

Immediate Impact of Prosthetic Graft Replacement of the Ascending Aorta on Circumferential Strain in the Descending Aorta

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WHAT THIS PAPER ADDS

The results of this study demonstrate graft induced increases in circumferential deformation and distensibility in the native descending aorta in the presence of unchanged haemodynamics and stroke volume after proximal aortic graft replacement, suggesting that replacing native aorta with a graft increases pulse pressure and energy propagation downstream to the graft. This observation may have important clinical implications and may be a mechanism contributing to the reported risk of aneurysm formation or expansion, dissection, or rupture of the aorta distal to graft.

Objectives: Prosthetic replacement of the ascending aorta (AA) can potentially modify energy propagation to the distal aorta and contribute to adverse aortic remodelling. This preliminary study employed intra-operative transoesophageal echocardiography (TOE) to assess the immediate impact of prosthetic graft replacement of the AA on circumferential strain in the descending aorta.

Methods: Intra-operative TOEs in patients undergoing AA graft replacement were analysed for circumferential strain, fractional area change (FAC), dimensions (end diastolic area [EDA], and end systolic area [ESA]) in the descending aorta immediately before and after graft replacement. Deformation was assessed via global peak circumferential aortic strain (CAS), together with pulse pressure corrected strain, time to peak strain (TTP), and aortic distensibility.

Results: Forty-five patients undergoing AA replacement with prosthetic graft (91% elective) were studied. Following grafting, descending thoracic aortic circumferential strain increased ($6.3 \pm 2.8\%$ vs. $8.9 \pm 3.4\%$, $p = .001$) paralleling distensibility ($5.7 [3.7-8.6] 10^{-3} \text{ mmHg}$ vs. $8.5 [6.4-12.4] 10^{-3} \text{ mmHg}$, $p < .001$). Despite slight increments in post graft left ventricular ejection fraction (LVEF) ($52.3 \pm 10.8\%$ vs. 55.0 ± 11.9 , $p < .001$), stroke volume was similar ($p = .41$), and magnitude of increased strain did not correlate with change in stroke volume ($r = -.03$, $p = .86$), LVEF ($r = .18$, $p = .28$), or pulse pressure ($r = .28$, $p = .06$). Descending aortic size (EDA $4 [2.7-4.6] \text{ cm}^2$ vs. $3.7 [2.5-5] \text{ cm}^2$, $p = .89$; ESA $4.3 [3.2-5.3] \text{ cm}^2$ vs. $4.5 [3.3-5.8] \text{ cm}^2$, $p = .14$) was similar pre- and post graft. In subgroup analysis, patients with cystic medial necrosis had a significantly higher post procedure CAS than patients with atherosclerotic aneurysms ($9.7 \pm 3.5\%$ vs. $7.0 \pm 2.3\%$, $p = .03$).

Conclusions: Prosthetic graft replacement of the AA increases immediate aortic circumferential strain of the descending aorta, particularly in patients with cystic medial necrosis. Our findings suggest that grafts augment energy transfer to the distal aorta, a potential mechanism for progressive distal aortic dilation and/or dissection.

Keywords: Strain, Echocardiography, Aortic aneurysm, Prosthetic grafts

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INTRODUCTION

Prosthetic graft replacement of the ascending aorta (AA) is widely used to treat patients with AA aneurysm and dissection. While AA replacement prevents dissection within the grafted segment, the long term risk of dilatation or dissection of the distal aorta persists after initial surgical repair.¹⁻⁵ One reason for this risk may stem from the impact of surgical grafts

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on aortic physiology: prosthetic grafts lack the normal distensibility of native aortic tissue, thereby losing the capacity to absorb part of the cardiac stroke volume transmitted into the aorta.⁶ This is known to induce changes proximal to the graft, including increased ventricular afterload and decreased coronary perfusion.⁷ Similarly, prior studies have shown that increased aortic compliance reduces left ventricular (LV) energy expenditure⁸ — supporting the notion that non-compliant ascending aortic grafts confer potentially deleterious sequelae with respect to both LV and descending aortic physiology. Whereas replacement of the AA with a prosthetic graft alters the diameter and mechanical properties of the aortic wall, the immediate impact of prosthetic replacement of the ascending aorta on pulsatile wall mechanics distal to aortic grafts is not well understood.

