Aortic Diameter ≥5.5 cm Is Not a Good Predictor of Type A Aortic Dissection

Observations From the International Registry of Acute Aortic Dissection (IRAD)

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Background—Studies of aortic aneurysm patients have shown that the risk of rupture increases with aortic size. However, few studies of acute aortic dissection patients and aortic size exist. We used data from our registry of acute aortic dissection patients to better understand the relationship between aortic diameter and type A dissection.

Methods and Results—We examined 591 type A dissection patients enrolled in the International Registry of Acute Aortic Dissection between 1996 and 2005 (mean age, 60.8 years). Maximum aortic diameters averaged 5.3 cm; 349 (59%) patients had aortic diameters <5.5 cm and 229 (40%) patients had aortic diameters <5.0 cm. Independent predictors of dissection at smaller diameters (<5.5 cm) included a history of hypertension (odds ratio, 2.17; 95% confidence interval, 1.03 to 4.57; $P=0.04$), radiating pain (odds ratio, 2.08; 95% confidence interval, 1.08 to 4.0; $P=0.03$), and increasing age (odds ratio, 1.03; 95% confidence interval, 1.00 to 1.05; $P=0.03$). Marfan syndrome patients were more likely to dissect at larger diameters (odds ratio, 14.3; 95% confidence interval, 2.7 to 100; $P=0.002$). Mortality (27% of patients) was not related to aortic size.

Conclusions—The majority of patients with acute type A acute aortic dissection present with aortic diameters <5.5 cm and thus do not fall within current guidelines for elective aneurysm surgery. Methods other than size measurement of the ascending aorta are needed to identify patients at risk for dissection. **(*Circulation*. 2007;116:1120-1127.)**

Key Words: aneurysm ■ aorta ■ aortic aneurysm ■ diagnosis

Relatively rare compared with other causes of cardiovascular death, acute ascending aortic dissection continues to defy our attempts to predict or prevent it. Aortic dissection is often described as catastrophic because the majority of patients present with severe intense pain accompanied by rapidly developing complications that may include acute aortic insufficiency, congestive heart failure, acute coronary occlusion, pericardial effusion and tamponade, stroke, syncope, limb ischemia, renal insufficiency, shock, rupture, and death. Despite better diagnostic imaging methods and newer surgical techniques for treatment, the mortality of type A aortic dissection ranges from 14% to 30% and still averages ≈25%.[1–3]

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Identification of patients at risk for aortic dissection is difficult. Established clinical risk factors are systemic hypertension (widespread in the general population) and aortic dilation or aneurysm, which can only be found with diagnostic imaging. Even patients with Marfan syndrome, Ehlers-Danlos syndrome, familial aortic aneurysm, or congenitally bicuspid aortic valve who are known to be at increased risk for dissection often go unrecognized until they present with an acute aortic syndrome.

Received March 16, 2007; accepted July 3, 2007.

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Guest Editor for this article was William R. Hiatt, MD.

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Circulation **is available at http://circ.ahajournals.org** **DOI: 10.1161/CIRCULATIONAHA.107.702720**

The mainstay of prevention of aortic dissection, aside from treatment of hypertension, is elective aortic surgery in patients with dilated ascending aortas. Guidelines for timing of aortic root repair are based on clinical observations by experienced clinicians and surgeons and a consensus based on clinical series and patient characteristics.[4–9] Consensus exists that surgery to prevent rupture or dissection of the ascending thoracic aortic aneurysm should be recommended when the ascending aortic diameter reaches 5.5 cm for non-Marfan patients and 4.5 cm in Marfan patients.[4]

Our observation that many dissection patients do not seem to have markedly dilated aortas at the time of presentation led us to examine the aortic diameters in the large patient database of the International Registry of Acute Aortic Dissection (IRAD). We sought to determine the utility of aortic size as a marker of risk for aortic dissection by determination of how many patients who present with acute type A aortic dissection have an aortic diameter <5.5 cm, the size for which elective repair of aortic aneurysm would generally be recommended.

