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Aortic Size Assessment by Noncontrast Cardiac Computed Tomography: Normal Limits by Age, Gender, and Body Surface Area

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OBJECTIVES To determine normal limits for ascending and descending thoracic aorta diameters in a large population of asymptomatic, low-risk adult subjects.

BACKGROUND Assessment of aortic size is possible from gated noncontrast computed tomography (CT) scans obtained for coronary calcium measurements. However, normal limits for aortic size by these studies have yet to be defined.

METHODS In 4,039 adult patients undergoing coronary artery calcium (CAC) scanning, systematic measurements of the ascending and descending thoracic aorta diameters were made at the level of the pulmonary artery bifurcation. Multiple linear regression analysis was used to detect risk factors independently associated with ascending and descending thoracic aorta diameter and exclude subjects with these parameters from the final analysis. The final analysis groups for ascending and descending thoracic aorta included 2,952 and 1,931 subjects, respectively. Subjects were then regrouped by gender, age, and body surface area (BSA) for ascending and descending aorta, separately, and for each group, the mean, standard deviation, and upper normal limit were calculated for aortic diameter as well as for the calculated cross-sectional aortic area. Also, linear regression models were used to create BSA versus aortic diameter nomograms by age groups, and a formula for calculating predicted aortic size by age, gender, and BSA was created.

RESULTS Age, BSA, gender, and hypertension were directly associated with thoracic aorta dimensions. Additionally, diabetes was associated with ascending aorta diameter, and smoking was associated with descending aorta diameter. The mean diameters for the final analysis group were 33 ± 4 mm for the ascending and 24 ± 3 mm for the descending thoracic aorta, respectively. The corresponding upper limits of normal diameters were 41 and 30 mm, respectively.

CONCLUSIONS Normal limits of ascending and descending aortic dimensions by noncontrast gated cardiac CT have been defined by age, gender, and BSA in a large, low-risk population of subjects undergoing CAC scanning. (J Am Coll Cardiol Img 2008;1:200–9) © 2008 by the American College of Cardiology Foundation

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Thoracic aortic aneurysm is a common, potentially lethal, but treatable disease, particularly if detected before dissection or rupture. Accurate assessment of aortic size is a key component in this detection and in guiding therapeutic decisions. Multiple imaging modalities are available for assessing the thoracic aorta, including X-ray angiography, transesophageal echocardiography (TEE), computed tomography (CT), and magnetic resonance imaging (MRI). Although all of these modalities have diagnostic value, CT has evolved to be the mainstay of evaluation owing to its accuracy and reproducibility, as well as its speed, simplicity, and true 3-dimensional capabilities.

To distinguish the normal from the enlarged aorta, it is necessary to standardize the values of "normal" aortic dimensions. Due to fundamental differences in the imaging techniques, normal limits of thoracic aorta dimensions for CT are needed. Little has been published regarding normal limits for thoracic aortic dimensions with CT (1–3). Moreover, to our knowledge, no publications to date have reported these limits with gated, noncontrast chest CT studies—the studies commonly used for coronary artery calcium (CAC) scanning. Therefore, our aim was to determine normal limits for ascending and descending thoracic aorta diameters in a large population of asymptomatic, low-risk adult subjects undergoing CAC scanning.

METHODS

We examined the noncontrast gated CT findings of 4,387 patients, age 26 to 92 years, free of known clinical coronary heart disease (CHD), who underwent CAC scanning during the period from July 2004 to March 2007 at Cedars-Sinai Medical Center. Subjects were self-referred (n = 44, 1%), referred by their physician (n = 3,308, 75%), or recruited as part of ongoing research (EISNER [Early Identification of Subclinical Atherosclerosis using NoninvasivE Imaging Research]-1, -2, and -3) protocols (n = 1,035, 24%). An additional 547 patients with incomplete data were excluded (e.g., missing height, missing weight, and/or aorta diameter not measured). The study was approved by the Cedars-Sinai Medical Center Institutional Review Board.

Data collection. Information regarding the presence of categorical cardiac risk factors was collected in every patient through written questionnaires. Systemic arterial hypertension was defined as a documented history of high blood pressure. Current smoking or history of smoking was defined as positive smoking status. Hypercholesterolemia was defined according to the National Cholesterol Education Program (4) guidelines: low-density lipoprotein (LDL) ≥100 mg/dl and Framingham risk score ≥20%, 2 or more risk factors and LDL ≥130 mg/dl, or LDL ≥160 mg/dl. All lipid measurements were within 1 week of the CT study. Subjects were classified as having diabetes if they carried an established diagnosis of diabetes mellitus made by a physician and/or were receiving treatment with insulin or oral hypoglycemic agents, or if their measured fasting glucose was >126 mg/dl. Family history of coronary artery disease (CAD) was defined as a CAD event occurring in a first-degree relative (men age ≤65 years and women age ≤55 years). Weight and height were also obtained and body mass index (BMI) and body surface area (BSA) were calculated using the Mosteller (5) method. Among the 4,387 subjects, 348 subjects with missing clinical data (56, 111, 269, and 277 cases of missing information about smoking, diabetes mellitus, dyslipidemia, and hypertension, respectively) were also excluded.

