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Exercise stress echocardiogram for the evaluation of change in the ventricular–arterial interaction after thoracic endovascular aortic repair

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Key question

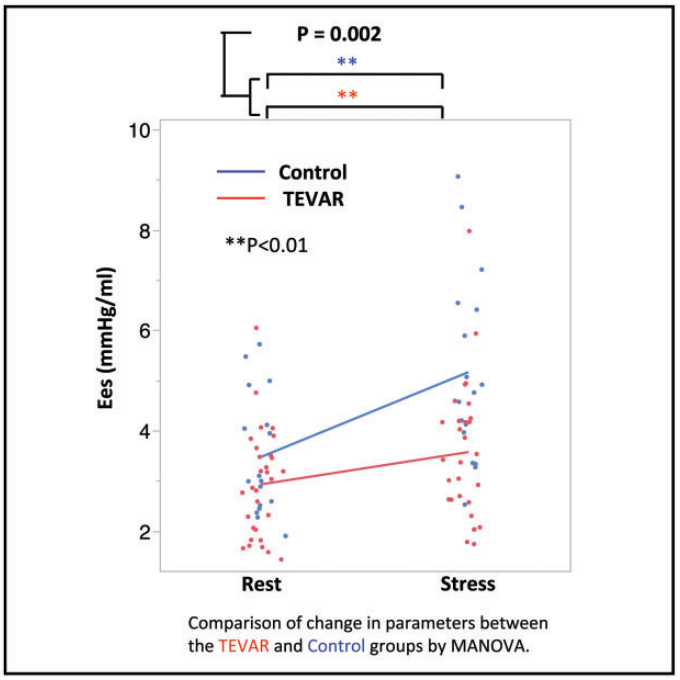
How does a thoracic stent graft influence cardiac function?

Key finding(s)

Arterial elastance is elevated in the endovascular repair group only. The increase in end-systolic elastance was more remarkable in the control group.

Take-home message

A stress echocardiogram exposed influences of thoracic endovascular aortic repair in suppressing left ventricular contractile capacity.



Comparison of change in parameters between the TEVAR and Control groups by MANOVA.

Abstract

OBJECTIVES: The aim of this study was to explore, with exercise echocardiography, the potential impact of thoracic endovascular aortic repair (TEVAR) on the ventricle–aorta coupling, based on the hypothesis that the interaction between the ventricle and aorta may be influenced by an increase in aortic stiffness due to the stent graft.

METHODS: Of the patients who underwent isolated TEVAR for thoracic aortic diseases between April 2016 and December 2016, changes found in haemodynamic and echocardiographic parameters between the control ($n = 17$) and TEVAR ($n = 30$) groups were analysed by a stress echocardiogram.

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RESULTS: The end-systolic elastance significantly increased with stress in both groups [from 3.0 (2.5–4.5) mmHg/ml to 4.8 (3.7–6.5) mmHg/ml, $P < 0.001$, in the control group and from 2.9 (2.0–3.5) mmHg/ml to 3.4 (2.6–4.2) mmHg/ml, $P < 0.001$, in the TEVAR group]. The arterial elastance significantly elevated only in the TEVAR group [from 1.6 (1.3–1.8) mmHg/ml to 1.7 (1.5–2.0) mmHg/ml, $P = 0.007$] and arterial elastance/end-systolic elastance (ventricular–arterial coupling) significantly decreased only in the control group [from 0.5 (0.4–0.7) to 0.4 (0.3–0.5), $P = 0.002$, in the control group and from 0.6 (0.4–0.8) to 0.5 (0.4–0.6), $P = 0.10$, in the TEVAR group]. In the control group, the change in end-systolic elastance and ventricular–arterial coupling tended to be greater ($P = 0.002$ and 0.07).

CONCLUSIONS: An exercise echocardiogram showed the underlying influences of TEVAR on the interaction between the heart and aorta. TEVAR may have the potential to suppress left ventricular contractile capacity and increase cardiac afterload during exercise.

Clinical trial registration number: A201770-01.