Methods

The inception and structure of IRAD has been previously described.[10] Data were obtained from hospital records of 591 patients with acute type A aortic dissection enrolled in IRAD between 1996 and 2005. Patients were identified at hospital presentation or by searches of discharge diagnosis records and surgical and echocardiography laboratory databases for all cases of acute dissection. Acute type A dissection was defined as any dissection that involved the ascending aorta in a patient who presented within 14 days of symptom onset. Traumatic dissection was excluded from IRAD. Data were collected on standardized forms with standard definitions.

Information on 290 clinical variables was collected, such as patient demographics, medical history, clinical presentation, physical findings, imaging studies, medical and surgical management, and in-hospital mortality. Maximum aortic diameters in the ascending aorta were measured by computed tomography, transesophageal echocardiography, magnetic resonance imaging, and/or angiography at the time of presentation. If multiple imaging tests were performed on a patient, the test that reflected the largest aortic diameter was selected by the site investigator and used for analysis. Images were read independently by experienced clinical radiologists, angiographers, and echocardiographers at each tertiary center. Maximum aortic diameter was taken from cross-sectional tomograms perpendicular to the long axis of the ascending aorta. Data were collected at presentation or by retrospective physician review of hospital records and were forwarded to the IRAD coordinating center at the University of Michigan. All forms were checked for face validity and analytical internal validity.

Data Analysis

Data analysis was performed with the use of SPSS statistical analysis software (SPSS Inc., Chicago, Ill.). Summary statistics that compare the 2 groups (aortic size <5.5 cm and ≥5.5 cm) are presented as frequencies and percentages for categorical variables and mean±SD for continuous variables. In all cases, missing data were not defaulted to negative, and denominators reflect only cases reported. Univariate associations with dissection at diameters <5.5 cm among clinical variables were obtained with χ^2 or 2-sided Fisher exact tests for nominal variables and Student t test for continuous variables. Forward stepwise logistic regression analysis was performed with variables that reached $P<0.20$ on univariate testing to determine independent predictors of dissection at aortic diameter <5.5 cm.

The 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.

Results

Demographics and Clinical History

The average patient age was 60.8±14.4 years old, two thirds were male, and 71.2% had a history of hypertension (Table 1). Nearly 5% of patients had Marfan syndrome, 4.2% had bicuspid aortic valves, 12.3% had a history of aortic valve disease, 12.4% had known aortic aneurysms, and 5.3% had a history of prior coronary artery bypass surgery.

Presenting Symptoms

Among the patients with aortic diameters <5.5 cm, more back pain, more radiating pain, more abrupt onset of pain, and more neurological deficits were present (Table 2). On presentation, 32% of patients had hypertension, 12.8% were in shock, and

TABLE 1. Demographics and Clinical History

	All	Ascending <5.5 cm	Ascending ≥5.5 cm	*P*
No. (%)	591	349 (59.1)	242 (40.9)	
Demographics				
Age, n (SD)	60.8 (14.4)	60.5 (13.6)	61.2 (15.5)	0.61
Male, n (%)	390 (66.0)	226 (64.8)	164 (67.8)	0.45
History				
Hypertension, n (%)	407 (71.2)	247 (72.4)	160 (69.3)	0.41
Marfan syndrome, n (%)	28 (4.9)	11 (3.2)	17 (7.5)	0.02
Known aortic aneurysm, n (%)	70 (12.4)	41 (12.0)	29 (12.9)	0.75
Prior aortic dissection, n (%)	22 (3.9)	15 (4.4)	7 (3.1)	0.44
BAV (n=383), n (%)*	16 (4.2)	6 (2.6)	10 (6.5)	0.06
Aortic valve disease, n (%)	68 (12.3)	30 (9.0)	38 (17.4)	0.003
Diabetes mellitus, n (%)	22 (3.9)	14 (4.2)	8 (3.6)	0.72
AVR, n (%)	32 (5.8)	14 (4.3)	18 (7.9)	0.08
CABG, n (%)	29 (5.3)	9 (2.8)	20 (8.8)	0.002
Catheterization/angiography, n (%)	34 (9.2)	15 (6.9)	19 (12.5)	0.07

*Presence or absence of BAV not included in early data collection (i.e., in first 108 patients). BAV indicates bicuspid aortic valve; AVR, prior aortic valve replacement; and CABG, coronary artery bypass surgery.

