artery is associated with peripheral diastolic blood pressure and pulse pressure

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Abstract: Patients with atherosclerotic cerebrovascular disease appear to have larger common carotid arterial diameters and lower carotid flow velocities compared to those without the disease. The index of arterial stiffness is strongly related to the extent of atherosclerosis and cardiovascular events. The objective of this study was to elucidate the association between morphologic or hemodynamic parameters of the carotid artery and the vascular hemodynamic parameters of patients without atherosclerotic cardiovascular disease (ASCVD). From patients who underwent carotid ultrasonography and non-invasive, semi-automated, radial artery applanation tonometry (Omron HEM-9000AI) in the Department of Internal Medicine at St. Vincent's Hospital from July 2011 to June 2015, 388 subjects (195 male, mean age, 55±12 years) without ASCVD were enrolled in this study. After multivariate analysis, diastolic blood pressure (BP) (β=-0.231, 95% Cl -0.003 to 0.000, P=0.019) and peripheral pulse pressure (PP) (β=0.230, 95% CI 0.000 to 0.003, P=0.016) were found to be associated with the resistance index after adjusting for age, gender, body mass index, fasting plasma glucose, HbA1c, smoking, diabetes, hypertension and use of antihypertensive medication and lipid-lowering therapy. Additionally, diastolic BP (β=-0.189, 95% Cl 0.001 to 0.208, P=0.047) was correlated with end-diastolic velocity. After multivariate analysis, Intima-media thickness and diameter were associated with several vascular hemodynamic parameters. Blood flow velocity of the carotid artery influences peripheral BP or PP rather than the index of arterial stiffness. However, morphological parameters are associated with arterial stiffness and systemic hemodynamic factors. Although further studies are required, multiparametric assessment might contribute to a better understanding of the structural, functional, and hemodynamic changes of the carotid artery.

Keywords: Blood flow velocity, carotid artery, arterial stiffness, atherosclerotic cardiovascular disease

Introduction

Increased arterial intima-media thickness (IMT) and presence of carotid plaque have been positively associated with atherosclerotic cardiovascular disease (ASCVD), and these results have been used to improve predictions for diagnosis of ASCVD [1-4]. ASCVD is a descriptive term rather than a specific diagnosis and is applied to patients with a history of acute coronary syndrome, myocardial infarction, stable or unstable angina, coronary or other arterial revascularization procedures, stroke, or peripheral arterial disease (PAD) presumed to be of atherosclerotic origin [5]. Increased IMT is also associated with increased arterial stiffness. In a previous study, increased brachial-ankle pulse wave velocity (PWV) and carotid femoral PWV were strongly associated with development of atherosclerosis and increased risk of CV events and mortality [6]. Additionally, the Strong Heart Study showed that non-invasively determined central blood pressure (BP), another index of arterial stiffness, was strongly correlated with extent of atherosclerosis and incidence of CV events [7].

The structure and function of the carotid artery are interrelated with local and systemic hemodynamic characteristics. Although carotid morphologic parameters (carotid diameter and IMT) are conventionally examined simultaneous to hemodynamic parameters in the carotid artery, few studies have considered all of these morphological, functional, and hemodynamic parameters. In previous cross-sectional stud-

ies, patients with atherosclerotic cerebrovascular disease appeared to have larger common carotid arterial diameters and lower carotid flow velocities [8]. These authors also reported that low common carotid end diastolic velocity (EDV) and peak systolic velocity (PSV) were independently associated with ischemic heart disease and stroke [9]. However, the relationships between these hemodynamic parameters of the carotid artery and risk factors for ASCVD have not gained much attention. Furthermore, the relationships between blood flow velocity in the carotid vessels and the indices of arterial stiffness according to CV risk factors have not yet been fully established.

The objective of this study was to identify the associations between morphologic or hemodynamic parameters of the carotid artery and vascular hemodynamic factors in patients without ASCVD.

