Aortic Root Remodeling Over the Adult Life Course

Longitudinal Data From the Framingham Heart Study

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Background—Aortic root remodeling in adulthood is known to be associated with cardiovascular outcomes. However, there is a lack of longitudinal data defining the clinical correlates of aortic root remodeling over the adult life course.

Methods and Results—We used serial routine echocardiograms in participants of the Framingham Heart Study to track aortic root diameter over 16 years in mid to late adulthood and to determine its short-term (4 years; n=6099 observations in 3506 individuals) and long-term (16 years; n=14 628 observations in 4542 individuals) clinical correlates by multilevel modeling. Age, sex, body size, and blood pressure were principal correlates of aortic remodeling in both short- and long-term analyses (all $P \leq 0.01$). Aortic root diameter increased with age in both men and women but was larger in men at any given age. Each 10-year increase in age was associated with a larger aortic root (by 0.89 mm in men and 0.68 mm in women) after adjustment for body size and blood pressure. A 5-kg/m^2 increase in body mass index was associated with a larger aortic root (by 0.78 mm in men and 0.51 mm in women) after adjustment for age and blood pressure. Each 10-mm Hg increase in pulse pressure was related to a smaller aortic root (by 0.19 mm in men and 0.08 mm in women) after adjustment for age and body size.

Conclusions—These longitudinal community-based data show that aortic root remodeling occurs over mid to late adulthood and is principally associated with age, sex, body size, and blood pressure. The underlying basis for these differences and implications for the development of cardiovascular events deserve further study. **(*Circulation*. 2010;122:884-890.)**

Key Words: aorta ■ epidemiology ■ echocardiography ■ prevention ■ remodeling ■ risk factors

Arterial remodeling with aging is widely recognized to play a key role in the pathogenesis of cardiovascular disease.[1] Numerous cross-sectional[2–5] and postmortem[6] studies have demonstrated an association between increasing age and dilatation of the proximal (central) aorta with underlying age-related changes in the aortic media such as reduced elastin content, elastin fractures, collagen deposition, and calcification. An enlarged aortic root has been shown to be a marker of cardiac and extracardiac target organ damage,[7] which is associated with cardiovascular events and mortality.[8] The cross-sectional correlates of aortic root size include age, anthropometric factors (sex, body size), and cardiovascular risk factors (mainly hypertension).[2–5] These factors have been postulated to interact in a complex fashion over the life course to affect vascular structure and function.[1]

Clinical Perspective on p 890

In contrast to the wealth of cross-sectional data on aortic root size, surprisingly few longitudinal data exist on the evolution of aortic root size over the adult life course and the clinical correlates of tracking of aortic size. Such longitudinal data are needed to define the natural history of aortic remodeling in adulthood and to understand factors influencing aging-associated aortic root dilatation. Accordingly, we evaluated the clinical correlates of long-term longitudinal tracking of aortic root size and short-term changes in aortic root size in a large sample from the community. For this purpose, we assessed factors influencing both short-term change (4 years) and long-term tracking (16 years) of aortic root size using multilevel modeling and aortic root measurements obtained over mid to late adulthood in the Framingham Offspring Study. We hypothesized that age, sex, body size, and blood pressure would also be the key correlates of longitudinal tracking and short-term changes in aortic root size. We further hypothesized that age and sex would interact with other clinical covariates to influence aortic remodeling over the adult life course.

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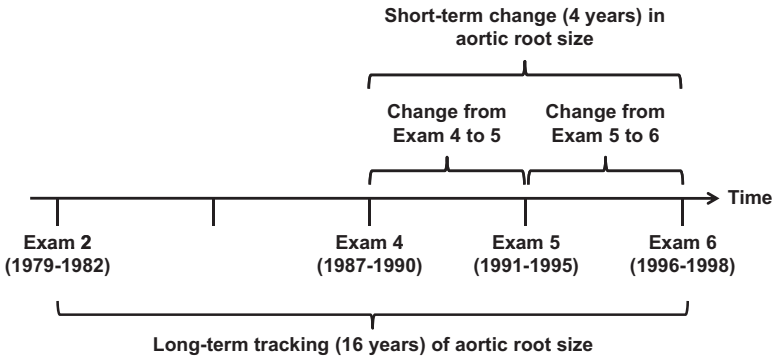

Figure 1. Study design. Routine echocardiograms were used to track aortic root diameter in participants of the Framingham Heart Study. Short-term (4 years) change in aortic root diameter was evaluated in 3506 individuals with at least 2 consecutive echocardiograms (6099 observations). Long-term (16 years) tracking of aortic root size was performed in 4542 individuals who attended up to 4 serial echocardiograms (14 628 observations).

