

Age-Associated Elongation of the Ascending Aorta in Adults

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OBJECTIVES To determine whether human aorta lengthens with aging and to evaluate the impact of the hypothesized aortic elongation on pulse wave velocity (PWV) measurements.

BACKGROUND Although it is generally thought that the aorta becomes tortuous with aging, there has been no systematic study to date in healthy adults to determine if this is so. Such age-related aortic elongation may be a confounding factor for the PWV measurement in elderly people.

METHODS Arterial lengths were computed by the 3-dimensional transverse magnetic resonance image arterial tracing of the aorta and carotid and iliac arteries in 256 apparently healthy adults (age 19 to 79 years).

RESULTS The ascending aorta was greater with advancing age ($r = 0.72$), whereas the lengths of the descending aorta and carotid and iliac arteries were not associated with age. The elongation of the ascending aorta was associated with the corresponding increases in aortic PWV ($\beta = 0.50$) and brachial/aortic pulse pressure ratio ($\beta = 0.24$), which is an index of pulse wave amplification. The straight distance between carotid and femoral sites (car-fem), the most popular arterial length measurement, overestimated the aortic length measured with the magnetic resonance image by $\sim 25\%$. The most accurate arterial length estimation was the distance obtained by subtracting carotid length from the car-fem, with $<5\%$ difference from the magnetic resonance image-measured length. Because the ascending aorta was omitted or subtracted from the length estimation in PWV, the impact of age-related elongation of the aorta on PWV was small.

CONCLUSIONS The aorta lengthens with age, even in healthy humans, due primarily to the elongation of the ascending aorta. Age-related aortic elongation has little impact on PWV measurements, as the ascending aorta, which undergoes lengthening with age, is not included in the arterial length measurements. (J Am Coll Cardiol Img 2008;1:739–48) © 2008 by the American College of Cardiology Foundation

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The incidence and prevalence of vascular disease increase steeply with advancing age, and advancing age itself unequivocally confers the major risk (1). A currently prevalent view is that changes in vascular structure and function occur with the aging process and alter the substrate on which specific pathological disease mechanisms become superimposed (2). Cross-sectional studies have demonstrated that wall thickening and

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dilation or enlargement of central elastic arteries are prominent structural changes that occur within large arteries (e.g., aortic and carotid arteries) with advancing age (3–5). Noninvasive studies in humans as well as histochemical analyses of animals indicate that the arterial wall thickness increases markedly even in a population with a low incidence of atherosclerosis and in beagles who do not develop atherosclerosis (6–8). These cross-sectional morphologies of central elastic arteries have been studied relatively extensively in relation to the arterial remodeling with aging. However, little information is available regarding the longitudinal morphological changes in the aorta (i.e., the aortic length) (9). It is generally thought that the aorta and large elastic arteries become longer and tortuous with aging (10–12). However, there are no systematic scientific studies to date to support such a claim. Additionally, the available information

was derived from case studies of cardiac patients and autopsy samples, and it is unclear whether these changes are a specific manifestation of the aging process or reflect a chronic adaptation to pathology. Moreover, because the artery is under constant modulatory influence by autonomic nervous system activity and humoral factors and is known to retract when excised (11), its in situ length may be markedly different from in vitro length derived from the autopsy. Accordingly, the primary aim of the present cross-sectional study was to determine whether the human aorta lengthens with advancing age.

Another characteristic feature of the aging process is vascular stiffening. There is a growing awareness that the cushioning function of cardiothoracic arteries plays an important role in the pathogenesis of cardiovascular disease (13). The most widely used approach for determining central arterial stiffness in humans is aortic pulse wave velocity (PWV),

which is calculated using the time delay and the distance traveled by a pulse between the carotid artery and the femoral artery (14). Even though the measurement of time delay required for the calculation of PWV has been performed automatically using a variety of algorithms to detect the “foot” of the blood pressure waveforms, the measurement of the distance component still relies on a rough estimation of the aortic length on the body surface using a tape measure. Because of the uncertainty associated with the body surface measurements, a variety of methods have been proposed to approximate the aortic length. Currently, it is not known how accurate these superficial measurements are to the actual length of the arterial segments. Additionally, if the aorta and elastic arteries were to become tortuous with aging (11,12), it may be a confounding factor for the evaluation of arterial stiffness in the elderly patients. Thus, the secondary aim of the present study was to evaluate the impact of the hypothesized age-related elongation of the aorta on PWV measurement.

