Changes in intraoperative aortic strain as detected by ultrasound elastography in patients following abdominal endovascular aneurysm repair

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ABSTRACT

Objective: Predicting success after endovascular aneurysm repair (EVAR) of abdominal aortic aneurysms (AAAs) relies on measurements of aneurysm sac regression. However, in the absence of regression, morphometric analysis alone is insufficient to reliably predict the successful remodeling of AAAs after EVAR. Biomechanical parameters, such as pressure-normalized principal strain, might provide useful information in the post-EVAR AAA assessment. Our objective was to assess the feasibility of our novel ultrasound elastography (USE) technique to detect changes in the aortic wall principal strain in patients who had undergone EVAR and determine the temporal nature of the biomechanical changes in the aorta.

Methods: USE images were obtained from patients undergoing elective EVAR intraoperatively, immediately before and after endograft implantation, and at their 30-day follow-up. The maximal mean principal strain ($\varepsilon_{r,PP}$) for each scan was assessed using our novel technique, which uses a finite element mesh to track the frame-to-frame displacements of the aortic wall over one cardiac cycle. The $\varepsilon_{r,PP}$ was then divided by the pulse pressure at the time of the scan to produce a pressure-normalized strain measurement ($\varepsilon_{r,PP}$/PP), a surrogate for tissue stiffness. Paired $t$ tests were used to compare the pre- and postoperative $\varepsilon_{r,PP}$/PP and the postoperative and 30-day $\varepsilon_{r,PP}$/PP. Patient 30-day sac regression and endoleak data were collected by a review of 30-day follow-up computed tomography scans.

Results: USE analysis of the data from 12 patients demonstrated a significant reduction in aortic wall $\varepsilon_{r,PP}$/PP (average, 0.191% ± 0.09%/kPa vs 0.087% ± 0.04%/kPa; $P$ = .002) immediately after graft implantation, with a nonsignificant change in the $\varepsilon_{r,PP}$/PP (0.091% ± 0.04%/kPa vs 0.102% ± 0.05%/kPa; $P$ = .47) from postoperatively to 30-day follow-up. This represents an average 46.5% reduction after stent placement, with a nonsignificant 18.1% increase at 30-day follow-up. All the patients showed sac stability, except for two patients who had demonstrated 7.3-mm and 6.8-mm sac regressions.

Conclusions: Our analysis has demonstrated that the presented USE technique is a feasible method for detecting significant reductions in aortic $\varepsilon_{r,PP}$/PP intraoperatively after EVAR. We found that patients undergoing EVAR will experience large reductions in the $\varepsilon_{r,PP}$/PP intraoperatively after graft implantation, with stabilization found at their 30-day follow-up. These preliminary data have shown that an intraoperative $\varepsilon_{r,PP}$/PP reduction could be a promising correlate of post-EVAR aneurysm remodeling. Our results have also suggested that endograft design likely plays a large role in determining the aneurysm biomechanical changes immediately after implantation. (J Vasc Surg Cases Innov Tech 2022;8:762-9.)

Keywords: Abdominal aortic aneurysm; Elasticity imaging techniques; Endovascular procedures; Ultrasonography
understood that aneurysm sac regression is a marker of better postoperative outcomes, the mechanism by which regression occurs has not yet been definitively established. Additionally, the appropriate treatment of patients in the absence of sac regression is less clear as aneurysm sac stability has been shown to be less benign than previously thought. It has also been shown and stated in the Society for Vascular Surgery United States guidelines that the risk of rupture can remain after EVAR even in the absence of sac expansion or an endoleak. These studies suggest that assessment of the AAA diameter and growth alone might be insufficient to determine the rupture risk after EVAR and that remodeling is likely more complex than simple morphometric analysis, involving a combination of both physical and biologic processes. Additionally, although sac regression has been associated with better outcomes, we have no well-established predictors that can be used directly after implantation to assess whether sac regression will occur.

To address these limitations, multiple studies have investigated potential biomechanical markers of AAA pathology and have demonstrated that a decreased elastic modulus and decreased beta stiffness (both measures of AAA wall stiffness) are associated with increased rupture risk. Thus, AAAs prone to rupture will show decreased stiffness and increased compliance compared with AAAs not prone to rupture. These biomechanical parameters can be applied to assess a patient’s risk of post-EVAR rupture, with the hypothesis that an increased modulus after EVAR will be indicative of a decreased risk of rupture and beneficial AAA collagen remodeling with sac regression. However, traditional imaging methods cannot provide insight into these biomechanical changes occurring in the aortic wall, and novel, noninvasive methods are needed to assess these properties.