Methods

Study Design

The design, sampling strategy, and recruitment of the Framingham Offspring Study have been detailed previously.[9] Briefly, since initiation of the study in 1971, participants have been examined at the Framingham Heart Study clinic approximately every 4 to 8 years. At each visit, participants undergo a comprehensive review of their cardiovascular risk profiles and anthropometric data, as well as a targeted physical examination. Blood pressure was measured in the left arm of the seated subject with a mercury column sphygmomanometer. All procedures are performed according to standardized study protocols. All participants provided written informed consent, and protocols were approved by the Boston University Medical Center Institutional Review Board.

Echocardiographic Measurement

Transthoracic echocardiograms were performed at offspring examination cycles 2 (1979 to 1982), 4 (1987 to 1990), 5 (1991 to 1995), and 6 (1996 to 1998). All echocardiograms were performed by trained technicians using standardized protocols. M-mode images of the aortic root were obtained with 2-dimensional echocardiographic guidance. Aortic root diameter was measured from the M-mode tracings with the leading edge to leading edge technique, as recommended by the American Society of Echocardiography.[10] Aortic root measurements obtained in this fashion were highly reproducible, as systematically assessed at the sixth examination cycle.[11]

To evaluate the progression of aortic root size in mid to late adulthood, observations were included if participants were within limits of the age group of interest (25 to 74 years) at the time of echocardiography. Significant valve disease (244 observations), defined as greater than mild valve disease at examination cycle 6, was excluded. Short-term (4-year) change in aortic root diameter was evaluated in participants who had at least 2 consecutive echocardiograms (Figure 1). Data on the change in aortic root diameter over any 4-year period (examination 4 to 5 or examination 5 to 6) were pooled to maximize statistical power (n=6099 observations in 3506 participants). Long-term (over the course of 16 years) tracking of aortic root size was evaluated in a total of 4542 participants who attended any of the examinations of interest, giving a total of 14 628 observations. The number of data points from each examination cycle that contributed to the analyses is available online (see the Table in the online-only Data Supplement).

Statistical Analyses

Correlates of Short-Term Change in Aortic Root Diameter

Generalized estimating equations were used to model the change in aortic root diameter over 4 years in which the dependent variable was change in diameter between examination cycles 4 and 5 and between cycles 5 and 6 (≈4-year intervals) (Figure 1), and predictor variables were obtained at the earlier of 2 consecutive examination cycles. Clinical covariates, selected on the basis of reported cross-sectional associations with aortic root size,[2–5] included age, sex, body mass index (BMI), systolic blood pressure, diastolic blood pressure, antihypertensive therapy, smoking status, and diabetes mellitus (model 1). Because peak (systolic blood pressure) and trough (diastolic blood pressure) blood pressures provide only a limited view of vascular hemodynamics, mean arterial pressure (MAP) and pulse pressure (PP) were also derived as indexes of steady-flow (MAP) and pulsatile (PP) components of blood pressure.[12] Thus, MAP and PP were included in place of systolic and diastolic blood pressures in a second multivariable model (model 2), along with the other clinical covariates. Both models also were adjusted for examination cycle and baseline aortic root size. Main effects were investigated, as well as biologically plausible interactions between age, sex, and other clinical covariates, by including appropriate terms in the multivariable models.

Correlates of Long-Term Tracking of Aortic Root Diameter

Multilevel modeling was used to model individual growth curves for aortic root diameter over the long term (up to 16 years). This analytic method is applicable to hierarchical data structures and is therefore well suited for analysis of data that vary both on the patient-level and over time. Furthermore, the method allows maximization of the number of observations that can be analyzed because the analytic approach accommodates missing data at some of the serial examinations. Multilevel modeling of aortic root diameter was performed, incorporating the same set of covariates and interaction terms as used in the short-term analysis. Models were fit by direct entry of candidate variables and using the maximum likelihood approach. Nonlinear effects of age were tested but were not statistically significant. Random intercepts and random effects of age were used to account for variation in initial aortic root sizes and slopes for age, respectively, in different participants. Regression coefficients, their corresponding SEs, and P values are reported. In addition, the change in aortic root diameter for a clinically meaningful increment of each significant predictor variable was calculated and is presented to aid in the interpretation of the data.