Keywords: Thoracic endovascular aortic repair • Stress echocardiography • Ventricle–aorta coupling • Cardiac afterload

INTRODUCTION

Thoracic endovascular aortic repair (TEVAR) is an established option for the treatment of thoracic aortic diseases with excellent outcomes being reported in several studies [1–3]. However, one infrequently observed complication of TEVAR, acute heart failure and chronic heart failure, is not recognized as a major complication, yet it should be investigated because the mechanism and risk factors are undetermined. TEVAR is a less invasive and effective option for avoiding complications due to open surgery with thoracotomy. Stent grafts may influence the elasticity of the native aorta, and its brief deployment induces a drastic geometric change in the aortic lesion, which has the potential to affect the cardiac afterload. When investigating the interaction between the left ventricle and the aorta, not only does the cardiac systolic function and diastolic function need to be evaluated but also the cardiac afterload.

To evaluate the accurate ventricular–arterial interaction, a pressure–volume analysis is generally used, but validated non-invasive approaches have also evolved [4, 5]. Left ventricular (LV) end-diastolic and end-systolic volume (EDV and ESV) and stroke volume (SV) are non-invasively obtained using echocardiography. Therefore, end-systolic elastance (Ees) and arterial elastance (Ea) in the pressure–volume loop can be calculated, and the ratio of Ea/Ees [ventricular–arterial coupling (VAC)] strongly predicts cardiovascular performance and efficiency [6–9]. Additionally, stress echocardiography is an effective technique to evaluate a more sensitive reaction of the heart during exercise, and it is used to evaluate asymptomatic valvular diseases and underlying exacerbation factors [10–13].

Herein, on the basis of the hypothesis that the ventricle–aorta interaction may be influenced by a drastic geometrical change as a result of TEVAR, the aim of this study was to explore the potential impact of TEVAR on the ventricle–aorta interaction using exercise echocardiography.

PATIENTS AND METHODS

Cohort and data collection

Of the patients who underwent isolated TEVAR for thoracic aortic aneurysm, including 13 for aortic dissection between April 2016 and December 2016, a cohort of 30 patients was enrolled in our study. In the 13 patients with aortic dissection, the reason for treatment intervention was malperfusion in 2 cases and aneurysmatic enlargement in 11 cases. The period from onset was 90 (12.5–165) days. Patients with residual aneurysm, significant valvular diseases, lack of sufficient physical ability and a poor echocardiographic image were excluded. Data collection was performed as approved by the institutional review board, and this study was approved by the institutional ethics committee in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki and its later amendments. Consent was obtained from all patients. As a comparison group, data of hypertensive patients without abnormal findings after detailed examinations, including an exercise echocardiogram, were used ($n = 17$). To reduce the influence of a hypertensive heart, patients with hypertension were selected as a comparison cohort. Change in haemodynamic and echocardiographic parameters evaluated by stress echocardiography was compared between the normal and TEVAR groups. Preoperative patient characteristics are presented in Table 1, and there were no significant differences between the groups. In the TEVAR group, devices used included 10 Gore TAG thoracic endoprostheses (WL Gore and Associates, Flagstaff, AZ, USA), 2 TX2 thoracic endovascular grafts (Cook Inc., Bloomington, IN, USA) and 6 Valiant thoracic stent grafts (Medtronic Cardiovascular, Santa Rosa, CA, USA). A combination of TX2 and Valiant thoracic stent grafts was used in 1 patient. In 9 patients, 2 pieces of stent graft were joined, the diameter of which ranged from 28 mm to 45 mm. The mean length of the devices for the treatment was 172 ± 44 mm, ranging from 100 mm to 274 mm, and the proximal landing areas were zone 1 for 2 patients, zone 2 for 8 patients, zone 3 for 11 patients, T4 for 1 patient, T5 for 1 patient, T6 for 4 patients, T8 for 1 patient, T9 for 1 patient and T10 for 1 patient [14].