Methods

Study population

The study population consisted of subjects referred for carotid artery ultrasonography between July 2011 and June 2015. Among subjects who simultaneously underwent carotid artery ultrasonography and non-invasive, semiautomated, radial artery applanation tonometry (HEM-9000AI; Omron Healthcare, Kyoto, Japan) in the Department of Internal Medicine at St. Vincent's Hospital, 388 (195 male, mean age, 55±12 years) were enrolled in this study. Exclusion criteria included current atrial fibrillation (because radial tonometry is not accurate in these patients), major systemic illness (e.g., chronic inflammatory disease, active malignancy), and a history or findings of ASCVD including acute coronary syndrome, myocardial infarction, stable or unstable angina, coronary or other arterial revascularization, stroke, or PAD (brachial-ankle index \leq 0.9, using VP-1000; Omron Healthcare, Kyoto, Japan). There was no industry involvement in the design, performance, or analysis of the study. The Ethics Committee of the hospital approved the use of clinical data for this study, and all patients provided written informed consent.

Assessment of intima-media thickness

The morphologic and hemodynamic parameters of the common carotid arteries were evalu-

ated using a Vivid Seven ultrasound machine (GE Medical Systems, Horten, Norway) with a linear array transducer frequency of 12-MHz and a color Doppler frequency of 6.5-MHz. With the patients in the supine position with slight hyperextension and rotation of the neck to the contralateral side in order to optimize image quality, depth control was fixed at 4 cm. Ultrasonographic images were scanned at end diastole (defined as the R wave of an electrocardiogram). The mean IMT of the common carotid artery (CCA) was measured over a 1-cmlong segment of the CCA that was considered to not contain any plaque (i.e., no perceivable protrusion of the artery wall into the lumen) [10]. The intra- and inter-observer reliabilities for the IMT measurements were 0.965 and 0.900, respectively. Inner diameter (InDIA) was defined as the distance between the leading edge of the echo produced by the intima-lumen interface of the near wall and the leading edge of the echo produced by the lumen-intima interface of the far wall. The outer diameter (OutDIA) was the mean of the maximal distance between the adventitia-to-periadventitia interface and the periadventitia-to-adventitia interface on the far wall at the point of the beginning of the dilation of the bifurcation bulb [11].

PSV and EDV were measured using continuouswave Doppler examination in the CCA 2-4 cm proximal to the bifurcation. For all participants, the scan-head was applied longitudinally for at least three cardiac cycles to obtain blood flow velocity measurements. Of the consequence of initial forward flow, the highest velocity during systole was identified as the PSV, and the lowest velocity during diastole was defined as the EDV. The highest value of the minimum of three measurements was recorded. In spectral analysis, falsely high values caused by the post stenotic jet or low insonation angles far cranially were excluded. The resistance index (RI) was calculated as (PSV-EDV)/PSV. The minimal EDV and PSV and the maximal RI in the CCA were used in this study.

Measurement of central and brachial blood pressure

After at least 10 min of rest and with the subject seated, the brachial BP was measured using an HEM-907 automatic cuff oscillometric device (Omron Healthcare). The average of two readings was used to determine systolic BP,

Table 1. Baseline characteristics of the 388 participants without atherosclerotic cardiovascular disease

Age, years 55±12 Male, number (%) 195 (50.3) Weight, kg 66.18±12.7 Height, cm 163±11 Body Mass Index, kg/m² 24.7±3.5 Faction algorithms of larger of larger and (dl.) 136.151
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Fasting plasma glucose, mg/dL 126±51
Hemoglobin A1c, % 7.7±2.2
Total Cholesterol, mg/dL 198±42
Triglyceride, mg/dL 146±132
High Density Lipoprotein cholesterol, mg/dL 45±11
Low Density Lipoprotein cholesterol, mg/dL 111±37
Smoking, number (%) 133 (34.3)
Diabetes mellitus, number (%) 143 (36.9)
Hypertension, number (%) 182 (46.9)
ACEI or ARB (%) 102 (26.3)
Calcium-channel blockers (%) 82 (21.1)
Diuretics (%) 28 (7.2)
Beta-blockers (%) 21 (5.4)