In the present study, we used US elastography (USE) to examine the biomechanical changes of the aortic wall based on measures of pressure-normalized AAA wall strain (εr+PP). Because modulus is defined by the slope of a stress-strain curve (stress/strain), εr+PP, which is a measurement of the mean strain normalized by dividing by the pulse pressure (stress), can represent the proportional inverse of the elastic modulus. Therefore, as the stiffness (elastic modulus) decreases in the aneurysm, the εr/PP will increase. A beneficial reduction in the εr+PP would, thus, be proportional to a beneficial increase in the elastic modulus and can serve as a surrogate to predict for future rupture or remodeling. εr/PP can be easily measured using USE, which calculates this parameter from the displacement of the aortic wall during physiologic pulsation. Thus, this is a quantitative method analogous to surgeons placing their hand on a patient’s abdomen and feeling for the degree of aneurysm pulsatility before and after endograft placement.

We hypothesize that USE can be used intraoperatively to predict whether aneurysm remodeling would occur in the future. We also believe this could be a future modality to assist in the analysis of AAA after EVAR in the absence of sac regression when treatment and surveillance decisions might be less clear. Therefore, the aim of the present pilot study was as follows: (1) to demonstrate the feasibility of an intraoperative USE method to determine the εr+PP in patients undergoing EVAR; and (2) to determine the temporal biomechanical changes that occur within a short follow-up interval after EVAR.

METHODS

Study design. We performed a single-center, descriptive, pilot study of patients undergoing EVAR for nonruptured infrarenal AAAs. The study included three USE imaging examinations, two intraoperatively before and after graft implantation and the third at a 30-day follow-up. The USE images were analyzed, and the εr+PP was calculated to assess the differences pre- and postoperatively and at 30 days after the procedure. Additional demographic and imaging information was recorded from the patients’ electronic medical records. The institutional review board office of the University of Rochester reviewed and approved the present study and informed consent process.

Patient recruitment. All the enrolled patients were recruited through the vascular surgery division of the University of Rochester Medical Center surgery department from June 2021 to January 2022. Patients were recruited based on a diagnosis of a nonruptured, infrarenal AAA requiring EVAR as determined by one of the division’s vascular surgeons. The exclusion criteria included any vulnerable populations; patients with human immunodeficiency virus, tuberculous, or coronavirus disease 2019; patients who had required emergent EVAR for a ruptured AAA; and patients who had experienced complications during the procedure, including rupture, conversion to open repair, or death. All enrolled patients provided written informed consent before EVAR. All enrolled patients met the strict instructions for use criteria for surgeon-selected endograft implantation.

US examination protocol. US imaging was conducted using either the Ultrasonic Sonix-Touch US System (BK Medical, Burlington, MA) or the Ultrasonic Sonix-Tablet US System and an Ultrasonic C7-3/50 convex transducer. B-mode US at a frequency of 5 MHz was used to visualize the abdominal aorta, in transverse orientation, at the point of the maximal diameter, as determined by the sonographer. The sector and depth settings were set to maximize the recorded frame rate to ≥50 frames per second.

The day of their procedure, the patients underwent intraoperative imaging. A 10-second breath hold was used, with a ventilation hold on the ventilator or...
The user-defined abdominal aortic wall, to track frame-to-frame displacements for a full cardiac cycle to measure the $\varepsilon_{r+}/PP$.

