# Intra-aneurysm sac pressure measurements after endovascular aneurysm repair: Differences between shrinking, unchanged, and expanding aneurysms with and without endoleaks

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***Objective:*** **Our objective was to study intra-aneurysm pressure after endovascular aneurysm repair (EVAR) in shrinking, unchanged, and expanding abdominal aortic aneurysms (AAAs) with and without endoleaks.**
***Methods:*** **Direct intra-aneurysm sac pressure measurement (DISP) by percutaneous translumbar puncture of the AAA under fluoroscopic guidance was performed 46 times during the follow-up of 37 patients (30 men; median age, 73 years [range, 58-82 years]; AAA diameter: median, 60 mm [range, 48-84 mm]). Three patients were included in two different groups because DISP was performed more than once with different indications. Tip-pressure sensors mounted on 0.014-inch guidewires were used for simultaneous measurement of systemic and AAA sac pressures. Mean pressure index (MPI) was calculated as the percentage of mean intra-aneurysm pressure relative to the simultaneous mean intra-aortic pressure.**
***Results:*** **Median MPI was 19% in shrinking (11 patients), 30% in unchanged (10 patients), and 59% in expanding (9 patients) aneurysms without endoleaks. Pulse pressure was also higher in expanding (10 mm Hg) compared with shrinking (2 mm Hg; $P < .0001$) AAAs. Four of the nine patients with expanding AAAs underwent five repeated DISPs later in the follow-up, and MPIs were consistently elevated. Seven of the 10 patients with unchanged AAAs without endoleaks underwent further computed tomography follow-up after DISP; 2 expanded (MPI, 47%-63%), 4 shrank (MPI, 21%-30%), and 1 remained unchanged (MPI, 14%). Type II endoleaks (6 patients, 7 DISPs) were associated with wide range of MPI (22%-92%). Successful endoleak embolization (n = 4) resulted in pressure reduction.**
***Conclusions:*** **Intra-aneurysm sac pressure measurement is an important adjunctive for EVAR evaluation, possibly allowing early detection of failures. High pressure is associated with AAA expansion and low pressure with shrinkage. Type II endoleaks can be responsible for AAA pressurization, and successful embolization appears to result in pressure reduction. (J Vasc Surg 2004;39:1229-35.)**

The goal of endovascular aneurysm repair (EVAR) of infrarenal abdominal aortic aneurysms (AAAs) is to avoid rupture through the exclusion of the aneurysm from both blood flow and systemic pressure. The achievement of this goal is usually determined indirectly by imaging of the aneurysm size and endoleaks.

Attempts have been made at direct assessment of EVAR by intra-aneurysm pressure measurements, mainly intraoperatively. Successful EVAR at least 1 year after the operation has been shown to lead to reduction of intra-aneurysm pressure and pulsatility.[1] On the contrary, endoleaks can be associated with near-systemic intra-aneurysm pressure within the endoleak channel.[2,3] It has also been demonstrated that aneurysm pressurization and expansion can occur in the absence of endoleaks, perhaps as a result of pressure transmission through thrombus.[4-8]

The aim of this study was to analyze the relation between intra-aneurysm pressure and AAA diameter changes after EVAR both with and without endoleaks.

## METHODS

Between November 1993 and August 2003, 329 patients underwent EVAR for AAA at our institution. Since October 2000, 37 of these patients (30 men/7 women; median age, 73 years [range, 58-82 years]; preoperative AAA diameter: median, 60 mm [range, 48-84 mm]) underwent 46 direct intra-aneurysm sac pressure measurements (DISPs). This technique was initially used and reported[1] in AAAs that had shrunk in diameter in the absence of endoleaks at least 1 year after EVAR. In the present study 11 patients with the same characteristics are included. DISP was also performed in patients displaying expanding (nine patients) or unchanged aneurysm diameter after EVAR (10 patients) without endoleaks. Four of the nine patients with expanding aneurysms underwent five repeated DISPs later in the follow-up. Six patients (seven DISPs) with type II endoleaks and four patients after type II endoleak emboli-

