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ORIGINAL ARTICLE

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Ascending aortic elongation and the risk of dissection†

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Abstract

OBJECTIVES: Unlike aneurysm formation, the role of ascending aortic elongation in the pathogenesis of Type A aortic dissection (TAD) is largely unclear. We investigated the morphology of healthy, dissected and predissection aortas with a focus on ascending aortic length.

METHODS: We retrospectively compared clinical and computer tomography angiography (CTA) data from TAD patients ($n = 130$), patients who developed a TAD in the further clinical course (preTAD, $n = 16$) and healthy control patients who received a CTA for non-aortic emergencies ($n = 165$). The length of the ascending aorta was defined as the distance between the sinotubular junction (STJ) and the brachiocephalic trunk (BCT) at the central line, the outer and inner curvature as well as the direct distance in the frontal and sagittal planes. Additionally, the aortic diameters were analysed.

RESULTS: In the healthy controls, we found a positive correlation of age with the aortic diameter ($r = 0.57$) and aortic length ($r = 0.42$). The correlation of the respective parameters with the body size was negligible ($r < 0.2$). The median ascending aortic diameter at the height of the pulmonary artery in TAD (50 mm) was significantly ($P < 0.001$) larger compared with the respective diameter of the healthy aortas (34 mm). The diameter of the preTAD aortas (40 mm) was also significantly larger compared with the healthy controls. These proportions were similar in all the aortic diameters. The midline length of the healthy ascending aortas was 71 mm. In the preTAD and TAD aortas, the same values were 81 mm and 92 mm, respectively (both $P < 0.001$). We evaluated the linear distance between the STJ and the BCT in the frontal plane as an easy-to-measure parameter of aortic length. In the TAD aortas (108 mm) and preTAD aortas (97 mm), this distance was significantly longer compared with the healthy aortas (84 mm).

CONCLUSIONS: Aortic diameter might not be an optimal parameter to predict dissection. Most aortas dissect at diameters below 55 mm. Both the TAD and preTAD aortas were elongated compared with the healthy controls. Thus, aortic elongation may play a role in the pathogenesis of and may be a risk factor for TAD.

Keywords: Aorta • Aneurysm • Aortic elongation • Aortic dissection • Computed tomography

INTRODUCTION

The ascending aortic transverse diameter is the only accepted morphological risk factor for Stanford type A aortic dissection (TAD) and triggers indications for preventive surgery [1]. However, most TADs develop in aortas with normal diameters and the process of dissection results in significant morphological changes, i.e. aneurysm formation [2]. Wall stress is regarded as the major pathogenic factor in aneurysm formation and dissection [3, 4]. However, if disproportionate circumferential wall stress and wall stability is the predominant pathophysiological mechanism, a longitudinal (vertical) rupture in the aortic intima should be expected, which is mostly not the case [3, 5, 6]. Unlike the transverse (circumferential) dilatation of the aorta, which is synonymous to aneurysm formation, longitudinal dilatation, termed elongation and the longitudinal impact of wall stress [7] have been largely neglected in pathogenic models of TAD. Ascending aortic elongation is frequently observed, but poorly studied. No normal values have been established. In theory, aortic elongation potentially leads to the loss of longitudinal elasticity and a risk of intimal rupture, which may explain the usually horizontal direction [3, 5, 6] of a TAD entry.

On the basis of these theoretical considerations and experimental data [7], we hypothesize that aortic elongation represents a pathological entity and risk factor for aortic dissection. Measurements of aortic length may be useful in identifying patients at risk for TAD. Thus, the aim of the present study was to compare and characterize the computed tomography angiography (CTA)-based morphology of healthy aortas, dissected aortas and aortas that dissected

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in the clinical course following a CTA for non-aortic indications (predissection).

MATERIALS AND METHODS

Patient groups and clinical data

We retrospectively compared three groups of patients. We analysed all of the patients treated for TAD in our clinic between January 2006 and January 2015 (9 years; $n = 146$). We identified patients who had received an adequate CTA of the aorta before the actual dissection occurred; those patients ($n = 16$) formed the preTAD group. The median time interval between the predissection CTA in the preTAD group and the actual event of dissection was 4.96 months; the shortest time interval was 4 days, the longest 2.46 years.

The remaining 130 patients formed the TAD group. We also screened all patients diagnosed in our emergency department with an adequate CTA between March 2014 and March 2015 because of non-aortic emergencies ($n = 177$). Expectedly, these predominantly included younger adults. Because the youngest patient in the TAD group was 23 years old, we excluded all patients in the all-comers emergency department group who were younger than 23 years ($n = 12$). This resulted in a healthy control group of 165 patients with non-aortic emergencies and with similar demographics compared with the TAD group.

