Changes in Noninvasive Arterial Stiffness and Central Blood Pressure After Endovascular Abdominal Aneurysm Repair

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Abstract

Purpose: To evaluate the impact of elective endovascular aneurysm repair (EVAR) on the carotid-femoral pulse wave velocity (cfPWV) and central pressure waveform, through 1-year follow-up. **Materials and Methods:** A tonometric device was used to measure cfPWV and estimate the central pressure waveform in 20 patients with an infrarenal abdominal aortic aneurysm scheduled for elective EVAR. The evaluated central hemodynamic parameters included the central pressures, the augmentation index (AIx), and the subendocardial viability ratio (SEVR). AIx quantifies the contribution of reflected wave to the central systolic pressure, whereas SEVR describes the myocardial perfusion relative to the cardiac workload. Measurements were performed before EVAR, at discharge, and 6 weeks and 1 year after EVAR. **Results:** CfPWV was increased at discharge (12.4 ± 0.4 vs 11.3 ± 0.5 m/s at baseline; p=0.005) and remained elevated over the course of 1-year follow-up (6 weeks: cfPWV = 12.2 ± 0.5 m/s; 1 year: cfPWV = 12.2 ± 0.7 m/s, p<0.05). After an initial drop in systolic central pressure at discharge, all the central pressures increased thereafter up to 1 year, without significant differences compared with baseline. The same was observed for the AIx and SEVR. **Conclusion:** Endovascular aortic aneurysm repair caused an increase in pulse wave velocity compared with baseline, which remained elevated through 1 year follow-up, which may be related to an increased cardiovascular risk. However, no differences in central pressure, augmentation index, and subendocardial viability ration were observed during follow-up.

Keywords

abdominal aortic aneurysm, augmentation index, central blood pressure, endovascular aneurysm repair, pulse wave velocity, pressure wave analysis, subendocardial viability ratio

Introduction

Endovascular repair (EVAR) is the preferred treatment for most infrarenal abdominal aortic aneurysms (AAAs).[1] Short-term results of EVAR are superior to those of open repair in terms of 30-day mortality. However, long-term results show that EVAR is associated with higher long-term all-cause and cardiovascular mortality, more reinterventions, and a higher secondary rupture rate compared with open repair.[2–5] There's however a catch-up mortality after 2 years, since survival rates within the first 2 years after surgery are higher for endovascular repair. So, the natural course after the initial 2 years after surgery is a higher mortality rate (within the 2-to 12-year time window in the randomized controlled trials). If the first 2 years are taken into account, overall mortality is equal. The catch-up mortality is also explained by an increased survival after EVAR and

therefore more patients at risk for cardiovascular events (the patients already had generalized cardiovascular disease at the start). One possible explanation is a potential effect of the endograft material on the pressure wave propagation along the arterial tree. Pressure waveforms consist of an incident forward wave ejected by the left ventricle and a reflected backward wave. A mismatch in elastic properties

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due to the presence of an endograft in the aorta locally alters arterial stiffness and can thereby cause additional wave reflections, increasing myocardial afterload.[6]

Peripheral pressure waves can be recorded noninvasively, reliably, and reproducibly, with applanation tonometry of the radial artery.[7,8] Carotid-to-femoral pulse wave velocity (cfPWV) can be recorded, which is a direct method to measure arterial stiffness and is also an important predictor of cardiovascular outcome independent of brachial blood pressure.[9] Literature is inconclusive about the effect of AAA on the cfPWV, but there is evidence for an increase in cfPWV after EVAR in the early phase after surgery,[10–14] and there is evidence that this increase is absent after open surgical aneurysm repair.[10,15] After EVAR, alterations in arterial stiffness and wave reflections might provide insight in the adverse effects on myocardial function and thereby whether these alterations might lead to the adverse cardiovascular risk profile on long term. Data are scarce on whether the initial increase after EVAR in PWV changes or stabilizes over time.

Additionally, central pressure waveforms can be synthesized from the peripheral waveform with a generalized radial-to-aorta transfer function.[16] From these central waveforms the central pressures can be derived, as well as the augmentation index (AIx), and subendocardial viability ratio (SEVR). AIx quantifies the contribution of reflected wave to the central systolic pressure. SEVR describes the myocardial perfusion relative to the cardiac workload.[17] So far, limited and conflicting evidence is available on effect of AAA on central pressure wave morphology.[18–21] Both central systolic pressure and AIx have independent predictive value for cardiovascular outcome.[22–26]

The aim of this study is to provide insight in changes through 1-year follow-up in cfPWV and central pressure hemodynamics after EVAR.