Finally, to illustrate the expected changes in aortic root diameter over the life course according to risk factor profile,[13,14] we modeled separate growth curves for 4 hypothetical groups: participants without hypertension and without obesity (blood pressure, 125/75 mm Hg; BMI, 25 kg/m^2), participants with hypertension but without obesity (blood pressure, 160/100 mm Hg; BMI, 25 kg/m^2), participants with obesity but without hypertension (blood pressure, 125/75 mm Hg; BMI, 35 kg/m^2), and participants with both hypertension and obesity (blood pressure, 160/100 mm Hg; BMI, 35 kg/m^2).

All analyses were performed with SAS software (SAS Institute Inc, Cary, NC). Statistical significance was determined at values of $P<0.05$.

All authors had full access to and take full responsibility for the integrity of the data. All authors have read and agree to the manuscript as written.

Table 1. Baseline Characteristics of Study Samples Used in Short-Term (4 Years) and Long-Term (16 Years) Longitudinal Analyses of Aortic Root Diameter

	Sample for Short-Term Analyses (n=3506)		Sample for Long-Term Analyses (n=4542)	
Variable	Men (n=1671)	Women (n=1835)	Men (n=2187)	Women (n=2355)
Age, y	52±10	51±10	46±10	45±10
Weight, kg	85.3±13.5	68.3±14.5	83.0±12.9	65.5±13.8
Height, m	175.3±6.7	161.9±6.1	175.6±6.9	161.9±6.2
BMI, kg/m^2	27.7±3.9	26.0±5.4	26.9±3.8	25.0±5.1
Systolic blood pressure, mm Hg	129±17	124±20	127±16	120±17
Diastolic blood pressure, mm Hg	81±10	76±10	81±9	76±9
MAP, mm Hg	97±11	92±12	96±11	90±11
PP, mm Hg	48±13	47±14	46±12	44±12
Hypertension, %	40.6	29.2	31.2	20.4
Antihypertensive treatment, %	20.1	14.4	13.0	10.6
Diabetes mellitus, %	7.3	4.2	4.8	2.5
Smoking, %	23.3	22.7	34.6	36.2
Baseline aortic root size, crude, mm	34.0±3.7	29.5±3.1	32.8±3.2	28.4±2.9

Values are mean±SD when appropriate. Characteristics shown are from the first of 2 consecutive examinations for the short-term sample and the first eligible examination for the long-term sample.

Results

Baseline Characteristics

The study sample consisted of middle-aged men and women (Table 1). Participant characteristics are described at the first examination of any 4-year period between consecutive examinations in the pooled short-term sample and at the first available examination of the 16-year period for the long-term sample (Figure 1).

Correlates of Short-Term Change in Aortic Root Diameter

After adjustment for baseline aortic root size, examination cycle, and antihypertensive therapy, short-term (4 years) increases in aortic root diameter were associated with older age, male sex, and larger body size (increasing BMI) (Table 2). Interestingly, divergent effects of blood pressure were observed, even after adjustment for the effect of antihypertensive medications: Lower systolic blood pressure and higher diastolic blood pressure were associated with short-term increases in aortic root diameter (Table 2, model 1). Accordingly, aortic root dilation over 4 years was related to a lower pulsatile component (PP) but higher steady-flow component (MAP) of blood pressure at baseline (Table 2, model 2).

Correlates of Long-Term Tracking of Aortic Root Diameter

Consistent with the analysis of short-term change in aortic root diameter, significant correlates of mean aortic root diameter over 16 years (Table 3) were older age, male sex, higher BMI, higher diastolic blood pressure (Table 3, model 1), or lower PP and higher MAP (Table 3, model 2) after adjustment for antihypertensive therapy and examination cycle. Similar results were observed among hypertensive participants alone (not shown).

