EDITORS' INTRODUCTION

Trans-Atlantic Debate: External Diameter for Abdominal Aortic Aneurysm (AAA) Size Versus Volume

Accurate measurement of abdominal aortic aneurysms is necessary to predict rupture risk and, more recently, to follow aneurysm sac behaviour following endovascular repair. Up to this point aneurysm diameter has been the most common measurement utilised for these purposes. Although aneurysm diameter is predictive of rupture, accurate measurement is hindered by such factors as aortic tortuosity and interobserver variability, and it does not account for variations in morphology, such as saccular aneurysms. Additionally, decreases in aneurysm diameter do not completely describe the somewhat complex remodelling seen following endovascular repair of aortic aneurysms.

Measurement of aneurysm volume has the advantage of describing aneurysm morphology in a multidimensional fashion, but has not been readily available or easily measured until recently. This has changed with the introduction of commercially-available software tools that permit quicker and easier-to-perform volume measurements. Whether it is time for volume to replace, or compliment, diameter is the subject of the current debate.

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Part One: For the Motion. External Diameter for AAA Size

INTRODUCTION

Abdominal aortic aneurysm (AAA) is an abnormal dilation of the abdominal aortic wall, common in 6% of men and 1% of women >65 years old.[1–4] The most catastrophic consequence of AAA is aortic rupture, which results in high morbidity and mortality.[5] The ability to predict the likelihood and timing of rupture would be useful when planning operative intervention to prevent death from acute aortic events. Traditionally, aneurysm diameter has been used as a method of predicting of aneurysm progression, as opposed to other techniques, such as volume or wall stress, which have limited utility in clinical practice. We believe AAA diameter is more important than AAA volume in determining rupture risk and cardiovascular health.

ESTABLISHING AAA DIAMETER AS THE GOLD STANDARD

The use of initial AAA diameter measurement as a prognostic factor for cardiovascular events and AAA progression was reported in the early literature by Szilagyi et al.[6] in 1972 in a study where they examined the outcome of patients who were turned down for elective open repair over a 19-year period. The main cause of death for 90 patients turned down for elective surgery was coronary artery disease (37/90, 41%); the second cause of death was ruptured AAA (25/90, 28%). In these patients, 40% had an AAA that ruptured within 1 year, and the likelihood of rupture as the cause of death was greater in large aneurysms (>6 cm) compared with small (<6 cm) ones (42.5% vs. 31.1%). In addition, they found that large aneurysms were more likely to have a fatal rupture earlier than those with small aneurysms (71.8% vs. 39.1%).[6]

Since this early work, contemporary authors have also found the importance of aneurysm size in predicting outcome,[7–11] and diameter measurements of the infrarenal aorta have become important in the management of the disease. Two randomized controlled trials evaluating the role of surveillance versus early intervention in the operative management of small aneurysms chose aortic diameter, measured on screening ultrasound, as the method for determining the threshold size for intervention.[12,13] Also, four randomised controlled trials evaluating population-based screening for the prevention of aortic-related death used diameter measurements from ultrasound studies to determine the need for further evaluation and treatment of patients with aneurysms.[1–4] As a result of this work, the American College of Cardiology/American Heart Association 2005 Practice Guidelines for the management of patients with peripheral arterial disease published in 2006[14] recommend surgical repair as class I for patients with AAAs measuring ≥5.5 cm in diameter.

As technology has progressed, it has become apparent that the imaging modality used to measure diameter is also an important variable to consider. All screening studies have relied on ultrasound as a measure of diameter. Duplex ultrasound is an excellent modality for surveillance of an AAA in an asymptomatic patient.[15,16] Blois[17] conducted a prospective observational study to assess the efficacy of an office-based, family physician-administered ultrasound

examination of AAA screening, with comparison of the difference in AAA diameter measured between resident physicians trained in an emergency unit and ultrasonography technicians. The office-based ultrasound scan had both a sensitivity and a specificity of 100%, with a difference of only 2 mm in diameter between two groups. It was concluded that AAA screening with measurement of AAA diameter can be performed safely in the office by family physicians trained in the use of ultrasound technology, with no significant difference in techniques or time taken compared with vascular surgeons.