Materials and Methods

This study was designed as a prospective single center study. The study was approved by the regional Medical Ethics Committee (CMO-2016-2431) and the local institutional review board. The study protocol was registered in *clinicaltrials.gov* (NCT03469388). The study was conducted in accordance with the principles of the Declaration of Helsinki and Good Clinical Practice guidelines.

Study Population

Patients with an infrarenal AAA scheduled for elective EVAR and aged ≥18 years were consecutively approached and enrolled in the study after providing written informed consent. Patients were excluded in case of a life expectancy ≤2 years, a psychiatric or other condition hampering informed consent, the presence of an irregular pulse, the presence of peripheral arterial disease (ankle-brachial index <0.9 or obstruction validated on imaging), a ruptured, symptomatic or mycotic AAA, and/or participation in another clinical trial. The choice of endograft was based on anatomical features and decided upon in a local vascular consensus meeting. All endografts were implanted according to instructions for use.

Noninvasive Pulse Wave Velocity Measurements and Pressure Wave Analysis

The SphygmoCor device (AtCor medical Pty Ltd, Sydney, Australia) was used to perform cfPWV and pressure waveform measurements. The cfPWV was measured according the instructions for use of the SpygmoCor at the right carotid and femoral artery after at least 10 minutes of rest. The direct distance $\times$ 0.8 was used to calculate cfPWV according to the guidelines.[27] The built-in generalized radial-to-central-aorta transfer function (validated in multiple studies as summarized by Weber et al[28] in 2014) was used to produce the synthesized central pressures and obtain arterial stiffness parameters. The tonometer was placed on the radial artery just above the wrist and recorded at least 10 pressure waves with a sample rate of 128 Hz. The device automatically finished recording after collection of 10 subsequent sufficient quality waves (quality index >0.9). Radial peak pressures were calibrated against mean arterial pressure (calculated as $0.6 \times$ diastolic pressure $+ 0.4 \times$ systolic pressure) at the brachial artery, determined as the mean of the second and third readings of 3 conventional brachial cuff measurements.

The assessed pressure wave analysis parameters included the systolic, diastolic, and mean pressures, the AIx and the SEVR. The AIx was normalized for heart rate of 75 beats per minute.

Measurements were performed before surgery (baseline), at discharge, and during follow-up at 6 weeks and 1 year with patients in supine position. Patients were instructed to be in a fasting state according to the recommendations by the expert consensus document.[29]

Statistical Analysis

Normality was determined based on visual inspection of the normality graphs and tested using the Shapiro-Wilk test. Baseline characteristics are presented as median and interquartile range (IQR). cfPWV values were corrected for mean arterial pressure (MAP). Changes over time were tested using analysis of variance (ANOVA) for repeated measures or Kruskal-Wallis, if assumptions for ANOVA were violated. P values <0.05 were considered as significant. Statistical analyses were performed using IBM SPSS Statistics (SPSS version 25.0 for windows, IBM Corporation, Armonk, NY, USA).
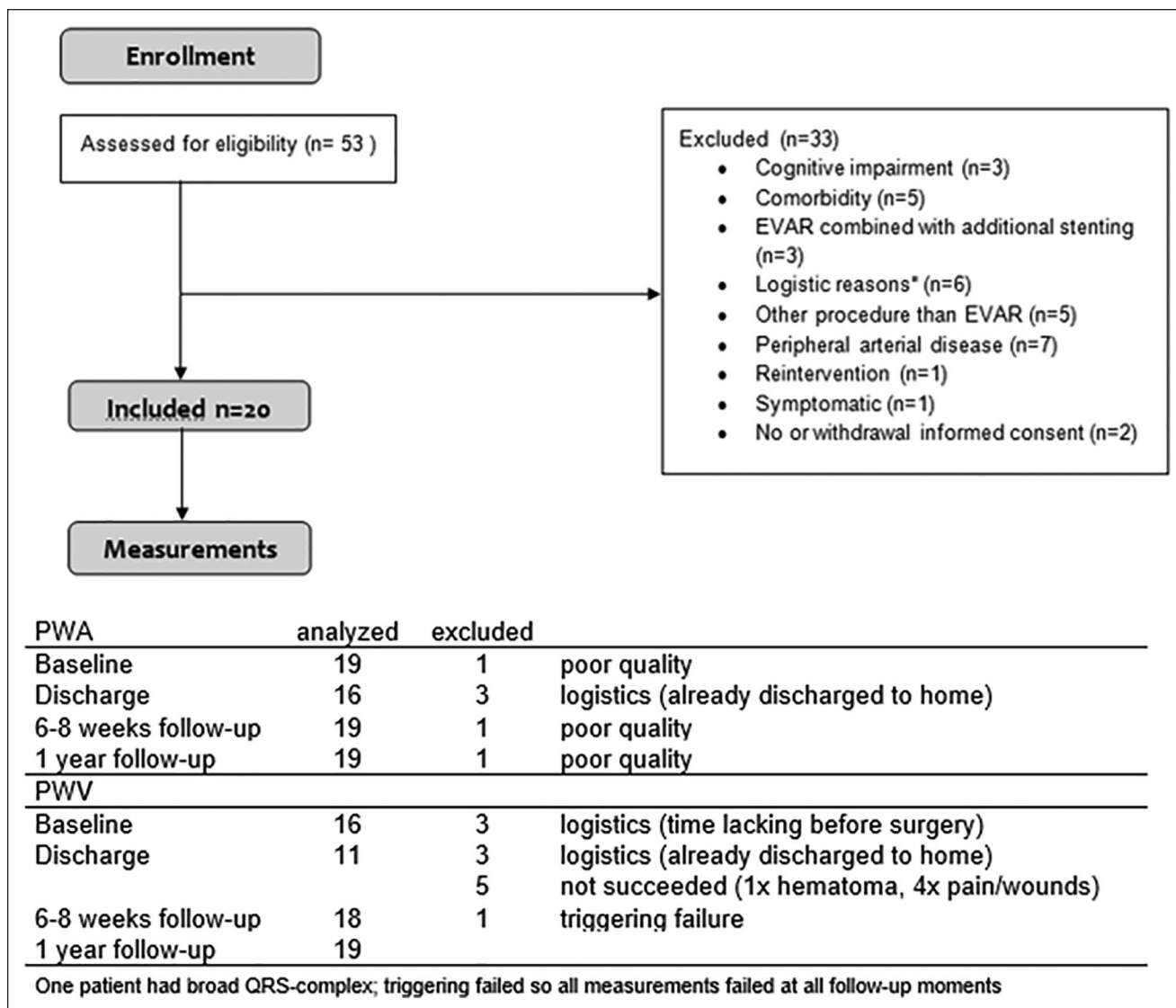
Figure 1. Inclusion flowchart. PWA, pressure wave analysis; PWV, pulse wave velocity; EVAR, endovascular aneurysm repair.
*Logistic reasons included the nonavailability of researcher to perform measurement at scheduled operating room and inability to schedule preoperative measurements with patients.