Interactions Between Age, Sex, and Other Clinical Covariates

Interactions were observed between age and sex, age and BMI, BMI and sex, and blood pressure and sex. To facilitate

Table 2. Correlates of Short-Term (4 Years) Change in Aortic Root Diameter (in mm)

	Model 1 (Modeling BP as SBP and DBP)			Model 2 (Modeling BP as PP and MAP)		
	Regression Coefficient	SE	*P*	Regression Coefficient	SE	*P*
Age, 52 y*	0.030	0.004	<0.0001	0.030	0.004	<0.0001
Male sex	1.796	0.082	<0.0001	1.796	0.082	<0.0001
BMI	0.051	0.007	<0.0001	0.051	0.007	<0.0001
SBP	−0.134	0.053	0.01	…	…	…
DBP	0.209	0.051	<0.0001	…	…	…
PP	…	…	…	−0.162	0.045	0.0004
MAP	…	…	…	0.157	0.043	0.0002
Antihypertensive treatment	0.193	0.095	0.04	0.193	0.095	0.04

BP indicates blood pressure; SBP, systolic BP; and DBP, diastolic BP. Regression coefficients represent the change in mean difference in aortic root diameter (in mm) between examination cycles every 4 years per 1-SD difference in each continuous predictor variable (1-unit difference in age and BMI) or presence versus absence of each categorical predictor variable. Models were also adjusted for examination cycle and baseline aortic root size.

*Age was centered at the mean of all participants at all exams (52 years) to reduce multicolinearity between regression coefficients.

Table 3. Correlates of Long-Term (16 Years) Longitudinal Tracking of Mean Aortic Root Diameter

	Model 1 (Modeling BP as SBP and DBP)			Model 2 (Modeling BP as PP and MAP)		
	Regression Coefficient	SE	*P*	Regression Coefficient	SE	*P*
Age, 52 y*	0.1175	0.01426	<0.0001	0.1172	0.01426	<0.0001
Male sex	2.654	0.4063	<0.0001	2.490	0.3962	<0.0001
BMI, kg/m^2	0.1011	0.00890	<0.0001	0.09860	0.00880	<0.0001
SBP, mm Hg	−0.0071	0.05388	0.9	…	…	…
DBP, mm Hg	0.1879	0.04995	0.0002	…	…	…
PP, mm Hg	…	…	…	−0.1174	0.04396	0.008
MAP, mm Hg	…	…	…	0.2699	0.03259	<0.0001
Antihypertensive treatment	0.2224	0.07477	0.003	0.2184	0.07475	0.004
Age, 52 y×men	0.02066	0.00611	0.0007	0.01957	0.00608	0.001
Age, 52 y×BMI	−0.0020	0.00053	0.0002	−0.0019	0.00053	0.0002
BMI×men	0.05433	0.01469	0.0002	0.06041	0.01430	<0.0001
SBP×men	−0.1928	0.07978	0.02	…	…	…
DBP×men	0.2040	0.07086	0.004	…	…	…
PP×men	…	…	…	−0.1442	0.06127	0.02

Abbreviations as in Table 2. Regression coefficients represent the change in mean aortic root diameter (in mm) per 1-SD difference in each continuous predictor variable (1-unit difference in age and BMI) or presence versus absence of each categorical predictor variable. Models were also adjusted for examination cycle.

*Age was centered at the mean of all participants at all exams (52 years) to reduce multicolinearity between regression coefficients.

the interpretation of the interaction terms, we calculated the expected effect of each variable of interest on tracking of aortic root diameter over 16 years (Table 4) in men and women, accounting for its main effect and its interactions. Each 10-year increase in age was associated with a larger predicted aortic root diameter of 0.89 mm in men and 0.68 mm in women (both with an average BMI of 25 kg/m^2) after adjustment for blood pressure and antihypertensive therapy. A 5-kg/m^2 increase in BMI was associated with a larger predicted aortic root diameter in men (0.78 mm) than in women (0.51 mm) after adjustment for age and blood pressure. Each 10-mm Hg increase in diastolic blood pressure was associated with a larger predicted aortic root diameter in men (0.39 mm) than in women (0.19 mm Hg) after adjustment for all other clinical covariates. The predicted effect of systolic blood pressure was small in both sexes. Of note, each

Table 4. Interaction of Clinical Correlates of Longitudinal Aortic Root Tracking Over a 16-Year Period