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**Table I.** Summary of DISP in AAAs without endoleaks

| *AAA diameter change* | *No. of patients* | *Follow-up before DISP (mo)* | *AAA diameter (mm)* | *ΔØ AAA (mm)* | *AAA pressure* | | |
|---|---|---|---|---|---|---|---|
| | | | | | *Systolic/diastolic/mean (mm Hg)* | *Pulse pressure (mm Hg)* | *MPI (%)* |
| Shrinking | 11 | 19 (14-44) | 54 (34-63) | −9 (−21, −6) | 19/18/19 (16-35)/(13-33)/(15-31) | 2 (0-6) | 19 (12-39) |
| Expanding | 9 | 38 (13-100) | 57 (52-76) | 6 (+5, +11) | 67/60/63 (48-112)/(31-87)/39-97) | 10 (3-30) | 59 (27-98) |
| Unchanged | 10 | 18 (1-94) | 60 (47-84) | 0 (−3, +4) | 32/26/29 (13-64)/(12-57)/(13-61) | 6 (1-12) | 30 (14-63) |

Values are presented as median with 5th and 95th percentiles between parentheses.
*AAA*, Abdominal aortic aneurysm; ΔØ, diameter change; *MPI*, mean pressure index.

zation underwent DISP. Three of the 37 patients were included in different groups because they were measured at two different occasions. In two patients measurements were performed before and after type II endoleak embolization; in the third patient DISP was performed first while a type II endoleak was present and 2 years later after spontaneous seal of the endoleak when the AAA had expanded.

Anatomic suitability for translumbar AAA puncture for DISP was determined from axial computed tomography (CT) scan performed in the month before DISP. It was defined as a large enough aneurysm sac that allowed a safe translumbar needle placement without entering the peritoneal cavity or running a high risk of damaging the stent graft.

The study was approved by the Institutional Ethics Committee, and all patients gave their informed consent before the procedure.

**Imaging.** Pre-EVAR imaging consisted of digital subtraction angiography and a spiral CT before and after iodinated contrast enhancement. Imaging follow-up after EVAR included contrast-enhanced spiral CT with delayed scan at 1 month and yearly thereafter. At the time of DISP an aortogram with selective angiography of superior mesenteric artery and hypogastric arteries was done if a type II endoleak was suspected. To avoid overestimation errors caused by vessel tortuosity, AAA diameter was measured in axial CT scans as the perpendicular to the maximum diameter or the minor axis when the aneurysm cross-section appeared elliptical. AAA diameter changes ≥5 mm after at least 1 year were considered significant.[9]

Diameter changes used for comparison with DISP pressure in this study were calculated to express the diameter evolution before DISP. In shrinking AAAs the diameter change was calculated as the difference between the diameter at the time of DISP and the preceding maximum AAA diameter, regardless of whether that maximum diameter occurred before or after EVAR. The reverse was used to calculate diameter change for expanding AAAs by using the difference between the diameter at the time of DISP and the preceding minimum aneurysm diameter. For instance, if there was an initial aneurysm diameter decrease after EVAR and then it increased back to the pre-EVAR level, the AAA diameter change was calculated as the difference between the smallest diameter and the diameter at the time of DISP. Diameter changes were considered as positive in expanding aneurysms and negative in the shrinking ones.

**DISP technique.** The technique of DISP is described in detail elsewhere.[1]. It consisted of percutaneous translumbar puncture of the AAA sac under fluoroscopic guidance with the down-the-barrel technique. Iodinated contrast medium was always injected into the aneurysm sac, ie, aneurysmography, to confirm the needle position inside the AAA and to obtain any additional information such as the possible presence of previously undiagnosed endoleaks. On no occasion were previously undiagnosed endoleaks demonstrated by direct injection of iodinated contrast medium into the aneurysm sac (aneurysmography).

Pressure was measured simultaneously within the stent graft lumen and the AAA sac. The pressure sensor for systemic pressure measurement was inserted transfemorally with a modified Seldinger technique and passed into the stent graft lumen. It was a tip-pressure sensor premounted on a 0.014-inch guidewire with a 3-cm floppy tip (PressureWire; RADI Medical System AB, Uppsala, Sweden). A similar wire with the tip shortened by the manufacturer to 1 mm was used for intra-aneurysm pressure measurement. The shorter tip allowed a more accurate placement within the AAA sac. The measurements were only considered valid when there was both a decrease in pressure on withdrawal of the pressure sensor from the AAA sac into the retroperitoneal space, and the recalibration after the measurement was within 5 mm Hg of the initial zero.