Table 2. Arterial hemodynamic parameters of the 388 participants without atherosclerotic cardiovascular disease

	All participants (n=388)
Peripheral, mmHg	
Systolic Blood Pressure, mmHg	133±18
Diastolic Blood Pressure, mmHg	80±12
Pulse Pressure, mmHg	53±14
Pulsatile Stress	3855±1169
Heart Rate, beats/min	73±12
Central	
Late systolic peak pressure (SBP2), mmHg	122±20
Radial Augmentation Index, %	79±13
Pulse Pressure, mmHg	42±15
Pulsatile Stress	3030±1010
Common carotid artery features	
Maximal Intima-media thickness, mm	0.84±0.26
Mean Intima-media thickness, mm	0.69±0.27
Outer diameter, mm	7.56±3.36
Inner diameter, mm	6.11±3.01
Peak-systolic velocity, cm/s	67.5±18.4
End-diastolic velocity, cm/s	19.7±7.0
Resistive index	0.71±0.08

diastolic BP, mean arterial pressure, pulse pressure (PP), and pulsatile stress (PS).

Peripheral PS was defined as the product of heart rate and peripheral PP. Next, the radial pulse wave was recorded using a model HEM-9010Al automated applanation tonometer (Omron Healthcare). The device consists of a sensor unit, pulse measurement unit, and personal computer. The watch-shaped sensor unit has a pressure sensor with an array of 40 microtransducer elements on its inner surface. Once the sensor is applied on the left wrist over the radial artery, the device automatically flattens the artery, adjusts the applanation hold-down pressure, and selects an optimal sensing element to appropriately record the pulse wave. The obtained pressure signals are digitized at 500 Hz inside the pulse measurement unit and then transmitted to the personal computer [12]. In this study, radial arterial pressure waveforms were recorded for 30 s. Radial augmentation index (RaAlx) was calculated as follows: [(second peak radial systolic pressure (SBP2)-diastolic pressure)/(first peak radial systolic pressurediastolic pressure)*100] [13]. Central PS was calculated as the product of heart rate and radial PP.

Systemic hypertension was defined as systolic pressure \geq 140 mmHg and/or diastolic pressure \geq 90 mmHg based on more than three measurements or as current use of antihypertensive drugs.

Clinical and biochemical assessment

Blood specimens were collected after a 12- to 14-hour fast (8:00 p.m. to 9:30 a.m.) in order to reduce the influence of circadian variation. Total cholesterol (TC) and triglyceride (TG) concentrations were measured using standard enzyme methods. High-density lipoprotein (HDL) cholesterol was measured after precipitation of very low-density lipoprotein and lowdensity lipoprotein (LDL) with phosphotungstic acid, and LDL was calculated using the Friedewald formula. Fasting glucose level was enzymatically determined using the hexokinase method. A blood sample from every patient was drawn and centrifuged within 30 min. The serum samples were stored at -80°C, and high

sensitivity C-reactive protein (hs-CRP) was determined using an immunoturbidity assay

Blood flow velocity in the carotid artery

Table 3. Correlation coefficients between cadiovascular risk factors and intima-media thickness (IMT), outer diameter (OutDIA), inner diameter (InDIA), peak systolic velocity (PSV), end-diastolic velocity (EDV), and resistance index (RI)