We performed a systematic investigation to address these aims. First, we measured the lengths of the aorta and carotid and iliac arteries using the 3-dimensional artery tracing on transverse magnetic resonance images (MRI). Before doing so, these length measurements were validated by the custom-made phantom. Second, we compared the MRI-derived arterial length with the body surface measurements typically performed for the PWV in the Japanese population. Then the same measurements were applied to a U.S. population to determine whether there were any systematic racial differences in the measurement of the aortic length. Our overall hypotheses were that the aorta would elongate with aging and that the lengthening of the aorta would underestimate aortic PWV in older adults.

METHODS

Participants. We studied 256 apparently healthy adults (130 men and 126 women, age 19 to 79 years). Participants were recruited from the city and its surrounding community using flyers, direct mailings, and e-mail ads (with proper approval). Women who were pregnant or participants with implants that are electrically, magnetically, or mechanically active, with intracranial aneurysm clips, with cardiovascular disease, or with epileptic seizures, or claustrophobic symptoms were excluded. A total of 27 patients were taking prescribed anti-hypertensive (n = 24), cholesterol lowering (n = 6),

ABBREVIATIONS AND ACRONYMS

car = carotid artery

fem = femoral artery

MRI = magnetic resonance image

PP = pulse pressure

PWV = pulse wave velocity

ssn = suprasternal notch

umb = umbilicus

diabetic ($n = 4$), and other (e.g., thyroid hormone, anticoagulation) ($n = 8$) medications. Data were collected both at the National Institute of Advanced Industrial Science and Technology in Japan and at the University of Texas at Austin in the U.S. This study was reviewed and approved by the institutional review boards of both institutions. All potential risks and procedures of the study were explained to the participants, and they gave their written informed consent to participate in the study.

Experimental protocol. All measurements were performed after 3 h of fasting and an abstinence of caffeine. Participants were studied under supine resting conditions in a quiet, temperature-controlled room (24°C to 26°C).

Measurements. ARTERIAL LENGTH. Using a 1.0-T (Magnetom Impact, Siemens Japan, Tokyo, Japan) or 3.0-T MRI (Signa Exite HD, GE Medical Systems, Milwaukee, Wisconsin) system, transverse images were taken at end diastole (gradient echo method; echo time/repetition time: 11.0/4.2 or 3,400/73.7 ms; flip angle: 25° or 90° ; field of view: 400 mm; slice thickness: 5 mm; interslice gap: 0 mm). The arterial length was computed by 3-dimensional tracing of the artery with image analysis software (MRicro 1.40, Chris Roden, Columbia, South Carolina). The ascending and descending aortic lengths were defined as distances from the sinus of Valsalva to the top of the aortic arch and from the top of the aortic arch to the level of common iliac bifurcation. Carotid arterial length was defined as a distance from the origin of left common carotid artery to the level of the carotid arterial pressure-recording site. Iliac arterial length was defined as a distance from the level of common iliac bifurcation to the level of femoral arterial pressure-recording site. Prior to data collection, the accuracy of the 3-dimensional artery tracing was verified using the custom-made phantom simulating the human aorta ($n = 14$, length: 608 to ~ 700 mm, diameter: 7 mm) for both MRI systems. The mean measurement errors for both 1.0-T and 3.0-T systems were similar at $0.3 \pm 0.2\%$ and $0.1 \pm 0.1\%$, respectively.

The following body surface lengths typically measured in the calculation of aortic (carotid-femoral) PWV were assessed: car-fem (15), (car-fem) - (ssn-car) (16), (ssn-fem) - (ssn-car) (17), (ssn-umb-fem) - (ssn-car) (18). In these measurements, "car-fem" is the straight distance between the carotid (car) and the femoral (fem) artery recording sites, "ssn-fem" is the straight distance between the suprasternal notch (ssn) and the femoral artery site, "ssn-car" is the straight distance

between the suprasternal notch and the carotid artery site, and "ssn-umb-fem" is the combined distances from the suprasternal notch to the umbilicus (umb) and from the umbilicus to the femoral site. When appropriate (e.g., assessing its relation with age), these segmental lengths were also adjusted for height or torso length (to standardize for the overall size). The horizontal distance between the suprasternal notch and the umbilicus was defined as the torso length. Aortic length used to calculate PWV_{MRI} was the distance derived by subtracting the carotid artery from the combined distance from the aortic arch to the iliac artery to the femoral recording site.