We recorded the following demographical and clinical parameters: dates of birth and index CTA, body height and weight and sex. We screened the files for a diagnosis of hypertension and the presence of three or more of the following drugs in the chronic medications: β-blockers, angiotensin converting enzyme inhibitors, angiotensin II receptor antagonists, central and peripheral vasodilators and calcium channel blockers, but not diuretics. The presence of three or more of the above-mentioned drugs indicated patients with long-standing, massive hypertension.

This study was reviewed and approved by our local ethics committee (No. 076/2015R). Written informed consents of patients were not necessary because of the purely retrospective observational character of the study.

Computed tomography

CTA studies were performed with standard equipment, including a second-generation Dual-Source CT scanner (Somatom Definition Flash, Siemens Healthcare, Erlangen, Germany). Established contraindications to CT imaging were also applied, including impaired renal function (serum creatinine >1.3 mg/dl), allergy to iodinated contrast or hyperthyroidism. A non-ionic, high-iodinated contrast bolus was generally tailored to body habitus (~100 ml, >350 mg/ml iodine) and injected at high flow rates (>4–5 ml/s). In the majority of the cases, an automated bolus triggering with a region of interest located in the ascending aorta was used. In this retrospective analysis, we included subjects in whom CTA images in transverse, frontal and sagittal reconstructions with a maximum thickness of 3 mm were available.

Image analysis

For image analysis, a commercially available PACS viewer software was used (Centricity, GE Healthcare, Chalfont St Giles, UK). All measurements were performed manually by experienced physicians (Tobias Krüger and Oksana Forkavets) applying the graphical measuring tools of the software. The individual studies were randomly assigned to one of the investigators. Randomly, 35 patients were reviewed by both investigators to ensure a low interobserver variability of the acquired data.

For reporting aortic diameters at certain landmarks (Fig. 1), we followed international guidelines [1, 8]. In the transverse planes, the diameters of the ascending and descending aorta were measured at the height of the pulmonary artery (PA) bifurcation. In the frontal plane, the diameters of the aorta at the sinotubular junction (STJ) and at the origin of the brachiocephalic trunk (BCT) were marked on the screen and measured. With the use of these landmarks, the length of the ascending aorta was measured using the graphical measuring tools. The same approach was used to analyse the length and diameters of the aortas in the sagittal plane. The aortic length was measured at the outer curvature, the central line and inner curvature of the aorta in the frontal and sagittal reconstructions. Additionally, we recorded the direct distance between the outer aspect of the STJ and the origin of the BCT in a straight line in the frontal and sagittal planes. These virtual parameters depend on the aortic length and are easy to measure.

We also recorded the morphology of the aortic arch in the sagittal plane: in the Type I arch configuration, the highest and reversal point of the arch is between the supra-aortic vessels; in the Type II arch configuration, this point is distal to the left subclavian artery [9, 10].

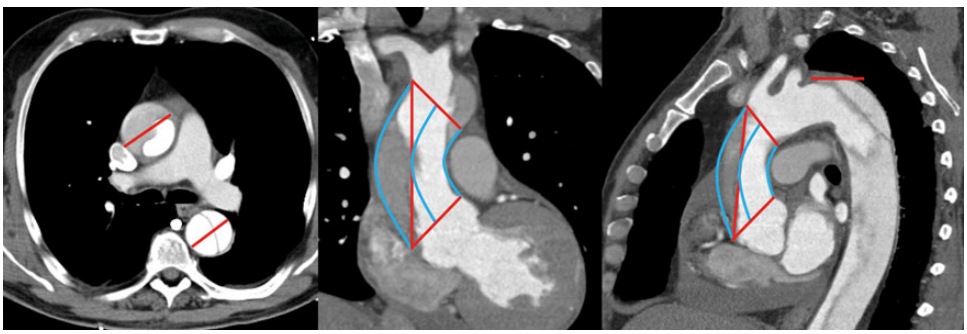

Figure 1: Measurements of aortic diameters and length: left: transverse plane: diameter of the ascending and the descending aorta at the height of the PA . Middle and right: frontal plane and sagittal plane: ascending aortic diameter (red) at the height of the STJ and the BCT. Distance between the STJ and the BCT at the central line, outer and inner curvature (blue). The direct distance between the STJ and the BCT as a virtual but easy-to-measure parameter of the ascending aortic length (red). Right: the height of the reversal point of the aortic arch. PA: pulmonary artery; STJ: sinotubular junction; BCT: brachiocephalic trunk.