CT imaging protocol. The CT studies were performed on 1 of 4 CT scanners: 2 electron beam computed tomography (EBCT) scanners (GE Imatron, C-150 or e-Speed, GE Medical Systems, San Francisco, California), a 16-slice multidetector computed tomography (MDCT) scanner (Brilliance CT scanner, Philips, Cleveland, Ohio), and a dual source computed tomography (DSCT) scanner (Somatom Definition, Siemens, New York, New York). For EBCT, we used a protocol of 3-mm slice thickness, 50- or 100-ms exposure time, 130 kVp, 630-mA tube current, either a 300- or 350-mm field of view for reconstruction, and a sharp reconstruction kernel. For MDCT, the protocol was 2.5-mm slice thickness, 140 kVp, 168-mA tube current, 250-ms exposure time, and a 350-mm field of view. For DSCT, the protocol was 3-mm slice thickness, 140 kVp, 150-mA tube current, 200-ms exposure time, and a 350-mm field of view. Prospective electrocardiogram triggering at the heart rate-dependent percentage of the R-R interval was used for all scanners. The CT images were acquired in the craniocaudal direction from immediately inferior to the aortic arch to the level of the diaphragm in a single breath-hold for all

ABBREVIATIONS AND ACRONYMS

BMI = body mass index
BSA = body surface area
CAC = coronary artery calcium
CAD = coronary artery disease
CHD = coronary heart disease
CT = computed tomography
DSCT = dual source computed tomography
EBCT = electron beam computed tomography
LDL = low-density lipoprotein
MDCT = multidetector computed tomography
MRI = magnetic resonance imaging
TEE = transesophageal echocardiography

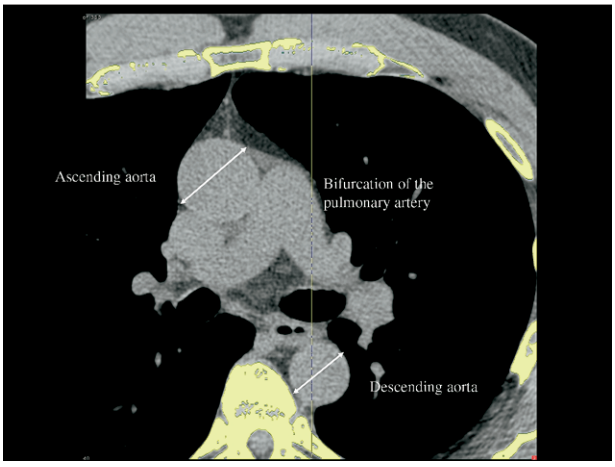

Figure 1. Thoracic Aorta Measurements

Transaxial slice at the lower level of the pulmonary artery bifurcation from a computed tomography coronary artery calcium scan in a patient with normal aortic dimensions showing the method for deriving the ascending and descending aortic dimensions. For interpretation, the images were transferred to a dedicated workstation (NetraMD, ScImage, Los Altos, California). The **white arrows** represent outer wall thoracic ascending aortic diameter and thoracic descending aortic diameter measurements perpendicular to the axis of rotation of the aorta.

examinations. For interpretation, the images were transferred to a dedicated workstation (NetraMD, ScImage, Los Altos, California). Systematic measurements of the outer aortic wall perpendicular to the axis of rotation of the aorta (Fig. 1) in the axial plane at the lower level of the pulmonary artery bifurcation in both the ascending and descending aorta were made by a licensed radiology technician experienced in cardiac CT. Measurements in excess of 3.5 cm were verified by an imaging cardiologist (D.S.B., J.D.F., L.E.J.T., or S.W.H.). Ascending and descending aorta cross-sectional areas were calculated from the measured diameter and indexed to BSA.

Statistical analysis. All continuous variables were assessed for normality using the Shapiro-Francia test and assessed visually by inspection of histograms and standardized normal probability (P-P) plots. Continuous variables were compared using the *t* test for 2 groups and categorical variables were compared using the Pearson chi-square statistic.

Using multiple linear regression analysis, we sought to detect parameters that are associated with ascending and descending thoracic aorta size and to exclude patients with these parameters from the final analysis groups; therefore, the final analysis group would include only patients without parameters that influence the aorta size and can be called essentially "normalized." We employed a 2-stage model. A main effects model, which included potentially clinically important predictors (i.e., age, gender, smoking, family history of CHD, diabetes, dyslipidemia, and hypertension), was developed in stage I and a main effects plus interactions model in stage II. Separate models were developed for ascending and descending aorta. Although either BMI or BSA formulas can be used for body size, BSA was chosen as the adjusting body size variable for all subsequent analyses. This is because BSA was previously found to have a greater association with thoracic aortic diameter than BMI does (6,7), and BSA was the body size variable that entered into selection models most frequently.