DISP pressure values were the result of automatic calculation of the mean of readings obtained during 10 consecutive heart cycles. Readings included were performed while the pressure sensor was located within the thrombus approximately mid-distance between the stent graft and the AAA wall. Mean pressure index (MPI) was calculated as the percentage of the mean intra-aneurysmal pressure relative to the simultaneous mean systemic pressure.

**Embolization of type II endoleaks.** Endoluminal embolizations were performed with coils and/or radiopaque glue (Hystoacryl; Braun, Tuttlingen, Germany and Lipiodol; Laboratoire Guerbet, Aulnay-Sous-Bois, France) whenever considered indicated. For translumbar embolization radiopaque glue was used exclusively either through the needle or through a catheter.

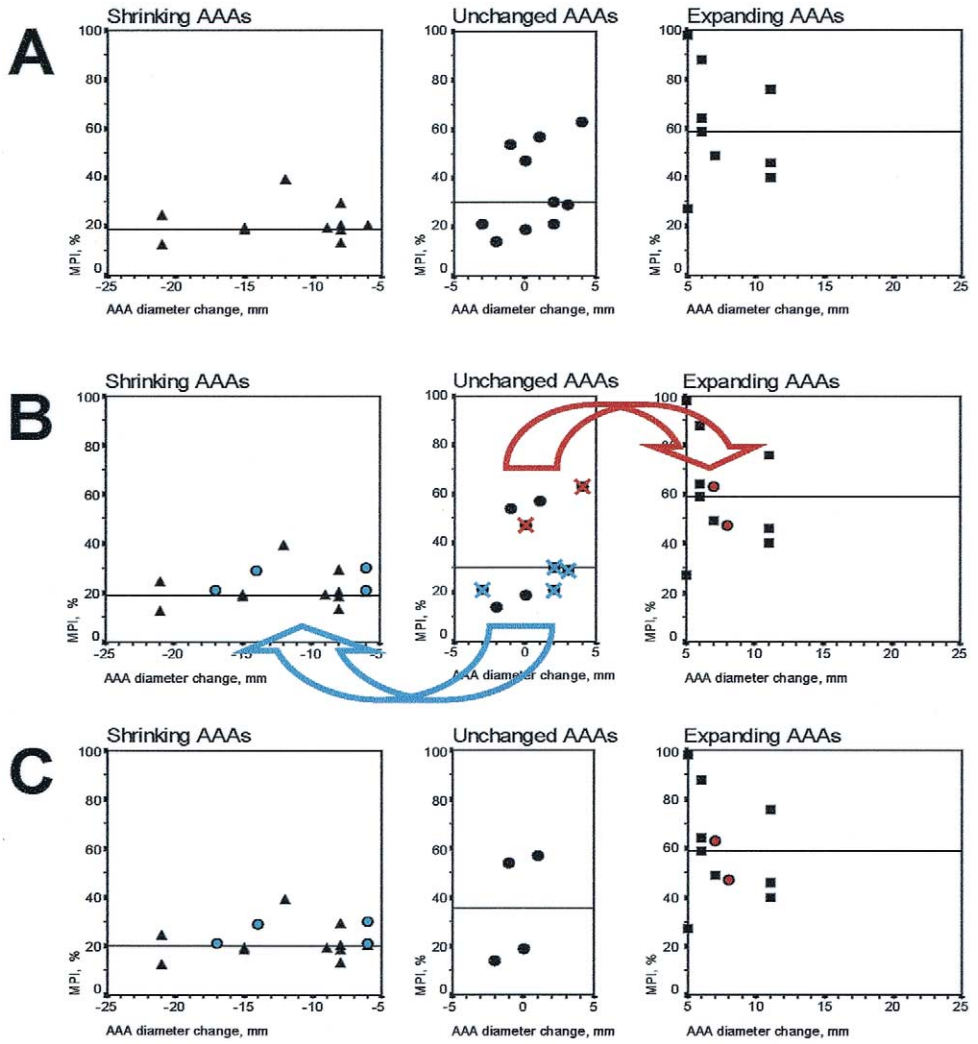


**Fig 1. A,** MPI versus AAA diameter change at the time of DISP. *Triangles, circles, and squares* represent patients with AAAs that shrank, remained unchanged, or expanded in diameter, respectively, before DISP. *Horizontal lines* represent median MPI in each group (19% for shrinking, 30% for unchanged, and 59% for expanding). **B,** AAA diameter evolution during CT follow-up after DISP in patients with initially unchanged AAA. *Arrows* illustrate the tendency of the diameter change (expanding or shrinking). *Crosses* represent patient status at the time of DISP, and *circles* represent the same patients after continued follow-up. Two patients had AAA expansion *(red-color crosses, circles, and arrow)*, and four patients had AAA shrinkage *(blue crosses, circles, and arrow)*. Of the remaining four patients with unchanged diameter at the time of DISP, only one patient underwent further CT follow-up after DISP (remained unchanged). **C,** MPI vs AAA diameter changes after the continued CT follow-up. Median MPI of patients with shrinking and expanding aneurysms did not change significantly from the initial values (20% for shrinking and 59% for expanding AAAs). *AAA*, Abdominal aortic aneurysm; *MPI*, mean pressure index.

**Statistics.