To minimize the effects of body contours (e.g., large stomach and bust) on body surface, we measured the straight horizontal distances using a special segmometer specifically designed for PWV studies (Rosscraft Anthropometric Calipers, Surray, Canada) instead of superficial tracing using a tape measure.

AORTIC PWV. Carotid and femoral artery pulse waves were obtained using arterial applanation tonometry incorporating an array of 15 micropiezoresistive transducers placed on the carotid and femoral arteries (VP-2000, Colin Medical, San Antonio, Texas) as previously described (19). The time delay was measured automatically with the foot-to-foot method. Carotid-femoral PWV were subsequently calculated from the time delay and a variety of arterial path lengths measured with the body surface measurement ($\text{PWV}_{\text{car-fem}}$, $\text{PWV}_{(\text{car-fem}) - (\text{ssn-car})}$, $\text{PWV}_{(\text{ssn-fem}) - (\text{ssn-car})}$, and $\text{PWV}_{(\text{ssn-umb-fem}) - (\text{ssn-car})}$) and by 3-dimensional artery tracing on MRI (PWV_{MRI}).

CAROTID AUGMENTATION INDEX. The carotid augmentation index was calculated as pressure wave above its systolic shoulder (ΔP) divided by pulse pressure (19,20). The foot and the first and second peaks of the systolic pressure of the carotid arterial pressure waveforms were automatically detected by using algorithms of the measurement device based on a band-pass filtering (5 to 30 Hz) and fourth derivatives (19).

BLOOD PRESSURE AND HEART RATE. Brachial blood pressure and heart rate were measured with oscillometric pressure sensor cuffs and electrocardiograms (VP-2000, Colin Medical, San Antonio, Texas). Aortic blood pressure was estimated from the second peak of the radial artery pressure wave (21) with a validated applanation tonometry-based automated radial blood pressure measurement device (HEM-9010AI, Om-

Table 1. Selected Subjects Characteristics

	Japanese	U.S.
Male/female, n	115/117	9/15
Age, yrs	54 ± 15	43 ± 18*
Height, cm	162 ± 9	172 ± 9*
Body mass, kg	60 ± 11	73 ± 16*
Body mass index, kg/m ²	22.9 ± 2.8	24.4 ± 3.7
Torso length, mm	363 ± 21	359 ± 23
Ascending aortic length, mm	75 ± 20	62 ± 16*
Descending aortic length, mm	395 ± 23	396 ± 24
Carotid arterial length, mm	112 ± 14	121 ± 14*
Iliac arterial length, mm	210 ± 26	218 ± 27
Arterial path length, mm	493 ± 35	493 ± 32

Data are mean ± SD. *p < 0.05 versus Japanese. Arterial path length = (descending aortic length) + (iliac arterial length) – (carotid arterial length).

ron Healthcare, Kyoto, Japan). The brachial/aortic pulse pressure (PP) ratio was obtained as an index of pulse wave amplification (11).

Statistical analyses. Univariate and partial correlation and regression analyses were performed to determine relations of interests. Stepwise forward multiple-regression analyses were used to determine independent physiological correlates of the age-associated changes in arterial length. Before the analyses, only variables that showed significant univariate correlations were included in the model. Analysis of variance was used to compare arterial path lengths measured with the body surface measurement and by the 3-dimensional artery tracing on MRI. In the case of a significant F value, a post-hoc test using the Newman-Keuls method identified significant differences among mean values. The racial differences were determined by the unpaired *t* test and analysis of covariance. All data are reported as mean ± SD. Statistical significance was set a priori at *p* < 0.05.

RESULTS

Participant characteristics. Selected participant characteristics and hemodynamic variables are presented in Tables 1 and 2. Mean length of ascending aorta was shorter in U.S. participants compared with the Japanese population due to younger mean age of the U.S. participants. Although the U.S. participants had significantly longer carotid arterial length compared with Japanese participants, this difference was abolished when height was taken into account.