Additionally, regression diagnostics (statistical methods used for quality assurance of regression models) (8), including checking the assumptions of linear regression, analysis of residuals, checking for multicollinearity, model specification, and model validation using a jackknifing procedure, were used to assess the stage I models. The most influential outliers or high leverage subjects were thus identified and then excluded as part of the normalization process.

Using the stage I models, all 2×2 interactions were tested for significance at the relaxed <0.10 alpha level, both singly and against their main effects. Additionally, the significant interaction terms were tested against each other using stepwise model selection to produce the stage II models. All potential interactions were investigated. Regression diagnostics were done on stage II models, as described for stage I. None of the predictors were shown to be collinear, except when interaction terms were introduced. Standardized regression coefficients were used to assess the relative contribution of the predictors in both stage I and II models. Based on these analyses, we were able to identify single predictors or interactions that had both significant ($p < 0.05$) and strong influence on aortic size (nonstandardized beta coefficient >0.5 in absolute value, meaning either >0.5 mm or <-0.5 mm). Subjects with influential predictors or manifesting high leverage on the model's diagnostics were later excluded from the final analysis group.

For the final analysis, we grouped the subjects by gender, age, and BSA for the ascending and descending aorta, separately. We calculated the mean aortic diameter, standard deviation, and upper normal limit (mean + 2 standard deviations) for each group. We also used linear regression models to create BSA versus aortic diameter nomograms based on the former stratification method. Because

Table 1. Clinical Characteristics of the Initial Study Population

	Overall (n = 4,039)	Female Patients (n = 1,529)	Male Patients (n = 2,510)	p Value
Age (yrs)	55.0 ± 10.2	57.5 ± 9.7	53.5 ± 10.2	<0.0001
BSA (m^2)	1.9 ± 0.3	1.7 ± 0.2	2.1 ± 0.2	<0.0001
BMI (kg/m^2)	26.4 ± 4.7	25.1 ± 5.3	27.2 ± 4.2	<0.0001
Smoking	1,626 (40.3%)	645 (42.2%)	981 (39.1%)	0.05
Family history of CAD	1,443 (35.7%)	608 (39.8%)	835 (33.3%)	<0.001
Diabetes	192 (4.8%)	82 (5.4%)	110 (4.4%)	0.16
Dyslipidemia	1,444 (35.8%)	477 (31.2%)	967 (38.5%)	<0.001
Hypertension	1,083 (26.8%)	396 (25.9%)	687 (27.4%)	0.31
Ascending aorta (mm)	33.2 ± 4.1	31.8 ± 3.7	34.0 ± 4.1	<0.0001
Descending aorta (mm)	24.6 ± 3.0	23.0 ± 2.6	25.6 ± 2.8	<0.0001

BMI = body mass index; BSA = body surface area; CAD = coronary artery disease.

previous reports used aortic diameter and area inconsistently, we also calculated aortic area. Finally, we created a formula for calculation of predicted aortic size by age, gender, and BSA. The upper limit of normal was chosen as 2 standard deviations above the mean.

All data were analyzed using Stata version 8 (StataCorp, College Station, Texas), Analyze It version 1.7 (Analyse-it Software, Leeds, England), and SPSS version 12 (SPSS Inc., Chicago, Illinois).

RESULTS

Table 1 shows the clinical characteristics of the population. Male patients were significantly different from female patients in all parameters other than prevalence of diabetes and hypertension.

Table 2 shows the stage I models of potential clinical predictors of aortic diameter for the ascending and descending aorta, separately. Both models found that age, gender, BSA, diabetes, and hypertension were significant predictors of aortic diameter. Smoking was an independent predictor only of descending aortic diameter.

Stage II models (significant stage I variables + interactions) are presented in Table 3. For both the ascending and descending aorta, age, BSA, diabetes, hypertension, and an interaction between age and male gender were significant predictors of aortic diameter. Interactions between BSA and smoking, BSA and hypertension, and hypertension and dyslipidemia were found to be exclusively associated with descending aortic diameter.

For the ascending aorta, hypertension and diabetes mellitus were found in stage II models to be significant and influential parameters; therefore, 1,225 subjects with hypertension and/or diabetes were excluded from the final analysis. For the de-

Table 2. Stage I Models: Potentially Clinically Important Predictors of Aortic Diameter

	Ascending Aorta n = 4,039 Adjusted R^2 = 0.28			Descending Aorta n = 4,035 Adjusted R^2 = 0.45		
Predictor	Unstandardized Coefficient (Standardized)	Standard Error	p Value	Unstandardized Coefficient (Standardized)	Standard Error	p Value
Age	0.15 (0.38)	0.006	<0.001	0.13 (0.44)	0.004	<0.001
Male gender	1.18 (0.14)	0.15	<0.001	1.58 (0.26)	0.01	<0.001
BSA*	1.22 (0.30)	0.07	<0.001	1.12 (0.38)	0.05	<0.001
Smoking	0.05 (0.005)	0.11	0.69	0.23 (0.04)	0.07	0.001
Family history of CAD	−0.09 (−0.01)	0.11	0.45	−0.03 (−0.005)	0.07	0.66
Diabetes	−0.65 (−0.03)	0.26	0.01	−0.33 (−0.02)	0.17	0.05
Dyslipidemia	0.07 (0.008)	0.11	0.57	−0.06 (−0.01)	0.07	0.39
Hypertension	0.76 (0.08)	0.13	<0.001	0.37 (0.06)	0.08	<0.001
Constant	14.58	0.62	<0.001	7.71	0.40	<0.001

*Per 0.25 increments.
Abbreviations as in Table 1.

