Intra-aneurysm Sac Pressure in Patients with Unchanged AAA Diameter after EVAR

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Abdominal aortic aneurysm (AAA); Endovascular aneurysm repair (EVAR); Pressure; Endotension

Abstract  Objective: To study intra-aneurysm sac pressure and subsequent abdominal aortic aneurysm (AAA) diameter changes in patients without endoleaks that remain unchanged in AAA diameter more than 1 year after endovascular aneurysm repair (EVAR).

Methods: A total of 23 patients underwent direct intra-aneurysm sac pressure (DISP) measurements 16 months (IQR: 14–35 months) after EVAR. Tip-pressure sensors were used through translumbar AAA puncture. Mean pressure index (MPI) was calculated as the percentage of mean intra-aneurysm pressure relative to the simultaneous mean intra-aortic pressure. Aneurysm expansion or shrinkage was assumed whenever the diameter change was >5 mm. Values are presented as median and interquartile range.

Results: In 18 patients, no fluid was obtained upon AAA puncture (group A). In five patients, fluid was obtained (group B). In group A, follow-up continued for 29 months (IQR: 15–35 months) after DISP; five AAAs shrank, 10 remained unchanged and three expanded (MPIs of 26% (IQR: 18–42%), 28% (IQR: 20–48%) and 63% (IQR: 47–83%) and intra-sac pulse pressures of 3 mmHg (IQR: 0–5 mmHg), 4 mmHg (IQR: 2–8 mm Hg) and 12 mmHg (IQR: 6–20 mmHg), respectively, for the three subgroups). MPI and intra-sac pulse pressures were higher in AAAs that subsequently expanded (P = 0.073 and 0.017, respectively). MPI and pulse pressure correlated with total diameter change (r = 0.49, P = 0.039 and r = 0.39, P = 0.109, respectively). Pulse pressure had a greater influence than MPI on diameter change (R² = 0.346, P = 0.041, beta standardised coefficient of 0.121 for MPI and 0.502 for pulse pressure). Similar results with stronger, and significant correlation to pulse pressure were obtained when relative diameter changes were used (r = 0.55, P = 0.017). In group B, MPI and AAA pulse pressure were 32% (IQR: 18–37%) and 1 mmHg (IQR: 0–6 mmHg), respectively. After 36 months (IQR: 21–38 months), one AAA shrank, three continued unchanged while one expanded.

Conclusions: AAAs without endoleak and unchanged diameter more than 1 year after EVAR will often continue unchanged. Expansion can eventually occur in the absence of intra-sac fluid accumulation and is associated with higher and more pulsatile intra-sac pressure. However, in patients with intra-sac fluid, expansion can occur with low intra-sac pressures.

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Endovascular aneurysm repair (EVAR) was introduced as a less invasive option for the treatment of abdominal aortic aneurysms (AAAs). The theoretical premise of EVAR is that a successful treatment will exclude the AAA sac from the systemic arterial pressure and circulation. The outcome after EVAR has, nevertheless, been traditionally evaluated by imaging methods since intra-aneurysm sac pressure has been difficult to measure. The imaging follow-up assesses changes in the AAA diameter and the presence of endoleaks. AAA diameter expansion after EVAR is considered a clinical failure, as opposed to AAA shrinkage that is taken as treatment success. Studies on intra-aneurysm sac pressure have reinforced these concepts by showing a significantly higher AAA sac pressurisation in expanding AAAs than in the shrinking in the absence of an endoleak. However, clinical reports have also shown the possibility of AAA expansion, with varying degrees of pressurisation, due to the accumulation of translucent/whitish intra-sac fluid, that is, hygroma formation. All AAA expansions in the absence of an endoleak are included in the currently accepted definition of endotension. However, although the denomination of endotension implies an increased tension within the aneurysm sac, the definition does not mention the degree of AAA sac pressurisation. Furthermore, preliminary results have shown that intra-sac pressure may also be increased while the AAA continues unchanged in diameter. A subsequent aneurysm expansion may or may not occur. The significance of AAAs unchanged in diameter without endoleak following EVAR is, therefore, more uncertain. Unchanged AAA diameter has, nevertheless, been accepted as a treatment success since there is no proof of a failure.

The aim of this study was to evaluate intra-aneurysm sac pressure in patients with unchanged AAA diameter for at least 1 year after EVAR.