TABLE 2. Presenting Symptoms

Presenting Symptoms, n (%)	All	Ascending <5.5 cm	Ascending ≥5.5 cm	*P*
Chest pain	476 (82.8)	286 (83.6)	190 (81.5)	0.52
Anterior	382 (75.6)	232 (75.8)	150 (75.4)	0.91
Posterior	207 (44.2)	128 (45.4)	79 (42.5)	0.53
Pain severity				
Mild	46 (9.8)	20 (7.1)	26 (13.8)	0.02
Severe	328 (69.8)	201 (71.3)	127 (67.6)	0.39
Worst ever	96 (20.4)	61 (21.6)	35 (18.6)	0.43
Pain in head or neck	133 (24.4)	87 (26.9)	46 (20.8)	0.10
Back pain	252 (45.8)	165 (50.8)	87 (38.7)	0.005
Abdominal pain	138 (25.0)	88 (27.1)	50 (22.1)	0.19
Migrating	89 (16.6)	59 (18.7)	30 (13.6)	0.12
Radiating	176 (32.4)	121 (37.3)	55 (25.0)	0.003
Leg pain	57 (10.6)	39 (12.2)	18 (8.2)	0.14
Abrupt onset	490 (88.3)	307 (93.0)	183 (81.3)	<0.001
Syncope	107 (19.0)	69 (20.5)	38 (16.7)	0.25
Other neurological deficit	52 (9.3)	40 (12.1)	12 (5.3)	0.007
Febrile	12 (3.4)	9 (4.4)	3 (2.1)	0.37

26% had clinical signs of pulse deficits (Table 3). Among the signs of aortic dissection, there was little to distinguish between patients with smaller or larger diameters of the ascending aorta apart from more symptoms of cerebral malperfusion in the patients with smaller diameters and more congestive heart failure in the patients with larger diameters (both $P=0.05$).

Diagnostic Imaging Studies

Chest x-ray findings were similar in both groups with 69% reported to have a widened mediastinum. Significantly more patients with dissections at diameters <5.5 cm had a normal chest x-ray (12.1% versus 6.8%, $P=0.05$) (Table 3).

Patients had an average of 1.85 aortic imaging studies. CT was the most frequent first imaging modality performed in 64% of patients, echocardiography, usually transesophageal echo, in 31% of patients, angiography in 4% of patients, and magnetic resonance in 1% of patients. The second imaging modality was echocardiography in 57% of patients, CT in 23% of patients, magnetic resonance in 9% of patients, and angiography in 12% of patients.

Aortic Diameters

Among all 591 patients with acute type A aortic dissection, the mean ascending aortic diameter was 5.3 cm and the median was 5.0 cm with a wide distribution (2 to 10 cm) (Figure 1). Nearly 60% of the 591 patients had diameters <5.5 cm, which is the current guideline for elective aortic aneurysm surgery, and 40% of patients had aortic diameters <5 cm. Compared with the group with aortic diameters of <5.5 cm, the cohort with larger diameters included more patients with Marfan syndrome, more bicuspid aortic valve, aortic valve disease, and prior coronary artery bypass surgery patients (all $P<0.05$), but the number of patients with prior known aortic aneurysm was not different between groups. The exclusion of Marfan and bicuspid aortic valve patients had no significant effect on the diameter results: mean diameter, 5.2 cm; median diameter, 5.0 cm. Independent predictors of dissection with an aortic diameter <5.5 cm were a history of hypertension, radiating pain, younger age, and the absence of Marfan syndrome (Table 4). We examined risk factors in the patients with normal diameters (<4.0 cm) and found that 51% had none of the known risk factors (hypertension, Marfan, or bicuspid valve) for aortic dissection. Mortality was not associated with diameter and did not vary significantly across the 10 diameter categories (Figure 2).