Results

Twenty patients were included between May 2017 and August 2018 (Figure 1). Baseline characteristics are depicted in Table 1. In 13 (65%) patients, an Endurant II (Medtronic, Santa Rosa, CA), in 6 (30%) patients (30%), an Excluder (W.L. Gore and associates, Flagstaff, AZ) and in 1 (5%) patient, an AFX (Endologix, Irvine, CA) endoprosthesis was implanted.

Not all measurements in all patients succeeded as shown in Figure 1. In total three pressure wave analysis measurements were of insufficient quality. In 3 patients, both measurements were not performed because of logistics reasons, including the nonavailability of researcher to perform

measurement at scheduled operation time and inability to schedule preoperative measurements with patients. One PWV measurement did not succeed because of failing triggering, and five measurements did not succeed because of hematoma and/or pain at the groin.

cfPWV and Pressure Wave Analysis

The cfPWV was significantly increased at discharge, when compared to baseline (12.4±0.4 vs 11.3 ± 0.5 m/s, p=0.005). During follow-up there were no further changes and the levels remained significantly higher relative to baseline through 1-year follow-up (p=0.519, one-year 12.2±0.7 m/s compared with baseline).

Table 1. Baseline Patient and Aneurysm Characteristics.[a]

Patient Characteristics	Median (Q3–Q1) or n (%)
Age (years)	75.0 (76.5–68.0)
Male gender, n (%)	17 (85.0)
Body mass index (kg/m^2)	26.2 (29.2–24.6)
Systolic blood pressure (mm Hg)	151.5 (164.0–135.0)
Diastolic blood pressure (mm Hg)	85.0 (95.5–76.0)
Current smoker, n (%)	2 (10.0)
History of smoking, n (%)	15 (75.0)
Hypertension, n (%)	15 (75.0)
Hyperlipidemia, n (%)	18 (90.0)
Statin use, n (%)	19 (95.0)
Anticoagulant therapy, n (%)	19 (95.0)
ASA classification, n (%)	
2	5 (25.0)
3	14 (70.0)
4	1 (5.0)
Medical history	
Cardiac disorder, n (%)	
Arrhythmia	2 (10.0)
Congestive heart failure	1 (5.0)
Coronary artery disease	11 (55.0)
Myocardial infarction	5 (25.0)
Coronary artery bypass grafting	4 (20.0)
Percutaneous coronary intervention	5 (25.0)
Stroke, n (%)	1 (5.0)
Transient ischemic attack, n (%)	4 (20.0)
Family history of AAA, n (%)	7 (35.0)
Renal insufficiency, n (%)	8 (40.0)
Diabetes mellitus, n (%)	3 (15.0)
Chronic obstructive pulmonary disease, n (%)	2 (10.0)
Cancer, n (%)	8 (40.0)

Abbreviations: AAA, abdominal aortic aneurysm; ASA, American Society of Anesthesiologists physical status.
[a]Continuous data are presented as the medians and interquartile ranges (Q3–Q1); categorical data are given as the counts (percentage).