Table 3. Stage II Models: Stage I Models + Interactions

Predictor	Ascending Aorta (n = 4,038) Adjusted R^2 = 0.28			Descending Aorta (n = 4,034) Adjusted R^2 = 0.45		
	Unstandardized Coefficient (Standardized)	Standard Error	p Value	Unstandardized Coefficient (Standardized)*	Standard Error	p Value
Age	0.13 (0.32)	0.009	<0.001	0.11 (0.39)	0.006	<0.001
Male gender	−0.87 (−0.10)	0.66	0.19	0.19 (0.03)	0.43	0.66
BSA*	1.23 (0.30)	0.07	<0.001	1.29 (0.43)	0.06	<0.001
Smoking†	0.03 (0.004)	0.11	0.77	2.11 (0.35)	0.55	<0.001
Family history of CAD	−0.09 (−0.01)	0.11	0.46	−0.03 (−0.004)	0.07	0.72
Diabetes	−0.66 (−0.03)	0.26	0.01	−0.33 (−0.02)	0.17	0.05
Dyslipidemia	0.08 (0.01)	0.11	0.46	−0.16 (−0.03)	0.09	0.07
Hypertension	0.78 (0.09)	0.13	<0.001	1.81 (0.27)	0.67	0.003
Age, male	0.04 (0.24)	0.01	0.001	0.03 (0.22)	0.007	0.001
BSA,† smoking	—	—	—	−0.24 (−0.31)	0.07	0.001
Hypertension,† BSA	—	—	—	−0.20 (−0.24)	0.08	0.01
Hypertension,† dyslipidemia	—	—	—	0.37 (0.04)	0.16	0.02
Constant	15.91	0.74	<0.001	7.41	0.53	<0.001

*Per 0.25 increments; †past or current smoking.
Abbreviations as in Table 1.

scending aorta, hypertension and smoking were significant and influential parameters; therefore, 2,286 patients with hypertension and/or smoking were excluded from the final analysis. We additionally excluded 193 and 55 cases of outliers for the ascending aorta and descending aorta, respectively, after determining that they were influential and had high leverage and, thus, were likely to be abnormal. The final analysis groups included 2,952 subjects (1,147 female and 1,805 male subjects) and 1,931 subjects (736 female and 1,195 male subjects) for the ascending and descending aorta, respectively.

The mean aortic diameters for the final analysis group in the combined genders were 33 ± 4 mm and 24 ± 3 mm for ascending and descending thoracic aorta, respectively. The corresponding up-