Discussion

These data demonstrate that, among patients with acute type A aortic dissection in IRAD, aortic diameter at presentation was <5.5 cm in the majority of cases, and <5.0 cm in 40% of cases. Currently established indications for surgical repair of aortic aneurysm would therefore not include the majority of IRAD patients who present with acute type A aortic dissection, even if those patients had undergone aortic imaging prior to presentation. Aortic size at presentation was not related to mortality, but hypertension, patient age, and nature of symptoms at presentation were associated with aortic size.

The mortality of type A acute aortic dissection over the past 20 to 30 years has not improved dramatically, despite better imaging methods and improved surgical management techniques. In 2 recent large series, type A dissection continues to carry a high (22% to 25%) mortality, whereas mortality for elective ascending aortic aneurysm surgery was reported as low as 1.5% to 2.5%.[2,3,11,12]

The reported 30% to 50% short-term mortality in patients with aortic aneurysm diameters >6 cm was, until fairly recently, the basis for recommendation of elective aortic surgery at 6 cm. Davies and colleagues followed 304 patients with unoperated thoracic aortic aneurysms (dissection free at presentation) with

TABLE 3. Chest X-Ray Findings and Signs of Aortic Dissection

	All	Ascending <5.5 cm	Ascending ≥5.5 cm	*P*
Chest x-ray findings				
Normal, n (%)	51 (10.0)	37 (12.1)	14 (6.8)	0.05
Widened mediastinum, n (%)	353 (69.1)	207 (67.6)	146 (71.2)	0.39
Abnormal aortic contour, n (%)	255 (51.5)	145 (48.8)	110 (55.6)	0.14
Abnormal cardiac contour, n (%)	137 (27.8)	77 (26.0)	60 (30.5)	0.28
Displacement/calcification of aorta, n (%)	37 (7.5)	24 (8.1)	13 (6.6)	0.54
Pleural effusion, n (%)	77 (15.7)	42 (14.2)	35 (17.9)	0.27
Signs of aortic dissection				
Presenting hemodynamics				
hypertensive, n (%)	179 (32.3)	107 (32.7)	72 (31.6)	0.78
normotensive, n (%)	249 (44.9)	141 (43.1)	108 (47.6)	0.30
hypotensive, n (%)	80 (14.4)	53 (16.2)	27 (11.9)	0.17
shock, n (%)	71 (12.8)	43 (13.1)	28 (12.3)	0.78
cardiac tamponade, n (%)	30 (5.3)	18 (5.4)	12 (5.2)	0.93
First BP systolic mean, n (SD)	129.6 (38.2)	130.9 (39.8)	127.8 (35.8)	0.34
First BP diastolic mean, n (SD)	74.6 (21.4)	74.2 (21.7)	75.0 (21.0)	0.68
Murmur of aortic insufficiency, n (%)	243 (45.3)	138 (43.3)	105 (48.2)	0.26
Pulse deficits, n (%)	139 (26.3)	84 (26.6)	55 (25.9)	0.87
Pericardial friction rub, n (%)	12 (2.2)	4 (1.2)	8 (3.6)	0.08
CVA, n (%)	44 (7.6)	32 (9.3)	12 (5.1)	0.06
Ischemic peripheral neuropathy, n (%)	17 (3.1)	14 (4.3)	3 (1.3)	0.05
Ischemic spinal cord damage, n (%)	11 (2.0)	8 (2.5)	3 (1.4)	0.54
Ischemic lower extremity, n (%)	54 (9.8)	37 (11.4)	17 (7.6)	0.14
Coma/altered consciousness, n (%)	64 (11.6)	41 (12.5)	23 (10.1)	0.38
Congestive heart failure, n (%)	27 (4.9)	11 (3.4)	16 (7.1)	0.05