The imaging data were analyzed by the study team. For each imaging examination, the radiofrequency data were converted to a B-mode cine loop, and the interpreter isolated the frames of one full cardiac cycle with the most maximal displacement during pulsation and an absence of transducer probe movement, patient movement, or respiratory displacement. The onset of systole was chosen according to the frame that showed the smallest luminal area directly before aortic expansion, and the end of diastole was determined using the same method after maximum deformation of the aorta. Once the frames had been chosen, the interpreter marked the borders that would constitute the region of interest for finite element mesh construction, with the first border representing the lumen of the vessel and the second border the outer wall of the vessel (Fig 1). Notable thrombus was included in the region selection, because it has been shown that thrombus affects strain and modulus calculations and can be difficult to distinguish from the aortic wall using US.19 The displacement of each node within this mesh was then tracked from frame to frame to calculate the principle strain of each element within the mesh. The average strain of all the elements within the mesh were then calculated to give the mean principal strain for a given frame. The mean principal strains over each frame were then graphed (Fig 2), and the maximal mean principal strain ($\varepsilon_{r+}$) was determined. Next, parametric imaging was used to show the regional principal strain within the entire aorta in the frame of the $\tau_{pp}^+$ (Fig 3). The $\tau_{pp}^+$ was then divided by the pulse pressure taken at the time of the scan to produce the $\varepsilon_{r+}/PP$. From the beginning of the scan to determination of the final $\varepsilon_{r+}/PP$ value required 30 to 60 minutes.
Additional data collection. The demographic data (eg, age, sex, race, ethnicity, smoking status, body mass index), medical history (eg, family history of AAAs, hypertension, hyperlipidemia, diabetes mellitus, cardiac arrhythmia, coronary artery disease, percutaneous coronary intervention, open heart surgery, chronic obstructive pulmonary disease, chronic kidney disease, stroke, hepatic disease, hypercoagulability, cancer), medications (eg, statin, angiotensin-converting enzyme inhibitor, angiotensin receptor blocker, beta-blocker, antiplatelet agent, anticoagulant drug, tacrolimus, cyclosporine), 30-day follow-up data (eg, endoleak, endograft complications), and intraoperative data (eg, endograft type used, presence of endoleak at case conclusion) were all obtained from the patients’ electronic medical records. The presence of endoleaks or complications at case conclusion were assessed using the completion angiogram. The presence of endoleaks or complications at 30-day follow-up were assessed using the 30-day CTA. The AAA sizes were recorded from the preoperative and 30-day CTA scans. The AAA sizes were determined by the study team using the short axis of the transverse aorta at the point of maximal diameter. In accordance with the Society for Vascular Surgery reporting standards, the interobserver and intraobserver variability of aneurysm diameter measurements on CTA scans is 2 to 5 mm, and, as such, 5 mm should represent a significant diameter change.20,21 In the present study, we considered aneurysm sac regression or expansion to be a change in the diameter of $\geq$5 mm from the preoperative CTA to the 30-day CTA.

Statistical analysis. The statistical analysis consisted of two-tailed paired t tests between the preoperative $\overline{P_{r}}$/PP and postoperative $\overline{P_{r}}$/PP and between the postoperative $\overline{P_{r}}$/PP and 30-day $\overline{P_{r}}$/PP. A two-tailed paired t test was also used to compare the differences between the preoperative and 30-day AAA sizes. All statistical analyses were conducted using Microsoft Excel, version 2111 (Microsoft Corp, Redmond, WA). The level of significance was determined by a P value $<$ .05.

RESULTS
A total of 15 patients had provided written informed consent between June 2021 and December 2021. Of the 15 patients, 3 were excluded from the present study because of the poor quality of the USE examinations related to an excessive body mass index or difficult bowel anatomy, which had obscured the image resolution. Thus, the data from 12 patients were analyzed pre- and postoperatively, and the data from a subset of 9 patients were analyzed completely through their 30-day follow-up. The patient demographic data are listed in Table I.

Pressure-normalized strain results. Analysis of the average $\overline{P_{r}}$/PP showed a significant reduction immediately after endograft implantation (average, 0.191% ± 0.09%/kPa vs 0.087% ± 0.04%/kPa; P = .002). The average percent change from before to after implantation was 46.5%. In the subset of nine patients with follow-up CTA scans available, no significant change was found in the $\overline{P_{r}}$/PP between postimplant scans and 30-day follow-up scans (average, 0.091% ± 0.04%/kPa vs 0.102% ± 0.05%/kPa; P = .47; Fig 4). We found that all aneurysms continued to show some low level of strain.
after EVAR and that this strain was heterogeneous around the circumference of the aortic wall, as shown by parametric imaging (Fig 3).

Follow-up clinical results. Of the nine patients with analysis completely to their 30-day follow-up, two had had type II endoleaks on the completion angiogram at case conclusion. In one of these patients, the endoleak had resolved on the 30-day CTA, with a 76.7% reduction in the $\epsilon/PP$ from postoperatively to 30 days. The other had a continued type II endoleak with an additional endograft kink found on the 30-day CTA, with a 42.8% increase in the $\epsilon/PP$ from postoperatively to 30 days. Three other patients had type II endoleaks found on the 30-day CTA without previous endoleak findings on the completion angiogram, with an average 32.4% increase in the $\epsilon/PP$ from postoperatively to 30 days. Thus, four patients had either a type II endoleak or a graft complication found on the 30-day CTA, with an average $\epsilon/PP$ increase of 35.0% from postoperatively to 30-day follow-up. No type I or type III endoleaks were found. Four patients had no endoleaks or complications found on the completion angiogram or follow-up CTA. These patients had an average $\epsilon/PP$ increase of 25.0% from postoperatively to the 30-day follow-up.