Table 4. Ascending and Descending Aortic Diameters by Gender, Age, and BSA

Age (yrs)	BSA (m^2)	Ascending (mm)* (n = 2,952)		Descending (mm)* (n = 1,931)	
		Female Patients (n = 1,147)	Male Patients (n = 1,805)	Female Patients (n = 736)	Male Patients (n = 1,195)
<45	<1.70	28.4 ± 2.7, 33.8 (22.6–39.8)	28.6 ± 2.2, 33.0 (26.0–32.1)	20.2 ± 1.4, 23.0 (17.9–23.2)	20.9, NA (19.8–23.0)†
	1.70–1.89	30.0 ± 2.2, 34.4 (26.0–34.2)	30.1 ± 3.1, 36.3 (24.9–37.7)	21.4 ± 1.6, 24.6 (17.8–24.0)	22.6 ± 2.0, 26.6 (18.6–27.4)
	1.90–2.09	29.8 ± 2.6, 35.0 (26.7–35.6)	30.9 ± 2.7, 36.3 (21.8–38.3)	20.3 ± 1.2, 22.7 (18.5–22.0)	23.3 ± 1.7, 26.7 (18.5–26.8)
	>2.1	31.3, NA (31.3)†	32.3 ± 3.0, 38.3 (25.6–41.0)	21.9, NA (21.9)†	24.3 ± 2.0, 28.3 (20.3–30.1)†
45–54	<1.70	29.6 ± 2.8, 35.2 (21.9–36.9)	31.0 ± 3.8, 38.6 (26.0–36.3)	21.1 ± 1.6, 24.3 (17.1–24.6)	22.0 ± 1.1, 24.2 (20.6–23.9)
	1.70–1.89	31.4 ± 2.9, 37.2 (24.7–39.7)	31.7 ± 3.2, 38.1 (21.9–39.0)	22.2 ± 1.6, 25.4 (19.1–26.1)	23.5 ± 2.0, 27.5 (19.2–30.1)
	1.90–2.09	32.5 ± 3.2, 38.9 (26.7–40.4)	33.1 ± 3.3, 39.7 (26.7–41.7)	23.6 ± 1.8, 27.2 (19.8–26.0)	24.8 ± 2.2, 29.2 (19.8–31.4)
	>2.1	34.4 ± 3.1, 40.6 (29.4–39.0)	34.4 ± 3.1, 40.6 (25.3–42.5)	23.9 ± 2.2, 28.3 (21.0–29.4)	25.8 ± 1.9, 29.6 (20.5–30.8)
55–64	<1.70	31.1 ± 2.9, 36.9 (24.6–39.1)	31.5 ± 2.4, 36.3 (28.0–34.9)	22.3 ± 1.8, 25.9 (18.5–27.4)	23.1 ± 1.5, 26.1 (20.6–26.0)
	1.70–1.89	31.8 ± 2.6, 37.0 (26.5–37.6)	33.5 ± 3.1, 39.7 (28.0–41.7)	23.3 ± 1.9, 27.1 (19.8–27.4)	25.2 ± 1.7, 28.6 (21.9–30.2)
	1.90–2.09	33.0 ± 3.0, 39.0 (26.7–40.3)	34.6 ± 3.3, 41.2 (26.0–43.1)	24.0 ± 1.9, 27.8 (20.5–28.0)	25.9 ± 2.0, 29.9 (20.6–31.1)
	>2.1	35.4 ± 3.3, 42.0 (30.1–43.8)	36.1 ± 3.5, 43.1 (28.0–52.0)	25.5 ± 3.1, 31.7 (19.9–32.8)	27.2 ± 2.2, 31.6 (20.5–34.2)
≥65	<1.70	32.5 ± 2.5, 37.5 (27.4–37.6)	33.9 ± 2.3, 38.5 (32.2–37.6)	23.4 ± 1.8, 27.0 (19.8–28.7)	25.3, NA (23.9–28.0)†
	1.70–1.89	33.4 ± 2.9, 39.2 (26.7–44.4)	35.0 ± 3.0, 41.0 (28.7–42.0)	24.6 ± 1.4, 27.4 (21.9–27.4)	26.8 ± 2.8, 32.4 (21.9–37.0)
	1.90–2.09	34.3 ± 4.2, 42.7 (28.0–43.8)	35.8 ± 3.2, 42.2 (28.7–43.1)	25.2 ± 1.9, 29.0 (22.6–29.4)	27.0 ± 2.0, 31.0 (22.6–30.9)
	>2.1	32.8, NA (30.8–34.9)†	36.8 ± 2.8, 42.4 (32.1–48.5)	26.0 ± 1.9, 29.8 (23.3–29.4)	28.5 ± 2.0, 32.5 (25.1–33.5)

*Values are expressed as mean ± 1 SD, upper limit (range). †In cases when there were fewer than 6 patients in a group, SD was not calculated and the upper limit of normal was not computed.
Abbreviations as in Table 1.