Central hemodynamic related parameters through the first year after EVAR are depicted in Table 2 and Figure 2. The central systolic pressure significantly decreased at discharge compared with baseline (137.2±13.6 vs 143.0±15.2 mm Hg, p=0.046), without significant decreases in the other parameters. At 6-week and 1-year follow-up, the central pressures returned to the baseline values (141.2±15.9 mm Hg, p=0.819 compared with baseline; and 147.0±18.1 mm Hg, p=0.090, respectively). The AIx and SEVR decreased after treatment (25.4±8.9 vs 32.3±8.9, p=0.003; and 141.6±15.9 vs 155.6±28.9, p=0.046, respectively), which returned to baseline values at 6 weeks (AIx = 29.7±7.6, p=0.225 compared with baseline; and SEVR = 154.5±29.5, p=0.637 compared with baseline) and 1-year follow-up (AIx = 28.7±8.0, p=0.157 compared with baseline; and SEVR = 159.4±27.3, p=1.000 compared with baseline).

Clinical Outcome

During follow-up, 7 adverse events occurred, including postoperative fever of unknown origin (n=4), wound infection

(n=2), and bleeding of access site requiring surgical correction (n=1). All were early complications, anticipated, and related to EVAR. At 1-year follow-up, a type II endoleak was reported in 7 patients, without other types of endoleaks. Comparison of those with and without type II endoleak at 1-year follow-up showed no differences in any of the measured parameters.

Discussion

In the current study, EVAR was related to an increase in cfPWV, which sustained through 1-year follow-up, which could be related to an increased cardiovascular risk. However, no differences in central pressure, AIx, and SEVR were observed during follow-up.

The baseline characteristics show that cfPWV and blood pressure were higher than reference values for this age group,[30] reflecting the expected preexistent adverse cardiovascular risk profile in patients with AAA.

In addition, cfPWV further increased with about 1 m/s after EVAR, which sustained through 1-year follow-up. The

Table 2. Changes in Pulse Wave Velocity and Pressure Wave Parameters Over Time.

Mean (SD)	Baseline	Discharge	6 Weeks	1 Year
Pulse wave velocity (m/s)	11.3 (0.5)	12.4 (0.4)[#]	12.2 (0.5)[#]	12.2 (0.7)[#]
Heart rate (beats/min)	61.4 (10.8)	71.9 (11.8)[#]	64.7 (12.6)[*#]	62.3 (10.3)
Central systolic pressure (mm Hg)	143.0 (15.2)	137.2 (13.6)[#]	141.2 (15.9)	147.0 (18.1)
Central diastolic pressure (mm Hg)	83.1 (10.0)	79.0 (9.3)	85.4 (8.6)[*]	88.2 (10.8)
Central mean pressure (mm Hg)	107.2 (10.8)	102.6 (9.6)	107.7 (9.1)[*]	112.1 (12.8)
Peripheral systolic pressure (mm Hg)	145.3 (19.4)	139.5 (12.6)	141.6 (17.2)	150.4 (19.5)[*]
Peripheral diastolic pressure (mm Hg)	81.4 (9.9)	77.9 (9.2)	82.8 (10.5)[*]	87.6 (10.7)[#]
Peripheral mean pressure (mm Hg)	105.7 (12.9)	98.22 (10.3)[#]	104.1 (11.5)	110.7 (14.2)
Augmentation index	32.3 (8.9)	25.4 (8.9)[#]	29.7 (7.6)	28.7 (8.0)
Subendocardial viability ratio	155.6 (28.9)	141.6 (15.9)[#]	154.0 (29.5)[*]	159.4 (27.3)

