Evaluation of Common Carotid Stiffness via Echo Tracking in Hypertensive Patients Complicated by Acute Aortic Dissection

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Objectives—To evaluate the common carotid stiffness via echo tracking in patients with hypertension and acute aortic dissection (AD) and to investigate the independent predictors for the occurrence of AD in hypertensive (HP) patients.

Methods—Fifty HP patients complicated by acute AD (AD group), 50 HP patients without AD (HP group), and 50 age-matched healthy volunteers (control group) were enrolled to assess the common carotid stiffness index ($\beta$), single-point pulsed wave velocity ($\text{PWV}_\beta$), and arterial compliance (AC) via echo tracking.

Results—The intima-media thickness, diameter, $\beta$ and $\text{PWV}_\beta$ of the common carotid artery (CCA) in the AD group were significantly higher than those in the HP and control groups, whereas AC in the AD group was significantly lower ($P < .05$). In a multivariate logistic regression analysis, the systolic blood pressure (SBP; odds ratio [OR], 2.316; 95% confidence interval [CI], 2.033–2.563; $P < .001$), $\beta$ (OR, 2.140; 95% CI, 1.931–2.367; $P < .001$), $\text{PWV}_\beta$ (OR, 1.212; 95% CI, 1.004–1.397; $P = .023$), and AC (OR, 0.565; 95% CI, 0.339–0.654; $P < .001$) were significantly related to the occurrence of AD in HP patients. The area under the curve values for the AC, SBP, $\beta$, and $\text{PWV}_\beta$ were 0.822, 0.806, 0.778, and 0.741, respectively, and the area under the curve was up to 0.943 when these parameters were combined.

Conclusions—The compliance of the CCA decreased, and the stiffness of the CCA increased significantly in HP patients complicated by AD. The AC, $\beta$, and $\text{PWV}_\beta$ of the CCA, together with the SBP, were independent predictors of the occurrence of AD in HP patients.

Key Words—aortic dissection; arterial stiffness; echo tracking; hypertension

Acute aortic dissection (AD) is a critical and life-threatening cardiovascular disease with a lethality rate of 1% to 2% per hour after onset of symptoms in patients without treatment. Aging, smoking, hypertension, aortic aneurysm, pregnancy, and genetic diseases such as Marfan disease, Loeys-Dietz syndrome, and Ehlers-Danlos syndrome are the common risk factors for AD. According to a study by the International Registry of Acute Aortic Dissection in 2000, hypertension is considered the most important risk factor for AD, as almost 80% of patients with AD had hypertension as well. Pathologic studies have confirmed that arterial remodeling and media degeneration are the basic vascular structural changes in hypertension. Moreover, the suddenly increased blood...
pressure will exert an abnormal shear force on the arterial wall, which may cause disruption of the degenerated media and eventually lead to the occurrence of AD.4 The diameter of the arterial lumen is generally considered an indicator of arterialremodeling.5 With the aortic diameter increasing, the risk of AD increases accordingly. Consequently, the maximumaortic diameter has been adopted as the criterion for prophylactic surgical interventions in current practice guidelines.6 However, most patients who presented with AD had aortic diameters below the recommended criterion (55 mm).7 It seems that the aortic diameter is an insufficient parameter to identify individuals at risk of an aortic catastrophe. In this regard, other parameters should be found as supplements for a personalized risk assessment and clinical decision making.

In large conduit arteries, the vascular compliance and stiffness usually change along with the arterial remodeling. The pulsed wave velocity (PWV) was recommended as a noninvasive “reference standard” for assessments of arterial stiffness according to the guidelines.5,8 However, the exfoliated intima flapping in the arterial lumen of patients with AD might influence the propagation of the pulsed wave, which limits its application for evaluating arterial stiffness in AD. Echo tracking (ET) is a high-resolution ultrasound technique used to assess the compliance and stiffness of large peripheral arteries, especially the common carotid artery (CCA).9 It provides the parameters of arterial compliance (AC), the arterial stiffness index (β), and the single-point pulsed wave velocity (PWVβ), which have been confirmed as sensitive indicators of early microstructural and functional changes in the arterial wall.10 Therefore, we aimed to evaluate the common carotid stiffness via ET and to investigate the predictive value of the common carotid stiffness for the occurrence of AD in hypertensive (HP) patients.