Despite its accessibility and the high specificity and sensitivity of ultrasound for the detection of AAAs in asymptomatic patients,[18,19] measurements provided can be user-dependent. For example, Beales et al.[20] reviewed the variation in intraobserver repeatability and interobserver reproducibility among studies of ultrasound screening of aneurysms in the published literature. Intraobserver repeatability coefficients for anteroposterior aortic diameter measurements in aortas were 1.6—4.4 mm among the five of the nine studies with usable outcome data included in the analysis and the reported interobserver reproducibility was less than 5 mm. An additional four studies reported poor interobserver reproducibility (range: −2 to 5.2 to −10.5 to 10.4 mm). Thus, ultrasound has been supplanted by other imaging modalities for clinical indications that require more than simple screening or diagnostic purposes.

Computed tomography (CT) angiography is a powerful tool for estimating the diameter of an AAA with great accuracy. Kauffmann et al.[21] estimated intra- and interobserver reproducibility of the maximal AAA diameter among four investigators using intraclass correlation coefficient (ICC) calculation. The intraobserver ICC of AAA maximal diameter was estimated at 0.997. Overall intraobserver ICC of the maximal AAA diameter was estimated at 0.995. The CT scan has been established in clinical practice as the gold standard for imaging and measurement of the aorta. In current practice, use of post-processing software allows for an accurate and reproducible method of measuring aneurysm diameter,[22] making the current initial diameter measurements the most accurate in the history of aortic imaging.

PHYSIOLOGIC BASIS FOR USING EXTERNAL DIAMETER FOR AAA SIZE

Once an aneurysm has developed, the tension on its wall increases in accordance with a fundamental hydraulic principle that was demonstrated 200 years ago by French astronomer Laplace. This principle considers wall tension to be directly proportional to intraluminal pressure and to radius: this law applies most accurately to a sphere with an infinitely thin wall. Sumner et al.[23] expanded on the principle that wall thickness is inversely proportional to tension. By measuring the protein composition focused on the collagen and elastin aorta specimens in 13 AAA patients they found that, as the aneurysm expands, wall thickness decreases resulting in increased tension and ultimate rupture. Later, Varduaki et al.[24] conducted a prospective study of the relationship between AAA growth rates and the risk of rupture of AAA using measurements taken from longitudinal aneurysmal growth data from screening studies in Chichester and Huntington, UK. They found a strong correlation between AAA initial diameter and AAA growth, with more rapid growth in large aneurysms (≥50 mm in diameter). Aortic diameter was assumed to change exponentially over time, and could be described using the following equation:

$$\text{Expected diameter} = \alpha \times e^{\beta t'}$$

where α is the estimated initial diameter, β is the estimated coefficient, and t is follow-up time (years). The exponential function ensures that absolute growth increases with diameter.

Conway et al.[25] analysed the outcome of all patients ($n = 106$) referred with AAAs >5.5 cm in diameter who were turned down for elective open repair, and determined the cause of death and risk of rupture in all patients over a 10-year period. In this study, 76 patients (71.7%) died after 10 years. Patients with AAAs >7.0 cm lived a median of 9 months. A ruptured aneurysm was certified as cause of death in 36% of patients with AAAs of 5.5—5.9 cm in diameter, in 50% of patients with an AAA of 6.0—7.0 cm in diameter, and in 55% of patients with an AAA diameter of ≥7.0 cm. Although retrospective in nature, this study appears to support the hypothesis that increasing aortic diameter is predictive of aortic-related death.

Aortic-related death is not the only outcome correlated with aneurysm diameter. Duncan et al.[26] conducted the prospective cohort study of long-term outcomes in men ($n = 8146$, aged 65—74 years) for AAA in relation to aortic diameter, morbidity, and mortality. All-cause mortality was significantly associated with aortic diameter: 512 (7.2%) men in the ≤24 mm group died compared with 69 (0.3%) in the 25—29 mm group and 73 (17.6%) in the ≥30 mm group. The mortality risk in men with an aneurysm or with an aorta measuring 25—29 mm was significantly higher than in men with an aorta of ≤24 mm. Men with AAAs and those with aortas measuring 25—29 mm also had an increased risk of subsequent hospital admissions compared with those men with an aorta diameter of ≤24 mm (adjusted hazard ratio 6.7, 99% confidence interval 3.4—13.2).