Table 1: Patient demographics

Variables	Control group ($n = 17$)	TEVAR group ($n = 30$)	P-value
Age (years), median (IQR)	74 (68–78)	69 (64–76)	0.15
Female gender, n (%)	7 (41.2)	4 (13.3)	0.07
Hypertension, n (%)	17 (100)	27 (90.0)	0.29
Hyperlipidaemia, n (%)	13 (76.5)	15 (50.0)	0.12
Diabetes mellitus, n (%)	5 (29.4)	6 (20.0)	0.49
Peripheral vascular disease, n (%)	1 (5.9)	1 (3.3)	>0.999
COPD, n (%)	0 (0)	1 (3.3)	>0.999
Beta-blocker intake, n (%)	7 (41.2)	21 (70.0)	0.07
Diuretic intake, n (%)	1 (5.9)	6 (20.0)	0.40
ACEI or ARB intake, n (%)	12 (70.6)	13 (43.3)	0.13

ACEI: angiotensin-converting enzyme inhibitor; ARB: angiotensin II receptor blocker; COPD: chronic obstructive pulmonary disease; IQR: interquartile range; TEVAR: thoracic endovascular aortic repair.

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Baseline quantitative echocardiography

Standard transthoracic echocardiography was performed on all patients in the left-lateral decubitus position by experienced sonographers before and after symptom-limited exercise, using a commercially available ultrasound machine (Aplio Artida, Toshiba Medical Systems Corporation, Tochigi, Japan). Standard data, such as ejection fraction (EF), diastolic and systolic dimensions and tricuspid regurgitation pressure gradient (TRPG), were obtained from the official echocardiographic report. Apical 4-chamber LVEDV and LVESV were obtained using the biplane method of disks as recommended by the American Society of Echocardiography [5]. SV was measured from the LV outflow tract area × LV outflow tract velocity time integral by the pulse wave Doppler method. The end-systolic pressure was estimated as 0.90 × systolic pressure, measured by a manual blood pressure cuff [4]. Effective Ea was defined as the ratio of end-systolic pressure to SV. Ees was calculated as mean blood pressure/LVESV. VAC was estimated by the Ea/Ees ratio [15, 16].

Exercise stress echocardiography

Exercise echocardiography was performed during semisupine bicycle exercise with the ergometer table tilted to 20° at 1–2 months after surgery. The initial workload was set at 25 W, followed by continuous increments of 25 W every 4 min, depending on the patient's physical condition. The duration of the exercise was measured, and the maximal exercise tolerance was expressed as estimated metabolic equivalents. Predicted exercise capacity was calculated in accordance with described nomograms [predicted metabolic equivalents; in men = 18 - (0.15 × age), in women = 14.7 - (0.13 × age)] [17]. Standard imaging windows were used, including parasternal long and short axes, as well as apical 4-chamber, 2-chamber and long-axis views. Echocardiographic images were digitally recorded and downloaded to an imaging server for offline analysis. A 12-lead electrocardiogram (ECG) was continuously recorded, and blood pressure was measured every 2 min. Patients were encouraged to exercise until exhaustion or the appearance of symptoms. The end points were as follows: (i) maximal heart rate reached (defined as the formula: 220 - age); (ii) systolic blood pressure <80 mmHg or >220 mmHg, or diastolic blood pressure >110 mmHg; (iii) ischaemia detected by ECG (>1 mm horizontal or downsloping ST depression 0.08 s after the J-point compared with the resting ECG); (iv) ventricular arrhythmias (>6 premature ventricular contractions per minute or complex arrhythmia); (v) rapid new atrial arrhythmias; and (vi) symptoms.

Statistical analysis

Continuous data are presented as median and the interquartile values (the first quartile to the third quartile). Continuous data were compared with the Mann–Whitney test. Categorical variables are given as a count and percentage of patients and compared using the χ^2 test. When any expected frequency was less than 1, or 20% of expected frequencies were ≤5, the Fisher's exact test was used. During the exercise test, the Wilcoxon signed rank test was used to evaluate the change between the rest and loading status in the normal and TEVAR groups, and changes in haemodynamic and echocardiographic parameters between the normal and TEVAR groups were analysed by multivariate analysis of variance. A P-value of <0.05 was considered statistically significant. All data were analysed using the Statistical Analysis Systems software JMP 12.0 (SAS Institute Inc., Cary, NC, USA).