Methods

Patients

Twenty-three patients (21 men, two women; 72 years (IQR: 69–77 years)) with unchanged AAA diameters after EVAR underwent direct intra-aneurysm sac pressure (DISP) measurement at 16 months (IQR: 14–35 months) postoperatively. Patient and AAA characteristics as well as stent grafts used are described in Table 1.

Patient selection for the study was based on the absence of an identifiable endoleak in an AAA with unchanged diameter at least 1 year after EVAR. Furthermore, patients had to be anatomically suitable for direct translumbar AAA puncture. Nine of these patients have been included in a previous report. Drainage of fluid upon puncturing of the AAA sac was used as a criterion for patient group analysis (Table 2). The study received the approval of the regional ethical committee, and all patients gave informed consent for the procedure.

Study setting

All procedures were carried out in a tertiary university centre between November 2000 and October 2006, where EVAR has been performed since 1993 in more than 1000 patients due to abdominal, thoracic and thoraco-abdominal aneurysms. During the study period, 413 patients underwent EVAR of AAA. Anatomical suitability for the translumbar puncture of the AAA was 90%. Furthermore, 75% of the patients were not candidates for this study since they exhibited AAA shrinkage 1 year after EVAR.

Imaging

Computed tomography (CT) scans were done preoperatively and at least yearly after EVAR. Additional CT scans were performed whenever indicated. Preoperative CT was biphasic with scanning before and after intravenous non-ionic iodinated contrast enhancement. The postoperative CT scans were triphasic with an additional delayed scan. All CT scans were reconstructed with 0.75–3 mm axial slices. All patients underwent an extra CT a month before DISP. A digital subtraction angiography was performed at the time of DISP. The presence of an endoleak was an absolute exclusion criterion and was assessed by follow-up and pre-DISP CT scan and digital subtraction angiography at the time of DISP.

AAA diameters were measured by the same observer on axial CT reconstructions using the shortest transverse diameter at the widest portion of the aneurysm. Diameter changes were calculated to express the diameter evolution over time until any re-intervention was performed. Diameter changes were calculated by subtracting the first diameter to the latest. Diameter changes were considered significant when equalling or exceeding 5 mm, with AAAs being grouped into shrinking (≤5 mm), unchanged

### Table 1 Characteristics of patients.

<table>
<thead>
<tr>
<th>Group</th>
<th>No. of patients</th>
<th>Median (IQR)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male/Female</td>
<td>21/2</td>
<td></td>
</tr>
<tr>
<td>AAA diameter (mm)</td>
<td>60 (53–69)</td>
<td></td>
</tr>
<tr>
<td>AAA diameter change until DISP (mm)</td>
<td>0 (0–3)</td>
<td></td>
</tr>
<tr>
<td>Timing DISP (months)</td>
<td>16 (14–35)</td>
<td></td>
</tr>
<tr>
<td>Stent grafts</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Zenith</td>
<td>21d</td>
<td></td>
</tr>
<tr>
<td>Vanguard</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Home-made</td>
<td>1</td>
<td></td>
</tr>
</tbody>
</table>

a Cook Europe A/S, Bjaeverskov, Denmark.
b Boston Scientific, Natick, MA.
c Based on full thickness Dacron graft (Vascutek Ltd., Inchinnan, Scotland) sutured to Gianturco Z- stainless steel stents (Cook Europe A/S, Bjaeverskov, Denmark) along all the stent-graft.
d Including 3 fenestrated Stent grafts and 1 iliac-branched.
(<5 mm and <5 mm) and expanding (≥5 mm) as recommended in the reporting standards for EVAR. Relative diameter changes were determined by calculating the ratio between the absolute diameter change and the original AAA diameter.

**Pressure measurement system and DISP technique**

Patients were considered anatomically suitable for DISP when a translumbar puncture of the AAA sac was considered possible without risking viscera or stent graft integrity. The CT scan performed in the month before DISP was used for this evaluation.