Materials and Methods

Study Population
This was a cross-sectional study conducted in accordance with the tenets of the Declaration of Helsinki and was approved by the Clinical Research Ethics Committee of the Second Affiliated Hospital of Dalian Medical University. Fifty consecutive HP patients who had a diagnosis of acute AD by computed tomographic angiography between January 2018 and October 2019 were enrolled as the AD group. The inclusion criteria included a hypertension history of more than 1 year and brachial systolic blood pressure (SBP) of 130 mm Hg from at least one side when AD was diagnosed. Another 50 primary HP patients were selected as the HP group. The inclusion criterion was brachial blood pressure of 130/80 mm Hg in more than 3 consecutive measurements.11 Based on the patients’ clinical features and their physical, laboratory, and imaging examinations, diseases of secondary hypertension, diabetes, coronary artery disease, valvular disease, connective tissue disease (Marfan disease, Loeys-Dietz syndrome, and Ehlers-Danlos syndrome), aortic aneurysm, trauma, vascular inflammation, and aortic coarctation were excluded. Fifty healthy volunteers who underwent physical, laboratory, and imaging examinations and had no any systemic diseases were recruited as the control group. The participants were age and sex matched in each group. All of them were from the Second Affiliated Hospital of Dalian Medical University and consented to the clinical trial.

Carotid Ultrasound Examinations and ET Analysis
The carotid ultrasound examinations were performed with an SSD-α7 ultrasound system (Aloka Co, Ltd, Tokyo, Japan) equipped with ET analysis software. The participants were in a supine position, breathing calmly. The CCA was detected with a UST-5548 linear transducer (5–12 MHz) and an electrocardiogram was recorded simultaneously. The CCA that communicated with the true lumen and had no exfoliated intima was selected for further assessment. Then the ipsilateral brachial blood pressure was measured first. When the longitudinal view of the CCA was clearly visible, the intima-media thickness (IMT) was measured. Echo tracking was started with a frame rate of more than 60 frames per second. The sampling gate was placed 1.5 cm below the bifurcation and adjusted perpendicular to the wall of the CCA. The trace lines were set in the media-adventitia of the proximal and distal walls of the CCA on the image, respectively. Then the motion curve of the carotid arterial wall was recorded by M-mode imaging. When the SBP and diastolic blood pressure (DBP) from the ipsilateral brachial artery were input into the software, the
diameter of the CCA and arterial stiffness parameters of AC, $\beta$, and PWV$\beta$ were calculated automatically (Figure 1).

**Repeatability and Reproducibility of Ultrasound Measurements**

Fifteen participants from the HP group and 15 from the AD group were randomly selected and assessed for the intraobserver and interobserver variability of ultrasound measurements. To assess intraobserver repeatability, the same observer repeated the measurement 5 minutes after the initial measurement. To assess interobserver reproducibility, measurements were performed by another observer, who was blinded to the first observer’s findings.

**Statistical Analyses**

SPSS version 18.0 software (IBM Corporation, Armonk, NY) was used for all data analyses. Nominal variables were presented as total numbers and compared by the $\chi^2$ test. Continuous variables were presented as mean ± standard deviation. A 1-way analysis of variance comparison was performed after the homogeneity test. Differences between groups were analyzed by the least significant difference method. A multivariate logistic regression analysis was performed to explore the independent variables associated with the occurrence of AD in HP patients. The parameters of $\beta$, PWV$\beta$, AC, SBP, DBP, maximum diameter ($D_{\text{max}}$), minimum diameter ($D_{\text{min}}$), and IMT were set as independent variables in the model. The results

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**Figure 1.** Echo tracking for the HP patients complicated with AD. A, The sampling gate was placed 1.5 cm below the bifurcation, and the trace lines were set in the media-adventitia of the proximal and distal walls of the CCA, respectively, from the 2-dimensional view on the left side. The wall motion curve of the CCA with electrocardiography simultaneously recorded was shown in the M-mode on the right side. B, Parameters of CCA stiffness were calculated and shown automatically on the screen.