Ageing and arterial length. There were no systematic differences in the data obtained in Japan and the U.S., so the data were combined. As shown in Figure 1, the ascending aortic length was positively

and strongly associated with age (*r* = 0.72; *p* < 0.0001). Lengths of the descending aorta and the carotid and iliac arteries were not related to age. When the arterial lengths were adjusted for torso length using partial correlation analyses, the descending aorta was weakly correlated with age (*r* = 0.13; *p* < 0.05). The length of the ascending aorta was not related to height or torso length. The arterial path length measured with the MRI (i.e., the distance derived by subtracting carotid artery from the top of the aortic arch to the femoral artery site) was not associated with age (*r* = 0.08; *p* = NS).

Blood pressure, arterial stiffness, and arterial length.

The relation between arterial length and blood pressure (BP) was determined in a subset of 228 participants who underwent measurements of aortic and brachial blood pressures. The ascending aortic length was positively and significantly associated with brachial diastolic BP (*r* = 0.42), aortic systolic BP (*r* = 0.47), aortic PP (*r* = 0.37), brachial/aortic PP ratio (*r* = –0.44), carotid augmentation index (*r* = 0.44), and PWV_{MRI} (*r* = 0.58) (Table 3, Fig. 2). When the influence of age was taken into account using partial correlation analyses, the relation between the ascending aortic length and PWV_{MRI} remained statistically significant (*p* < 0.05), whereas brachial/aortic PP was no longer significantly associated with the ascending aortic length. Stepwise multiple regression analyses revealed that PWV_{MRI} (beta = 0.47) and brachial/aortic PP ratio (beta = –0.32) were significant, independent physiological determinants of the ascending aortic length. The lengths of the descend-

Table 2. Hemodynamic Variables of Subjects

Heart rate, beats/min	60 ± 8
Brachial systolic BP, mm Hg	124 ± 16
Brachial diastolic BP, mm Hg	75 ± 11
Brachial mean BP, mm Hg	94 ± 13
Brachial PP, mm Hg	49 ± 9
Aortic systolic BP, mm Hg	115 ± 19
Aortic PP, mm Hg	40 ± 12
Brachial/aortic PP ratio, U	1.27 ± 0.31
Carotid augmentation index, %	16.4 ± 19.3
PWV _{MRI} , cm/s	860 ± 199
Ascending aortic length, mm	74 ± 20
Descending aortic length, mm	395 ± 23
Carotid arterial length, mm	113 ± 14
Iliac arterial length, mm	211 ± 26
Arterial path length, mm	493 ± 35

BP = blood pressure; PP = pulse pressure; PWV_{MRI} = aortic pulse wave velocity obtained with arterial path length measured with the 3-dimensional artery tracing on magnetic resonance image.