(-1)	IMT	OutDIA	InDIA	PSV	EDV	RI
Age, years	0.225**	0.046	0.027	-0.342**	-0.317**	0.123**
Body Mass Index, kg/m ²	0.025	0.025	0.012	0.048	-0.006	0.062
Fasting plasma glucose, mg/dL	-0.036	-0.027	-0.033	0.094	-0.066	0.158**
Hemoglobin A1c, %	-0.131	-0.189*	-0.185*	0.132	-0.048	0.211**
Total Cholesterol, mg/dL	-0.057	0.008	0.012	0.033	0.071	-0.041
Triglyceride, mg/dL	-0.053	0.035	0.059	0.017	0.042	-0.078
High Density Lipoprotein cholesterol, mg/dL	-0.116*	-0.021	-0.023	-0.105	-0.006	-0.068
Low Density Lipoprotein cholesterol, mg/dL	0.068	-0.004	-0.022	0.095	0.11	-0.024
Systolic Blood Pressure, mmHg	0.125*	0.009	0.008	-0.145**	-0.147**	0.065
Diastolic Blood Pressure, mmHg	-0.021	-0.004	0.004	-0.059	0.120*	-0.226**
Peripheral Pulse Pressure, mmHg	0.190**	0.012	-0.004	-0.152**	-0.324**	0.311**
Peripheral Pulsatile Stress	0.137**	-0.027	-0.019	-0.104**	-0.206**	0.190**
Heart Rate, beats/min	-0.051	-0.057	-0.017	0.02	0.113*	-0.152**
Late systolic peak pressure (SBP2), mmHg	0.118*	0.018	0.011	-0.191**	-0.101*	-0.037
Central Pulse Pressure, mmHg	0.176**	0.027	0.012	-0.207**	-0.235**	0.138**
Central Pulsatile Stress	0.156**	-0.004	-0.002	-0.178**	-0.176**	0.079
Radial Augmentation Index, %	0.087	0.034	0.027	-0.183**	0.029	-0.205**
IMT, mm		0.097	0.052	0.071	0.032	0.052
OutDIA, mm	0.097		0.966**	-0.055	-0.077	-0.046
InDIA, mm	0.052	0.966		-0.034	0.081	-0.168**
PSV, cm/s	0.071	-0.055	-0.034		0.699**	0.035
EDV, cm/s	0.032	-0.007	0.081	0.699**		-0.686**
RI	0.052	-0.046	-0.168**	0.035	-0.686**	

^{**}p< 0.01, *p< 0.05.

system (Liatest; Stago, Asnieres sur Seine, France), with an interassay variability coefficient of variation of 6.25%.

Statistical analyses

Continuous variables are presented as mean ± standard deviation, and categorical variables are presented as absolute and relative frequencies (%). Pearson's correlation was used to determine the associations between morphologic and hemodynamic parameters of the carotid artery, central hemodynamics, and risk factors at baseline. The independent predictors of morphologic and hemodynamic parameters were examined with multivariate regression analysis after adjustment. All reported p-values were two-sided, and p-values ≤ 0.05 were considered to indicate statistical significance. All statistical analyses were conducted in SAS 9.1 statistical software (SAS Institute, Cary, North Carolina, USA).

Results

Baseline clinical characteristics are shown in **Table 1**. The mean age of the study subjects was 55±12 years, and 195 (50.3%) subjects were men. Some patients demonstrated hypertension [HBP (n=182, 46.9%)], diabetes mellitus [DM (n=143, 36.9%)], or smoking (n=133, 34.3%). Angiotensin converting enzyme (ACE) inhibitors or angiotensin receptor blockers (n=102, 26.3%), calcium channel blockers [CCB (n=82, 21.1%)], diuretics (n=28, 7.2%), or betablockers (n=21, 5.4%) were prescribed in patients without ASCVD.

Table 2 shows the peripheral and central hemodynamic parameters and the morphologic and hemodynamic parameters of the carotid artery.

Table 3 shows the correlation coefficients between traditional risk factors, peripheral and central hemodynamic parameters, and morphologic and hemodynamic parameters of the

Table 4. Associations between morphologic parameters of common carotid artery and index of peripheral or central hemodynamics using multivariate analysis