Statistical analysis

Continuous data are described as the median with the first (Q1) and third (Q3) quartile and range (minimum and maximum) and presented with box-and-whiskers plots. Categorical data are described as percentages. Pearson's product–moment coefficient was used to determine the correlation between continuous variables. For inferential statistical comparisons of continuous variables, a Mann–Whitney rank sum test was used. Categorical variables were compared using a χ^2 test. All reported *P*-values were two-sided, and *P*-values of ≤ 0.05 were considered to indicate statistical significance. SSPS (SSPS 20.0, IBM Corp., Amonk, NY, USA) and Excel (MS Excel 2010, Microsoft, Redmond, WA, USA) were used for all analyses and data presentation.

RESULTS

Aortic ageing

To investigate the physiological age- and body size-dependent changes in the ascending aortic morphology, we analysed the aortic dimensions in the patients who received a CTA in our emergency department because of non-aortic emergencies. Figure 2 displays the ascending aortic diameters at the height of the PA bifurcation (blue) as a function of the patients' age, revealing a gradual and steady increase in diameter. The aortic diameter at the PA bifurcation was strongly correlated with age ($r = 0.57$), whereas it was not associated with body height, body weight or body-mass-index (BMI) ($r = 0.08$; $r = 0.22$; and $r = 0.19$, respectively). This increase with age is also observed at the other diameters of the aorta, namely at the STJ ($r = 0.38$), BCT ($r = 0.56$) and in the descending aorta ($r = 0.52$). No relevant correlations were found with body dimensions for either of these diameters (data not shown).

The measurements of aortic length (Fig. 2), central line length in the frontal plane (red) and in the sagittal plane (green), have similar steady increases with age. In the frontal and sagittal projections, the central line length was strongly correlated with age (both $r = 0.42$). All the correlation coefficients for both parameters with body height, body weight and BMI were far below $r < 0.2$.

Similarly, the easy-to-measure linear STJ–BCT distances in the frontal plane ($r = 0.47$) and the sagittal plane ($r = 0.43$) were correlated with age, whereas correlations with body height, body weight and BMI were weak ($r = 0.14$; $r = 0.18$ and $r = 0.10$, respectively).

Aortic dimensions and hypertension

In our emergency department cohort, we analysed the aortic dimensions based on the presence of arterial hypertension. The aortic diameter and length were significantly larger in the hypertensive patients compared with the non-hypertensive patients. The results are presented in Table 1. However, the differences in the measurements of the diameter were more profound compared with the differences in the measurements of the length.

No significant differences in the aortic dimensions were found between the patients suffering from massive hypertension defined

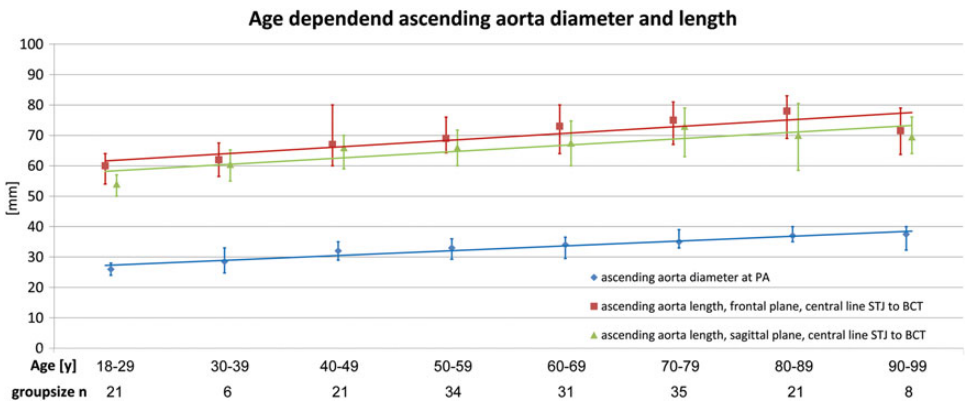

Figure 2: Age-dependent ascending aortic diameter and length. The dots represent the median, and the whiskers represent the first and third quartiles (Q1 and Q3).