	Tracking of Aortic Root Diameter, Mean (95% CI), mm	
	Model 1 (Modeling BP as SBP and DBP)	Model 2 (Modeling BP as PP and MAP)
Men vs women with age=52 y, BMI=25 kg/m^2, SBP=125 mm Hg, DBP=75 mm Hg, PP=49 mm Hg	3.9780 (3.8180–4.1380)	3.9970 (3.8378–4.1562)
Effect of age (per 10-y increase) in		
Men with BMI=25 kg/m^2	0.8888 (0.79–0.9876)	0.8815 (0.7830–0.9800)
Women with BMI=25 kg/m^2	0.6822 (0.5844–0.7800)	0.6858 (0.5880–0.7836)
Effect of BMI (per 5-kg/m^2 increase) in		
Men, age=52 y	0.7772 (0.6596–0.8947)	0.7951 (0.6790–0.9111)
Women, age=52 y	0.5055 (0.4183–0.5927)	0.4930 (0.4068–0.5792)
Effect of SBP (per 10-mm Hg increase) in		
Men	−0.1090 (−0.1722–−0.0458)	…
Women	−0.0040 (−0.0616–0.0536)	…
Effect of DBP (per 10-mm Hg increase) in		
Men	0.3925 (0.2910–0.4940)	…
Women	0.1882 (0.0902–0.2862)	…
Effect of PP (per 10-mm Hg increase) in		
Men	…	−0.1860 (−0.2524–−0.1196)
Women	…	−0.0830 (−0.1443–−0.0217)
Effect of MAP (per 10-mm Hg increase)		0.2329 (0.1778–0.288)
Antihypertensive treatment	0.2224 (0.0759–0.3689)	0.2184 (0.0719–0.3649)

Abbreviations as in Table 2. Shown is the effect of each variable on long-term (16 years) tracking of mean aortic root diameter in the indicated subgroups after adjustment for all other clinical covariates.