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Dependent variable	Independent variable	β	95% CI	p value			
Outer Diameter	SBP	0.248	0.005 to 0.025	0.004			
	DBP	0.201	0.001 to 0.035	0.039			
	Peripheral PP	0.213	0.002 to 0.030	0.023			
	Peripheral PS	0.198	0.000 to 0.001	0.031			
	SBP2	0.231	0.003 to 0.022	0.009			
	Central PP	0.197	0.000 to 0.028	0.044			
	Central PS	0.245	0.000 to 0.001	0.013			
Inner Diameter	SBP	0.229	0.004 to 0.031	0.012			
	DBP	0.225	0.003 to 0.051	0.026			
	Peripheral PS	0.234	0.000 to 0.001	0.014			
	SBP2	0.222	0.003 to 0.029	0.017			
	Central PS	0.267	0.000 to 0.001	0.009			
IMT	SBP	0.193	0.000 to 0.006	0.036			
	Peripheral PP	0.288	0.002 to 0.011	0.003			
	Peripheral PS	0.325	0.000 to 0.001	0.001			
	SBP2	0.196	0.000 to 0.003	0.047			
	Central PS	0.251	0.000 to 0.001	0.017			

Adjusted for age, gender, body mass index, fasting plasma glucose, HbA1c, smoking, diabetes mellitus, hypertension, antihypertensive medication and lipid lowering therapy.

carotid artery using the bivariate Pearson's correlation analysis. IMT was positively correlated with age, systolic BP, peripheral PP, peripheral PS, SBP2, central PP, and central PS. PSV and EDV were negatively correlated with age, systolic BP, peripheral PP, peripheral PS, SBP2, central PP, central PS, and RaAlx. The RI was positively correlated with age, FBS, HbA1c, peripheral PP, peripheral PS, and central PP and negatively correlated with diastolic BP, HR, and RaAlx.

After multivariate analysis, diastolic BP (β =-0.231, 95% CI -0.003 to 0.000, P=0.019), and peripheral PP (β =0.230, 95% CI 0.000 to 0.003, P=0.016) were associated with RI after adjusting for age, gender, body mass index (BMI), FBS, HbA1c, smoking, DM, HBP, and use of antihypertensives and lipid-lowering therapy. Also, diastolic BP (β =-0.189, 95% CI 0.001 to 0.208, P=0.047) was correlated with EDV after adjusting for age, gender, BMI, FBS, HbA1c, smoking, DM, HBP, and use of antihypertensive medication and lipid-lowering therapy. After multivariate analysis, the outer and inner diameters were associated with several peripheral

and central hemodynamic parameters after adjusting for age, gender, BMI, FBS, HbA1c, smoking, DM, HBP, and use of antihypertensive medication and lipid-lowering therapy (**Table 4**). Also, IMT was correlated with peripheral and central hemodynamic parameters after adjusting for age, gender, BMI, FBS, HbA1c, smoking, DM, HBP, and use of antihypertensive medication and lipid-lowering therapy.

Figure 1 shows the relationships between hemodynamic or morphologic parameters of the carotid artery and age using linear regression analysis. PSV and EDV were negatively correlated with age; however, RI, IMT, OutDIA, and InDIA were positively correlated with age.

Subgroup analysis was performed according to identified risk factors. In patients with HBP [n=182 (46.9%)], diastolic BP (β =-0.226, 95% CI -0.953 to -0.044, P=0.032) was associated with PSV after adjusting for age, gender, BMI, FBS, HbA1c, smoking, DM, and use of antihypertensive medication and

lipid-lowering therapy according to multivariate analysis. Diastolic BP (β =-0.306, 95% CI -0.005 to 0.000, P=0.031) was associated with RI. Systolic BP (β =0.328, 95% CI 0.005 to 0.043, P=0.014), diastolic BP (β =0.393, 95% CI 0.011 to 0.075, P=0.009), SBP2 (β=0.358, 95% CI 0.007 to 0.039, P=0.007), and RaAlx ($\beta=0.319$, 95% CI 0.006 to 0.067, P=0.020) were associated with OutDIA. Systolic BP (β=0.294, 95% CI 0.002 to 0.049, P=0.033), diastolic BP $(\beta=0.307, 95\% \text{ CI } 0.000 \text{ to } 0.080, P=0.049)$ and SBP2 (β =0.273, 95% CI 0.000 to 0.041, P=0.047) were associated with InDIA. Systolic BP (β =0.330, 95% CI 0.001 to 0.007, P=0.012), peripheral PP (β =0.397, 95% CI 0.001 to 0.009, P=0.008), SBP2 (β =0.334, 95% CI 0.001 to 0.006, P=0.010), central PP (β=0.414, 95% CI 0.002 to 0.008, P=0.005), central PS (β =0.382, 95% CI 0.000 to 0.001, P=0.011), and RaAlx $(\beta=0.265, 95\% \text{ CI } 0.000 \text{ to } 0.009, P=0.051)$ were associated with increased IMT.