RESULTS

Change in blood pressure and heart rate evaluated by stress echocardiography

The duration of exercise was 9 (8–12) min and 10 (7–12) min in the TEVAR and control groups ($P = 0.93$), and the maximum exercise tolerance was 50 (25–75) W and 60 (40–75) W in the TEVAR and control groups ($P = 0.40$).

In the control group, heart rate [from 64 (56–73) to 112 (94–119), $P < 0.001$] and the systolic blood pressure and diastolic blood pressure [from 131 (118–143) to 185 (153–195), $P < 0.001$; and, from 75 (69–84) to 87 (76–94), $P < 0.001$] significantly elevated with stress. In the TEVAR group, similarly, heart rate [from 68 (62–72) to 100 (91–106), $P < 0.001$], and systolic blood pressure and diastolic blood pressure [from 125 (113–137) to 165 (151–179), $P < 0.001$; and from 71 (63–78) to 88 (81–95), $P < 0.001$] significantly increased with stress. There was no significant difference in the rate of increase in systolic blood pressure and diastolic blood pressure between the groups ($P = 0.36$ and 0.23). However, the rate of increase in heart rate between at-rest and during exercise was greater in the control group compared to the TEVAR group ($P = 0.002$) (Fig. 1, Supplementary Material, Table S1).

Change in haemodynamics evaluated by stress echocardiography

Baseline echocardiographic data are shown in Table 2. LV diastolic dimension and LV systolic dimension were significantly larger in the TEVAR group. There were no significant differences in the remaining parameters between the groups. In the control group, LVEDV increased during stress [from 78 (65–99) ml to 87 (68–101) ml, $P = 0.07$], although this did not reach statistical significance. LVESV, LVEF, systemic vascular resistance (SVR), SV and cardiac output (CO) were significantly changed by exercise [LVESV, from 29 (22–38) ml to 25 (18–30) ml, $P = 0.002$; LVEF, from 65% (60–69%) to 72% (68–75%), $P < 0.001$; SVR, from 21 (16–23) Wood to 12 (11–15) Wood, $P < 0.001$; SV, from 65 (58–80) ml to 81 (67–90) ml, $P < 0.001$; and CO, from 4.5 (3.3–5.2) l/min to 8.2 (7.5–10.1) l/min, $P < 0.001$]. In the TEVAR group, LVEDV, LVEF, SVR, SV and CO were significantly affected during exercise [LVEDV, from 87 (72–125) ml to 111 (83–141) ml, $P < 0.001$; LVEF, from 65% (62–68%) to 70% (65–75%), $P < 0.001$; SVR, from 17 (14–19) Wood to 12 (10–14) Wood, $P < 0.001$; SV, from 74 (64–88) ml to 90 (78–103) ml, $P < 0.001$; and CO, from 4.9 (4.5–5.8) l/min to 9.2 (7.4–10.2) l/min, $P < 0.001$]. However, LVESV did not significantly decrease [from 31 (27–43) ml to 32 (26–46) ml, $P = 0.56$]. The change with exercise was equivalent in SV and CO between the groups ($P = 0.67$ and 0.25, respectively), whereas there were significant differences in change in LVEDV, LVESV, LVEF and SVR between the groups ($P = 0.030$, 0.004, 0.044 and <0.001, respectively) (Fig. 2, Supplementary Material, Table S2).