All but two of the patients demonstrated AAA sac stability on the 30-day CTA. The average sac diameter changes at 30 days showed a nonsignificant decrease (61.6 $\pm$ 19.3 mm vs 59.5 $\pm$ 17.7 mm; $P = .12$). Two patients had sac regression. One patient was found to have a regression of 7.3 mm with a $\epsilon/PP$ decrease of 75.7% from pre- to postoperatively, but an increase of 11.9% from postoperatively to their 30-day follow-up. The second patient was found to have a regression of 6.8 mm, with a 61.1% decrease in the $\epsilon/PP$ from pre- to postoperatively and a 52.1% increase in the $\epsilon/PP$ from postoperatively to 30 days (Table II).

Table I. Patient characteristics (N = 12)

| Variable                        | Value          |
|---------------------------------|----------------|
| Demographics                    |                |
| Age, years                      | 75.0 $\pm$ 13.1|
| Female sex                      | 3 (25)         |
| Non-White race                  | 0 (0)          |
| Non-Hispanic ethnicity          | 12 (100)       |
| Smoking status                  |                |
| Current                         | 3 (25)         |
| Former                          | 6 (50)         |
| Never                           | 3 (25)         |
| BMI, kg/m$^2$                    | 26.0 $\pm$ 6.2 |
| Medical history                 |                |
| Family history of AAA           | 3 (25)         |
| Hypertension                    | 9 (75)         |
| Hyperlipidemia                  | 11 (91.7)      |
| Diabetes mellitus               | 1 (8.3)        |
| Cardiac arrhythmia              | 1 (8.3)        |
| CAD                             | 2 (16.7)       |
| CHF                             | 1 (8.3)        |
| PCI                             | 2 (16.7)       |
| Open heart surgery              | 1 (8.3)        |
| COPD                            | 2 (16.7)       |
| CKD                             | 0 (0)          |
| Stroke                          | 1 (8.3)        |
| Hepatic disease                 | 0 (0)          |
| Hypercoagulability              | 0 (0)          |
| Cancer                          | 6 (50)         |
| Medications                     |                |
| Statin                          | 11 (91.7)      |
| ACE inhibitor                   | 2 (16.7)       |
| ARB                             | 2 (16.7)       |
| Beta-blocker                    | 3 (25)         |
| Antiplatelet                    | 7 (58.3)       |
| Anticoagulation                 | 2 (16.7)       |
| Tacrolimus                      | 0 (0)          |
| Cyclosporine                    | 0 (0)          |
| Intraoperative data             |                |
| Type of endograft               |                |
| Excluder stent graft$^a$         | 10 (83.3)      |
| TREO stent graft$^b$             | 2 (16.7)       |

AAA, Abdominal aortic aneurysm; ARB, angiotensin receptor blocker; BMI, body mass index; CAD, coronary artery disease; CHF, congestive heart failure; CKD, chronic kidney disease; COPD, chronic obstruction pulmonary disease; PCI, percutaneous coronary intervention.

Data presented as mean $\pm$ standard deviation for continuous variables and number (%) for categorical variables.

$^a$W.L. Gore & Associates, Flagstaff, AZ.

$^b$Terumo Corp, Tokyo, Japan.
DISCUSSION

Main results. It has been accepted that AAA tissue is stiffer and experiences less baseline strain compared with normal aortic tissue. However, evidence has shown that AAAs that are more apt to rupture will show decreased stiffness compared with nonruptured AAAs. Wilson et al demonstrated that a 10% decrease in aortic wall elastic modulus conferred a 28% increase in the risk of rupture and that a reduction in elastic modulus and beta stiffness significantly reduces the time interval to AAA rupture. Therefore, a theory is that although AAAs will be stiffer than normal aortic tissue, at a certain point, the stress on the tissue will overcome its strength, leading to failure of tissue remodeling and resulting in less stiffness and increased compliance before rupture. These biomechanical markers can be monitored using noninvasive imaging and can be used in the assessment of AAAs after EVAR to predict the success or failure of the repair.