[a]Continuous data are presented as the means $\pm$ SD.
*p<0.05 compared with previous measurement. #p<0.05 compared with baseline.
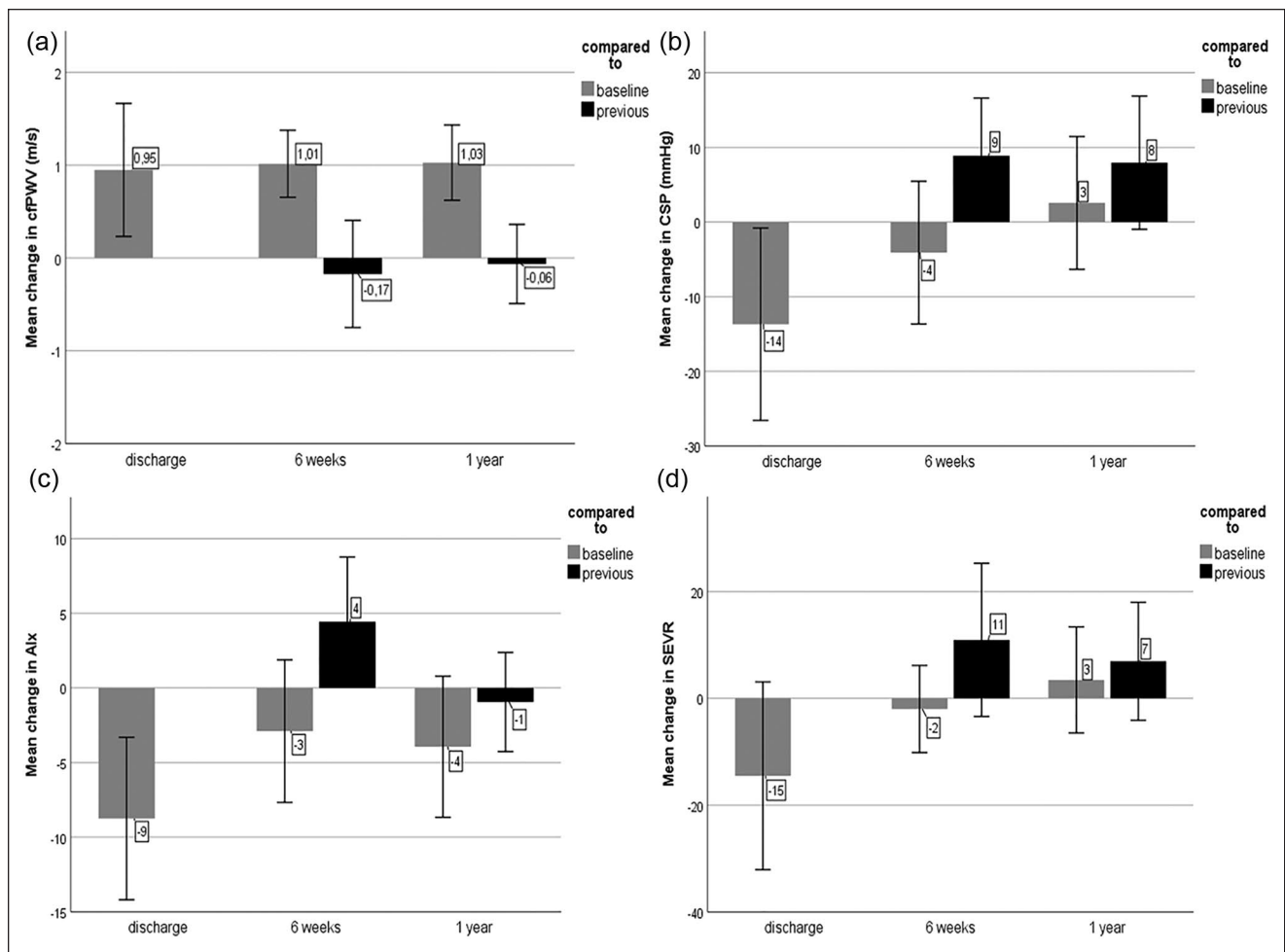

Figure 2. Differences in carotid-to-femoral pulse wave velocity (cfPWV; corrected for mean arterial pressure) in meters per second (a), central systolic pressure (CSP) in mm Hg (b), augmentation index corrected for heart rate of 75 beats per minute (AIx@HR75) (c), and subendocardial viability ratio (SEVR) (d) compared with baseline and with the previous time point. Presentation of mean differences in bars and 95% confidence interval in whiskers.
*Denotes p<0.05 compared with previous measurement and #denotes p<0.05 compared with baseline.

fact that the initial increase in cfPWV remains stable through 1-year follow-up is important information in the light of the increased cardiovascular risk in patients treated with EVAR. A pooled data analysis from 17,635 subjects in 16 studies showed that, after adjustment for age and sex, the increase in risk for cardiovascular events, cardiovascular mortality, and total mortality was 45%, 41%, and 22%, respectively, for an increase of 1 standard deviation change in log PWV. After adjustment for additional risk factors, the respective increases were 30%, 28%, and 17%.[9] To exemplify these results, for a 60-year old man who is a non-smoker, nondiabetic, and normo-lipidemic, a 1 m/s increase in PWV leads to a 7% increase of the hazard for cardiovascular events.[31] PWV integrates and reflects the long-term effect of the established, as well of the currently unknown, risk factors on the arterial wall, together with the genetic predisposition of the individual. Since most patients with an AAA have one or more cardiovascular risk factors, this risk might be even higher. An increase in PWV leads to an increased cardiovascular risk in the future, so it is important if the increase after EVAR is a temporarily increase or a permanent increase. If PWV would have decreased within a year after treatment; the short-term increase would not likely have led to an increased cardiovascular risk on the longer term. However, the current study showed a persistent increased PWV after EVAR.