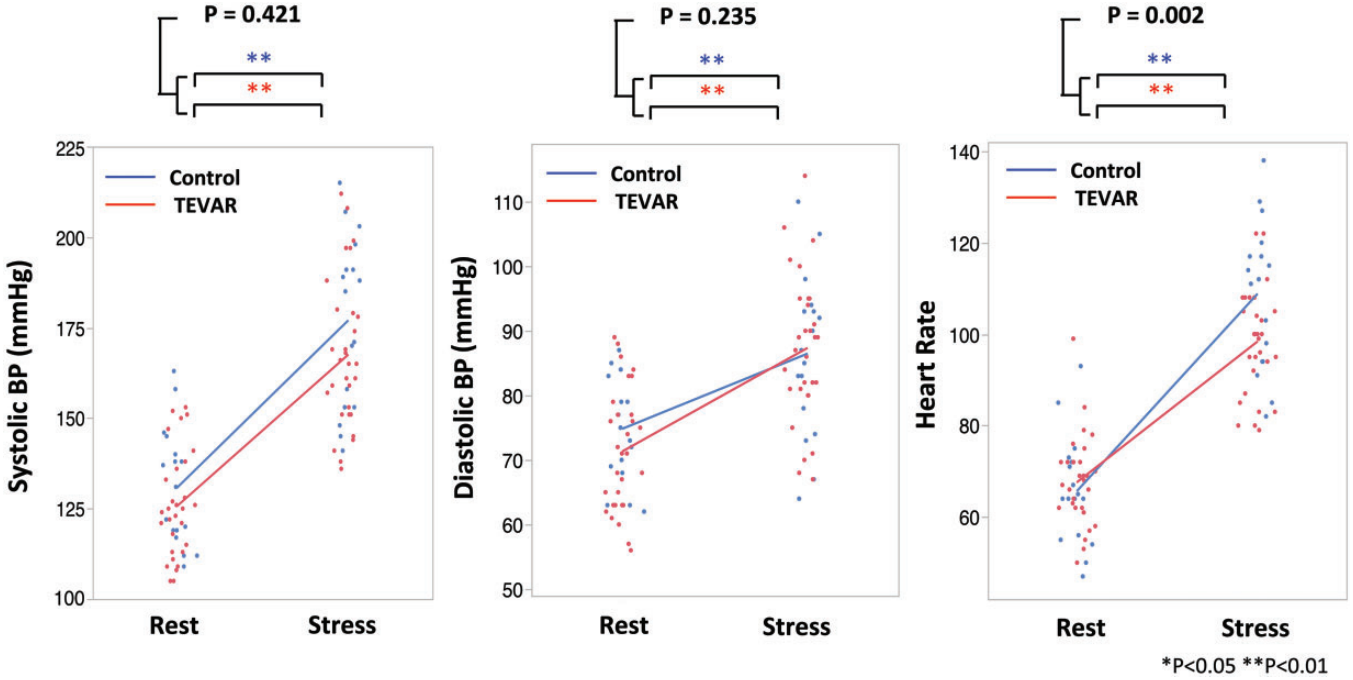

Figure 1: Change in BP and heart rate evaluated by stress echocardiography. BP: blood pressure; TEVAR: thoracic endovascular aortic repair.

Table 2: Preoperative echocardiographic data

Variables	Control group ($n = 17$), median (IQR)	TEVAR group ($n = 30$), median (IQR)	P-value
LVDD (mm)	44 (42–45)	48 (44–50)	0.012
LVDS (mm)	28 (26–29)	30 (28–32)	0.025
LVEF (%)	65 (63–68)	67 (62–68)	0.44
LAD (mm)	33 (31–39)	38 (36–42)	0.09
AI grade	1 (0–2)	1 (0.75–2)	0.36
MR grade	1 (1–1)	1 (1–2)	0.37
E/e′	10 (8–13)	11 (8–13)	0.50
E/A	0.8 (0.7–1.0)	0.9 (0.7–1.2)	0.56
TRPG (mmHg)	21 (18–26)	21 (18–25)	0.85

AI: aortic insufficiency; IQR: interquartile range; LAD: left atrial dimension; LVDD: left ventricular diastolic dimension; LVDS: left ventricular systolic dimension; LVEF: left ventricular ejection fraction; MR: mitral regurgitation; TEVAR: thoracic endovascular aortic repair; TRPG: tricuspid regurgitation pressure gradient.