In the present study, we sought to better understand the temporal changes of these biomechanical principles in the setting of EVAR using USE to measure the pressure-normalized strain ($\varepsilon_{PP}$) changes in the aortic wall before and after stent placement. We found that patients experienced significant decreases in aortic $\varepsilon_{PP}$ immediately after EVAR, without a continued decrease in the $\varepsilon_{PP}$ in the subset of patients with 30-day follow-up USE scans available.

Our findings are in line with those from most of the previous studies examining the biomechanical changes in the aorta after EVAR. Long et al using tissue Doppler imaging, found an increase in the elastic modulus and beta stiffness (proportional to a decrease in the $\varepsilon_{PP}$) from pre- to postoperatively (within 1 month). Malina et al measuring the pulsatile wall motion (PWM), found a reduced PWM of as high as 70% at 1 month after EVAR (similar to a finding of a decreased $\varepsilon_{PP}$, because strain is highly related to the PWM). Breken et al using their custom regional linear strain algorithm, also found a decrease in strain from pre- to postoperatively (2 days), with a relative reduction in both maximum strain and peak circumferential average strain of 41% and 68%, respectively.

Additionally, the finding of a stable $\varepsilon_{PP}$ from postoperatively to 30-day follow-up, determined by nonsignificant increases, was similar to previous findings. Thus, it seems that the aortic wall undergoes relatively small amounts of remodeling within a short follow-up and that most of the protection to the aortic wall will be provided by the endograft itself. Further studies and, potentially, more direct methods of biomechanical remodeling testing are required to confirm these ideas. Also, future studies are needed to confirm that no additional drastic strain reduction occurs after 30 days due to continued remodeling at further follow-up points.

We found a residual amount of strain in all patients, although at very low levels, after EVAR. This finding

Table II. Pressure-normalized strain, abdominal aortic aneurysm (AAA) size, and endoleak data for patients undergoing ultrasound elastography (USE)

| Pt. No. | Preop $\varepsilon_{PP}$, %/kPa | Postop $\varepsilon_{PP}$, %/kPa | 30 Days $\varepsilon_{PP}$, %/kPa | Difference, % change | AAA size, mm | Endoleak information |
|---------|-------------------------------|-------------------------------|-------------------------------|---------------------|-------------|----------------------|
| 1       | 0.126                         | 0.108                         | 0.154                         | $-14.54$            | 53.7        | $-2.2$ Type II endoleak on completion angiogram with continued leak and endograft kink on 30-day CTA |
| 2       | 0.144                         | 0.123                         | 0.133                         | $-14.57$            | 51.8        | $-0.5$ None |
| 3       | 0.349                         | 0.127                         | 0.188                         | $-63.73$            | 54.6        | $-3.8$ Type II endoleak on 30-day CTA |
| 4       | 0.172                         | 0.034                         | $-79.96$                     | $-14.57$            | 40.9       | $-3.7$ Type II endoleak on 30-day CTA |
| 5       | 0.345                         | 0.066                         | $-80.97$                     | $-14.57$            | 50.1        | $-3.7$ Type II endoleak on 30-day CTA |
| 6       | 0.172                         | 0.036                         | 0.050                         | $-79.22$            | 89          | $-1.9$ Type II endoleak on completion angiogram with resolution on 30-day CTA |
| 7       | 0.123                         | 0.129                         | 0.030                         | $-15.10$            | 100.3       | $-6.8$ None |
| 8       | 0.199                         | 0.048                         | 0.054                         | $-75.67$            | 56.8        | $-7.3$ None |
| 9       | 0.283                         | 0.129                         | $-54.32$                     | $-14.57$            | 100.3       | $-6.8$ None |
| 10      | 0.147                         | 0.057                         | 0.087                         | $-61.07$            | 54.4        | $-0.3$ None |
| 11      | 0.113                         | 0.108                         | 0.138                         | $-5.46$             | 43.6        | $-0.7$ Type II endoleak on 30-day CTA |
| 12      | 0.122                         | 0.081                         | 0.088                         | $-33.88$            | 14.57       | $-5.46$ None |

CTA. Computed tomography angiography; $\varepsilon_{PP}$, pressure-normalized strain measurement. Postop, postoperatively. Preop, preoperatively. Pt. No., patient number.

* A negative percent difference indicates a decrease in $\varepsilon_{PP}$, and a positive percent difference, an increase in $\varepsilon_{PP}$.