Furthermore, the study showed that adding PWV improved the 10-year risk classification of patients at intermediate risk for cardiovascular disease.[9] The 2013 European Society of Hypertension/European Society of Cardiology guidelines for the management of arterial hypertension recommended the assessment of PWV for the evaluation of subclinical organ damage in hypertensive patients, and suggests a cutoff of 10 m/s to discriminate between normal aortic elasticity and aortic stiffening.[27,32] For clinical practice, this suggests that cardiovascular risk stratification could be optimized for patients with an AAA by adding PWV measurements.

The increase in cfPWV after EVAR is in line with previous studies.[10–14,33] Other studies reported the immediate to early effect up to 6 weeks, with 2 reports showing an increased cfPWV at 6 months[11,13] and 1 study showing an increased cfPWV in 30 patients at 1 year after treatment.[12] There is evidence that different types of endografts could have differential effect on cfPWV after EVAR.[34,35] An increased cardiovascular mortality and morbidity after endovascular versus open surgical treatment has been shown by multiple studies.[2–5] Previous studies have suggested that alterations in wave reflections due to inserting a relatively stiff endograft could be an explanation for this increase in cardiovascular mortality.[11,34] It has been suggested that the increase in PWV, as a result of these alterations in wave reflections might be an explanation. Also, an increased inflammatory response[36] and an increased left ventricular load[12] have been suggested as possible causes. Larger studies are needed to elucidate this issue.

The central systolic pressure, AIx, and SEVR initially showed a decrease after EVAR. No new medications were started before discharge. Various other factors may have affected blood pressure during the discharge measurements, including anesthetics during surgery, the passive supine position in the ward, and increased compliance of anti-hypertensive use in the hospital setting. We have interpreted that anesthetics and blood regulation during the procedure might have affected the blood pressures. Also, patients were in supine position mostly after the procedure, which might also have had an effect on blood pressure. The same trend was observed for peripheral blood pressure.

No significant increase in central pressures and derived parameters was observed thereafter through 1-year follow-up. It could be hypothesized that waves will be reflected to the heart earlier and with increased magnitude when a relatively stiff endograft is inserted in an aneurysmatic abdominal aorta, resulting in increased central pressures and AIx. This would increase the ventricular after-load and systolic work. A previous study reported that each increase of 10 percentage points in AIx, increased the risk of all-cause and cardiovascular mortality by 51% and 48%, respectively in patients with end-stage renal disease.[37] A meta-analysis showed that an increase in AIx of 10% was associated with a 32% increase in cardiovascular event risk and a 38% increase in all-cause mortality risk.[24] The current analyses, however, showed no differences in mean and diastolic central pressure and AIx directly post-EVAR, except for a decreased systolic pressure. At 6-week and 1-year follow-up, no significant differences relative to before EVAR were observed. Our results therefore do not support the above hypothesis.

Previously, it was shown that the AIx decreased after both EVAR and open surgical repair, whereas the SEVR decreased after open surgical repair, but not after EVAR at 6-month follow-up.[38] Also, a decreased AIx after EVAR was reported 4 weeks after EVAR.[19] In contrast, others showed an increased AIx (+4%) after EVAR and a decreased AIx (−8.5%) after open surgical repair.[10] The use of different measurement devices and different types of endografts implanted, makes a comparison between studies difficult to perform. Only 1 study reported the SEVR values and described a decrease after open AAA repair that maintained up to 6-month follow-up, without differences after EVAR.[38] This appears to be in contrast with the significant decrease in SEVR at discharge, but this was followed by return to baseline at 6 weeks, in the current report.

The evidence of an increased PWV after AAA treatment has been accumulating, but data on the influence of AAA treatment on pressure wave parameters are scarce. So, we think that the hypothesis is confirmed with regard to PWV; however, more research is needed in larger patient groups with regard to pressure wave analysis.

The current study is limited by the small sample size. Additionally, PWV measurements cannot reliably be performed in some patients because of triggering problems with the electrocardiogram; in the current study in 1 patient the measurement did not succeed because of a broad QRS complex. After treatment, PWV measurements could not successfully be performed in some patients because of hematoma and pain in the groin.