Transoesophageal echocardiography (TOE) enables intra-operative monitoring of cardiovascular function in patients undergoing aortic surgery, and is widely used to guide intra-operative surgical decision making. Given that TOE is a key component of surgical assessment, evaluation of novel TOE technologies is of broad relevance. New advances in echo speckle tracking technology have enabled rapid, accurate, angle dependent assessment of regional myocardial deformation.⁹ Using the technique of speckle tracking echocardiography (STE), a given region of interest can be tracked between time points to assess strain, an index of displacement.¹⁰ A key advantage of strain is it that can be derived from post-processing of standard images, enabling new insights to be derived from pre-existing data, including intra-operative TOE acquired prior to and following aortic graft implantation. Strain has been predominantly used to assess ventricular physiology, and has been shown to improve assessment of left and right ventricular systolic function compared with visual wall motion and ejection fraction (EF).^{11–14} Given that the aorta demonstrates pulsatile systolic expansion, strain imaging also holds the potential to assess dynamic alterations in aortic physiology by measuring circumferential displacement of the vessel wall between end diastole and peak systole.¹⁵

This preliminary study employed TOE to assess native descending thoracic aortic mechanics in relation to proximal aortic graft surgery, and to test the hypothesis that prosthetic graft replacement of the proximal aorta augments strain in the native descending aorta. Secondary variables of pulse pressure adjusted strain, time to peak strain, aortic areas (systolic and diastolic), fractional area change, and aortic distensibility index were also evaluated.

MATERIAL AND METHODS

Study population

This study was conducted with approval of the Weill Cornell Institutional Review Board, which approved the use of pre-existing clinical imaging data as were analysed for research purposes. The population comprised patients undergoing elective or urgent replacement of the AA (excluding those with associated arch or root aneurysms and cases of aortic dissections) with a prosthetic (polyethylene terephthalate; PET) graft, for whom intra-operative TOE was performed

immediately prior to (pre-cardiopulmonary bypass [CPB]) and immediately following prosthetic graft implantation (post-CPB), after protamine administration and achievement of stable haemodynamics. Aortic surgery was performed at Weill Cornell Medical College (New York, NY) between 2015 and 2018. Patients were selected for inclusion based on availability of TOE datasets inclusive of descending aortic short axis images of adequate quality to assess aortic deformation pre- and immediately post-surgical graft implantation.

For the current study, primary (DICOM) datasets were retrieved from digital archives and re-analysed (blinded to demographic as well as cardiac structural/functional indices) for the purpose of evaluating strain evidenced changes in distal aortic physiology following proximal grafting.

Image acquisition

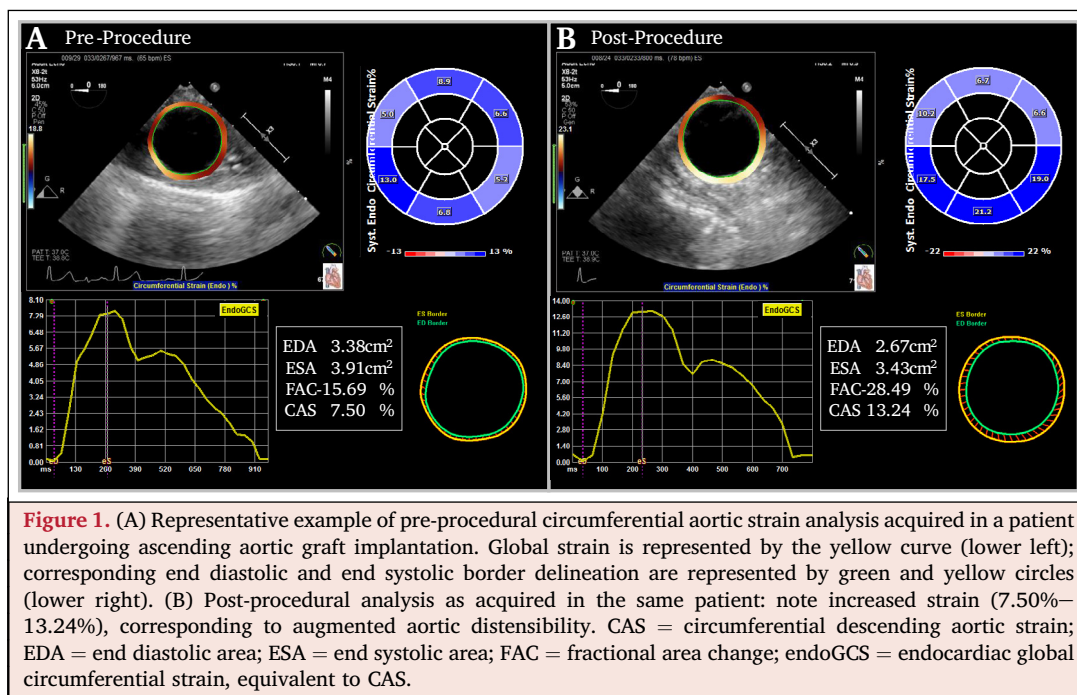
TOEs were acquired in accordance with European Society of Cardiology and American Society of Echocardiography (ESC/AASE) guidelines using commercially available equipment (IE33, Philips Medical Systems, Andover, MA, USA; EPIQ 7, Philips Medical Systems; and Vivid 7, GE Healthcare, Madison, WI, USA, ultrasound systems). All examinations included standard short axis views of the mid-descending aorta, which was targeted at the level of the LV outflow tract as acquired in a mid-oesophageal depth. Aortic imaging (before and after graft placement) was acquired at stable haemodynamic time points prior to and following CPB and protamine administration. Invasive blood pressure tracings at the approximate time of echocardiography were used to derive pulse pressure.