* A negative AAA size reduction indicates a decrease in AAA size.
suggests that even after EVAR, a small amount of force will be transferred through the endograft to the aortic wall of the aneurysm sac. This is further complicated by postprocedure thrombus formation in the aneurysm sac, which could explain the accompanied finding of small increases in the \( \frac{\sigma_{PP}}{PP} \) at the 30-day follow-up. Increased thrombus formation would allow for more effective transfer of force from physiologic pulsation through the combined endograft and sac thrombus to the aortic wall. This also highlights that the type of endograft used and the graft material likely play a tremendous role in aortic wall remodeling after EVAR because certain endograft architecture could maximally mitigate the transfer of these forces.

Two major limitations of previous studies investigating the biomechanical changes of the aorta after EVAR were the assumed wall homogeneity and the linearity of the measurements. The analyses reported by Long et al\(^\text{25}\) and Malina et al\(^\text{26}\) involved the selection of two points on opposing sides of the vessel, with subsequent linear displacement measurements of those points. This method assumes an axisymmetric tube and ignores the regional, asymmetric variation of the tissue properties in the aorta. This is concerning because a main contributor to stress on the AAA wall is the asymmetry of the vessel.\(^\text{25}\) The methods outlined by Brekken et al\(^\text{27}\) provide additional regionality to their measurements. However, as they confirmed, one of their study’s limitations was the measurement of only the linear strain of the vessel, which physiologically experiences strain from forces in multiple directions.

Therefore, for our imaging algorithm, we used a finite element mesh analysis to account for the asymmetry of the physiologic aorta and for the strain imposed on the wall in multiple directions.\(^\text{16}\) This not only allows for more accurate biomechanical measurements, but also allows us to better analyze the regional strain variations of the vessel. Using our parametric analysis methods we were able to confirm that \( \frac{\sigma_{PP}}{PP} \) is heterogeneous around the circumference of the vessel wall in all patients (Fig 3). This finding further emphasizes the idea that the asymmetry of the aortic vessel will result in large variations in the regional strain measurements and most likely plays a large part in the risk of rupture.\(^\text{22,28}\)

**Secondary clinical results.** Of our nine patients with data analyzed through their 30-day follow-up, three had had no evidence of an endoleak at case conclusion but were found to have a type II endoleak at follow-up, one was found to have a type II endoleak at case conclusion with persistence at follow-up (and an endograft kink), and one had had a type II endoleak at case conclusion with resolution at follow-up. In all five cases, the \( \frac{\sigma_{PP}}{PP} \) increased or decreased as expected (Table II). The patients with a type II endoleak at follow-up had slightly greater \( \frac{\sigma_{PP}}{PP} \) increases from post-operatively to 30 days (35% increase) compared with those without an endoleak at 30 days (25% increase). The patient with endoleak resolution was found to have a massive 76.7% reduction in their aortic wall \( \frac{\sigma_{PP}}{PP} \). Although interesting, these findings simply highlight the potential for our method to detect endoleaks. However, the sample size in the present study resulted in insufficient statistical power to make any adequate statement regarding the strength of the association between the \( \frac{\sigma_{PP}}{PP} \) level and the occurrence of endoleaks. Further research is required to investigate the usefulness of our method to assess risk of endoleak formation.

**Study limitations.** Our USE image analysis algorithm and calculations requires 30 to 60 minutes to complete. However, with further coding and the use of field programmable gate arrays to further automate the process, strain measurements could be produced in near real-time. One limitation inherent to USE analysis is the reliance on image quality. Patients with a larger body habitus or excessive bowel gas can be difficult to scan. Additionally, although the pre- and postoperative scans were conducted by the same sonographer, the 30-day follow-up scans were not. Skill variation between sonographers could have affected the findings. However, we believe this was significantly mitigated using a very basic image of the aorta that does not require advanced US skill. A patient-specific limitation is the inability for some patients to perform a 10-second breath hold. Patients with extensive respiratory comorbidities could experience difficulty holding their breath adequately to prevent physiologic displacement of the aorta. The 10-second breath hold is imperative to the analysis, which relies on the displacement of the aortic wall during pulsation. Additional displacement due to breathing will be registered by the algorithm and result in inaccurate strain readings. We believe this occurred with patient 10 owing to chronic obstructive pulmonary disease and caused the abnormally high postoperative to 30-day \( \frac{\sigma_{PP}}{PP} \) increase (Table II).