The pressure measurements were performed using wired tip-pressure sensors mounted on 0.014-inch guide wires (PressureWire4, RADI Medical AB, Uppsala, Sweden). The sensors were calibrated immediately before insertion, using saline solution according to the instructions for use provided by the manufacture. All procedures were performed under local anaesthesia using fluoroscopic guidance according to the previously described and validated DISP technique. The pressure sensors were directly connected to a digital interface registering the systemic and AAA sac pressures, simultaneously. All pressures were analysed as systolic, diastolic, mean and pulse pressures (Fig. 1B). The previously established mean pressure index (MPI) was calculated as the percentage of the mean intra-aneurysmal pressure relative to the simultaneous mean systemic pressure.

\[
\text{MPI} = \frac{\text{mean AAA pressure}}{\text{mean systemic pressure}} \times 100
\]

The calibration of the sensor was verified each time the sensor was completely withdrawn from the patient. A drift <5 mmHg by the end of the measurements compared with the initial calibration was required for the measurements to be considered of good quality. Otherwise, a new measurement was taken after recalibrating the sensor.

**Statistical analysis**

Normal distribution was not assumed. Values are presented as medians and interquartile range (IQR) between parentheses, if not stated otherwise. Considering the differing and reduced number of patients in the groups, non-parametric exact tests were used for comparisons, with

<table>
<thead>
<tr>
<th>Group</th>
<th>No. of patients</th>
<th>Age (years)</th>
<th>AAA Ø (mm)</th>
<th>Timing DISP (mo)</th>
</tr>
</thead>
<tbody>
<tr>
<td>No intra-sac fluid</td>
<td>18</td>
<td>73 (68–77)</td>
<td>60 (53–68)</td>
<td>17 (15–45)</td>
</tr>
<tr>
<td>With intra-sac fluid</td>
<td>5</td>
<td>71 (68–77)</td>
<td>60 (51–78)</td>
<td>14 (12–24)</td>
</tr>
</tbody>
</table>

Group division was done according to the presence or absence of intra-sac fluid upon AAA puncture.
Statistical analysis was done using SPSS 16.0.1 (SPSS Inc., Chicago, IL, USA).

Results

Twenty-three patients with unchanged AAA diameter after EVAR (21 male and two female; 72 (69–77) years old, Table 1) underwent DISP. Direct translumbar puncture of the AAA was successful in all patients and was not associated with any immediate complications. In five patients, the puncture was associated with fluid drainage, as opposed to the remaining 18 where there was no drainage upon AAA puncture.

Patient without intra-sac fluid

Eighteen patients (16 male and two female; Table 1) underwent DISP and no fluid was obtained upon puncture of the AAA sac. DISP was performed 17 months (IQR: 15–45 months) after EVAR and patients were followed up for 29 months (IQR: 15–35 months) afterwards. During this follow-up period, five AAs shrank in size, 10 remained unchanged and three expanded (Table 3). MPIs in these three subgroups were 26% (IQR: 18–42%), 28% (IQR: 20–48%) and 63% (IQR: 47–83%) (P = 0.073, Figs.2 and 3), respectively.

Pulse pressure in these groups were 3 mmHg (IQR: 0–5 mmHg), 4 mmHg (IQR: 2–8 mmHg) and 12 mmHg (IQR: 6–20 mmHg) (P = 0.017), respectively.

MPI and diameter change

There was a positive correlation between MPI and total diameter change during follow-up (r = 0.49, P = 0.039, Fig. 4). The regression analysis gave similar results when the relative diameter changes were used (r = 0.55, P = 0.017). Furthermore, the relative dependency of the diameter change on the pulse pressure seems to be greater than on MPI as assessed by multilinear regression (r² = 0.346, P = 0.041, beta standardised coefficient 0.502 for pulse pressure and 0.121 for MPI).

Intra-aneurysm sac pressure and relative diameter change

MPI and pulse pressure continued to correlate positively and in a similar way with the diameter changes when the analysis was done using the relative diameter changes instead of the absolute values (r = 0.44, P = 0.066 and r = 0.55, P = 0.017, respectively, Fig. 4). The regression analysis gave similar results when the relative diameter changes were used (r² = 0.368, P = 0.032, beta standardised coefficient 0.453 for pulse pressure and 0.194 for MPI).

Patient with intra-sac fluid

In five patients, fluid was obtained upon AAA puncture 14 months (IQR: 12–24 months) after EVAR. MPI in these patients was 32% (IQR: 18–37%) while intra-aneurysm sac pulse pressure was 1 mmHg (IQR: 0–6 mmHg). After a median follow-up of 36 months (IQR: 21–38 months), one aneurysm shrank (MPI of 32% and AAA pulse pressure of 1 mmHg), three remained stable in diameter (MPIs of 14%, 37% and 38% and AAA pulse pressure of 1, 1 and 12 mmHg) and one expanded (MPI of 22% and AAA pulse pressure of 0 mmHg).