Image analysis

Aortic strain. Aortic size and deformation were assessed on short axis images acquired perpendicular to the long axis of the mid-descending thoracic aortic wall. Images were acquired at high temporal frame rates (51 ± 15 Hz). Commercially available, vendor independent, software (TomTec, Unterschleissheim, Germany) was then used for aortic strain analyses. In brief, multiple (15–20) seed points were manually placed at equidistant locations throughout the circumference of the aortic wall as assessed at aortic end systole. Automated speckle tracking analysis was then performed, such that seed points were tracked on a frame to frame basis throughout the cardiac cycle (end diastole to end diastole) (Fig. 1A, lower left hand panel). Software generated outputs were then visually inspected frame by frame to allow the operator to ensure adequate aortic wall border tracking. If computer generated analysis was deemed suboptimal (e.g. discordant with visualised aortic contours), seed points were manually adjusted to optimise aortic wall vessel tracking.

Consistent with prior studies for which strain has been employed to assess vascular physiology,^{15,16} the aortic circumference was partitioned into six, 60° segments, with the segmental results averaged to derive the following global indices of aortic mechanics:

- circumferential aortic strain (CAS) — peak circumferential deformation of the aortic wall between systole and



diastole (measured as the relative (%) aortic area difference between these two time points; $[(\text{end systole} - \text{end diastole})/\text{end diastole}] \times 100$

- pulse pressure adjusted CAS – CAS normalised for pulse pressure (PP): $(\text{CAS}/\text{PP})^{18}$
- time to peak (TTP) strain – calculated as time interval between minimum and maximum circumferential aortic strain.

In addition to the above strain related indices, analyses included quantification of aortic end systolic area (ESA) and end diastolic area (EDA). Aortic area measurements were used to calculate fractional area change $(\text{FAC} = [\text{ESA} - \text{EDA}]/\text{EDA})$, together with difference in cyclic aortic area changes between pre- and post-graft time points $([\text{ESA pre} - \text{EDA pre}] - [\text{ESA post} - \text{ESA post}])$. In addition, the aortic distensibility index was calculated via an established equation; $(\text{ESA} - \text{EDA})/\text{EDA} \times (\text{SBP} - \text{DBP})$, where SBP represents systolic blood pressure, DBP diastolic blood pressure, and EDA and ESA the area of the descending aorta at end systole and end diastole.¹⁹

Fig. 1 provides a representative example of study related aortic analyses performed using TOE datasets acquired pre- and post-proximal aortic grafting.

Left ventricular function and remodelling

LV size and systolic function were calculated via biplane Simpson's method in accordance with established guidelines;²⁰ end diastolic and end systolic chamber volumes were used to derive EF and corresponding functional parameters (stroke volume [SV], cardiac output [CO]).

Haemodynamic assessments

Invasive blood pressure was measured via peripheral radial arterial catheters, which were placed in accordance with

established clinical practice at Weill Corenell Medical Centre. Pre-procedure blood pressure was acquired after anaesthesia ready and before heparin administration/CPB from the electronic anaesthesia record (CompuRecord, Phillips Medical Systems); post-procedure measurements were obtained approximately 30 min after CPB to prior to chest closure at the approximate time of the TOE exam. The heart rate was obtained pre- and post-graft based on review of corresponding TOE images used for strain analysis.

Statistical analyses

The Shapiro–Wilk test was used to assess normality of all continuous variables. Normally distributed data were presented as mean \pm standard deviation and skewed variables as median with interquartile ranges. Categorical variables were reported as counts and percentages.