Table 1: Aortic dimensions depend on the presence of arterial hypertension

	Normotensive	Hypertensive	*P*
n	108	69	
Ascending aortic dimensions	Median (Q1–Q3; range) (mm)	Median (Q1–Q3; range) (mm)	
Diameter at PA	32 (27–35; 21–45)	36 (33–40; 28–53)	<0.001
Length at central line, frontal plane	69 (60–78; 50–114)	73 (65–80; 48–98)	0.03
Length at central line, sagittal plane	65 (56–73; 39–98)	70 (60–76; 50–87)	0.02
Length as the direct distance from STJ to BCT, frontal plane	82 (74–88; 58–124)	87 (80–93; 64–109)	0.004
Length as the direct distance from STJ to BCT, sagittal plane	76 (67–83; 51–104)	80 (73–87; 56–104)	0.03
Descending aortic diameter at PA	23 (21–26; 14–32)	27 (21–26; 18–38)	<0.001

All of the measurements are in mm and presented as the median, first and third quartile and range.
PA: pulmonary artery; STJ: sinotubular junction; BCT: brachiocephalic trunk.

Table 2: Demographic variables in the TAD group and healthy controls

	TAD	Healthy controls	*P*
n	130	165	
Male	67.7%	65.5%	0.69
	Median (Q1–Q3; range)	Median (Q1–Q3; range)	
Age (years)	65 (56–74; 23–86)	63 (51–77; 23–96)	0.51
Body height (cm)	175 (168–180; 145–196)	171 (167–180; 152–196)	0.13
Body weight (kg)	80 (70–90; 50–132)	80 (70–90; 50–130)	0.52

All of the measurements are in mm and presented as the median, first and third quartile and range.
TAD: type A aortic dissection; PA: pulmonary artery; STJ: sinotubular junction; BCT: brachiocephalic trunk.

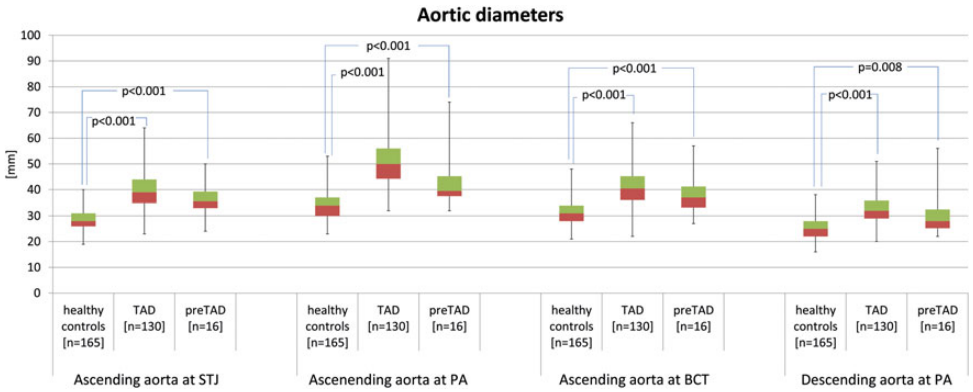

Figure 3: Diameters of the healthy, dissected and predissection aortas. The box-and-whiskers plots present the median, first and third quartile and range. TAD: type A aortic dissection; PA: pulmonary artery; STJ: sinotubular junction; BCT: brachiocephalic trunk.

by three or more antihypertensive drugs in their medication and the hypertensive patients with fewer drugs (data not shown).

However, it has to be taken into account that the patients with documented hypertension were significantly ($P < 0.001$) older (mean 73 years; Q1–Q3 65–80 years, range 48–91 years) than those without hypertension (mean 51 years; Q1–Q3 40–65 years; range 18–96 years). Therefore, it cannot be decided if the morphological changes are a result of hypertension or a patient's age.

Aortic dimensions in the undissected and dissected aortas

Table 2 shows the demographic data of the Type A dissection patients and patients with healthy aortas (controls). No relevant differences were found with respect to gender, age and body dimensions.

The small cohort of the preTAD patients ($n = 16$) consisted of 75% of men. The median age was 66 years (Q1–Q3 53–72 years; range 41–85 years), median height 176 cm (Q1–Q3 170–178 cm; range 162–188 cm) and median weight 76 kg (Q1–Q3 72–85 kg; range 60–115 kg). With respect to these parameters, and considering the critically small group size, there were no significant differences between the preTAD and the TAD or control group.