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**Table 1.** Baseline Characteristics of the Participants

| Characteristic   | Control (n = 50) | HP (n = 50) | AD (n = 50) | $P$  |
|------------------|------------------|------------|-------------|------|
| Age, y           | 55.00 ± 9.64     | 55.09 ± 8.86 | 55.19 ± 9.23 | .908 |
| Male             | 25 (50)          | 30 (60)    | 35 (70)     | .160 |
| Smoking          | 20 (40)          | 24 (48)    | 26 (52)     | .728 |
| Height, m        | 1.68 ± 0.12      | 1.71 ± 0.48 | 1.72 ± 0.41 | .181 |
| Weight, kg       | 71.26 ± 10.80    | 71.55 ± 7.32 | 72.22 ± 6.47 | .360 |
| BMI, m/kg$^2$    | 25.75 ± 7.51     | 24.46 ± 2.05 | 24.41 ± 1.72 | .896 |
| SBP, mm Hg       | 128.52 ± 14.38   | 137.39 ± 20.57$^a$ | 161.66 ± 178.92$^{ab}$ | <.001 |
| DBP, mm Hg       | 75.00 ± 8.15     | 7723 ± 6.71$^a$ | 81.16 ± 9.83$^{ab}$ | <.001 |

Continuous variables are presented as mean ± SD. Nominal variables are presented as total number (percent). Comparisons were made with a 1-way analysis of variance. BMI indicates body mass index.

$^a$Significant differences compared to the control group.

$^b$Significant differences compared to the HP group.
were described as the odds ratio (OR) and 95% confidence interval (CI). Receiver operating characteristic curves were used to determine the sensitivity and specificity of independent predictors of AD. The Bland-Altman test and intraclass correlation coefficient (ICC) was used to show reliability in this method. Statistical significance was at the .05 level.

**Results**

**Characteristics of the Population**

The AD group included 35 male and 15 female participants (18 cases of Stanford type A and 32 cases of Stanford type B; age range, 39–73 years). The HP group included 30 male and 20 female participants.

**Table 2. Parameters of CCA Ultrasound and Arterial Stiffness**

| Parameter | Control (n = 50) | HP (n = 50) | AD (n = 50) | P |
|-----------|-----------------|-------------|-------------|---|
| Dmax, mm  | 7.56 ± 1.97     | 7.95 ± 1.05a| 8.60 ± 0.78ab| <.001|
| Dmin, mm  | 6.81 ± 0.97     | 7.31 ± 0.82a| 8.13 ± 0.67ab| <.001|
| IMT, mm   | 0.96 ± 0.41     | 1.47 ± 0.51a| 1.80 ± 0.47ab| <.001|
| β         | 8.48 ± 1.02     | 9.49 ± 1.87a| 13.13 ± 4.15ab| <.001|
| PWVβ, m/s | 710 ± 1.73      | 8.89 ± 1.87a| 11.79 ± 3.71ab| <.001|
| AC, mm²/kPa| 2.53 ± 0.64    | 1.67 ± 0.26a| 1.22 ± 0.46ab| <.001|

Continuous variables are presented as mean ± SD. Comparisons were made with a 1-way analysis of variance.

aSignificant differences compared to the control group.
bSignificant differences compared to the HP group.

**Table 3. Intraobserver and Interobserver Variability of Measurements**

| Parameter | Intraobserver | Variability | Interobserver | Variability |
|-----------|---------------|-------------|---------------|-------------|
|           | ICC           | 95% CI      | ICC           | 95% CI      |
| β         | 0.909         | 0.816–0.956 | 0.858         | 0.724–0.930 |
| PWVβ      | 0.788         | 0.602–0.893 | 0.723         | 0.493–0.858 |
| AC        | 0.781         | 0.589–0.890 | 0.818         | 0.655–0.909 |
| IMT       | 0.818         | 0.657–0.908 | 0.781         | 0.589–0.890 |

**Figure 2. Variability of ultrasound measurements. A. Bland-Altman plot of intraobserver variability. B. Bland-Altman plot of interobserver variability.**
those in the control and HP groups, the $D_{\text{max}}$, $D_{\text{min}}$, and IMT of the CCA in the AD group were significantly higher ($P < .05$). The common carotid stiffness parameters of $\beta$, PWV, and AC of the 3 groups were significantly different ($P < .05$). The $\beta$ and PWV were significantly higher than those in the HP and control groups, whereas AC in the AD group was significantly lower than that in the other groups ($P < .05$).