Differences between groups were assessed by the Student *t* test for normally distributed variables and Mann–Whitney U or Wilcoxon test for non-normally distributed variables. Correlation coefficients were used to test magnitude of associations between continuous variables. Two sided $p < .05$ was considered statistically significant. Inter- and intra-observer reliability was assessed by intraclass correlation coefficient (ICC) analysis calculated together with its corresponding 95% confidence interval (95% CI) on 20% of randomly selected patients in the total population. Sensitivity analysis for four subgroups were defined based on (1) pathological diagnosis findings (cystic medial necrosis vs. atherosclerotic), (2) dimension of the aneurysm (below vs. above or equal the median of 5.4 cm), (3) dimension of the implanted graft (below vs. above or equal the median of 28 mm), and (4) elective or urgent surgery. Pathological diagnosis was performed by an expert cardiovascular pathologist blinded to the study at the time of surgery. Statistical analysis was performed using SPSS 24.0 (SPSS Inc., Chicago, IL, USA).

RESULTS

Population characteristics

The population comprised 45 patients who underwent isolated elective or urgent replacement of the AA with a prosthetic graft. Table 1 reports clinical characteristics of study participants, including pathology, aneurysm size, graft size, risk factors related to aortic disease and indications for surgery.

Left ventricular performance indices

LV chamber size and function as quantified immediately prior to and following proximal aortic graft implantation are reported in Table 2. As shown, LV stroke volume was similar before and after the procedure ($p = .41$). Slight decrements in LV end systolic volume (42.2 ± 15.5 mL vs. 39.5 ± 18.6 mL, $p = .01$) were accompanied by a mild increase in post-procedure LVEF (52.3 ± 10.8 mL vs. 55.0 ± 11.9 mL, $p < .001$).

Regarding haemodynamic indices, Table 2 also shows that heart rate increased post-procedure (62 [56.7–74.2] to 80 beats/min [71–82], $p = .001$), but that invasive blood pressure indices were similar (all $p = \text{NS}$).

Graft induced aortic strain

Table 3 reports strain related variables as measured in the descending aorta together with conventional indices of aortic size. As shown, absolute global circumferential strain increased 1.4 fold, as did pulse pressure normalised strain and fractional area change between aortic end diastole and end systole (all $p < .001$): when assessed as a categorical variable, 89% (40/45) of patients manifested some increase in post-procedure global circumferential strain (vs. pre-procedure baseline), and strain increased by $\geq 50\%$ in nearly half (44% [20/45]) of patients. The magnitude of increased strain did not correlate with change in LV stroke volume ($r = .15$, $p = .36$), LVEF ($r = .18$, $p = .29$), or heart rate ($r = .03$, $p = .84$). As shown in Fig. 2,

magnitude of increased strain (both CAS and pulse pressure corrected CAS) correlated with the difference in absolute area change from pre-graft and post-graft status ($r = .56$ and $.72$ respectively, both $p < .001$).

Table 3 also demonstrates that increased native aortic strain was paralleled by increased aortic distensibility ($p \leq .001$). Proximal aortic graft induced alterations in distal native aortic distensibility paralleled the increase in pulsatile descending aortic cross sectional area from end diastole to end systole (Δ area = 0.2 cm^2 , $p < .001$). Regarding temporal indices, time to peak strain decreased post grafting ($p = .02$), consistent with post-procedure increments in heart rate. When differences between aortic pathology were considered, post-procedure global circumferential strain was significantly higher in patients with cystic medial necrosis than atherosclerotic disease ($9.7 \pm 3.5\%$ vs. $7.0 \pm 2.3\%$, $p = .03$). There was no difference in post procedure global circumferential strain when subgroups were compared based on median native aortic aneurysm size ($9.5 \pm 3.6\%$ vs. $8.0 \pm 3.0\%$, $p = .14$), median graft size ($9.5 \pm 3.4\%$ vs. $8.5 \pm 3.5\%$, $p = .35$), or elective vs. urgent surgery ($9.1 \pm 3.5\%$ vs. $6.2 \pm 1.4\%$, $p = .17$).

The intraclass coefficient for pre-procedure global circumferential strain showed good agreement for both inter- and intra-observer measurements (ICC = $.76$ [95% CI 0.04–0.94] and ICC = 0.78 [95% CI 0.11–0.95] respectively); post-procedure global circumferential strain measurements demonstrated excellent agreement (ICC = 0.95 [95% CI 0.80–0.98] and ICC = 0.85 [95% CI 0.27–0.97]) respectively for inter- and intra-observer measurements.