The aortic diameters differed significantly between the TAD patients and control group without aortic pathologies. Figure 3 depicts the aortic diameters in the different aortic regions. At all four measuring points of the thoracic aorta, the diameters of the dissected aortas significantly exceeded those of the control group. However, the numerically largest differences were found at the height of the PA. Here, the median diameter of the dissected aortas was 50 mm (Q1–Q3 44–56 mm; range 32–91 mm). In the healthy aortas, the median diameter was 34 mm (Q1–Q3 30–37 mm; range 23–53 mm).

The median ascending aortic diameter at the PA bifurcation in the preTAD group was 40 mm (Q1–Q3 38–45 mm; range 32–74 mm), also significantly larger than the diameter of the healthy control aortas. With 36 mm at both the STJ (Q1–Q3 33–39 mm; range 24–50 mm) and BCT (Q1–Q3 33–41 mm; range 27–57 mm), these diameters of the preTAD aortas were also significantly ($P < 0.001$) larger than the respective values in the control group but insignificantly smaller than the respective values in the dissected aortas.

Similar to the aortic diameters, the parameters of the aortic length showed significant differences between the healthy and dissected aortas in the frontal and the sagittal planes.

In the frontal plane (Fig. 4), the central line length of the healthy control group aortas was 71 mm (Q1–Q3 64–80 mm; range 47–114 mm) whereas, in the dissected aortas, the corresponding length was 92 mm (Q1–Q3 84–102 mm; range 57–141 mm), which is significantly ($P < 0.001$) longer. Similarly, the lengths of the outer and inner curvatures were significantly different between the healthy and dissected aortas (Fig. 4).

In the sagittal plane, the differences in ascending aortic length were also evident (Fig. 5). The median central line length of the healthy ascending aorta in the sagittal plane was 66 mm (Q1–Q3 59–75 mm; range 39–98 mm) versus 87 mm (Q1–Q3 76–96; range 51–135 mm) in the dissected aortas ($P < 0.001$). The measures of the inner and outer curvatures also differed significantly (Fig. 5).

Remarkably, the preTAD aortas were also significantly longer compared with their healthy control counterparts. The median

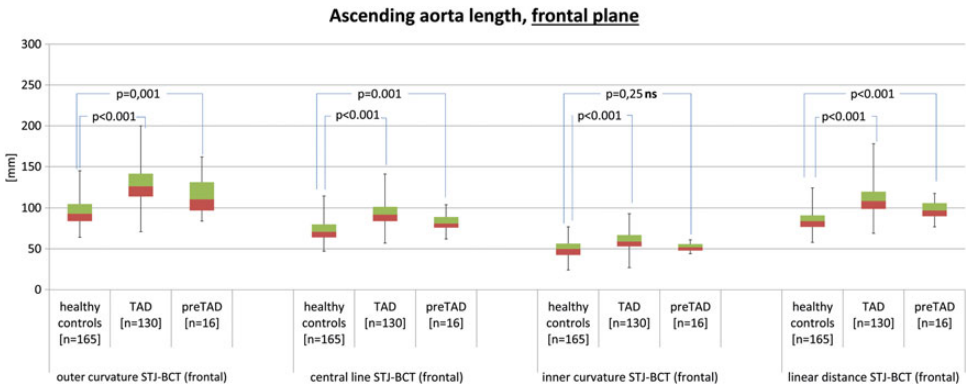

Figure 4: Length of the ascending aorta. The healthy, dissected and predissection aortas. Length of the outer curvature, central line, inner curvature and the direct distance between the STJ and the BCT in the frontal plane view. TAD: type A aortic dissection; PA: pulmonary artery; STJ: sinotubular junction; BCT: brachiocephalic trunk.

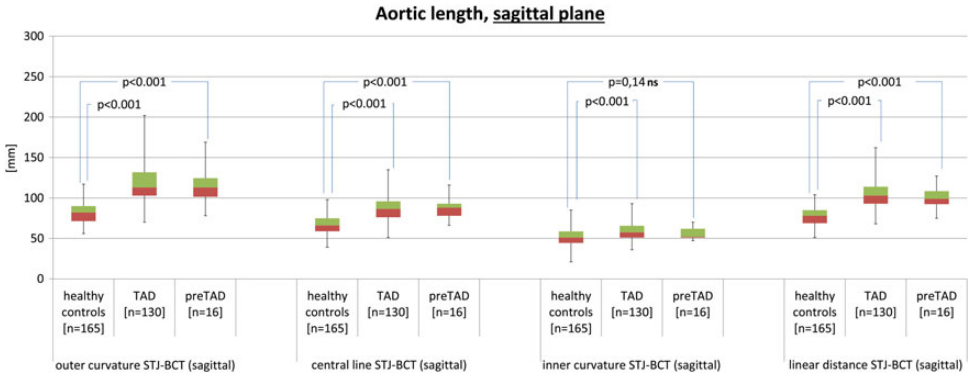

Figure 5: Length of the ascending aorta. The healthy, dissected and predissection aortas. The length of the outer curvature, central line, inner curvature and direct distance between the STJ and the BCT in the sagittal plane view. TAD: type A aortic dissection; STJ: sinotubular junction; BCT: brachiocephalic trunk.