In patients with DM [n=143 (36.9%)], peripheral PP (β =0.251, 95% CI 0.000 to 0.003, P=0.035) was associated with RI after adjusting for age, gender, BMI, FBS, HbA1c, smoking,

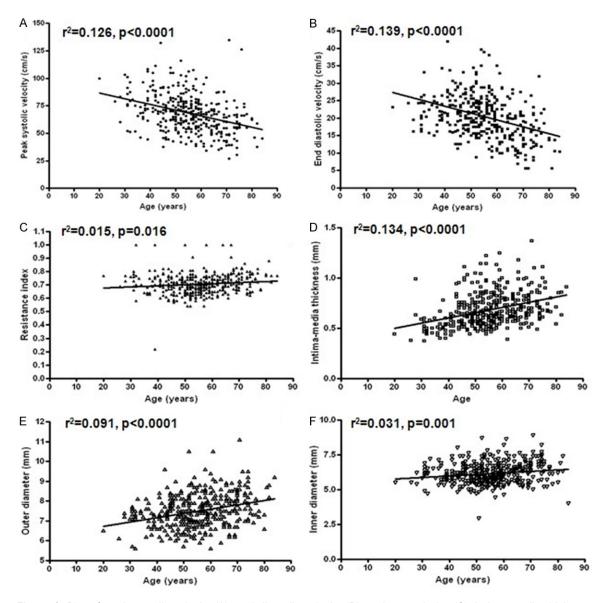


Figure 1. Plot of peak systolic velocity (A), end diastolic velocity (B), resistance index (C), intima-media thickness (D), out diameter (E), and inner diameter (F) in the relation to age of patient without atherosclerotic cardiovascular disease using the linear regression analysis.

HBP, and use of antihypertensive medication and lipid-lowering therapy according to multivariate analysis. Systolic BP (β =0.272, 95% CI 0.005 to 0.032, P=0.007), peripheral PP (β =0.318, 95% CI 0.008 to 0.044, P=0.005), SBP2 (β =0.251, 95% CI 0.003 to 0.027, P=0.015), central PP (β =0.294, 95% CI 0.005 to 0.039, P=0.010), and central PS (β =0.254, 95% CI 0.000 to 0.001, P=0.027) were associated with OutDIA. Systolic BP (β =0.281, 95% CI 0.007 to 0.042, P=0.006), peripheral PP (β =0.275, 95% CI 0.005 to 0.054, P=0.018), peripheral PS (β =0.271, 95% CI 0.000 to 0.001, P=0.016), SBP2 (β =0.255, 95% CI

0.004 to 0.036, P=0.015), central PP (β =0.248, 95% CI 0.002 to 0.047, P=0.036), and central PS (β =0.300, 95% CI 0.000 to 0.001, P=0.010) were associated with InDIA. Peripheral PP (β =0.268, 95% CI 0.001 to 0.012, P=0.026) and peripheral PS (β =0.301, 95% CI 0.000 to 0.001, P=0.009) were associated with increased IMT.

Discussion

The objective of this study was to elucidate the associations between morphologic or hemodynamic parameters of the carotid artery and vas-

cular hemodynamic factors in patients without ASCVD. The role of central and peripheral artery stiffness on hemodynamic or morphological parameters of the carotid artery is contentious. The present study demonstrates that blood flow velocity in the carotid vessels is associated with brachial diastolic BP and PP but not central hemodynamics after adjusting for traditional cardiovascular risk factors. However, IMT and lumen diameter of the carotid artery are correlated with peripheral and central hemodynamic parameters. Therefore, the assessment of vascular hemodynamic factors might play a useful role in detecting hemodynamic or morphologic changes of the carotid arteries in patients without ASCVD.