DISCUSSION

This is the first study to evaluate aortic distensibility in the descending thoracic aorta after prosthetic replacement of the AA using 2D STE. Key findings are that after prosthetic replacement of the AA, there is significant acute increase of circumferential strain and corrected peak global circumferential strain in the descending aorta, paralleling concomitant increases in absolute and fractional descending aortic area change as well as aortic distensibility. As stroke volume was unchanged post graft replacement and magnitude of increase in strain did not correlate with changes in LV function, the observed increase in circumferential strain distal to the graft and increased distension of the thoracic aorta are probably related to augmented energy propagation to the distal aorta due to the presence of a non-compliant proximal graft which cannot absorb forward stroke volume.

Regarding the mechanism of the findings, it should be noted that whereas this study did not assess compliance within AA grafts, previous studies have reported that prosthetic grafts have reduced compliance compared with the native aorta.²¹ Based on this, it is likely that graft induced impairments in vascular compliance reduce blood accumulation in the AA, resulting in augmented propagation of systolic flow (vs. physiological/pre-graft conditions) to the descending aorta. Consistent with this concept, compliance mismatch has been shown to impact on cardiovascular physiology both

Table 1. Baseline characteristics

Age, years	57.0 \pm 16.4
Male gender	27 (60.0%)
Family history of aneurysm	4 (8.9%)
Prior or current smoker	22 (48.8%)
Chronic pulmonary disease	4 (8.9%)
Pathology	
Atherosclerotic	9 (20%)
Cystic medial necrosis	32 (71.1%)
Unknown	4 (8.9%)
Diabetes mellitus	5 (11.1%)
Heart failure*	12 (26.6%)
Renal insufficiency†	2 (4.4%)
Hypertension	31 (68.9%)
Previous stroke	4 (8.9%)
Peripheral vascular disease	2 (4.4%)
Urgent surgery	3 (6.7%)
Ascending aorta aneurysm size – cm	5.5 \pm 0.8
Ascending aorta graft size – mm	27.2 \pm 2.4

Discrete indices reported as absolute number n (%), continuous indices as mean \pm standard deviation.

* New York Heart Failure Association Class ≥ 2 .

† Serum creatinine >1.5 mg/dL.

Table 2. Conventional imaging and haemodynamic variables

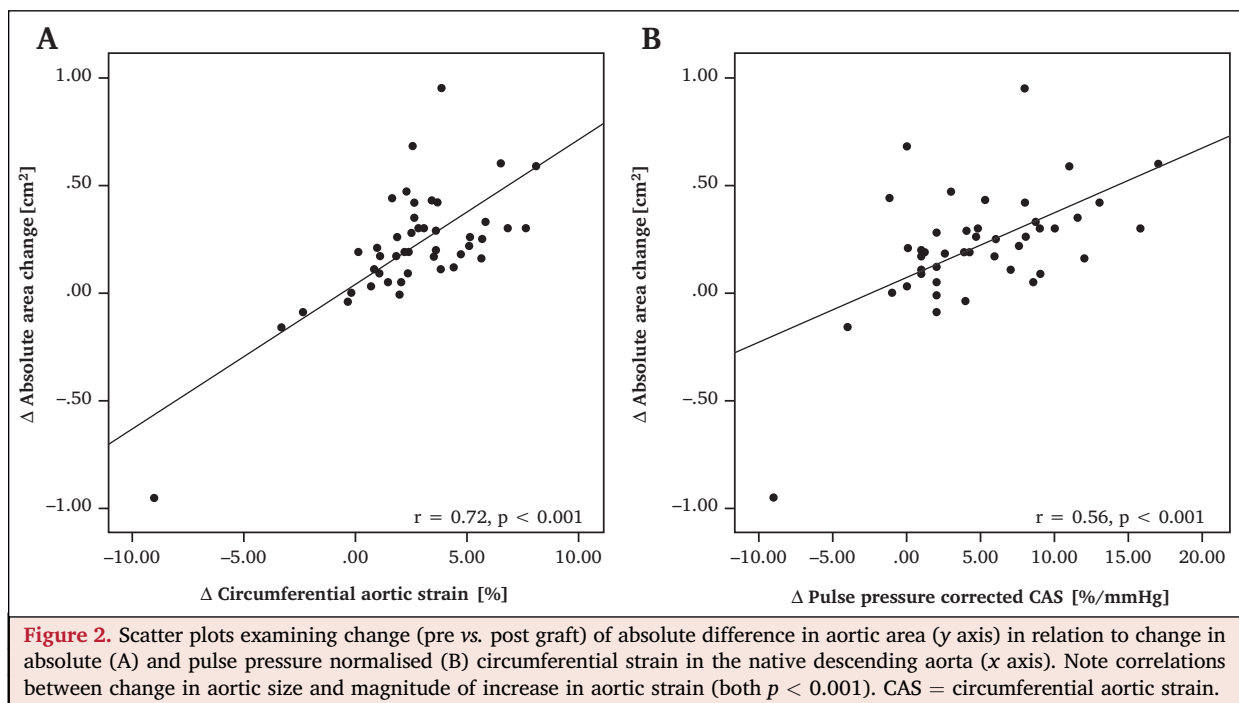
	Before graft placement	After graft placement	Δ	p
<i>LV function and chamber size</i>				
End diastolic volume, mL	81.1 (69.3–104.6)	82.1(65.9–96.4)	-5.3 (-13.2 to 4.7)	.06
End diastolic volume index mL/m ²	43.7 (36.4–52.2)	42.1 (33.7–51.1)	-2.9 (-6.3 to 2.5)	.07
End systolic volume, mL	42.2 ± 15.5	39.5 ± 18.6	-2.7 ± 10.5	.01
End systolic volume index mL/m ²	21.7 (16.1–27.3)	17.5 (12.6–26.9)	-2.2 (-5.0 to 1.58)	.02
Ejection fraction, %	52.3 ± 10.8	55.0 ± 11.9	2.7 ± 5	.003
Stroke volume, mL	44.3 (34.8–53.9)	43.9 (36.4–51.1)	-0.7 (-4.5 to 2.5)	.41
Cardiac output, L/min	3.1 (2.1–3.9)	3.4 (2.8–4.3)	0.42 (-0.04 to 1.06)	>.01
<i>Haemodynamic indices</i>				
Systolic blood pressure – mmHg	110.2 ± 9.9	109.5 ± 8.25	-0.6 ± 6.3	.37
Diastolic blood pressure – mmHg	59.1 ± 7.6	56.4 ± 9.7	-2.7 ± 9.6	.06
Pulse pressure – mmHg	51.1 ± 10.1	53.2 ± 8.9	2.1 ± 11.6	.18
Heart rate – bpm	62 (56.8–74.3)	80 (71–82)	11 (3.5–22.5)	.001