Change in the ventricular–arterial interaction evaluated by stress echocardiography

In the control group, Ea did not demonstrate significant changes during exercise [from 1.7 (1.5–2.2) mmHg/ml to 2.0 (1.7–2.4) mmHg/ml, $P = 0.11$], and Ees significantly increased and VAC significantly decreased with stress [Ees, from 3.0 (2.5–4.5) mmHg/ml to 4.8 (3.7–6.5) mmHg/ml, $P < 0.001$, and VAC, from 0.5 (0.4–0.7) to 0.4 (0.3–0.5), $P = 0.002$], whereas in the TEVAR group, Ea and Ees significantly increased [Ea, from 1.6 (1.3–1.8) mmHg/ml to 1.7 (1.5–2.0) mmHg/ml, $P = 0.003$; and Ees, from 2.9 (2.0–3.5) mmHg/ml to 3.4 (2.6–4.2) mmHg/ml, $P < 0.001$]. VAC did not change with stress exercise in the TEVAR group [from 0.6 (0.4–0.8) to 0.5 (0.4–0.6), $P = 0.10$]. When comparing ventricular–arterial interaction between groups, changes in Ea and VAC were not significantly different ($P = 0.85$ and $P = 0.07$, respectively) whereas the increase in Ees was significantly greater in the control group ($P = 0.002$) (Fig. 3, Supplementary Material, Table S3).

Change in diastolic function evaluated by exercise stress echocardiography

E velocity, *E/A* ratio, TRPG and *E/e′* were evaluated as markers of diastolic function. In the control group, *E* velocity, *E/A* ratio, TRPG and *E/e′* significantly increased [*E* velocity, from 73 (54–80) cm/s to 116 (105–145) cm/s, $P < 0.001$; *E/A* ratio, from 0.8 (0.7–1.0) to 1.1 (0.9–1.4), $P = 0.007$, TRPG, from 21 (19–25) mmHg to 44 (40–58) mmHg, $P < 0.001$; and *E/e′*, from 11.4 (7.7–12.9) to 13.3 (10.3–16.8), $P = 0.044$]. Similarly, all parameters significantly increased in the loading phase in the TEVAR group [*E* velocity, from 63 (53–81) cm/s to 102 (86–138) cm/s, $P < 0.001$; *E/A* ratio, from 0.9 (0.8–1.2) to 1.1 (0.9–1.5), $P < 0.001$; TRPG, from 22 (17–25) mmHg to 39 (34–47) mmHg, $P < 0.001$; and *E/e′*, from 10.7 (9.5–14.1) to 14.0 (10.9–15.6), $P = 0.004$]. Of these parameters, the elevation of *E* velocity was greater in the control group ($P = 0.07$), although this was not statistically significant. The change in the parameters is shown in Supplementary Material, Table S4.

DISCUSSION

We documented the impact of TEVAR on the ventricular–arterial interaction by evaluating the results of the stress echocardiogram between patients who underwent TEVAR and the control study group. Firstly, the exercise-loading test revealed that the heart rate significantly increased in the control group compared to the TEVAR group. Secondly, in the TEVAR group, LVESV and LVEF did not react during exercise compared to the control group. Finally, Ea was elevated only in the TEVAR group, which showed

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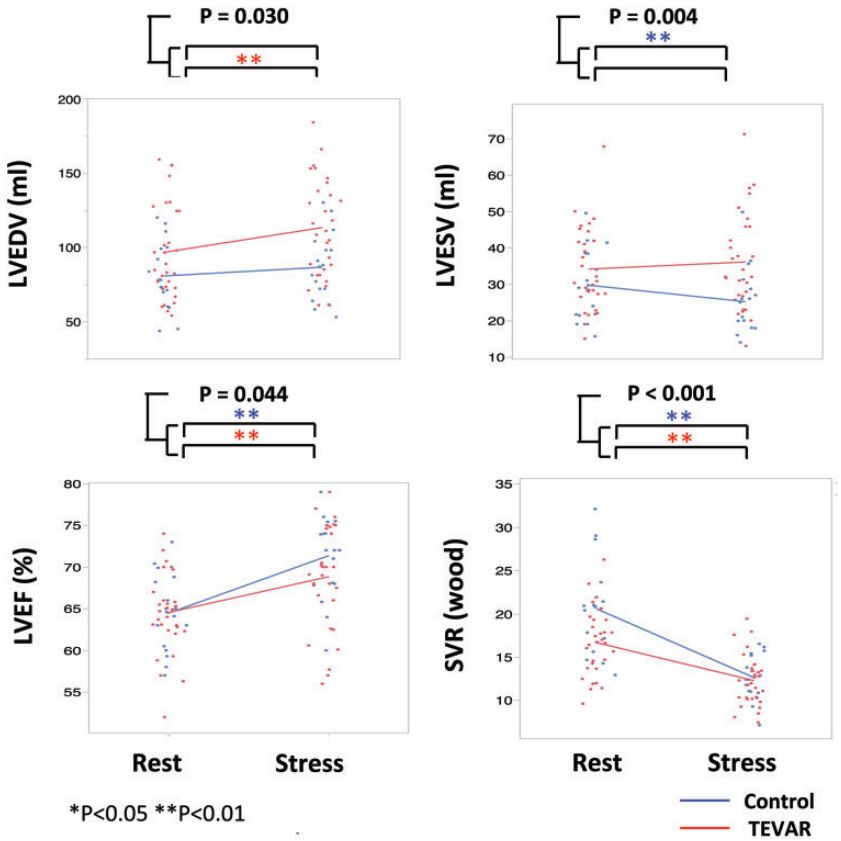