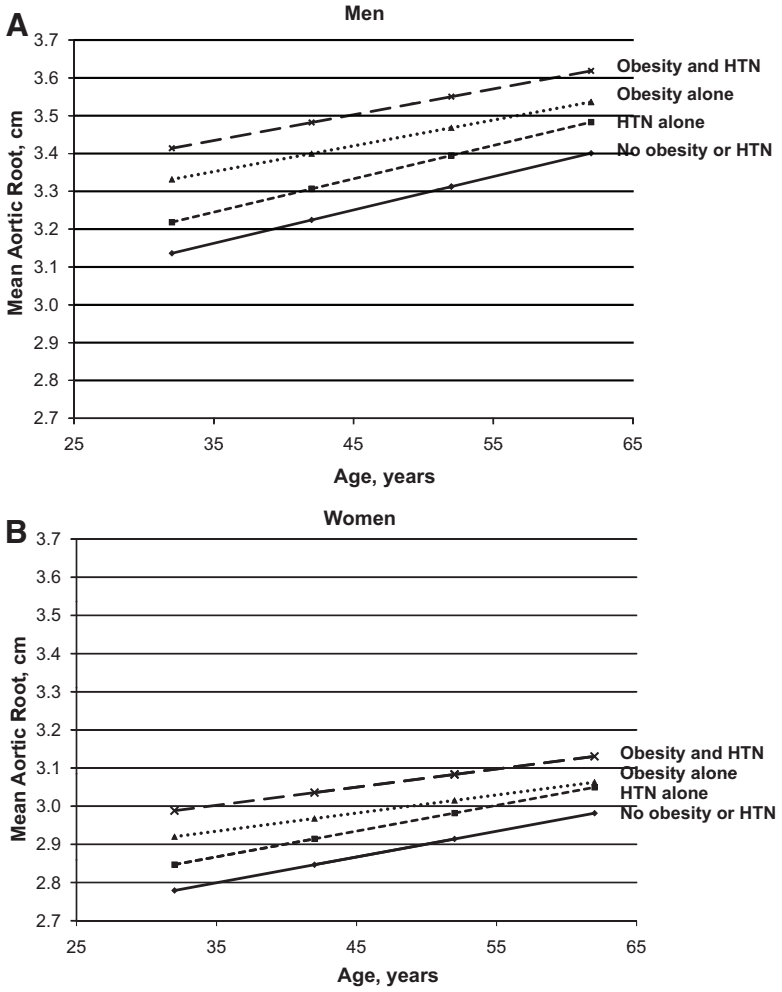

Figure 2. Predicted mean aortic root growth curves in men and women with and without obesity or hypertension. Sex-specific growth curves were modeled in men (A) and women (B), tracking mean aortic root diameter over 16 years in participants without hypertension (HTN) and without obesity (blood pressure, 125/75 mm Hg; BMI, 25 kg/m^2), participants with HTN but without obesity (blood pressure, 160/100 mm Hg; BMI, 25 kg/m^2), participants with obesity but without HTN (blood pressure, 125/75 mm Hg; BMI, 35 kg/m^2), and participants with both HTN and obesity (blood pressure, 160/100 mm Hg; BMI, 35 kg/m^2).

10-mm Hg increase in PP was related to a smaller predicted aortic root diameter in men (0.19 mm) and women (0.08 mm) after adjustment for age, BMI, and antihypertensive therapy.

Impact of Hypertension and Obesity on Tracking of Aortic Root Diameter

Figure 2 illustrates the expected mean aortic root diameter over the life course in modeled growth curves according to different clinical risk factor profiles. As shown, mean aortic root diameter enlarged over the mid to late adulthood period in both men and women, but at any given age, men had larger mean aortic roots compared with women. Furthermore, greater aortic dilatation was expected in the presence of hypertension or obesity, and the presence of both factors had a greater impact (steeper slope) on aortic root dilatation in men than women.

Discussion

Our study provides longitudinal community-based data on tracking of aortic root size over mid to late adulthood. Age, sex, body size, and blood pressure were principal correlates of aortic remodeling in both the short-term (4 years) and long-term (16 years) analyses. Separate components of hemodynamic load differed in their relation with aortic root size: Steady-flow load (MAP) was associated positively with aortic root diameter, whereas pulsatile load (PP) was associated negatively with aortic root diameter over the adult life course. Given the discordant effects of MAP and PP on aortic root diameter but their concordant contributions to systolic blood pressure, systolic blood pressure alone had a limited relationship to aortic root diameter. Furthermore, sex emerged as an important modifier of the associations of age, body size, and blood pressure with aortic root remodeling.

The importance of aortic remodeling over the adult life course and its role in the pathogenesis of cardiovascular disease have been the focus of considerable interest.[1,15–18] A modest increase in diameter of the proximal aorta with advancing age has been well established in cross-sectional[2–5] and postmortem[6] studies. Associated structural changes in the arterial wall have also been described.[19,20] Age-related increases in collagen synthesis within the aortic wall result in increasing proximal aortic stiffness because mechanical stresses, no longer buffered by elastin, are transferred to the less extensible collagen fibers.[16] PP, generally regarded as an index of arterial stiffening, is well known to increase with age and to be associated with adverse cardiovascular events, including heart failure.[21] Similarly, an enlarged aortic root has been associated with cardiac disease, adverse cardiovascular outcomes, and mortality.[7,8]

Despite widespread recognition of the significance of aortic remodeling and the wealth of cross-sectional data describing its clinical correlates, longitudinal data are scarce. Our present study in a large community-based cohort establishes that the aortic root size gradually increases in mid to late adulthood by 0.9 mm in men and 0.7 mm in women for each decade of life, assuming a normal BMI and adjusting for blood pressure. In modeled growth curves, the presence of hypertension or obesity was associated with greater mean aortic root size over time, with the greatest effect predicted when both risk factors were present. These results are consistent with previous cross-sectional studies in which age, sex, body size, and blood pressure were identified as key factors influencing aortic root size in adulthood.[2–5] The present longitudinal data extend previous studies by characterizing the contributions of these key factors to aortic dilatation over time and provide insight into interactions between various clinical factors that affect aortic root size over the life course, thus defining the natural history of aortic remodeling in adulthood.