Normally distributed data are presented as mean ± standard deviation and skewed variables as median with interquartile ranges. Δ = area, change in area between systole and diastole; bpm = beats per minute; LV = left ventricular.

Table 3. Descending (native) aortic strain

	Pre	Post	Δ	p value
End diastolic area, cm ²	4 (2.7–4.6)	3.7 (2.5–5.1)	-0.02 (-0.4 to 0.6)	.89
End systolic area, cm ²	4.3 (3.2–5.3)	4.4 (3.3–5.8)	0.12 (-0.3 to 0.9)	0.14
Δ area, cm ²	0.4 (0.3–0.6)	0.7 (0.5–0.8)	0.2 (0.1–0.3)	<.001
Fractional area change, %	12.05 (8.0–17.3)	19.0 (12.7–22.1)	5.7 (2.9–9.2)	<.001
Global circumferential strain, %	6.3 ± 2.8	8.9 ± 3.4	2.6 ± 2.9	.001
Time to Peak Strain TTP, ms	281.4 ± 63.9	210.6 ± 49.9	-70.8 ± 85.5	<.001
Δ Area/TTP, cm ² /s	1.7 (1.1–2.3)	3.2 (2.3–5.2)	1.6 (1.1–2.6)	<.001
PP/TTP, mmHg/s	180.4 (149.5–216.7)	259.1 (221.4–299.7)	67.5 (16.4–131.2)	<.001
Pulse pressure corrected strain (CAS/PP)	12.7 ± 5.3	17.5 ± 7.7	4.7 ± 5.1	.001
Distensibility, 10 ⁻³ mmHg	5.7 (3.7–8.6)	8.5 (6.4–12.4)	2.9 (1.2–5.8)	<.001

CAS = circumferential descending aortic strain; PP = pulse pressure; TTP = time to peak.



proximal and distal to the graft. Increased afterload, reduced coronary perfusion, and LV hypertrophy have been reported proximal to, and increased aortic impedance distal to, prosthetic grafts.^{6,22} Increased energy propagation has been hypothesised to be a contributor to pseudoaneurysm formations and distal aortic adverse remodelling.⁷ Animal models have linked compliance mismatch between prosthetic endovascular grafts and the native descending aorta to post-procedure complications such as dissection, aneurysmal dilatation, and rupture distal to the repair.^{6,23,24} Nauta et al.²⁵ assessed the impact of thoracic endovascular aortic repair on aortic strain using cardiac gated computed tomography and found that implantation of the prosthetic stent graft was associated with increased pulsatile longitudinal strain and circumferential strain proximal and distal to stented aortic segments. The authors hypothesised that this may contribute to stent graft related complications such as retrograde dissection, aneurysm formation, and rupture.