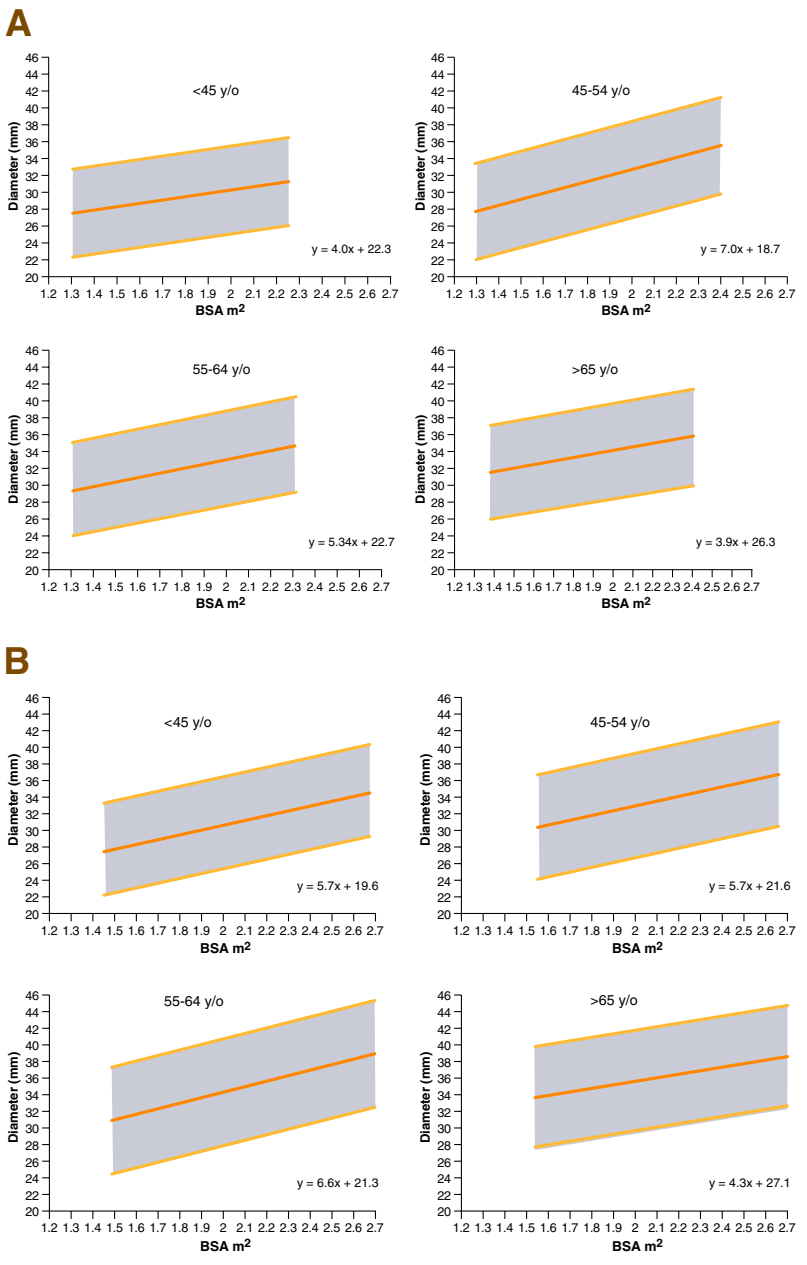

Figure 2. Predicted Ascending and Descending Thoracic Aortic Diameter by Gender, Age, and BSA

(A) Nomogram of normal value of thoracic ascending aorta—female patients. **(B)** Nomogram of normal value of thoracic ascending aorta—male patients. *Continued on next page.*

per limits of normal were 41 and 30 mm. The mean ascending and descending thoracic aortic diameters for females were 31.4 ± 3.2 mm and 22.6 ± 2.2 mm, respectively, and the corresponding upper limits of normal were 37.4 and 27.0. The mean ascending and descending thoracic aortic diameters for males were 33.5 ± 3.6 mm and 25.1 ± 2.5 mm, respectively, and the corresponding upper limits of normal were 40.7 and 30.1.

Table 4 details aortic diameter parameters stratified by gender, age, and BSA. In the final ascending aorta analysis group there were 787 (27%), 106 (4%), and 1 (0.04%) subjects with an ascending thoracic aortic diameter greater than 35, 40, and 50 mm, respectively. Nomograms for aortic diameter, ascending and descending, for gender and age group by BSA are shown in Figure 2. The formula for predicting ascending aortic diameter is 14.10 +