Strain is heterogeneous along the vessel wall. Thus, using our method, analysis of one cross-sectional view at one point might not be fully adequate to assess the biomechanics of the full AAA. Future studies could mitigate this by imaging multiple cross-sections along the length of the AAA. Additionally, other methods that might provide a more comprehensive analysis of the vessel, such as four-dimensional US or magnetic resonance imaging, are being explored.\(^\text{29,31}\) Finally, our study had a small sample size; thus, significant differences might have been present in the 30-day follow-up and endoleak analyses. However, the present study was too underpowered to detect them.
CONCLUSIONS

In the present study, we have demonstrated that the USE method proposed is a feasible technique for the intraoperative AAA analysis during EVAR. We have demonstrated that patients can experience large reductions in aortic wall \( \frac{f_{PP}}{PP} \) from pre- to postoperatively, with a relatively stable \( \frac{f_{PP}}{PP} \) from postoperatively to 30 days of follow-up, suggesting that choice of endograft and its material components might play a predominant role in AAA wall strain reduction and remodeling. Further investigation is needed to assess the ability of this USE method to detect significant endoleaks. However, our preliminary data have shown that \( \frac{f_{PP}}{PP} \) could be a promising correlate of the presence or resolution of type II endoleaks. Overall, the preliminary results from the present pilot study suggest that this method could be an additional tool in the assessment of AAAs after EVAR. We believe this could be a future tool to analyze AAA intraoperatively, with the goal of predicting whether beneficial remodeling will occur, in addition to helping with treatment decisions postoperatively in the absence of sac regression.