PARAMETERS BEYOND DIAMETER

Despite the widespread use of diameter measurements in clinical trials and its ease of ascertainment in clinical practice, several studies have also concluded that the diameter may not be reliable as rupture risk criterion and that it should be replaced by a more specific criterion.[27—29] Therefore, multiple studies have focused on patient-specific issues, such as peak wall stress[30—32] or intraluminal thrombus in AAA.[33—36] In the current era, these determinants of AAA progression and cardiovascular events

are still poorly understood and have thus proven to have limited clinical applicability over diameter measurement at the present time.

Certainly, patients with aneurysms measuring ≥10 cm that have not yet ruptured occasionally rise to the clinical horizon, which challenges a simple association between aneurysm diameter and the probability of rupture and death. Therefore, other parameters which might play a role in causing an aneurysm have been studied. One of the parameters is peak wall stress (PWS). Fillinger et al.[30] studied the differences in PWS between patients with elective and symptomatic or ruptured AAAs (mean diameter: 6.6 cm). They were able to measure PWS using three-dimensional computer models of AAA from CT scan data combined with an AAA geometry and blood pressure data analysis, which depicted the mechanical behavior of the AAA wall. This group found that PWS was significantly higher for patients who had ruptured, which meant that PWS was strongly correlated with AAA rupture. Speelman et al.[31] investigated the relationship between AAA diameter and PWS with the 99-percentile stress (defined as peak stress value in the AAA after exclusion of 1% total surface area with the highest stress). In the linear regression analysis between AAA diameter and 99-percentile stress, there was a moderate regression coefficient (99-percentile stress $= -75 + 7$*diameter; $R^2 = 0.75$, $p < .001$), which suggests a positive correlation between this measurement and AAA diameter. Vorp et al.[32] conducted the study of the mechanical wall stress in AAA to investigate the effect of AAA maximum diameter and asymmetric bulge on wall stress using three-dimensional computer models of AAA and concluded that the stress within the wall of the AAA and possibly the potential for rupture are as dependent on aneurysm shape and as they are on maximum diameter. Thus, there is a strong association between PWS and AAA maximum diameter. However, the methods for measurement of PWS are difficult to interpret in the clinical environment because they are not readily available for patient evaluation on a day-to-day basis.

Other parameters related to AAA growth have been described. The serum marker osteoprotegrin (OPG) is a biological marker associated with atherosclerosis and AAA. OPG is a secreted glycoprotein member of the tumor necrosis factor receptor superfamily and a key regulator of bone modelling,[37] and is also associated with cardiovascular diseases, such as carotid atherosclerosis and myocardial infarction.[38,39] Moran et al.[40] assessed the association between circulating concentration of OPG and the presence of AAA and growth with peroxisome proliferator-activated receptor (PPAR) gamma, and concluded that circulating concentrations of OPG are associated with AAA and with one *PPAR gamma* gene polymorphism, which were associated with AAA presence and growth. Koole et al.[41] studied the association between OPG and AAA diameter through analysis of OPG concentration in AAA intraoperative biopsy specimens. The concentration of OPG correlated positively with aortic diameter (<55 mm; 16.1, 55–70 mm; 21.9, >70 mm: 24.0 ng OPG/mg total amount of protein, $p = .020$). After adjustment for cardiovascular risk factors, OPG concentrations were independently associated with AAA diameter ($p = .001$; coefficient 0.189; standard error, 0.001). In this study of OPG association with AAA growth, OPG had no correlation with the ventral thickness of the thrombus ($r = 0.017$, $p = .082$) and intraluminal thrombus (ILT) volume ($r = 0.163$, $p = .148$). However, OPG had a borderline significant correlation with AAA diameter ($r = 0.218$, $p = .052$). At the current time, OPG is not being used in routine clinical practice.