central line length of the preTAD aortas was 81 mm (Q1–Q3 76–89 mm; range 62–104 mm) in the frontal ($P = 0.001$; Fig. 4) and 88 mm (Q1–Q3 78–93 mm, range 66–116 mm) in the sagittal plane ($P < 0.001$; Fig. 5).

However, the aortic length in the frontal and sagittal planes is somewhat difficult to measure. Therefore, we evaluated the linear distances between the STJ and the BCT in the frontal and sagittal projections (Fig. 1) as parameters for aortic length.

In the frontal plane, the median linear distance between the STJ and the BCT in the healthy controls was 84 mm (Q1–Q3 77–91 mm, range 85–124 mm). However, in the TAD and preTAD aortas, it was significantly ($P < 0.001$) longer with 108 mm (Q1–Q3 99–120 mm; range 69–178 mm) and 97 mm (Q1–Q3 90–106 mm, range 77–117 mm), respectively (Fig. 4).

In the sagittal plane, the median linear distance between the STJ and the BCT in the healthy controls was 78 mm (Q1–Q3 69–85 mm, range 51–104 mm). However, in the TAD and preTAD aortas, this parameter, as well, was significantly ($P < 0.001$) longer with 103 mm (Q1–Q3 93–114 mm; range 68–162 mm) and 99 mm (Q1–Q3 93–109 mm, range 75–127 mm), respectively (Fig. 5).

Aortic arch morphology in the undissected and dissected aortas

In the healthy control group, the highest point of the aortic arch, the reversal point (sagittal plane) was between the supra-aortic branches (Type I arch) in 77% of patients. In 23%, it was downstream from the left subclavian artery (Type II arch).

The patients with Type II arch morphology in the median were significantly ($P < 0.001$) older (77 years; Q1–Q3 68–83 years; range 51–94 years) compared with the patients with Type I arch morphology (57 years; Q1-Q3 48–72; range 23–96). Moreover, the Type II arch patients more often ($P < 0.001$) suffered from hypertension (68.4%) compared with the Type I arch population (31.5%).

Within the preTAD group, 10 of 16 patients (62.5%) had Type II aortic arch morphology. However, within the TAD group, 36.9% (48 of 130 patients) had a Type II arch, which is significantly (<0.001) more than the healthy control group.

DISCUSSION

Methodical considerations and limitations

We analysed the ascending aorta diameters and length in routine aortic CTA exams (Fig. 1). With this approach, we did not measure the anatomical 3D length of the ascending aorta. Instead, we measured the 2D projection in the frontal or sagittal plane. The disadvantage of this approach is that the lengths in the sagittal and frontal planes are not directly comparable. However, the advantage is the broad availability in routine imaging workflows. Future studies will entail more dedicated and advanced post-processing algorithms, including 3D modelling.

We compared the aortic dimensions of the three different groups of patients, namely the controls with the healthy aortas, TAD patients and preTAD patients. With respect to the demographic and body-size parameters, no relevant differences were present in the respective groups, indicating comparability. However, the inevitably

small group size of the preTAD group compared with the others has to be taken into account in the interpretation.

Additionally, the retrospective nature of data acquisition and the fact that we do not have long-term follow-up of the control group and consequently do not know how many people out of this cohort will develop TAD in the future make multivariate modelling impossible. Similarly, it is not possible to quantify the risk that is associated with aortic elongation or to differentiate the risk of transverse dilatation and elongation if both are present. However, this was not intended in the study design; the study was meant to provide a first insight into the topic of aortic elongation and its connection to TAD.

Aortic ageing

It is known that aortic diameters increase during a lifetime. In the literature, a growth rate of 0.7–0.9 mm per decade is described [1, 11–13]. In our data, we observed a similar increase of at least 1 mm per decade. However, the ascending aortic diameter (height of PA) increases by 35% between the third and the eighth decade of life. The gain in aortic length in the frontal and sagittal plane central lines was only 25 and 30%, respectively, during the same period. Thus, in physiological ageing, transverse dilatation seems to be the predominant direction of aortic growth. Consequently, it may be useful to develop age-adjusted nomograms for aortic dimensions. The morphology of the aortic arch, Type I or Type II, was also associated with patient age [10] and the prevalence of hypertension.