**Variability of Ultrasound Measurements**

The interobserver and intraobserver ICCs for the $\beta$, PWV, AC, and IMT were 0.86 and 0.91, 0.72 and 0.79, 0.78 and 0.82, and 0.82 and 0.78, respectively. The measurements had good repeatability and reproducibility. The data for the ICCs and 95% CIs are presented in Table 3 and Figure 2.

**Multivariate Logistic Regression Analysis of AD in HP Patients**

The multivariate logistic regression analysis revealed that the SBP, $\beta$, PWV, and AC were optimal predictors of the occurrence of AD in HP patients (SBP: OR, 2.316; 95% CI, 2.033–2.563; $\beta$: OR, 2.140; 95% CI, 1.931–2.367; PWV: OR, 1.212; 95% CI, 1.004–1.397; AC: OR, 0.565; 95% CI, 0.339–0.654; $P < .05$). The area under the curve (AUC) values of the AC, SBP, $\beta$, and PWV were 0.822, 0.806, 0.778, and 0.741, respectively. When these parameters were combined, the AUC was up to 0.943. According to the Youden index (sensitivity + specificity – 1), when AC was down to 1.6 mm$^2$/kPa, the sensitivity and specificity of the occurrence of AD in HP patients were 78% (39 of 50) and 88% (44 of 50); when the SBP was up to 148 mm Hg, the sensitivity and specificity were 94% (47 of 50) and 20% (10 of 50); when the $\beta$ was up to 11.15, the sensitivity and specificity were 88% (44 of 50) and 66% (33 of 50); and when the PWV was up to 10.15 m/s, the sensitivity and specificity were 64% (32 of 50) and 86% (43 of 50). The differences were statistically significant ($P < .05$; Table 4 and Figure 3).

**Discussion**

Acute AD is a critical cardiovascular disease frequently accompanied by devastating complications such as lethal malperfusion syndrome, aortic regurgitation,
cardiac failure, cardiac tamponade, and stroke.\textsuperscript{2,12} Although nearly 80% of individuals with AD have concomitant hypertension, the most HP patients, even severely HP ones, never have AD.\textsuperscript{13,14} In addition, a recent cohort study conducted by researchers from the Mayo Clinic showed that the mean age at onset of AD was 68.9 \pm 15.6 years in the United States.\textsuperscript{13} However, epidemiologic data from Chinese researchers showed that the mean age at onset of AD in China was much younger compared to the data from the International Registry of Acute Aortic Dissection (46.9 \pm 12.0 versus 61.5 \pm 14.6 years for type A and 50.6 \pm 12.3 versus 63.6 \pm 14.1 years for type B; \( P < .001 \)).\textsuperscript{14} The mean age at onset of AD in patients with hypertension in our study was 55.19 \pm 9.23 years, which was also younger than the onset ages reported in the United States and Europe.\textsuperscript{2,13} In view of so many younger HP patients complicated by AD, it is necessary to screen some indicators to predict the occurrence of AD in HP patients.

Arterial remodeling in hypertension is one of the essential vascular structural changes, which is characterized by an increased IMT (about +15%–40%) and dilated lumens of proximal elastic arteries.\textsuperscript{5} In our study, the IMT, \( D_{\text{max}} \) and \( D_{\text{min}} \) of the CCA in the AD group were much higher than those in the HP and control groups, indicating that arterial remodeling in HP patients complicated by AD is substantial. Previous investigations have shown that in the human arterial system, shear forces ranged from about 1 to 7 Pa in healthy individuals up to 10 Pa in HP patients and 13 Pa in those with AD.\textsuperscript{15} Increased shear force on the arterial wall can induce vascular smooth muscle cell relaxation in the early stage and later lead to media degeneration.\textsuperscript{5,16} If the intima and degenerative arterial wall are torn by the abnormally increased shear stress, the pulsatile blood flow will penetrate into the diseased media, leading to AD. According to current practice guidelines, the indicators for prophylactic aortic surgery to prevent this lethal complication are based on the aortic diameter, with a recommended threshold of 55 mm for nonsyndromic individuals.\textsuperscript{6} Furthermore, the controversy over whether the risk of AD in a moderately dilated ascending aorta (40–50 mm) has been sparsely evaluated has been presented sequentially.\textsuperscript{17,18} Therefore, some other indicators, other than the diameter alone, should be taken into account for clinical decisions.