Figure 2: Change in haemodynamics evaluated by stress echocardiography. LVEDV: left ventricular end-diastolic volume; LVEF: left ventricular ejection fraction; LVESV: left ventricular end-systolic volume; SVR: systemic vascular resistance; TEVAR: thoracic endovascular aortic repair.

an increase in vascular stiffness [18]. Although Ees significantly increased in both groups, the change was more remarkable in the control group. VAC (Ea/Ees) significantly decreased only in the control group. As a result, these findings indicate that TEVAR may have the potential to suppress LV contractile capacity and enhance cardiac afterload during exercise.

Although TEVAR is by far the most popular option for the treatment of thoracic aortic diseases, the physical effects resulting from anatomical change by TEVAR are not fully known. Essentially, aortic elasticity serves a critical function in damping the highly pulsatile flow from the left ventricle, known as the 'Windkessel effect' [19, 20]. TEVAR diminishes the normal physiological function and creates an non-physiological condition which can lead to various adverse events. Recently, the influence of TEVAR on the induction of aortic stiffening has been demonstrated [21–23], and the dramatic change that causes hypertension, increase in cardiac afterload and decrease in coronary perfusion has been observed [24–27]. An increase in aortic stiffening due to TEVAR is known; however, the impact on the aorta and heart is still unclear, since recognizable effects are not observed in most cases. TEVAR not only causes direct geometric changes to the stented aorta but also results in dynamic changes from the diastole to systole in the ascending aorta and stented aorta. Hirotsu *et al.* reported that after TEVAR, the ascending aorta increased in axial length and decreased in mean curvature from the diastole to systole. The increase in aortic stiffening after TEVAR is compensated by greater length and curvature changes in the ascending aorta [25]. On the other hand, Takeda *et al.* [27] reported that endovascular repair increased vascular stiffness and induced LV hypertrophy and diastolic dysfunction. Additionally, impaired dispensability was associated with lower exercise tolerance. These findings may advocate results yielded by our present study and should be recognized as an important phenomenon. However, it is difficult to identify risk factors when effects are asymptomatic. Therefore, stress echocardiography is thought to be an effective technique to evaluate the change in the ventricle–arterial coupling after TEVAR, although preoperative stress echocardiography cannot be performed in patients with aortic diseases. Additionally, there are many unanswered questions regarding the influences of preoperative native aortic condition (originally stiff or not), different location of endografts (distance from the heart), different device materials (strength of the radial force), different pathologies (dissection or not) and different systolic and diastolic cardiac function (potentially good or not). It is also unknown whether the adverse effects of TEVAR may lead to heart failure associated with clinical problems, as the heart can adapt after a longer follow-up period. Further evaluation of the influences and the potential impact of endovascular repair on the interaction between the heart and aorta is required.