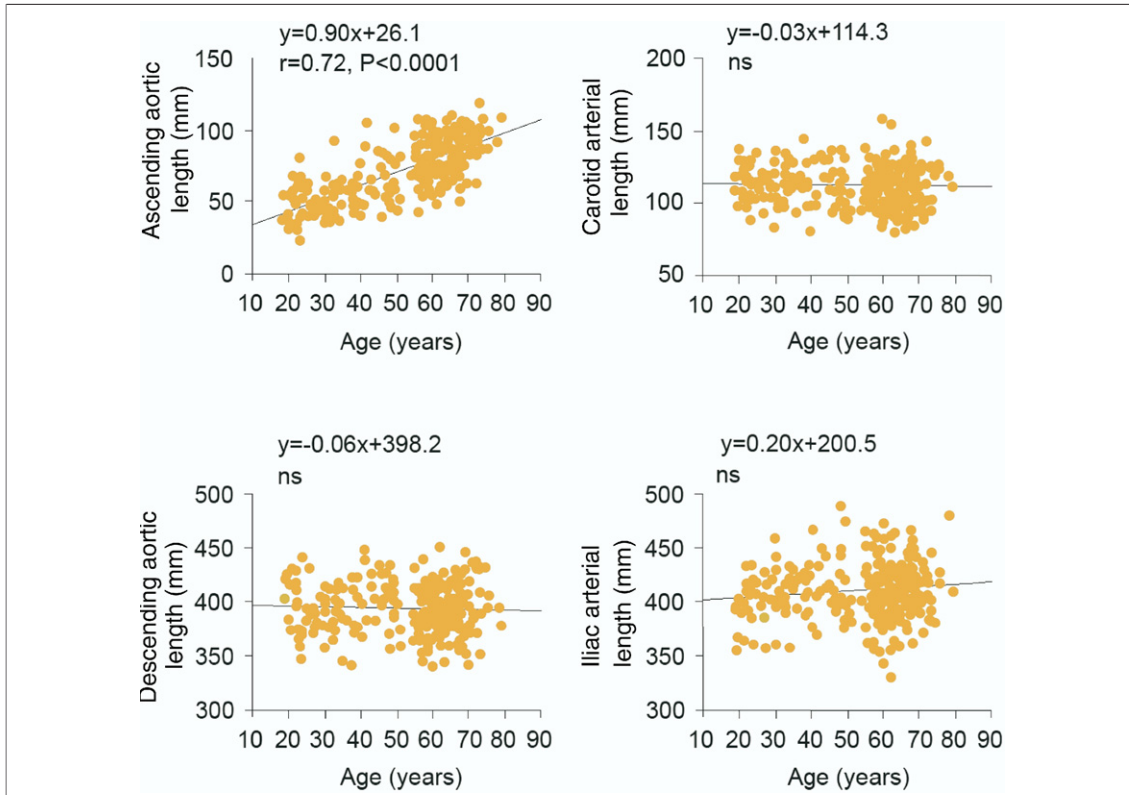


Figure 1. Changes in Aortic and Arterial Lengths With Age

The ascending aortic length was positively and strongly associated with age, whereas lengths of the descending aorta and the carotid and iliac arteries were not related to age.

ing aorta, carotid artery, and iliac artery were not associated with brachial or aortic BP.

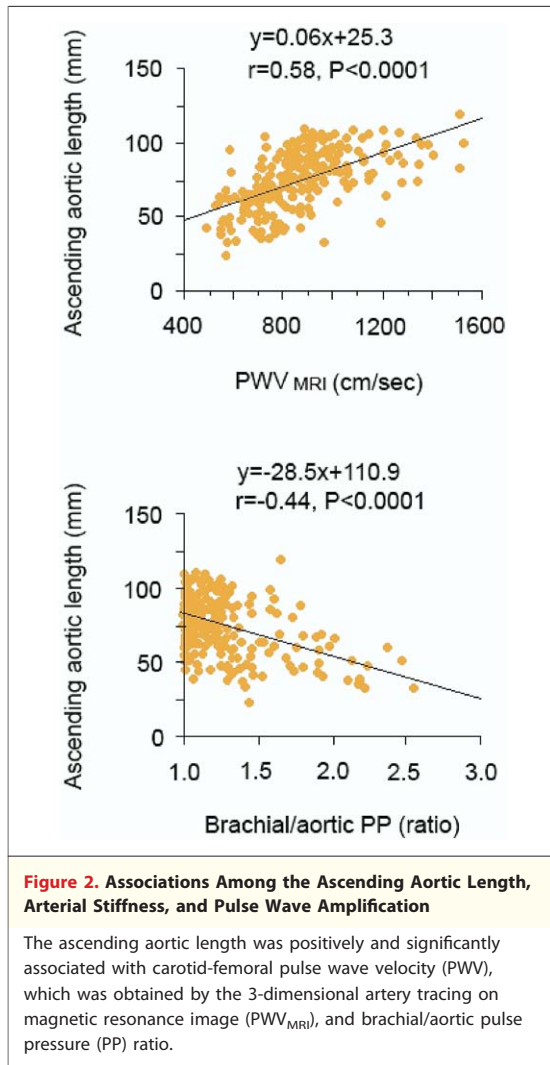
Arterial length for aortic PWV measurement: MRI versus body surface measurements. Table 4 displays arterial path length measured with the 3-dimensional artery tracing on MRI and with a segmometer on the body surface. The most popular way to

estimate aortic length, the car-fem length, was ~25% greater than the MRI-derived arterial length. The body surface length measured using the (car-fem) – (ssn-car) equation displayed the significantly smallest difference to the actual arterial length for carotid-femoral PWV measured with the MRI.