BP indicates blood pressure; CVA, cerebrovascular accident.

aortic diameters ≥3.5 cm, for a median of 32 months for the end points of rupture, dissection, and death.[12] In their series, the risk of dissection was twice that of the risk of rupture, and a fairly linear correlation existed between aortic diameter and risk of rupture. Interestingly, the risk of rupture increased by 50% when aortic diameters increased from a range of 4 to 4.9 cm to a range of 5 to 5.9 cm. The risk of aortic dissection, however, did not increase between 4 to 4.9 cm and 5 to 5.9 cm. Beyond 6 cm the risk of dissection rose again to parallel that of rupture, but at any given diameter dissection occurred at a higher rate than rupture.

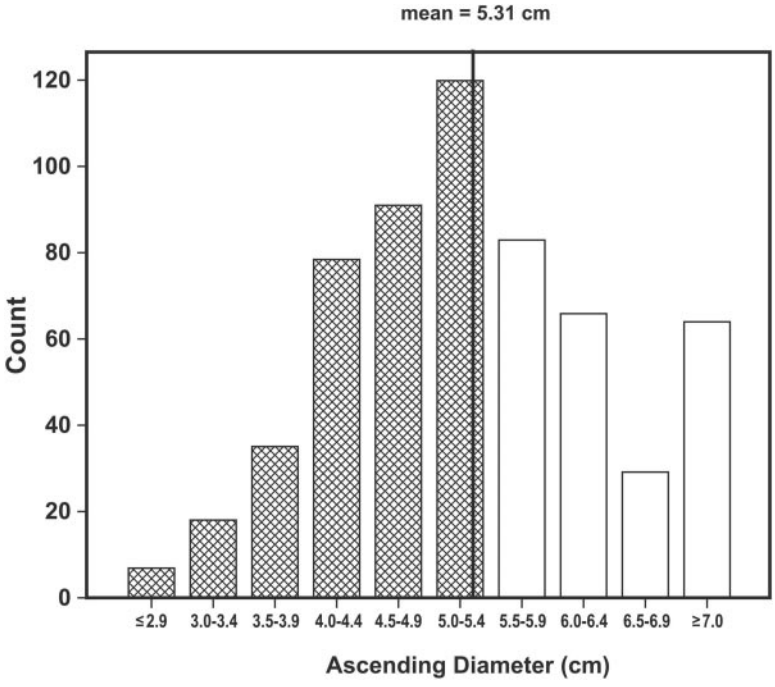

Figure 1. Distribution of aortic size at time of presentation with acute type A aortic dissection (cm). Shaded bars indicate 59% of patients with diameters <5.5 cm.

TABLE 4. Independent Predictors of Dissection at Diameters <5.5 cm

	Odds Ratio	95% Confidence Interval	*P*
History of hypertension	2.17	1.03 to 4.57	0.04
Radiating pain	2.08	1.08 to 4.0	0.03
Age	1.03	1.00 to 1.05	0.03

In those patients with an initial aortic diameter of >6 cm, the combined risk of rupture, dissection or death was >15% per year. Clearly, elective surgical repair or reconstruction is the preferred option if patients at risk can be identified. Most surgeons now recommend elective operation at aortic diameters of ≥5.5 cm.[5,12]

Aortic dissection has been most carefully studied in Marfan patients who have the highest lifetime risk of dissection.[13] In Marfan patients the threshold for aortic surgery has also been lowered from 5.5 to 4.5 cm on the basis of observations that 15% of Marfan patients dissect or rupture at 5 cm or less.[4] Of 158 Marfan patients operated on for aortic dissection, 46% had diameters <6.5.[4] Some investigators have suggested surgery at even smaller sizes in Marfan patients, especially if rapid expansion of the aorta has been observed (>0.5 cm/year) or in case of a family history of dissection.[4,14,15] Unfortunately, too often it is only when the patient suffers an acute dissection that the diagnosis of Marfan syndrome is made. Our finding that patients with Marfan syndrome were less likely to dissect at smaller diameters may seem to be at odds with this known risk of dissection. The present study was not a prospective study of aneurysm patients or Marfan patients, and this registry collected relatively few Marfan patients. Only patients with acute dissection were entered into our registry. It is clear that physicians have increasingly recognized the limited value of aortic size in clinical decision-making for Marfan patients. Our data suggest that aortic size has limited value in non-Marfan patients, too, because dilation is only one of many manifestations of structural weakness of the aortic wall.