Limitations

The present study included several limitations. Firstly, this was a retrospective study with a small sample size. In the present study, there were no significant correlations between the location, size, length of devices and echocardiographic data. However, the influences of the times of the tests, devices, location and size of

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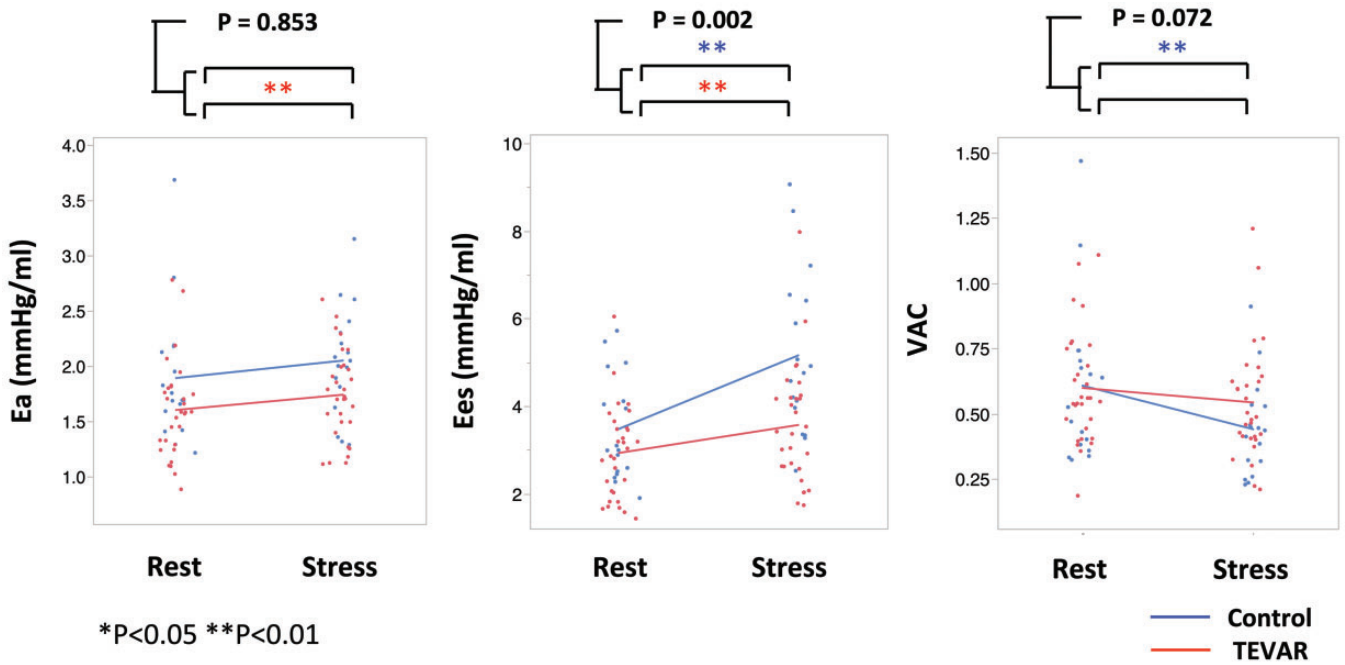

Figure 3: Change in ventricular–arterial interaction evaluated by stress echocardiography. Ea: arterial elastance; Ees: end-systolic elastance; TEVAR: thoracic endovascular aortic repair; VAC: ventricular–arterial coupling.

the stent graft on the results were not fully adjusted for. A second major limitation is selection bias associated with the preoperative condition of the aorta and heart. Although hypertensive patients were enrolled in the control group, baseline characteristics, including beta-blocker intake rate and echocardiographic parameters could influence the results. Therefore, the impact of TEVAR on the heart and aorta may not have been accurately evaluated. Preoperative and postoperative evaluation using the stress echocardiogram for patients with aortic diseases may be a more suitable option; however, patients with aortic diseases were contraindicated to a stress echocardiogram. Finally, the stress echocardiography cannot be performed on all patients and, consequently, cannot be used as a standard examination.

CONCLUSIONS

Exercise echocardiogram showed underlying influences of TEVAR on the interaction between the heart and aorta. Endovascular repair for thoracic aortic lesion may have the potential to suppress LV contractile capacity and enhance cardiac afterload during exercise.

SUPPLEMENTARY MATERIAL

Supplementary material is available at *EJCTS* online.

Conflict of interest: none declared.

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