The PWV is considered the reference standard for assessing arterial stiffness.\textsuperscript{5,8} Every increase of the PWV by 1 m/s indicates a 15% increase in the risk of cardiovascular disease.\textsuperscript{19} The carotid-femoral PWV and brachial-ankle PWV are widely used to assess the arterial stiffness, which depends on the distance and the time over which the wave travels. However, the exfoliated intima flapping in the aortic lumen of AD might affect the propagation of the pulsed wave, which limits the application of the carotid-femoral PWV or brachial-ankle PWV for evaluating arterial stiffness in AD. Nevertheless, the noninvasive ET technique is a good solution for assessment of AC and stiffness in AD because only a single point of the arterial wall needs to be detected. The properties of elasticity and stiffness, including AC, \( \beta \), and PWV\( \beta \), are automatically estimated by the Young elastic modulus.\textsuperscript{20} As a superficial large artery, the CCA is accessible to being examined. Although the heterogeneous elastic properties along the arterial tree create a stiffness gradient, for instance, the elasticity decreases from the proximal ascending aorta to the downstream iliac and femoral arteries, the AC and stiffness of the CCA are still good references for us to learn about the elasticity of large conduit arteries.\textsuperscript{15} Shingu et al\textsuperscript{21} investigated the stiffness and AC of the CCA in patients with chronic AD and found that there was no significant difference between the patients with a thoracic aortic aneurysm and chronic AD and the patients with coronary disease without AD. However, they did not compare them to HP patients and healthy control participants. In this study, we found that the \( \beta \) and PWV\( \beta \) of the CCA in the AD group were significantly higher than those in the HP and control groups, whereas AC was significantly lower than that in the HP and control groups, which indicated that the elasticity of the CCA in HP patients with AD was damaged much more severely than that in HP patients without AD. Deplano et al\textsuperscript{22} reported that the parameters \( \beta \) and the Peterson modulus measured in vivo were higher in patients with dissection than those in the control group, which was consistent with the in vitro histologic findings from the dissected samples. The elastin dislocations, reduced elastin density, and increased collagen density were shown in these dissected samples.

In addition, according to the logistic regression in our study, the AC, SBP, \( \beta \), and PWV\( \beta \) were screened
as independent predictors for the occurrence of AD in HP patients. Arterial compliance is a protective factor, whereas the SBP, β, and PWVβ are risk factors for AD in HP patients. Although the diameter and IMT are important indicators of arterial remodeling, the AC and stiffness of the CCA, which optimally reflect the elastic and stiffness properties of the proximal large arteries, are more sensitive for identifying the risk of AD. On account of these findings, an evaluation of common carotid stiffness in HP patients via ET is expected to be a new reference for a personalized risk assessment and clinical decision making. In addition, it might be used to screen medicines targeted for the improvement of the arterial stiffness to prevent the occurrence of AD in HP patients.

There were a few limitations in this study. First, this study was a cross-sectional study with a small sample size in a single center. Second, patients with hypertension were not classified on the basis of blood pressure, and medications for antihypertension treatment were also not considered in this study. Third, the ET technique is based on a high-resolution image; poor image quality or contouring of the carotid artery can affect the accuracy of the measurement. Although these factors may have some influence on the result, the noninvasive assessment of common carotid stiffness is undoubtedly helpful for the early warning signs of the occurrence of AD.

In conclusion, the compliance of the CCA decreased and the stiffness of CCA increased significantly in HP patients complicated by AD. The AC, β, and PWVβ of the CCA, together with the SBP, were independent predictors of the occurrence of AD in HP patients.