VOLUME FOR AAA SIZE

The use of aneurysm volume is another variable that has been considered as a predictor of rupture risk. AAA volume is usually composed of two components; one is a space of blood flow and another is ILT, which contains leucocytes, pro-inflammatory cytokines, and proteolytic enzymes, and is implicated in AAA development, progression, and ruptures by other studies.[33–36] ILT products are also released into the circulation, where they have potential to stimulate leucocytes and other changes that might promote atherosclerotic plaque activation and cardiovascular events.[42,43] Parr et al.[44] investigated the association of AAA volume and thrombus volume, as well as AAA initial diameter. In this study, annual AAA volume was positively correlated with thrombus volume ($r = 0.50$, $p = .001$). Annual AAA volume was also positively associated with AAA initial diameter ($r = 0.44$, $p = .006$). These trends were found in the correlation study with cardiovascular events, which means that not only thrombus volume but also AAA initial diameter strongly correlated with AAA aneurysm growth and cardiovascular events. Georgakarakos et al.[45] analysed the correlation between PWS and AAA geometric parameters in the presence of ILT. In this study, a positive correlation was observed between PWS [ΔPWS%] (the percentage change in PWS in the presence of ILT) and relative ILT volume ($p = .03$). PWS in the presence of ILT significantly correlated with the degree of centerline tortuosity ($r = 0.72$, $p = .003$) and AAA maximum diameter ($r = 0.88$, $p < .001$) (Fig. 1).

It was also found that the presence of ILT correlated with a significant reduction of PWS, which seemed to be a contrary result related to PWS, that is the more ILT was, the less the PWS was. Speelman et al.[46] investigated the effect of ILT on the PWS and growth rate of aneurysms using patient-specific models in wall-stress computations and found that a larger thrombus in AAA was associated with a higher AAA growth rate; however, the thrombus caused a significant reduction in wall stress, which was stronger for larger thrombi and higher elastic moduli. This result is consistent with the theory by Georgakarakos et al.[45] that greater ILT may mean lower peak wall stress. Regardless of these findings, the analysis of AAA volume related to PWS or ILT definitely necessitates sophisticated analysis software with three-dimensional CT, which is not yet clinically practical.

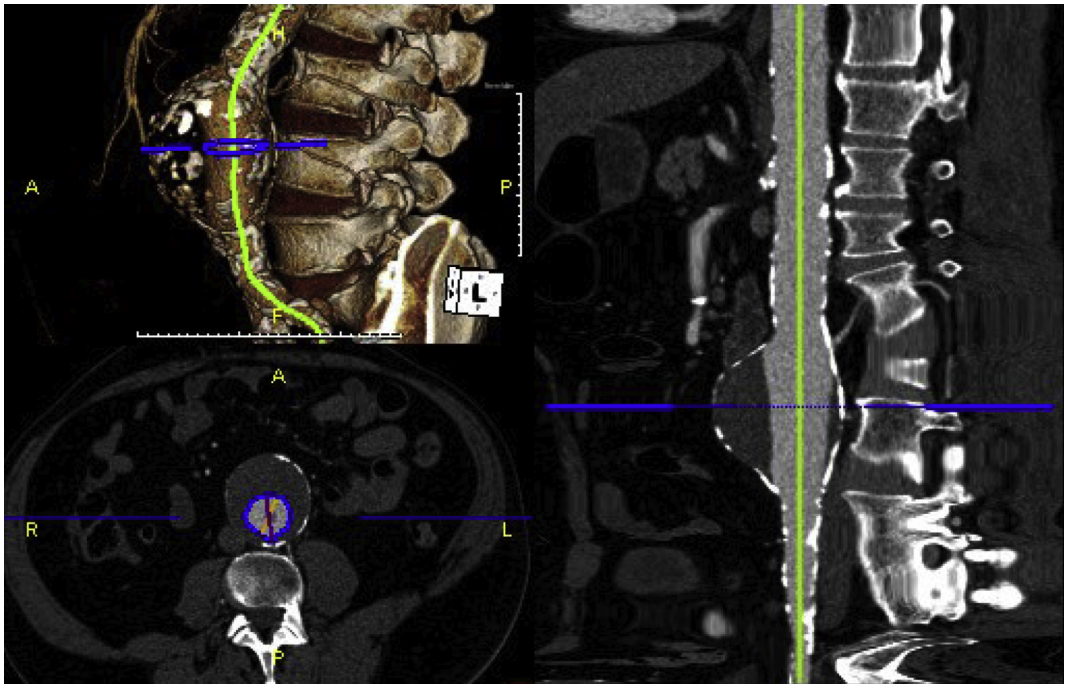

Figure 1. Diagram illustrating the use of centre-lumen of flow projections for accurate measurement of aortic aneurysms. All images originated from the Cleveland Clinic Foundation.

CONCLUSION

Despite its rudimentary nature, the diameter measurement of an aneurysm is superior to volume measurement for prognosis and operative planning.

CONFLICTS OF INTEREST

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