Table 3  Intra-aneurysm sac pressure.

<table>
<thead>
<tr>
<th>Diameter change after DISP</th>
<th>N</th>
<th>Follow-up before DISP (mo)*</th>
<th>AAA mean pressure (mm Hg)</th>
<th>AAA pulse pressure (mm Hg)</th>
<th>MPI (%)</th>
<th>Follow-up after DISP (mo)**</th>
</tr>
</thead>
<tbody>
<tr>
<td>No intra-sac fluid</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Shrinking</td>
<td>5</td>
<td>15 (14–17)</td>
<td>30 (18–46)</td>
<td>3 (0–5)</td>
<td>26 (18–42)</td>
<td>34 (20–34)</td>
</tr>
<tr>
<td>Expanding</td>
<td>3</td>
<td>14 (14–16)</td>
<td>56 (55–106)</td>
<td>12 (6–20)</td>
<td>63 (47–83)</td>
<td>28 (9–37)</td>
</tr>
<tr>
<td>With intra-sac fluid</td>
<td>5</td>
<td>14 (12–24)</td>
<td>27 (20–46)</td>
<td>1 (0–6)</td>
<td>32 (18–37)</td>
<td>36 (21–38)</td>
</tr>
</tbody>
</table>

Patients without intra-sac fluid upon AAA puncture were separated according to the AAA diameter change after DISP (changes were considered significant when ≥ 5 mm). *P = .036. **P = .803.

Image: Figure 2 Mean Pressure Index (MPI) versus AAA diameter change at the time of direct intra-aneurysm sac pressure measurement (DISP) in patients without intra-sac fluid upon AAA puncture (n = 18). All patients had unchanged AAA diameter at the time of DISP.
Discussion

EVAR-treated patients have been subjected to intensive follow-up programmes based on imaging methods assessing mainly the evolution of the AAA diameter and the presence of endoleaks. AAA expansion is considered a sign of treatment failure, while shrinkage is considered a success. Previous reports have confirmed the premise of EVAR by showing the association between depressurisation of the AAA sac and diameter shrinkage, and AAA expansion and high intra-sac pressure. However, high intra-sac pressure was also seen in aneurysms with unchanged diameter after EVAR that may eventually expand. An increased knowledge on the intra-sac pressure may, therefore, potentially allow us the early identification of patients developing AAA expansion later on.

The present study shows that there is a considerable group of patients with unchanged aneurysm diameter more than 1 year after EVAR that will continue unchanged, while a smaller proportion will shrink or expand later on. Furthermore, a period of unchanged AAA diameter may be insufficient to predict future diameter changes and this unchanged AAA diameter may or not be associated with the accumulation of intra-sac fluid. Intra-aneurysm pressure, especially pulse pressure, seems to be one of the determinants of subsequent expansion in patients without intra-sac fluid. This concurs with our previous studies where high intra-aneurysm sac pressure was measured in patients that already had demonstrable AAA expansion by the time of the pressure measurement. On the contrary, low non-pulsatile pressure was seen in patients with successful EVAR, that is, with AAA shrinkage and no endoleak. In the present study, statistical significance is not reached for MPI between the groups. This is most likely due to the small number of patients with expanding and shrinking AAAs. However, it reinforces the notion of the need for a more intensive follow-up in those patients with high intra-sac pressures. The previously suggested cut-off of 35%, beyond which MPI is considered high, appears to continue to be well justified.

The present study also indicates pulse pressure as a significant determinant of subsequent diameter evolution. This had been previously suggested by ultrasound- and MR-based studies showing that AAA pulsatility seems to decrease after EVAR, especially in the absence of endoleak. More recent studies have shown the possibility of assessing AAA pulsatility in vivo with MR and CT and its dependency on the intra-aortic pressure in non-operated aneurysms. Future studies will allow us to compare these imaging evaluations with intra-aneurysm pressure after EVAR and test the hypothesis generated by the present study. Pulse pressure seems even to be more relevant than MPI for the diameter changes in this particular group of patients as shown by the higher Beta Standardised Coefficient in the multilinear regression. However, pulse pressure and MPI do not seem to justify more than 35% of the

![Figure 3](image1.png)  
**Figure 3** MPI versus AAA diameter changes after the continued CT follow-up after DISP in patients without intra-sac fluid ($n=18$). The horizontal lines represent median MPI in patients with AAA that shrank (26%, $n=5$), continued stable (28%, $n=10$) or expanded in diameter (63%, $n=3$). Green, blue and red circles represent, respectively, patients whose AAA shrank, remained unchanged or expanded in diameter after DISP.