This study was designed as an explorative study and future studies could well include other pathologies and techniques, when the technique is proven to be valid. Furthermore, this study was not designed to be able to show a causal relation between the increase in PWV and future cardiovascular events; larger studies with specific design are needed to draw conclusions on this topic.

In hindsight, it might be better to not include the AFX but refrain to 1 single endograft or 2 in equally large groups. Additionally, we did not exclude patients with carotid disease on forehand. We had one patient with an occlusion of the right carotid artery; however, in this patient it was possible to measure the left carotid and left femoral, so this did not influence the results.

In the meanwhile, we have started a study in patients under surveillance for an AAA that has not reached the treatment threshold yet, in which these measures are performed at baseline (along with a carotid artery reactivity test; NCT03989011). Patients will be followed for 2 years. Besides, we have started a study in patients scheduled for treatment of their AAA (all treatment modalities; NCT04183426). In that study these measures are performed before treatment, and 6 weeks, 1 year, and 2 years after treatment. These studies will provide more answers.

Conclusions

Endovascular aortic aneurysm repair caused an increase in pulse wave velocity compared with baseline, which remained elevated through 1-year follow-up, which may be related to an increased cardiovascular risk.

However, no differences in central pressure, augmentation index, and subendocardial viability ration were observed during follow-up.

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References

1. Chaikof EL, Dalman RL, Eskandari MK, 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.
2. Li B, Khan S, Salata K, et al. A systematic review and meta-analysis of the long-term outcomes of endovascular versus open repair of abdominal aortic aneurysm. *J Vasc Surg*. 2019;70:954–969.e30.
3. Brown LC, Greenhalgh RM, Thompson SG, et al. Does EVAR alter the rate of cardiovascular events in patients with abdominal aortic aneurysm considered unfit for open repair? Results from the randomised EVAR trial 2. *Eur J Vasc Endovasc Surg*. 2010;39:396–402.
4. Brown LC, Thompson SG, Greenhalgh RM, et al. Incidence of cardiovascular events and death after open or endovascular repair of abdominal aortic aneurysm in the randomized EVAR trial 1. *Br J Surg*. 2011;98:935–942.
5. De Bruin JL, Baas AF, Buth J, et al. Long-term outcome of open or endovascular repair of abdominal aortic aneurysm. *N Engl J Med*. 2010;362:1881–1889.
6. van Noort K, Holewijn S, Schuurmann RCL, et al. Effect of abdominal aortic endoprostheses on arterial pulse wave velocity in an in vitro abdominal aortic flow model. *Physiol Meas*. 2018;39:104001.
7. Wilkinson IB, Fuchs SA, Jansen IM, et al. Reproducibility of pulse wave velocity and augmentation index measured by pulse wave analysis. *J Hypertens*. 1998;16(12 pt 2):2079–2084.
8. Filipovsky J, Svobodova V, Pecen L. Reproducibility of radial pulse wave analysis in healthy subjects. *J Hypertens*. 2000;18:1033–1040.
9. Ben-Shlomo Y, Spears M, Boustred C, et al. Aortic pulse wave velocity improves cardiovascular event prediction: an individual participant meta-analysis of prospective observational data from 17,635 subjects. *J Am Coll Cardiol*. 2014;63:636–646.
10. Lantelme P, Dzudie A, Milon H, et al. Effect of abdominal aortic grafts on aortic stiffness and central hemodynamics. *J Hypertens*. 2009;27:1268–1276.
11. Kadoglou NP, Moulakakis KG, Papadakis I, et al. Changes in aortic pulse wave velocity of patients undergoing endovascular repair of abdominal aortic aneurysms. *J Endovasc Ther*. 2012;19:661–666.
12. Takeda Y, Sakata Y, Ohtani T, et al. Endovascular aortic repair increases vascular stiffness and alters cardiac structure and function. *Circ J*. 2014;78:322–328.
13. Paraskevas KI, Bessias N, Psathas C, et al. Evaluation of aortic stiffness (aortic pulse-wave velocity) before and after elective abdominal aortic aneurysm repair procedures: a pilot study. *Open Cardiovasc Med J*. 2009;3:173–175.

14. Iwahashi T, Obitsu Y, Koizumi N, et al. Clinical comparison of two different types of bifurcated graft for postoperative baPWV and ABI. *Int Angiol*. 2009;28:232–237.

15. Gray C, Goodman P, Badger SA, et al. Endovascular aneurysm repair increases aortic arterial stiffness when compared to open repair of abdominal aortic aneurysms. *Vasc Endovascular Surg*. 2016;50:317–320.

16. Williams B, Lacy PS, Yan P, et al. Development and validation of a novel method to derive central aortic systolic pressure from the radial pressure waveform using an n-point moving average method. *J Am Coll Cardiol*. 2011;57:951–961.