The risk of dissection has also been related to aortic size in bicuspid aortic valve patients.[7] Svensson and colleagues noted that 12.5% of 40 bicuspid aortic valve patients with dissection had aortic sizes <5 cm estimated at the time of surgery.[7] This finding was similar to the reported proportion of Marfan patients who dissected at smaller aortic sizes. It is likely that some patients were first diagnosed with Marfan syndrome or, for that matter, bicuspid aortic valves at the time they presented with acute dissection. Thus, a series of dissection patients is not representative of the broader population of Marfan and bicuspid valve patients. This brings us back to the problem of identification of patients with inherent aortic weakness before a life-threatening complication occurs.

For the present study, we examined aortic size at the time of presentation, after dissection had occurred. No data exist that demonstrate that aortic sizes immediately before dissection are much smaller than after dissection. In studies that have reported aortic size at the time of surgery,[7,8] the presumption has been that the size of the aorta was, if anything, smaller before than after dissection occurs (i.e., before the layers of the wall separate and the dissecting hematoma spreads between the layers of the wall). The dynamic and unpredictable evolution of a dissection makes it difficult to generalize about the relative sizes of the aorta before and after dissection. In some cases the major finding is

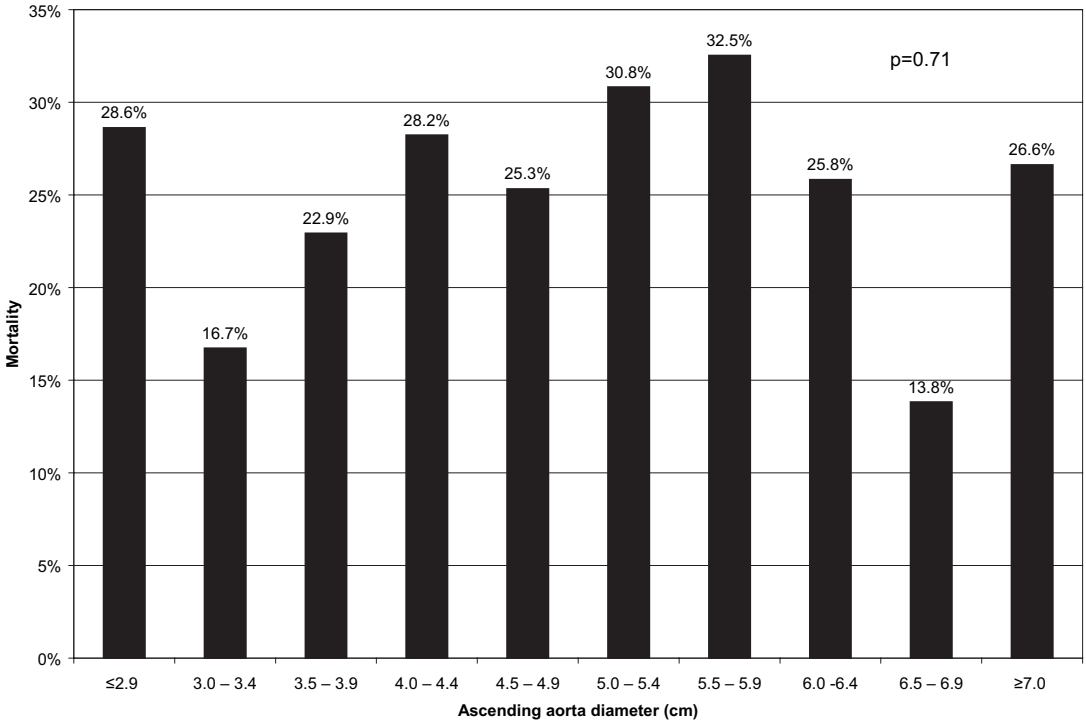

Figure 2. Mortality of acute type A aortic dissection by ascending aortic diameter (cm).