However, in the healthy aortas of any age, the diameter of the ascending aorta did not exceed 45 mm. The length between the STJ and the BCT did not exceed 100 mm in the central or direct line measurement irrespective of whether it was measured in the frontal or sagittal plane. In our opinion, larger values can be classified as pathological.

Correlations between the aortic dimensions and body size and weight were remarkably low.

Hypertension is one of the treatable risk factors for aortic dilatation and dissection. The patients with documented hypertension had larger aortic diameters and larger values of aortic length. However, the hypertensive patients in our study on average were older than the normotensive patients. Therefore, we cannot conclude if aortic dilatation was the result of hypertension or age.

Pathological aortic diameters

In the absence of connective tissue disorders, guidelines have triggers for prophylactic ascending aortic replacement at 55 mm [1]. In our preTAD group, the median ascending diameter was 39 mm (Q1–Q3 37–44 mm; range 32–74 mm). In fact, in just one of the 16 preTAD patients, the aorta exceeded this trigger diameter. Another aorta that was 53 mm was close to this value. The other 14 aortas dissected at diameters <50 mm. Moreover, 75% of the dissected aortas had ascending diameters <55 mm. This indicates that the 55 mm cut-off is an insufficient parameter to identify patients at high risk for dissection. Moreover, it is evident that during the process of acute dissection, a sudden dilatation of mainly the ascending aorta occurs [2]. Consequently, the sole diameter is a suboptimal discriminator between aortas at risk for dissection and those that are not. This reveals the need for other parameters and risk prediction models.

Pathological aortic elongation

We showed that the dissected aortas and aortas prior to dissection are significantly elongated compared with their healthy counterparts. In fact, 75% of the healthy aortas had a central line distance from the STJ to the BCT in the frontal plane of <80 mm, whereas in 50% of the preTAD and >75% of the TAD patients, the respective value was >80 mm. Similarly, in the sagittal plane, 75% of the healthy aortas had a central line length <75 mm, whereas in 75% of the preTAD and TAD aortas, the central line length exceeded 75 mm.

Furthermore, we evaluated the direct distance between the lateral aspect of the STJ and the BCT in the frontal and sagittal planes as easy-to-measure surrogate parameters of aortic length. In the frontal plane, this parameter in 75% of the healthy aortas was <90 mm. In 75% of the predissection aortas, it was >90 mm. In the dissected aortas, it was even >99 mm in 75% of the cases. In the sagittal plane, the direct distance between the STJ and the BCT was <85 mm in 75% of the healthy aortas and >91 mm in 75% of the preTAD and TAD aortas.

Consequently, these parameters of aortic length seem to be connected to morphological changes prior to or during aortic dissection.

Aortic elongation is a potential risk factor for aortic dissection. In mechanics and engineering, cylinder stress models (Laplace's law; Barlow's equation) include circumferential stress and axial stress of tubes. Pressurized vessels (i.e. steam boilers, gun barrels and cooked sausages) usually rupture into an axial/longitudinal direction because the circumferential stress at a given internal pressure is always twice as high as the longitudinal stress. On the contrary, an aortic dissection entry mostly runs in the circumferential direction [3, 5, 6]. This observation generated the hypothesis that the main trauma/stress must be in the longitudinal direction. Longitudinal elasticity is a major component of the physiological 'Windkessel' effect [7]. Therefore, elongation of the aorta may lead to the loss of longitudinal elasticity [13, 14] and increased stress on the intima.

Moreover, an asymmetric change in morphology seems to take place during dissection. In the outer curvature, the gain in length was 37% from the non-dissected to dissected aortas, whereas in the inner curvature it was just 16%. This asymmetric elongation is necessarily accompanied by a pronounced angulation of the ascending aorta, further increasing the longitudinal stress.

Future studies will have to reveal to what extent aortic elongation is the main risk factor for dissection.

Prediction of aortic dissection

Our dataset is not sufficient to establish aortic elongation as an independent risk factor for TAD nor to quantify the risk associated with a certain degree of elongation. This precludes prognostication.