![Figure 4](image2.png)  
**Figure 4** Correlation analysis between intra-aneurysm pulse pressure and the relative AAA diameter changes during the entire follow-up period in patients without intra-sac fluid ($r=.55$, $P=.017$). The relative diameter change was calculated relative to the original AAA diameter. Green, blue and red circles represent, respectively, patients whose AAA shrank, remained unchanged or expanded in diameter after DISP.
subsequent diameter evolution in patients with initially unchanged diameter. Factors other than pressure might, therefore, be important. Previous experimental publications have suggested the importance of the mechanical properties of the aneurysm wall, the AAA size and the type of graft. Stiffer aneurysms were associated with higher intra-sac pressures\(^\text{17}\) as opposed to larger aneurysms\(^\text{18}\) and stiffer grafts\(^\text{19}\) that have been associated with lower intra-sac pressures. In our series, the original AAA diameter does not seem to correlate to either MPI or to the intra-AAA pulse pressure. The stiffness of the stent graft does not seem to be important in our study since all stent grafts, with one exception, were based on the same stent graft. The differences can therefore be associated with idiosyncrasy of the properties of the AAA wall and intra-luminal thrombus that may condition the elastic recoil capacity and thereby inhibit the shrinkage even in a depressurised sac.

The accumulation of intra-sac fluid after EVAR has been previously associated with the formation of hygroma.\(^\text{6}\) Previous reports have associated this condition with the risk of AAA expansion with or without simultaneous pressurisation of the AAA sac.\(^\text{5}\) In our series, the accumulation of intra-sac fluid did not lead to significant pressurisation of the AAA sac and there was no relation between the intra-sac pressure and the subsequent diameter change. Patients accumulating intra-sac fluid have, therefore, a reduced benefit from a pressure measurement, since there seems to be no association between the level of intra-sac pressurisation and AAA diameter change. A direct puncture of the AAA may, nevertheless, have a good diagnostic value as it allows the identification of this group of patients with intra-sac fluid. This finding is especially important in expanding AAAs, since it allows the distinction between endotension without fluid accumulation and those with hygroma and concomitant diameter expansion. Endotension without fluid accumulation constitutes an indication for a prophylactic re-intervention even when patients are asymptomatic, while hygroma with diameter expansion is usually managed conservatively until patients become symptomatic.\(^\text{3}\) In these instances, the translumbar puncture may also have a therapeutic value since it leads to shrinkage of the AAA (unpublished data).

The low intra-sac pressure in patients with intra-sac fluid may be related to the timing of the puncture. The fluid will most likely only lead to a pressurisation of the sac when the volume increases beyond the level at which the distension capacity of the aneurysm wall is surpassed. We could not confirm this hypothesis in this study since all patients had low intra-sac pressures and our protocol did not register the exact volume drained from the AAA sac, which limits a proper analysis (from our experience the volume is usually in the range of 50–100 ml). Furthermore, the technique used cannot exclude the accumulation of small amounts of fluid in areas encased by a low-permeability thrombus not reached by the needle. This seems, nevertheless, less likely taking into consideration the relatively high volumes drained compared with the total AAA volumes reported in the literature.\(^\text{15,20}\)

Although we recognise the limitations to this study, these data have a scientific value in the understanding of the pathophysiology of aneurysm sac behaviour after EVAR. However, the clinical value of pressure measurements in patients with unchanged AAAs after EVAR is questionable at this moment. Direct puncture of the AAA may, nevertheless, be still advantageous in patients with overt expansion to identify the accumulation of intra-sac fluid. However, the periodic assessment of AAA size either by measuring diameters or volumes must continue to be considered the mainstay of the follow-up after EVAR.

In conclusion, AAAs without endoleak that are unchanged in diameter more than 1 year after EVAR will often remain unchanged. Subsequent expansion can occur and is associated with higher MPIs and intra-aneurysm sac pulse pressures. There seems to be a subgroup of patients where expansion is associated with the accumulation of intra-sac fluid without significant pressurisation of the AAA sac. The translumbar puncture of the AAA allows the identification of these patients, but the pressure measurement is of limited value, considering the low intra-sac pressure.

Conflict of Interest

None.

Acknowledgements

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