apparent compression of the true lumen by the blood pressure in the false lumen and only modest overall aortic enlargement. In experimental studies of human and porcine cadaver specimens, Williams et al demonstrated a 140% increase in circumference after they induced aortic dissection while maintaining physiological hydrostatic pressures in both true and false lumens.[16] Because dissection usually separates and extends within the medial layers of the aorta between the middle two thirds and the outer one third, wall stress usually increases on the aorta's outer layer, which in the presence of dissection may be only one third of its normal thickness. This reduction in wall thickness and the increase in wall stress frequently lead to expansion and rupture.

To address the question of aortic size prior to dissection, Neri and colleagues attempted to derive predissection aortic size from surgical specimens removed from 220 patients at the time of operation for acute type A dissection.[17] With the use of a unique surgical technique, they retrieved cylinders of fresh aortic tissue and measured the inner layer of the true aortic lumen with a correction for the absence of perfusion pressure. The median diameter by this unique method was 41.4 mm for the entire cohort. Furthermore, they found that only 10% of patients had frank aneurysms, defined as 50% above predicted aortic size. The range of sizes was broad, as in the present study.

Although size is a risk factor for acute dissection, what initiates the separation of aortic layers is not clear, and the pathogenesis of aortic dissection is complex. The absence of an intimal tear in ≈10% of aortic dissections has stimulated theories about the pathogenesis of dissection.[18] Examination of the structural changes show aortic medial degeneration with elastic fiber disruption, differential distribution of extracellular matrix proteins, and increased expression of metalloproteinases.[19,20] In bicuspid aortic valve patients, aortas are often structurally abnormal with disrupted extracellular matrix.[21,22] There may be similarities among other subgroups of aortic aneurysm patients. Familial aortic aneurysm and dissection patients have been shown to share mutations at other loci such as the TAA gene.[23–26] Whereas the genetic basis of Marfan syndrome lies in mutations of the fibrillin gene, the genetic underpinnings of aortic abnormalities in bicuspid aortic valve patients are as yet undefined. Truly exciting new developments in the understanding the role of transforming growth factor β signaling in the pathogenesis of aortic disease may allow modification and hopefully prevention of the structural weakness in Marfan patients.[13]

The association of hypertension and aortic dissection has long been known, and early studies showed that left ventricular dP/dT played a role in pathogenesis. In recent years investigators have studied biomechanics of the aorta to better understand the mechanisms that initiate dissection within the aortic wall.[27–30] These investigators are studying mechanical factors such as aortic root motion and the direction of twisting forces on the wall, which together may cause the aortic layers to separate, often under seemingly normal conditions.

The present finding that among the patient with aortic diameters ≥5.5 cm, 8.8% had prior coronary artery bypass surgery compared with only 2.8% of patients with less than 5 cm diameters ($P=0.002$) raises the question as to whether or not subclinical underlying aortic weakness predisposes to surgical trauma from cannulation or cross clamping leading to later dissection.

Although exciting progress is being made with regards to the pathogenesis of dissection, especially in the Marfan syndrome, effective methods of prevention remain elusive.[15] The increasing application of high-quality transthoracic echocardiography, chest CT, and magnetic resonance imaging will likely result in more incidental findings of mild aortic dilation with no established follow-up strategy. Given the relatively low incidence of aortic dissection in the general population, screening to detect thoracic aortic dilation is not practical.

The present study has several strengths as well as limitations. Among its strengths, it is unique in its presentation of diameter information from the largest reported series of acute dissections to date and adds to the current approach to dissection prevention with aortic diameter as a guide to elective surgery by confirming that type A aortic dissection may even occur at a nearly normal aortic diameter. Moreover, aortic size measurements were obtained after acute dissection and thus would tend to overestimate aortic size, which further supports the observation.