References

1. Gawinecka J, Schönrath F, von Eckardstein A. Acute aortic dissection: pathogenesis, risk factors and diagnosis. Swiss Med Wkly 2017; 147:w14489.
2. Hagan PG, Nienaber CA, Isselbacher EM, et al. The International Registry of Acute Aortic Dissection (IRAD): new insights into an old disease. JAMA 2000; 283:897–903.
3. Tanaka LY, Laurindo FRM. Vascular remodeling: a redox-modulated mechanism of vessel caliber regulation. Free Radic Biol Med 2017; 109:11–21.
4. Sato F, Kitamura T, Kongo M, et al. Newly diagnosed acute aortic dissection: characteristics, treatment modifications, and outcomes. Int Heart J 2005; 46:1083–1098.
5. Laurent S, Cockcroft J, Van Bortel L, et al. Expert consensus document on arterial stiffness: methodological issues and clinical applications. Eur Heart J 2006; 27:2588–2605.
6. Hiratzka LF, Bakris GL, Beckman JA, et al. 2010 ACCF/AHA/ATS/ACR/ASA/SCA/SIR/ST/SVM guidelines for the diagnosis and management of patients with thoracic aortic disease: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines, American Association for Thoracic Surgery, American College of Radiology, American Stroke Association, Society of Cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, Society of Interventional Radiology, Society of Thoracic Surgeons, and Society for Vascular Medicine. J Am Coll Cardiol 2010; 55:e27–e129.
7. Pape LA, Tsai TT, Isselbacher EM, et al. Aortic diameter > or = 5.5 cm is not a good predictor of type A aortic dissection: observations from the International Registry of Acute Aortic Dissection (IRAD). Circulation 2007; 116:1120–1127.
8. van Sloten TT, Schram MT, van den Hurk K, et al. Local stiffness of the carotid and femoral artery is associated with incident cardiovascular events and all-cause mortality: the Hoorn study. J Am Coll Cardiol 2014; 63:1739–1747.
9. Giannattasio C, Salvi P, Valbusa F, et al. Simultaneous measurement of beat-to-beat carotid diameter and pressure changes to assess arterial mechanical properties. Hypertension 2008; 52: 896–902.
10. Wu Y, Xie M, Zhang L, Lu X, Cheng X, Lv Q. Carotid intima-media roughness and elasticity in hypertensive patients with normal carotid intima-media thickness. J Ultrasound Med 2019; 38: 1545–1552.
11. Whelton PK, Carey RM, Aronow WS, et al. 2017 ACC/AHA/ABC/ACPM/AGS/APHA/ASH/ASPC/NMA/PCNA guideline for the prevention, detection, evaluation, and management of high blood pressure in adults: executive summary—a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. Hypertension 2018; 71:1269–1324.
12. Weiss S, Sen I, Huang Y, et al. Cardiovascular morbidity and mortality after aortic dissection, intramural hematoma, and penetrating aortic ulcer. J Vasc Surg 2019; 70:724–731.e1.
13. DeMartino RR, Sen I, Huang Y, et al. Population-based assessment of the incidence of aortic dissection, intramural hematoma, and penetrating ulcer, and its associated mortality from 1995 to 2015. Circ Cardiovasc Qual Outcomes 2018; 11:e004689.
14. Huang B, Chen Z, Lu H, et al. Influence of age on clinical presentation, therapeutic options, and outcome in Chinese patients with acute aortic dissection. Int Heart J 2019; 60:1373–1380.
15. Shi Y, Zhu M, Chang Y, Qiao H, Liu Y. The risk of Stanford type-A aortic dissection with different tear size and location: a numerical study. *Biomed Eng Online* 2016; 15:128.

16. Chistiakov DA, Orekhov AN, Bobryshev YV. Effects of shear stress on endothelial cells: go with the flow. *Acta Physiol (Oxf)* 2017; 219: 382–408.

17. Adriaans BP, Wildberger JE, Westenberg JJM, Lamb HJ, Schalla S. Predictive imaging for thoracic aortic dissection and rupture: moving beyond diameters. *Eur Radiol* 2019; 29:6396–6404.

18. Kim JB, Spotnitz M, Lindsay ME, MacGillivray TE, Isselbacher EM, Sundt TM III. Risk of aortic dissection in the moderately dilated ascending aorta. *J Am Coll Cardiol* 2016; 68: 1209–1219.

19. Liao J, Farmer J. Arterial stiffness as a risk factor for coronary artery disease. *Curr Atheroscler Rep* 2014; 16:387.

20. Niki K, Sugawara M, Chang D, et al. A new noninvasive measurement system for wave intensity: evaluation of carotid arterial wave intensity and reproducibility. *Heart Vessels* 2002; 17:12–21.

21. Shingu Y, Shiya N, Ooka T, et al. Augmentation index is elevated in aortic aneurysm and dissection. *Ann Thorac Surg* 2009; 87:1373–1377.

22. Deplano V, Boufi M, Gariboldi V, et al. Mechanical characterisation of human ascending aorta dissection. *J Biomech* 2019; 94:138–146.