Arterial distensibility has been employed increasingly as an index to study aortic physiology, including studies in which altered aortic flow propagation or vessel wall properties have been linked to changes in aortic resistance. Consistent with this concept, the group employed cine cardiac magnetic resonance imaging to demonstrate decreased distensibility of the descending aorta among patients with Marfan syndrome vs. normative controls ($p < .001$), paralleling a similar trend when Marfan patients were compared with those with bicuspid aortic valve ($p = .09$).²⁶ Prior studies have employed echo to assess vascular stiffness in the descending and abdominal aorta.^{18,26–28} For example, among a cohort of hypertensive patients, echo derived circumferential strain has been shown to be impaired compared with controls.²⁹ Two dimensional STE strain imaging of the aorta and vascular structures has also been used to predict aortic biomechanics. Emmott et al.³⁰ evaluated patients undergoing aortic resection and found that TOE derived stiffness from circumferential strain correlated well with aortal wall biomechanics and histopathology. Whereas strain can be quantified using an array of gating imaging techniques, a key advantage of echocardiography derives from its wide availability and ability to achieve high quality imaging in critically ill patients unable to tolerate closed space environments and breathing required for other imaging techniques such as computed tomography and magnetic resonance imaging. In this context, the findings are of broad relevance with respect to peri-operative aortic assessment.

Circumferential strain potentially may be influenced by intrinsic aortic wall properties, higher pulse pressure, and LV stroke volume.³¹ Baseline CAS was obtained in all patients and accounted for intrinsic aortic properties. Given equal arterial stiffness of a vessel, a higher pulse pressure should result in higher strain values.¹⁷ Therefore, a corrected circumferential aortic strain (CAS) was calculated (global CAS/pulse pressure). It has also been shown that vessel wall properties and systolic flow impact on global circumferential strain in the AA, and that LV stroke volume is a key determinant of altered aortic strain.³⁰ This effect was tested by

calculating stroke volume before and after graft replacement. There was no significant change in stroke volume between these time points, suggesting that changes in aortic deformation are due to increased pulse propagation after graft placement rather than an acute increase in LV stroke volume.

Several limitations should be acknowledged. The sample size of the study is limited and haemodynamic measurements may not have been recorded at the exact time of the echo image acquisition. Similarly, intra-operative medications that could affect cardiovascular haemodynamics and thus impact strain were not accounted for. On the other hand, it should be noted that blood pressure and LV stroke volume did not differ significantly between time points, supporting the notion that aortic loading conditions were not responsible for observed strain differences. Additionally, depth of image acquisition in the descending aorta may have differed slightly between examinations; this source of variability could potentially have affected observed differences in strain. Given that strain quantification is a semi-automated post-processing technique that can be impacted by image orientation and edge definition, limitations in image quality, and analytic software could have impacted the analyses. While the data extend on prior literature which has used conventional strain software similarly (designed for the LV) to assess aortic mechanics,¹⁷ it is possible that tailored aortic strain software would have provided greater precision with regard to assessment of graft induced changes in aortic physiology and possible that strain values would have differed using different echo equipment, concepts that should be tested in future research. Finally, the study did not entail serial imaging to assess longitudinal changes in aortic strain induced by surgical prosthetic graft implantation, or the impact of peri-operative changes in native aortic strain on risk of aortic dilation and/or dissection. Future, larger scale, longitudinal research is warranted to test these concepts in a prospective manner, including whether the predictive value of aortic strain varies based on the aetiology of aortic pathology.

In conclusion, results of this study demonstrate immediate graft induced increases in circumferential deformation and distensibility in the native descending aorta in the presence of unchanged haemodynamics and stroke volume after aortic graft replacement, suggesting that replacing native aorta with a graft increases the pulse pressure and energy propagation downstream to the graft. This may be a mechanism contributing to the reported risk of aneurysm formation or expansion, dissection, or rupture of the aorta distal to the graft.¹ Applied clinically, it is possible that patients with marked augmentation in aortic strain following grafting warrant close surveillance for changes in distal aortic remodelling (e.g. aneurysm expansion) or clinical events. The findings also highlight the need for further studies to test the impact of altered prosthetic graft design and novel surgical strategies (informed by pre-procedural imaging) to reduce post-operative strain in the descending aorta. Future prospective clinical research is warranted to test the longitudinal impact of graft induced alterations in native aortic mechanics among patients with sporadic and genetically mediated aortic aneurysms, as well as to test whether use of prosthetic grafts with increased

elasticity reduces native aortic strain and improves long term clinical outcomes for patients undergoing ascending aortic graft surgery.