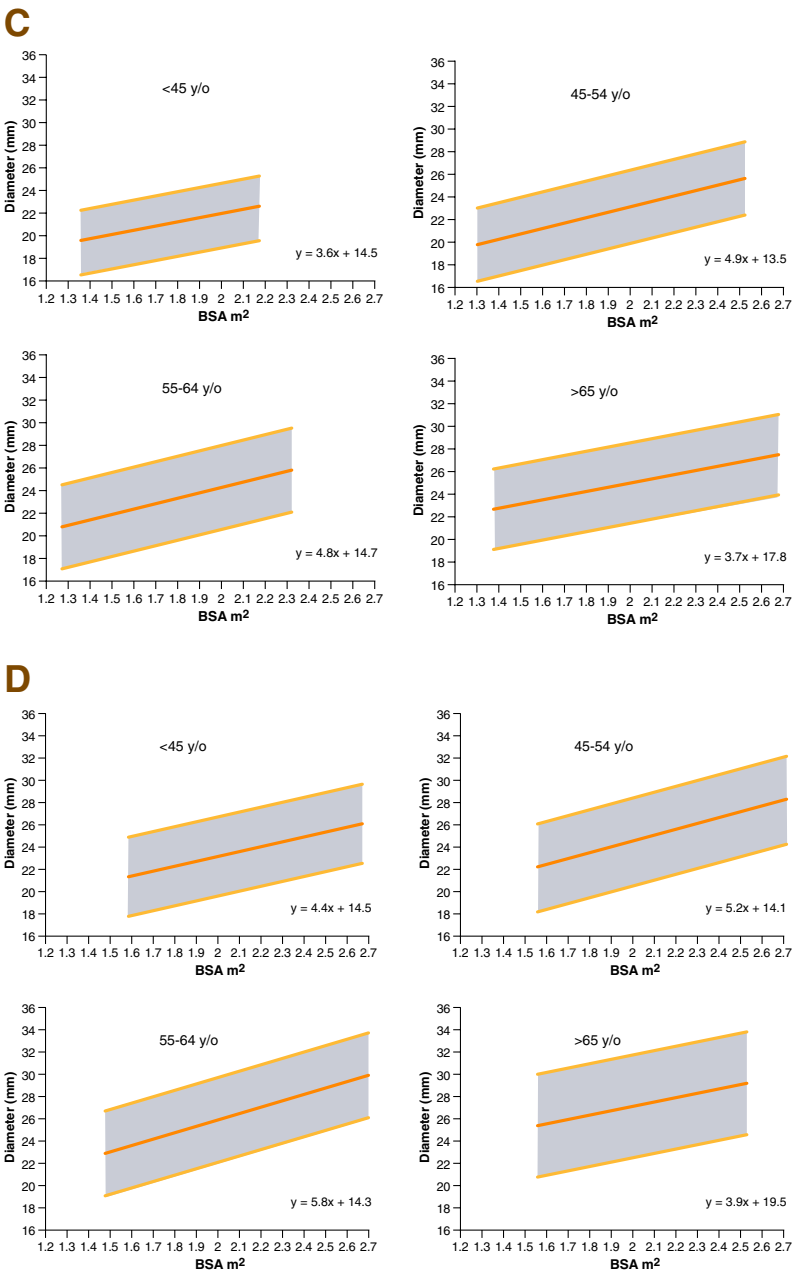

Figure 2. Predicted Ascending and Descending Thoracic Aortic Diameter by Gender, Age, and BSA

(C) Nomogram of normal value of thoracic descending aorta—female patients. **(D)** Nomogram of normal value of thoracic descending aorta—male patients. The **gold lines** represent the estimated mean thoracic ascending aorta diameter. The **orange lines** represent the estimated 95% normal confidence upper and lower limits. BSA = body surface area; y/o = years of age.

0.13 × age (years) − 1.09 (if male) + 0.04 × age (if male) + 5.80 × BSA; the formula for predicting descending aortic diameter is 7.73 + 0.11 × age (years) + 0.41 (if male) + 0.02 × age (if male) + 4.91 × BSA. Table 5 shows the ascending and descending aortic cross-sectional area measurements by BSA, gender, and age.

DISCUSSION

Measurement of thoracic aortic size is important in detecting aortic aneurysm. The present study demonstrates that measurements can be made by gated, noncontrast CT scans obtained for assessment of CAC.

Table 5. Ascending and Descending Aortic Cross-Sectional Area for BSA by Gender and Age (cm^2/m^2)*

Age (yrs)	Ascending Aorta (n = 2,952)		Descending Aorta (n = 1,931)	
	Female Patients (n = 1,147)	Male Patients (n = 1,805)	Female Patients (n = 736)	Male Patients (n = 1,195)
<45	3.9 ± 0.8, 5.5 (2.5–8.3)	3.8 ± 0.7, 5.2 (1.87–6.00)	2.0 ± 0.3, 2.6 (1.34–2.67)	2.1 ± 0.3, 2.7 (1.34–3.10)
45–54	4.4 ± 0.8, 6.0 (2.49–7.28)	4.3 ± 0.8, 5.9 (2.09–7.19)	2.2 ± 0.3, 2.8 (1.35–3.20)	2.4 ± 0.4, 3.2 (1.50–3.95)
55–64	4.7 ± 0.9, 6.5 (2.80–7.96)	4.7 ± 0.9, 6.5 (2.65–9.65)	2.4 ± 0.4, 3.2 (1.48–3.84)	2.6 ± 0.4, 3.4 (1.50–4.18)
≥65	5.2 ± 0.9, 7.0 (3.08–9.11)	5.1 ± 0.9, 6.9 (3.23–8.03)	2.7 ± 0.4, 3.5 (1.78–4.31)	3.0 ± 0.5, 4.0 (1.98–5.97)
Total	4.6 ± 0.9, 6.4 (2.49–9.11)	4.4 ± 1.0, 6.3 (1.87–9.65)	2.4 ± 0.4, 3.2 (1.34–4.31)	2.5 ± 0.5, 3.5 (1.34–5.97)

*Values are expressed as mean ± 1 SD, upper limit (range).

Relationship to previous studies. The mean aortic diameter and cross-sectional area found in the current study are comparable to the dimensions previously reported by CT, MRI, and echocardiography studies (Table 6) (1–3,6,7,9–12). Our findings that aortic dimension relates directly to age (2,3,6,7,9) and BSA confirm previous results (6,7). Interestingly, we also confirmed that the association between BSA and aortic diameter was stronger than that between BMI and aortic diameter (6,7).

Regarding the relationship between thoracic aortic dimensions and gender, previous works have reported that male gender is associated with a larger aortic diameter (3,6). In our study, however, male gender was a significant predictor only when interacting with age, such that older men have, on average, larger aorta than women of a similar age, but the difference is smaller for younger men and women (Table 3).

In addition to hypertension, smoking was found to be an independent predictor only of the descending aortic diameter. It has been suggested that the etiology of thoracic aneurysms differs between the ascending and the descending segments and that the pathogenesis of aneurysms in the descending thoracic aorta may more closely resemble that of the abdominal aorta aneurysms than that of ascending thoracic aneurysms (10). Because smoking has been shown to be the risk factor most strongly associated with abdominal aortic aneurysms, it is not surprising that the current study finds that smoking is also associated with larger descending thoracic aorta diameters (11).

The similarity of the findings of Guthaner et al. (1) and the current study suggests that at the level of the pulmonary artery, there is reasonable resemblance between gated noncontrast CT and nongated contrast CT measurements. Though the current study was not designed to address this issue, this observation, if further proven by additional studies, might suggest that the normal limits proposed in the current work can be used for contrast nongated studies too. Gated aorta studies, however, can provide additional information in the form of temporal diameter changes during the cardiac cycle.