The results of the present study are consistent with those of an earlier study that reported that both lumen diameter and wall thickness of the CCA increase with age in the general population [14]. These findings regarding morphologic parameters are similar to the results of several previous studies [7, 15]. Furthermore, even after adjusting for several risk factors and use of anti-hypertensive medication in the present study, increased IMT and lumen diameter of the CCA correlated with arterial stiffness in patients without ASCVD.

A previous study has reported that CCA shear stress decreased with age, and that BP and BMI were associated with increased IMT independent of other hemodynamic, clinical, or biochemical factors [16]. Few studies have described the relationship between blood flow velocity and arterial stiffness or hemodynamic index, as was focused on in the present study. Bai et al. pointed out that the incidence of embolic stroke was strongly associated with low EDV, higher RI of extracranial carotid arteries, and larger CCA luminal diameter [8]. However, these authors did not demonstrate correlations between hemodynamic parameters and arterial stiffness. In the present study, EDV and RI of the carotid vessels were associated with increased brachial diastolic BP and PP but not with change in central hemodynamics after adjusting for traditional cardiovascular risk factors and medication use.

Vascular aging is likely associated with vascular structural and functional alterations. Schnidt-Trucksass et al. reported that a lower EDV and higher RI might be characteristic of

aging [17]. In the present study, PSV and EDV were negatively correlated with age; however, RI, IMT, OutDIA, and InDIA were positively correlated with age. These findings are similar to the results of previous studies.

Changes in the morphological and functional characters of the carotid artery affect local and systemic hemodynamics. Accelerated and reflected waves meet the central arteries during early systole, which amplify aortic and ventricular pressures during early systole and reduced aortic pressure later. As a consequence, an increase in aortic stiffness leads to increase in central PP [18], which increases after load and induces left ventricular hypertrophy (LVH) and vascular damage within the carotids, brain, and kidneys, Mannami et al. demonstrated that outer and inner CCA diameters correlate with conventional CV risk factors. including high BP and IMT [11]. Another study reported that blood flow velocity and diastolic blood flow in the CCA was significantly decreased in hypertensive patients with LVH [19]. In the present study, structural parameters were associated with peripheral or central PP in all hypertensive or diabetic patients and in the group of subjects as a whole.

In this study, patients without ASCVD but with various risk factors or history of certain medical treatments, arterial stiffness and various parameters of the carotid artery were evaluated. However, there are some limitations to the present study. First, this study was cross-sectional, so any possible causal relationships observed between morphologic or hemodynamic parameters of the carotid artery and the parameters of arterial stiffness in patients without ASCVD must be fully established by subsequent prospective studies. Second, SBP2 and RaAlx were obtained directly from radial pressure waveforms instead of aortic or carotid pulse waves. Although this method is not the gold standard, SBP2 and RaAlx have been significantly associated with carotid stiffness parameters in an adult population with a wide age range and provide information equivalent to carotid Al measurements [20].

The results of the current study indicate that hemodynamic parameters in the carotid vessels are associated with peripheral diastolic PB and PP but not with the index of arterial stiffness after adjusting for age, gender, BMI, FBS,

HbA1c, smoking, DM, HBP, and use of antihypertensive medication and lipid-lowering therapy. However, IMT and lumen diameter of the carotid artery are correlated with peripheral and central hemodynamic parameters, in agreement with results observed in patients with HBP or DM. In conclusion, the results of this study suggest that blood flow velocity of the CCA influences peripheral blood pressure or pulse pressure rather than the index of arterial stiffness. However, morphological parameters are associated with arterial stiffness and systemic hemodynamic factors. Although further studies are required, multiparametric assessment contributes to a better understanding of structural and hemodynamic changes of the carotid artery.

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Disclosure of conflict of interest

None.

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Blood flow velocity in the carotid artery

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