Table 3. Univariate Correlation and Forward Step Multiregression Analyses for Predicting “Physiological” Determinants of the Ascending Aortic Length

	r	R ² Increment	Beta Coefficient
PWV _{MRI} , cm/s	0.58*	0.34	0.47*
Brachial/aortic PP ratio, U	-0.44*	0.05	-0.32*
Aortic PP, mm Hg	0.37*	0.01	-0.15
Brachial diastolic BP, mm Hg	0.42*	0.01	0.11
Aortic systolic BP, mm Hg	0.47*	(not entered)	
Carotid augmentation index, %	0.44*	(not entered)	
Brachial mean BP, mm Hg	0.40*	(not entered)	
Brachial systolic BP, mm Hg	0.37*	(not entered)	
Brachial PP, mm Hg	0.15		
Heart rate, beats/min	-0.09		

*p < 0.05. Total variance explained R² = 0.40 (p < 0.0001).
 Abbreviations as in Table 2.



All of the carotid-femoral PWV obtained with the body surface length measurements were significantly and linearly associated with PWV_{MRI} ($r = 0.96$ to 0.97) (Fig. 3). The PWV estimated with the (car-fem) – (ssn-car) equation was the closest to the line of identity in relation to the PWV_{MRI} with the mean \pm SD difference of 35 ± 41 cm/s. The Bland-Altman plots reveal that PWV obtained with the carotid-femoral length overestimated PWV_{MRI}, whereas PWV calculated with the (ssn-fem) – (ssn-car) equation and the (ssn-umb-fem) – (ssn-car) equation were underestimated PWV_{MRI}.

DISCUSSION

In the present study, we addressed 2 questions that are pertinent to vascular aging. First, we determined whether aging is associated with the lengthening or

elongation of the aorta and large conduit arteries. We found that the length of the ascending aorta increased significantly with advancing age, whereas the descending aorta did not demonstrate age-associated lengthening. The age-related elongation of the ascending aorta was associated with the corresponding increase in central arterial stiffness and pulse wave amplification, suggesting that the reduction in the elastic property as well as local pulsatile pressure might play a causative role. Second, we determined what impact the aortic elongation has on PWV, which is 1 of the most established measures of arterial stiffness. We found that age-related aortic elongation has little impact on PWV measurement because the segment of the ascending aorta is omitted from the arterial length measurements for carotid-femoral PWV. Moreover, the most accurate procedure in estimating aortic length appears to be the (car-fem) – (ssn-car) equation.

The present results indicated that the ascending aorta elongated markedly with aging even in adults without apparent cardiovascular disease. Interestingly, such marked elongation of the aorta was absent in the descending aorta or more peripheral arteries (i.e., carotid artery, iliac artery). What physiological mechanisms explain the age-related lengthening of the proximal aorta? Because the proximal aorta is the principal segment to absorb left ventricular ejection and dampen pulsatile flow, it is more likely to experience “material fatigue” with advancing age (11). We observed that aortic PWV and brachial/aortic PP ratio, an index of pulse wave amplification, were independent determinants of the age-related increase in ascending aortic length, suggesting the prominent roles of the decreased buffering function and the increased local pulse pressure for the longitudinal arterial remodeling. A number of structural changes in the arterial wall, including the thinning, splitting, fraying, and

Table 4. Arterial Path Lengths for PWV Measurement

	Arterial Path Length	Difference
MRI, mm	493 \pm 35	
car-fem, mm	621 \pm 37*	128 \pm 34†
(car-fem) – (ssn-car), mm	518 \pm 31*	25 \pm 26
(ssn-fem) – (ssn-car), mm	427 \pm 29*	–66 \pm 24†
(ssn-umb-fem) – (ssn-car), mm	458 \pm 30*	–35 \pm 24†

Data are mean \pm SD. MRI = arterial path length measured with the 3-dimensional artery tracing on magnetic resonance image. Difference = versus arterial path length of MRI. * $p < 0.0001$ versus arterial path length of MRI. † $p < 0.0001$ versus difference in (car-fem) – (ssn-car). car = carotid artery; fem = femoral artery; ssn = suprasternal notch; umb = umbilicus; other abbreviations as in Table 2.