CONFLICT OF INTEREST

None.

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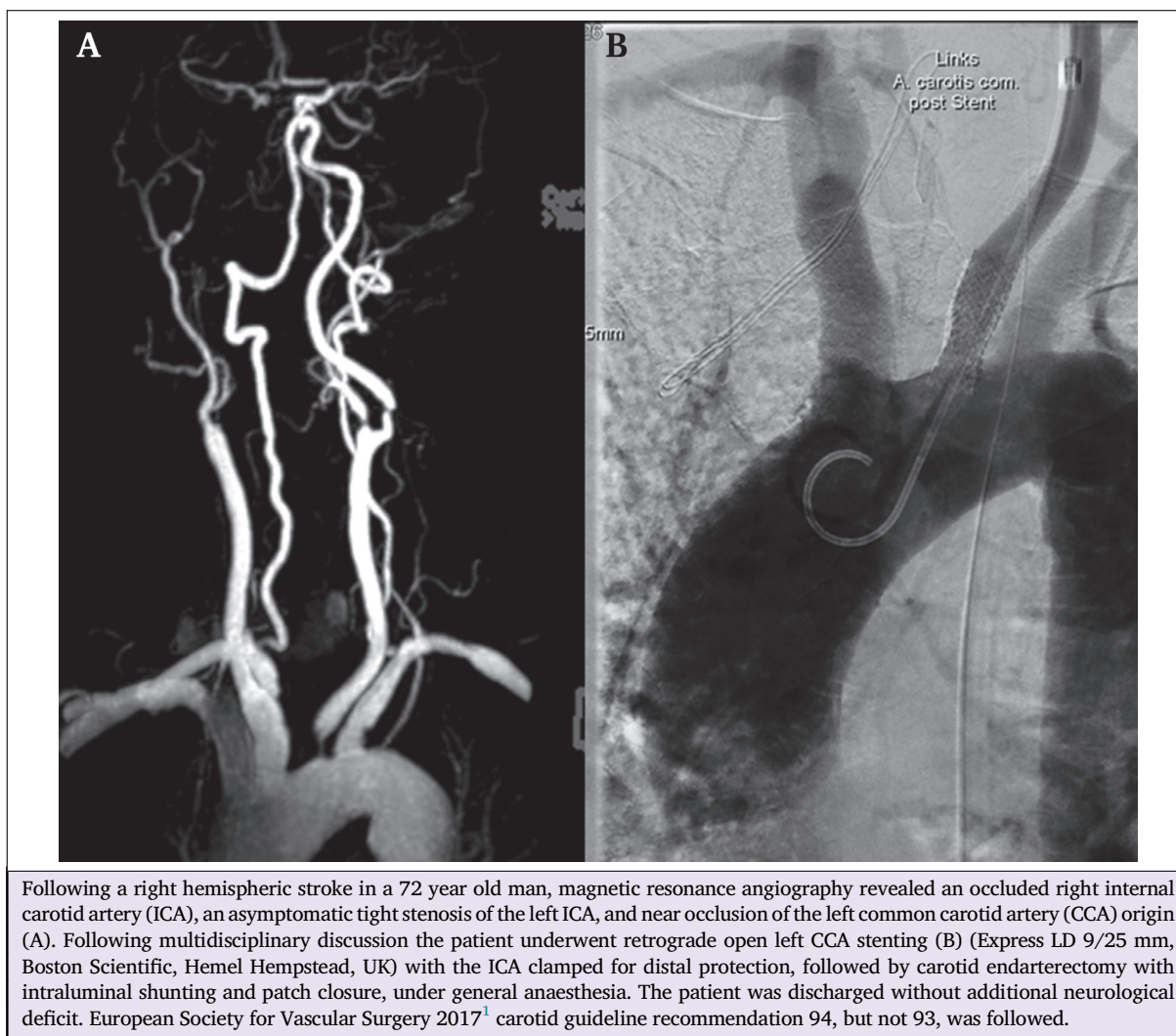
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COUP D'OEIL

Hybrid Management of Tandem Carotid Arterial Stenoses with Contralateral Carotid Occlusion

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