REFERENCES

1. U.S. Preventive Services Task Force. Owens DK, Davidson KW, Krist AH, Barry MJ, Cabana M, et al. Screening for abdominal aortic aneurysm: US Preventive Services Task Force recommendation statement. JAMA 2019;322:2221-8.
2. Dua A, Kuy S, Lee CJ, Upchurch GR Jr, Desai SS. Epidemiology of aortic aneurysm repair in the United States from 2000 to 2010. J Vasc Surg 2014;59:1512-7.
3. Buth J, Laheij RJ. Early complications and endoleaks after endovascular abdominal aortic aneurysm repair: report of a multicenter study. J Vasc Surg 2000;31(1):134-46.
4. Harris PL, Vallabhaneni SR, Becquemin JP, van Marrewijk C, Laheij RJ. Incidence and risk factors of late rupture, conversion, and death after endovascular repair of infrarenal aortic aneurysms: the EURESTAR experience. European Collaborators on stent/graft techniques for aortic aneurysm repair. J Vasc Surg 2000;32:759-49.
5. Chaikof EL, Blankensteijn JD, Harris PL, White GH, Zarins CK, Bernhard VM, et al. Reporting standards for endovascular aortic aneurysm repair. J Vasc Surg 2002;35:410-68.
6. Chaikof EL, Dalman RL, Eskandari MK, Jackson BM, Lee WA, Mansour MA, et al. The Society for Vascular Surgery practice guidelines on the care of patients with an abdominal aortic aneurysm. J Vasc Surg 2018;67:2-77.e2.
7. Wolf YG, Johnson BL, Hill BB, Rubin GD, Fogarty TJ, Zarins CK. Duplex ultrasound scanning versus computed tomographic angiography for postoperative evaluation of endovascular abdominal aortic aneurysm repair. J Vasc Surg 2000;32:142-8.
8. Parkinson F, Ferguson S, Lewis P, Williams IM, Twine CP, South East Wales Vascular Network. Rupture rates of untreated large abdominal aortic aneurysms in patients unfit for elective repair. J Vasc Surg 2015;61:1606-12.
9. Brown PM, Zeilt DT, Sobolev B. The risk of rupture in untreated aneurysms: the impact of size, gender, and expansion rate. J Vasc Surg 2003;37:280-4.
10. O’Donnell TFX, Deery SE, Boitano LT, Sircusue JJ, Schermerhorn ML, Scali ST, et al. Aneurysm sac failure to regress after endovascular aneurysm repair is associated with lower long-term survival. J Vasc Surg 2019;69:141-22.
11. Cho JS, Park T, Kim JY, Chaer RA, Rhee RY, Makaroun MS. Prior endovascular abdominal aortic aneurysm repair provides no survival benefit when the aneurysm ruptures. J Vasc Surg 2010;52:127-34.
12. Candell L, Tucker LY, Goodney P, Walker J, Okuhn S, Hill B, et al. Early and delayed rupture after endovascular abdominal aortic aneurysm repair in a 10-year multicenter registry. J Vasc Surg 2014;60:1146-53.
13. Di Martino ES, Bohra A, Vande Cept JP, Gupta N, Makaroun MS, Vorp DA. Biomechanical properties of ruptured versus electively repaired abdominal aortic aneurysm wall tissue. J Vasc Surg 2006;43:570-6. [discussion: 576].
14. Wilson K, Bradbury A, Whyman M, Hoskins P, Lee A, Fowkes C, et al. Relationship between abdominal aortic aneurysm wall compliance and clinical outcome: a preliminary analysis. Eur J Vasc Endovasc Surg 1998;15:472-7.
15. Wilson KA, Lee AJ, Lee AJ, Hoskins PR, Fowkes FG, Ruckley CV, et al. The relationship between aortic wall distensibility and rupture of infrarenal aortic aneurysms. J Vasc Surg 2003;37:112-7.
16. Mix DS, Yang L, Johnson CC, Couper N, Zarras B, Arabadis I, et al. Detecting regional stiffness changes in aortic aneurysmal geometries using pressure-normalized strain. Ultrasound Med Biol 2017;43:2372-94.
17. Richards MS, Doyley MM. Non-rigid image registration based strain estimator for intravascular ultrasound elastography. Ultrasound Med Biol 2017;43:935-33.
18. Chimenti RL, Flemister AS, Ketz J, Bucklin M, Buckley MR, Richards MS. Ultrasound strain imaging of Achilles tendon compressive strain patterns during dorsiflexion. J Biomech 2016;49:44.
19. Li ZY, U-King-Im J, Tang TY, Soh E, See TC, Gillard JH. Impact of calcification and intraluminal thrombus on the computed wall stresses of abdominal aortic aneurysm. J Vasc Surg 2008;47:928-35.
20. Aarts NJ, Schurink GW, Schultz Kool LJ, Bode PJ, van Baalen JM, Hermans J, et al. Abdominal aortic aneurysm measurements for endovascular repair: intra- and interobserver variability of CT measurements. Eur J Vasc Endovasc Surg 1999;18:475-80.
21. Lederle FA, Wilson SE, Johnson CR, Reinke DB, Littooy FN, Acher CW, et al. Variability in measurement of abdominal aortic aneurysms. Abdominal Aortic Aneurysm Detection and Management Veterans Administration Cooperative Study Group. J Vasc Surg 1995;21:945-S.
22. Vorp DA. Biomechanics of abdominal aortic aneurysm. J Biomech 2007;40:1887-902.
23. Wilson K, Whyman M, Hoskins P, Lee AJ, Bradbury AW, Fowkes FG, et al. The relationship between abdominal aortic aneurysm wall compliance, maximum diameter and growth rate. Cardiovasc Surg 1999;7:208-13.
24. Vande Cept JP, Sacks MS, Vorp DA. The effects of aneurysm on the biaxial mechanical behavior of human abdominal aorta. J Biomech 2006;39:1324-34.
25. Long A, Rouet L, Vitry F, Albertini JN, Marcus C, Clement C. Compliance of abdominal aortic aneurysms before and after stenting with tissue Doppler imaging: evolution during follow-up and correlation with tissue diameter. Ann Vasc Surg 2009;23:49-59.
26. Malina M, Lanne T, Ivaniec K, Lindblad B, Brunkwall J. Reduced pulsatile wall motion of abdominal aortic aneurysms after endovascular repair. J Vasc Surg 1998;27:624-31.
27. Breken R, Bang J, Odegaard A, Aasland J, Hemes TA, Myhre HO. Strain estimation in abdominal aortic aneurysms from 2-D ultrasound. Ultrasound Med Biol 2006;32:33-42.
28. Vorp DA, Raghavan ML, Webster MW. Mechanical wall stress in abdominal aortic aneurysm: influence of diameter and asymmetry. J Vasc Surg 1998;27:632-9.
29. Kolipaka A, Illapany VS, Kenyhercz W, Dowell JD, Co MR, Starr JE, et al. Quantification of abdominal aortic aneurysm stiffness using magnetic resonance elastography and its comparison to aneurysm diameter. J Vasc Surg 2016;64:966-74.
30. Cebull H, Soepriatna A, Boyle J, Rothenberger S, Goergen C. Strain estimation in abdominal aortic aneurysms from 2-D ultrasound. Ultrasound Med Biol 2006;32:33-42.
31. van Disseldorp EMJ, Petterson NJ, van de Vosse FN, van Sambeek MR, van der Wouden C, Zottola et al. 769

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