Table 6. Summary of All Previous Data Regarding Normal Limits for Thoracic Aortic Dimensions Using Any Modality

Modality	Author (Ref. #)	Year	Age Range (yrs)	Anatomical Landmark of the Ascending Aorta	Ascending Aorta Dimensions Diameter or Area/m^2 (N)	Descending Aorta Dimensions Diameter or Area/m^2 (N)	Comments
MRI	Kersting-Sommerhoff et al. (9)	1987	Young adults	Pulmonary artery level	30 ± 4 mm (20)	24 ± 4 mm (20)	Adopted also for CT (10)
	Mohiaddin et el. (11)	1990	10–>60	Pulmonary artery level	2.1–4.8 cm^2/m^2 (70)	1.1–2.8 cm^2/m^2 (70)	
Echo	Roman et al. (7)	1989	20–74	Proximal ascending aorta	30 ± 4 cm (68 male patients), 27 ± 4 (67 female patients)		TEE
	Drexler et al. (12)	1990	19–30	2 cm above the aortic sinus	3.6 ± 0.5 cm^2/m^2 (25)	1.9 ± 0.4 cm^2/m^2 (25)	TEE
	Agmon et al.(6)	2003		Pulmonary artery level	33 ± 4 (373)	26 ± 3 (373)	TEE
CT	Guthaner et al. (1)	1979		Pulmonary artery level	32 ± 5 mm (15)	25 ± 4 mm (15)	
	Aronberg et al. (2)	1984	21–≥61	Caudal to the aortic arch	35 mm (102)	26 mm (102)	
	Hager et al. (3)	2002	17–89	Caudal to the aortic arc "at maximal size"	31 ± 4 mm (70)	25 ± 4 (70)	
	Current study	2008	26–75	Pulmonary artery level	33 ± 4 mm, 4.5 ± 0.9 cm^2/m^2 (2,952)	24 ± 3 mm, 2.4 ± 0.5 cm^2/m^2 (1,930)	

Data are provided as reported in the original manuscript (some of the manuscripts provide only partial data). Diameter is given in millimeters and cross-sectional area in square centimeters.
CT = computed tomography; Echo = echocardiography; MRI = magnetic resonance imaging; TEE = transesophageal echocardiography.

The current study was not designed to provide prognostic data. However, a previous study (12) has shown that thoracic aortic dissection and rupture is related to aortic diameter and that the annual complication rate for ascending thoracic aorta diameter below 35 mm (the mean in the current study is 33 mm) and below 40 mm (the upper limit of normal in the current study was 41 mm) was 0% and 0.3%, respectively. Following the above-mentioned criteria, we found that only 4% of the patients in the final analysis group had more than 0.3% of annual risk for rupture.

In the current study, we used CT data from a low-risk population in a selective manner. Identification of clinical parameters that were associated with increasing aortic dimensions allowed exclusion of patients with these parameters from the final analysis group. A final analysis group that more closely represents a "normal" population was defined, as opposed to the original unselected asymptomatic population. In the future, when it is impractical to examine a large number of healthy subjects of various ages and both genders, this statistical approach to selecting a "normal" patient group may be appropriate to determine normal limits for anatomic or physiologic measurements.

Study limitations. We describe herein an approach to arrive at a "normal" sample from a "nearly normal" unselected group of patients. The major limitation of such an approach, in our opinion, is that if the risk factor profile of the initial unselected group is significantly biased away from normal, no amount of fine-tuning can make the sample "normative" and, therefore, fewer initial risk factors enable a better "normalization" process. The risk factor profile of our initial group is given in Table 1. When we compare the current study group with the American Heart Association—heart disease and stroke statistics—2007 update data (13) and with the MESA (Multi-Ethnic Study of Atherosclerosis) population (14), which included a cohort of patients with no CAD and a low-risk factor profile, we see that the current study group has a lower risk profile. Therefore, we believe that the initial group of the study is suitable for the whittling down process and the final group represents a truly normal sample.

Several other limitations should also be addressed. The dataset comprises a patient population from a single center. Although our initial unselected group consisted of a fairly large number of patients, owing to the whittling down and stratification process, in a few subgroups, we were not able to provide an upper normal limit because of low subject count. It is also noted that measurement of aortic diameters by noncontrast CT in the level of the pulmonary artery may not be representing the true short axis, and to measure the true short axis, contrast-enhanced study may be required. Lastly, because different scanners were employed during the course of this study, there could be some variability due to the calibration of the measurement itself. However, given the strict acceptance criteria applied to our CT scanners, this difference for aortic measurements is likely to be minimal.

CONCLUSIONS

Assessment of aortic size is possible from CT scans obtained for CAC measurements. Given the importance of detecting thoracic aortic aneurysm, consideration should be given to including these measurements as part of the clinical report of the CAC scan. The nomograms provided by our study could allow such a report to include a statement regarding the relationship of a patient's measurements to the expected upper normal for a given patient (15–17).

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