Blood pressures can be viewed as separate components of steady-flow (MAP) and pulsatile (PP) hemodynamic load. This separation is clinically meaningful because each component relates physiologically to different regions of the arterial tree (MAP relates predominantly to small-artery function; PP, to large-artery stiffness) and has differential effects on other physiological variables such as left ventricular mass.[12,22] Considerable debate surrounds the relationship between blood pressure components and aortic root size.[18,23–26] Cross-sectional studies have shown both direct[2,7,8] and indirect[3,4] associations but in general suggest that aortic root diameter increases with increasing diastolic blood pressure but decreases with increasing PP. This is counterintuitive to the classic notion that age-related elastic fragmentation, passive aortic dilatation, wall stiffening, and premature wave reflection lead directly to increasing PP with age. On the contrary, these observations have led to the hypothesis that a smaller aortic root may play a key role in the pathogenesis of systolic hypertension by introducing a mismatch between aortic root diameter and blood flow so that forward wave amplitude is increased.[23] Indeed, direct measurements of pulsatile hemodynamics in patients with systolic hypertension showed that although aortic root dilatation and stiffening occurred with increasing age, higher PP was associated with increased characteristic impedance and reduced rather than increased aortic root diameter.[23] However, in the only prospective study testing this hypothesis,[26] a smaller aortic root was not found to be associated with the incidence of hypertension in middle-aged adults from the community.

Thus, instead of being an antecedent cause of hypertension, it is conceivable that dynamic aortic remodeling occurs as an active, compensatory response to changing hemodynamic load. Outward remodeling of the aortic root may serve to limit the increase in PP as the aortic wall stiffens with age. "Blunted remodeling" may therefore explain the association between smaller aortic root size and increasing PP, as observed in this study and others.[3–5,18] Our results further suggest that sex is an important correlate and effect modifier of the extent of aortic remodeling in adulthood, with men displaying a greater propensity to outward remodeling of the aortic root compared with women. This is consistent with the detailed impedance analyses performed in the large Asklepios study[27] in which a cross-sectional hemodynamic study of adults 35 to 55 years of age showed that total arterial compliance decreased with age in women but not in men, leading to age-associated increases in arterial input impedance in women but not in men. Furthermore, characteristic impedance even decreased in men in this age range but was unchanged in women. Although the authors did not specifically relate these findings to measured sex-related differences in aortic root size, they derived a theoretical "effective cross-sectional aortic area" and suggested that the increase in effective cross-sectional area in men outweighed the increase in aortic wall stiffness and may therefore explain the decrease in characteristic impedance with age in men. Although our study cannot conclusively prove that blunted aortic remodeling causes increased PP, we provide convincing longitudinal evidence of sex-associated differences in aortic remodeling over time, whether with aging alone or with superimposed risk factors of obesity or hypertension. Further research is warranted to elucidate the underlying mechanisms for such differences. An understanding of the consequences of these differences on central PP, cardiac afterload, and ventricular remodeling may also provide insight into the predisposition of elderly hypertensive women to heart failure with preserved ejection fraction.

Limitations

We recognize that there is heterogeneity in remodeling characteristics across the arterial tree and that our observations are limited to aortic root diameter measured at the level of the sinuses of Valsalva. Although geometric complexities at this level may limit the precision of measurements, our measurements were highly reproducible.[11] Invasive hemodynamic measurements were not possible in this large community-based sample, and brachial blood pressure may be a suboptimal surrogate for central PP. Nonetheless, these longitudinal data add to our understanding of aortic remodeling over the adult life course by harnessing the unique availability of systematic routine measurements in the large community-based cohort of the Framingham Heart Study.

Conclusions

Aortic root remodeling occurs over mid to late adulthood and is related principally to age, sex, body size, and blood pressure. These factors interact in a complex fashion in their associations with aortic root remodeling over the life course. In particular, obese hypertensive individuals show the greatest dilatation, and women display less aortic root dilatation than men. The underlying basis for these differences and implications for the development of hypertensive heart disease and its sequelae such as heart failure with preserved ejection fraction deserve further study.

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Disclosures

None.

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CLINICAL PERSPECTIVE

Our study provides longitudinal community-based data on tracking of aortic root size over 16 years in mid to late adulthood. The aortic root gradually enlarges over the life course and is principally related to age, sex, body size, and blood pressure. These factors interact in a complex fashion in their associations with aortic root remodeling. In particular, obese individuals with hypertension show the greatest dilatation, whereas women display less aortic root dilatation than men. Furthermore, separate components of hemodynamic load differed in their relation with aortic root size: Steady-flow load (mean arterial pressure) was positively associated with aortic root diameter, whereas pulsatile load (pulse pressure) was inversely associated with aortic root diameter. The underlying basis for these differences and implications for the development of cardiovascular events deserve further study.