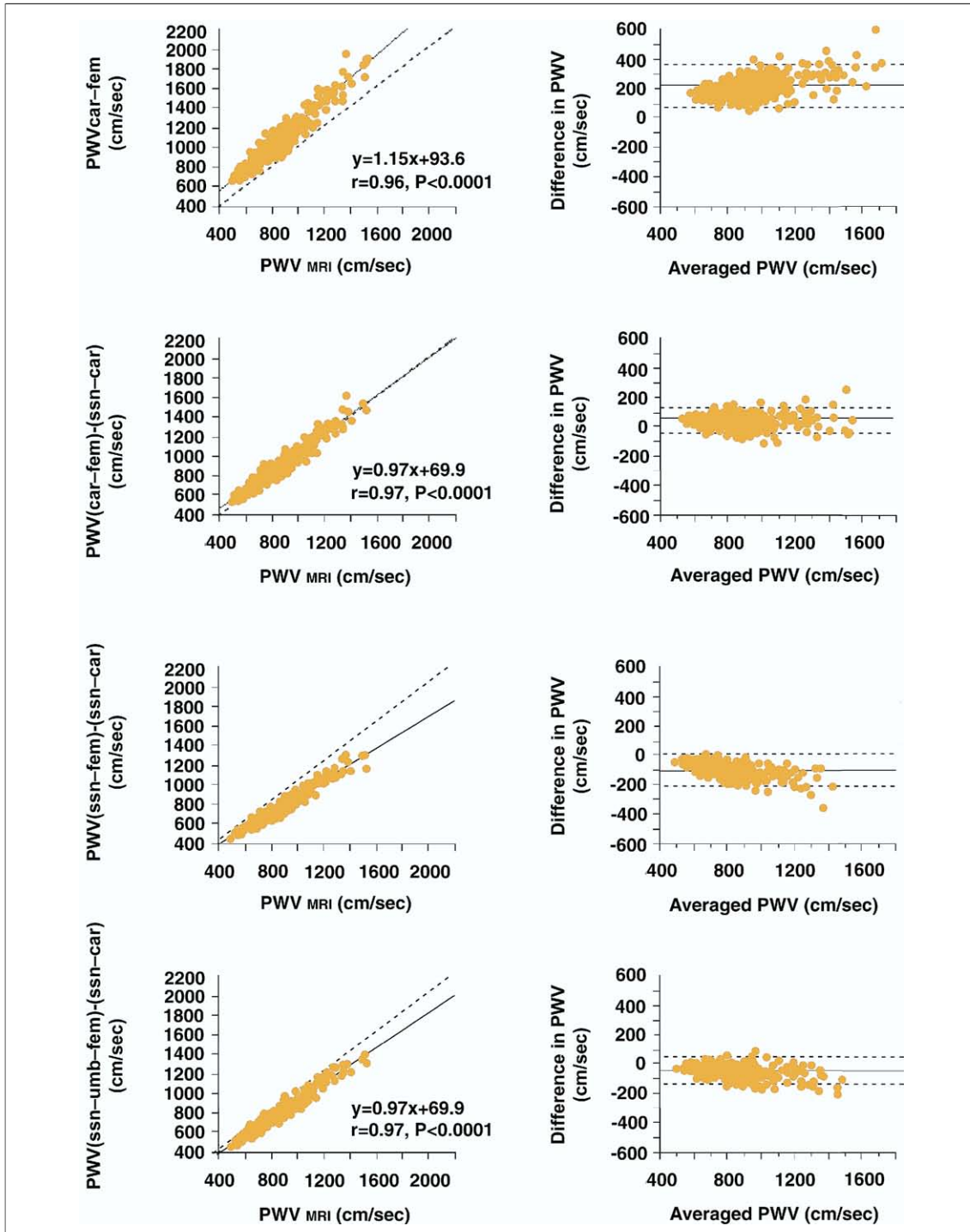


Figure 3. Comparisons of PWV

Scatter plots (left) and Bland-Altman plots (right). All of the carotid-femoral PWVs obtained with the body surface length measurements were significantly and linearly associated with that obtained by PWV_{MRI} . The PWV estimated with the (car-fem) – (ssn-car) equation was the closest to the line of identity in relation to the PWV_{MRI} with the mean \pm SD difference of 35 ± 41 cm/s. car = carotid artery; fem = femoral artery; ssn = suprasternal notch; umb = umbilicus; other abbreviations as in Figure 2.

fragmentation of elastin fibers as well as loss of orderly arrangement of elastin fibers and elastic laminae, have been reported with aging (11,22,23). These changes could, in turn, induce dilation, stiffening, thickening, and elongation of the artery attributable to fibrous remodeling. Because the ratio of elastic fibers to collagen fibers is highest in the proximal ascending aorta (24), the progressive changes in elastic fibers that occur with advancing age would be expected to have a greater effect on the proximal aorta. In this context, patients with Marfan syndrome, which is characterized by the connective tissue disorder, demonstrate pronounced dilation of the proximal aorta (25) as well as the decreased buffering function of large conduit artery (26,27). Similar to the present study focusing on the aging process, the vascular alterations associated with the Marfan syndrome manifested only in the proximal aorta as the aortic arch, abdominal aorta, and carotid and femoral arteries did not demonstrate such structural changes (25).