Although surveillance of patients with known aortic enlargement, aggressive treatment of hypertension, and elective surgery are established ways to avoid dissection, the need for better prognostication becomes obvious. Surgery based on aortic size alone will prevent only a minority of aortic dissections because diameter is not specific enough to identify the risk of dissection. Because even elective aortic surgery carries with it a significant mortality risk, it would be simplistic and inappropriate to merely recommend that the diameter at which surgery should be performed be lowered. To address the question of the optimal aortic size at which surgery should be performed, prospective multicenter registries of aneurysm patients, such as the recently launched National Institutes of Health Genetically Mediated Thoracic Aortic Conditions Registry (GENTAC) are needed. Such registries should separately track ascending and descending aneurysms. Research into pathogenetic mechanisms, genetic triggers, and a correlation between ultrasensitive imaging findings and risk of dissection will be the key to future prevention strategies.

Conclusions

The majority of patients with acute type A aortic dissection in our registry presented with aortic diameters <5.5 cm. Current surgical guidelines for thoracic aortic aneurysm repair (≥5.5 cm) would fail to prevent the majority of acute aortic dissections seen in this cohort. Even with more aggressive guidelines (<5 cm), preemptive aneurysm surgery would fail to prevent 40% of acute aortic dissections seen in our registry. Thus, aortic size is not a sufficient marker of risk for aortic dissection. Although the present data suggest that 60% of aortic dissection patients would not be prevented by current surgical guidelines, we do not recommend that the guideline be changed, but rather that the premise for the guideline continue to be examined. To prevent aortic dissection and its often catastrophic outcome, we need better risk predictors, probably genetic or biomarkers, or aortic functional studies to

identify patients at risk. Then we may need randomized treatment trials to move from risk prediction to prevention.

Sources of Funding

The present work was supported by the University of Michigan Faculty Group Practice, Biosite, Inc, and the Varbedian Fund for Aortic Research.

Disclosures

Dr Eagle has received research support from SanofiAventis, as well as honoraria from SanofiAventis and Pfizer. Dr Froehlich has received research support from the Mardigian Fund; honoraria from SanofiAventis, Pfizer, and Merck; and he has served as a consultant for Merck. Dr Nienaber has received research support from Medtronic and honoraria from Medtronic and Eli Lilly and Company. He has served as a consultant for Medtronic and Boston Scientific. The remaining authors report no conflicts.

Appendix

The International Registry of Acute Aortic Dissection (IRAD) Investigators

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CLINICAL PERSPECTIVE

Prompt diagnosis of acute aortic dissection continues to challenge clinicians. Clinical suspicion remains the strongest diagnostic tool. Once the diagnosis is considered, a number of accurate imaging tests are now widely available to confirm the diagnosis. Treatment approaches have evolved, with catheter-based techniques used with increasing frequency to treat type B (descending aorta) dissections. The greater challenge, however, is prevention. Prevention of dissection and rupture currently includes surgery or placement of stent grafts (depending on location) for aortic aneurysms, monitoring of patients with known connective tissue disorders and prior aortic surgery, and/or aggressive treatment of hypertension. The investigators of the International Registry of Acute Aortic Dissection asked the question, if the aortic diameter at presentation were the same as before dissection occurred, how many patients would have met current guidelines for prophylactic aortic surgical repair? The results show that nearly 60% of the 591 type A dissection patients enrolled in the International Registry of Acute Aortic Dissection had diameters <5.5 cm, and 40% had diameters <5.0 cm. It was assumed that, if they had been measured immediately before dissection occurred, aortic diameters would have been even smaller than at presentation because dissection causes an expansion of the aortic media and/or adventitia (information not available). Only a small number of patients are identified as high risk before aortic dissection. The results of the present descriptive study in a large population of patients highlight the need for predictors other than aortic size to identify patients at risk for aortic dissection.