When comparing the frequency spectrums of the diameters of healthy, dissected and predissection aortas in the box-and-whiskers plots in Fig. 3, another important issue becomes clear. Despite the statistically significant differences of the location parameters (i.e. median), the dispersion parameters (i.e. range, exemplified by the whiskers) substantially overlap. This region of overlap is essentially responsible for the suboptimal discrimination by diameter alone between the healthy and diseased aortas or between the aortas at risk or not at risk for dissection.

However, a similar region of overlap is present when comparing the parameters of length in the healthy and diseased aortas (see box-and-whiskers plots in Figs 4 and 5). Basically, the length parameters suffer from the same lack in discriminatory power between the patients at and not at risk for dissection.

Consequently, using parameters of aortic length as standalone measurements to identify patients at risk for TAD does not seem to have advantages over using the aortic diameter. Currently, it remains unclear to what extent it would be advantageous to use these parameters in combination.

This reveals the need of integrative 3D models comprising several parameters, i.e. diameters, lengths, volume, cuspidity, root and arch morphology and angulations, to better define a risk morphology of the aorta and to enhance accuracy of diagnosis and prognostication.

CONCLUSION

We showed that dissected aortas and aortas predissection are elongated compared with their healthy counterparts. Moreover, the concept of aortic elongation as a risk factor for dissection appears conclusive from a pathophysiological viewpoint. The length of the healthy ascending aorta does not exceed 100 mm in the central line (frontal and sagittal view) measurement or direct distance between the STJ and the BCT. Thus, these measures may represent a relevant additional risk factor for TAD. Further investigations are needed to assess the pathophysiological role and diagnostic value of aortic elongation.

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Conflict of interest: none declared.

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APPENDIX. CONFERENCE DISCUSSION

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Dr M. Czerny (Freiburg, Germany): I would like to ask you, because there is a certain dependency on diameter and length that the reason why these aortas were longer were due to the fact that they were already slightly larger. So I think that volumetry as another means to quantify morphological changes would be helpful in better getting into the issue. That is the first point.

The second point is, it would be interesting to know about the cuspidity in these patients, the morphology of the sinotubular junction, arch vessel variations such as bovine arches, isolated vertebral off springs, because it might also play a certain role.

Finally, I think that this work should be seen as a driving force to initiate screening programs focusing on that issue of length.

Dr Krüger: Dr Czerny, and I totally agree. My idea is, to get more parameters like aortic length, arch morphology and arch angulation. Sometimes we see these huge, elongated arches with sharp angulation. We should probably invent a model or develop a score to maybe better predict type A aortic dissection with these parameters.

Dr R. De Paulis (Rome, Italy): Is there any ratio between the length and the diameter indicating the patients at higher risk?

Dr Krüger: There is a correlation between aortic dilatation and aortic elongation. Sometimes dilated aortas are elongated, but also undulated and slim aortas may be elongated. The correlation between diameter and length was 0.5 or 0.6. But which parameter, elongation or dilatation is the stronger risk factor I cannot tell you precisely.

Dr De Paulis: So given the same diameter, is there a small risk if the aorta is longer or shorter?

Dr Krüger: Given the same diameter, the risk, in my opinion, is much higher if the aorta is longer and elongated.

Dr De Paulis: This is already a good piece of information, I think

Dr M. Shrestha (Hannover, Germany): What would your message be to the surgical community depending on this work, because you basically have 15 patients in the retrospective study, but would you advise us to do a CT scan in all patients or in certain high risk patients if you can define the high risk patients? What would be the message?

Dr Krüger: Well, we had not just 15 patients in the entire study, just the group of patients who have had a CT before the event of dissection contained 15 patients; The post dissection- and the healthy control groups had about 150 patients. The message is, that we probably should evaluate the measures of aortic length, as risk factors for type A, aortic dissection – I don't know which maybe is the best parameter, probably the central line length in 3D reconstruction—we should test this in a larger series, and in the future we maybe make the indication for prophylactic surgery also on aortic length.

Dr Shrestha: Yes, but the question is to do that you need a CT scan. So which group of patients would you advise to get a CT scan to find that out?

Dr Krüger: Patients with Marfan disease or with connective tissue disease, would be the first group. Patients who received CT's for other indications and were found to have an elongated aorta would form the second group. Also maybe, but I am not sure about that, patients with massive hypertension, because we found that hypertensive patients had longer aortas as well.

Dr T. Schachner (Innsbruck, Austria): Did you also index the aortic length with regard to body surface area or body mass index?

Dr Krüger: We found no proper correlations between aortic dimensions and body surface area or body mass index. The only correlation was with age and hypertension.