It is well established that elastic arteries undergo enlargement in diameter with advancing age (2,11). Aortic dilation is likely due to the failure of elastic fibers to sustain physiological hemodynamic stress and is thought to be an indication of mechanical failure and fatigue of biomaterials in the arterial wall (11) mediated by the interaction of various factors including metalloproteinases, integrins, and endothelial function (2). Unfortunately, aortic diameter was not measured in the present study. However, it is interesting to note that the extent of age-related change in the ascending aortic length observed in the present study (12% per decade) is substantially greater than that in the cross-sectional expansion of the ascending aorta (~3% per decade) reported in previous studies (28,29).

As the early detection and prevention of cardiovascular disease using arterial stiffness were widely promoted, the use of carotid-femoral PWV has been gaining popularity as the primary modality to assess arterial stiffness (15–17,30,31). Indeed, aortic PWV has been considered a gold standard index of arterial stiffness that has been directly linked with cardiovascular disease risk, mortality, and morbidity (15–17,30–33). The distance component (i.e., the length of the aorta) required for the carotid-femoral PWV calculation, however, is still being measured on the body surface using a tape measure. The most commonly used procedure is to measure the straight distance between the carotid and the femoral arterial pulse recording sites (15,30,31). However, this

procedure ignores the distance traveled by the pulse wave in the opposite direction. Indeed, we found that the straight body surface distance between the carotid and femoral recording sites overestimated the MRI-derived arterial length as much as 25%. Alternatively, carotid-femoral transit distance is estimated by measuring the straight distance from the carotid to femoral artery recording sites and then subtracting the length between the suprasternal notch and the carotid recording site (16,17). The body surface length measured this way was the closest to the arterial length measured with the MRI. Moreover, PWV estimated with this distance was the closest to the line of identity in relation to the PWV_{MRI} with the mean \pm SD difference of 35 ± 41 cm/s. Thus, the (car-fem) – (ssn-car) equation appears to be the most accurate way to estimate actual arterial length required for carotid-femoral PWV.

What impact does the age-related elongation of the ascending aorta exert on the measurement of carotid-femoral PWV? As described, carotid-femoral transit distance is estimated by the distance from the carotid to femoral artery recording sites minus the suprasternal notch to the carotid recording site distance to account for the parallel transmission along carotid arteries and aortic arch, where the pulse is traveling in the opposite direction. By doing so, the segment of the ascending aorta is dropped from the distance estimation. As such, the age-related aortic elongation has little impact on PWV measurement because the ascending aorta, which elongates with age, is not involved in the arterial paths typically estimated for aorta-femoral and/or carotid-femoral PWV measurements. It should be noted, however, that there are several reports showing the elongation of descending aorta in the patient population. For example, patients with claudication demonstrate tortuosity of the distal aorta and the iliac arteries (34). Additionally, in renal disease patients, the tortuosity of the abdominal aorta is reported to increase with increasing age (12).

Study limitations. Several limitations of the present study should be noted. First, because we conducted the cross-sectional study design, it is difficult to eliminate the possibility that genetic or other constitutional factors may have influenced our cross-sectional findings. Second, the sample size of the U.S. population was smaller than that of the Japanese population. Third, we collected the data at 2 different sites using different MRI machines that use different field strengths. However, both MRI systems were validated using the custom-made phantom prior to the study and mean measurement

errors for both 1.0- and 3.0-T systems were very small and similar ($0.3 \pm 0.2\%$ vs. $0.1 \pm 0.1\%$).

CONCLUSIONS

Our findings indicate that the ascending aorta lengthens with advancing age even in healthy humans. However, the age-related aortic elongation has little impact on PWV measurement as the ascending aorta is omitted from the arterial length

measurements. The estimation of arterial path length by measuring the distances obtained by subtracting the carotid arterial length from the distance of the carotid to femoral sites is closest to the MRI-measured arterial path length.

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