17. Avolio AP, Butlin M, Walsh A. Arterial blood pressure measurement and pulse wave analysis–their role in enhancing cardiovascular assessment. *Physiol Meas*. 2010;31:R1–R47.

18. Lyck Hansen M, Dahl Thomsen M, Rasmussen LM, et al. Abdominal aortic aneurysm, arterial stiffening and the role of the intraluminal thrombus. *Vasa*. 2015;44:349–353.

19. Lee CW, Sung SH, Chen CK, et al. Measures of carotid-femoral pulse wave velocity and augmentation index are not reliable in patients with abdominal aortic aneurysm. *J Hypertens*. 2013;31:1853–1860.

20. Beckmann M, Jacomella V, Kohler M, et al. Risk stratification of patients with peripheral arterial disease and abdominal aortic aneurysm using aortic augmentation index. *PLoS One*. 2015;10:e0139887.

21. Georgakarakos E. Estimating the influence of aortic-stent grafts after endovascular aneurysm repair: are we missing something? *Med Hypotheses*. 2016;97:26–30.

22. Boardman H, Lewandowski AJ, Lazdam M, et al. Aortic stiffness and blood pressure variability in young people: a multimodality investigation of central and peripheral vasculature. *J Hypertens*. 2017;35:513–522.

23. Wang KL, Cheng HM, Sung SH, et al. Wave reflection and arterial stiffness in the prediction of 15-year all-cause and cardiovascular mortalities: a community-based study. *Hypertension*. 2010;55:799–805.

24. Vlachopoulos C, Aznaouridis K, O'Rourke MF, et al. Prediction of cardiovascular events and all-cause mortality with central haemodynamics: a systematic review and meta-analysis. *Eur Heart J*. 2010;31:1865–1871.

25. Roman MJ, Devereux RB, Kizer JR, et al. Central pressure more strongly relates to vascular disease and outcome than does brachial pressure: the Strong Heart Study. *Hypertension*. 2007;50:197–203.

26. Muiesan ML, Salvetti M, Bertacchini F, et al. Central blood pressure assessment using 24-hour brachial pulse wave analysis. *J Vasc Diagn Intervent*. 2014;2014:141–148.

27. Van Bortel LM, Laurent S, Boutouyrie P, et al. Expert consensus document on the measurement of aortic stiffness in daily practice using carotid-femoral pulse wave velocity. *J Hypertens*. 2012;30:445–448.

28. Weber T, Wassertheurer S. Moving on—on average in the right direction? Noninvasive methods to estimate central blood pressure. *Hypertension*. 2014;63:665–667.

29. 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.

30. Reference Values for Arterial Stiffness' Collaboration. Determinants of pulse wave velocity in healthy people and in the presence of cardiovascular risk factors: 'establishing normal and reference values'. *Eur Heart J*. 2010;31:2338–2350.

31. Vlachopoulos C, Aznaouridis K, Stefanadis C. Aortic stiffness for cardiovascular risk prediction: just measure it, just do it! *J Am Coll Cardiol*. 2014;63:647–649.

32. Mancia G, Fagard R, Narkiewicz K, et al. 2013 ESH/ESC guidelines for the management of arterial hypertension: the Task Force for the Management of Arterial Hypertension of the European Society of Hypertension (ESH) and of the European Society of Cardiology (ESC). *Eur Heart J*. 2013;34:2159–2219.

33. Sekhri AR, Lees WR, Adiseshiah M. Measurement of aortic compliance in abdominal aortic aneurysms before and after open and endoluminal repair: preliminary results. *J Endovasc Ther*. 2004;11:472–482.

34. Kadoglou NP, Moulakakis KG, Papadakis I, et al. Differential effects of stent-graft fabrics on arterial stiffness in patients undergoing endovascular aneurysm repair. *J Endovasc Ther*. 2014;21:850–858.

35. van Herwaarden JA, Muhs BE, Vincken KL, et al. Aortic compliance following EVAR and the influence of different endografts: determination using dynamic MRA. *J Endovasc Ther*. 2006;13:406–414.

36. Kadoglou NP, Papadakis I, Moulakakis KG, et al. Arterial stiffness and novel biomarkers in patients with abdominal aortic aneurysms. *Regul Pept*. 2012;179:50–54.

37. London GM, Blacher J, Pannier B, et al. Arterial wave reflections and survival in end-stage renal failure. *Hypertension*. 2001;38:434–438.

38. Moloney MA, McHugh S, O'Donnell DH, et al. Comparison of arterial stiffness and microcirculatory changes following abdominal aortic aneurysm grafting. *Ir J Med Sci*. 2011;180:375–378.