** Normal distribution was not assumed. Values are presented as medians and 5th and 95th percentiles between parentheses when not stated otherwise. Mann-Whitney test was used for unpaired comparisons. Results were considered significant when $P < .05$. Exact $P$ values are presented whenever statistically significant. Absolute pressure values are described as systolic/diastolic (mean) in mm Hg. Statistical analysis was done with SPSS 11.5.1 software (SPSS Inc, Chicago, Ill).

## RESULTS

### Aneurysms without endoleaks

**Shrinking AAAs.** Eleven patients (9 men/2 women; age, 77 years [57-83 years]) with shrinking aneurysms after EVAR underwent DISP at 19 (14-44) months of follow-up. Ten of these patients were included in our initial study with DISP.[1] The median MPI was 19% (12%-39%), and the

**Table II.** Repeating DISP in patients with expanding aneurysm diameter after endovascular aneurysm repair

| *Patient* | *Follow-up before DISP (mo)* | *ΔØ AAA (mm)* | *AAA pressure* | |
|---|---|---|---|---|
| | | | *Systolic/diastolic (mean) (mm Hg)* | *MPI (%)* |
| 1 | 22 | +6 | 66/60 (63) | 59 |
| | 32 | +11 | 78/71 (74) | 60 |
| 2 | 24 | +6 | 86/76 (80) | 65 |
| | 56 | +14 | 53/46 (49) | 46 |
| 3 | 38 | +7 | 67/57 (51) | 49 |
| | 47 | +11 | 60/55 (57) | 60 |
| 4 | 39 | +11 | 109/87 (96) | 76 |
| | 45 | +10 | 88/73 (79) | 63 |
| | 49 | +10 | 64/52 (58) | 59 |

In first three patients DISP was repeated once and in the last patient twice. MPIs remained consistently higher than in shrinking AAAs. The first DISP for each patient in this table was used for comparative analysis with the other groups (Table I and Fig 1).
*DISP*, Direct intra-aneurysm sac pressure measurement; ΔØ, diameter change; *AAA*, abdominal aortic aneurysm; *MPI*, mean pressure index.

intra-aneurysm pulse pressure was 2 mm Hg (0-6 mm Hg) in these patients (Table I).

**Expanding AAAs.** Nine patients (7 men/2 women; age, 74 years [71-80 years]) with expanding aneurysm diameter after EVAR underwent DISP at a median 38 months (13-100 months) of follow-up (Table I). Median MPI was 59% (27%-98%), which was higher than in shrinking aneurysms ($P < .0001$; Fig 1, *A*). The intra-aneurysm systolic (67 mm Hg [48-112 mm Hg]), diastolic (60 mm Hg [31-87 mm Hg]), mean (63 mm Hg [39-97 mm Hg]), and pulse (10 mm Hg [3-30 mm Hg]) pressures were also higher compared with shrinking aneurysms ($P < .0001$).

In four of the nine patients with expanding aneurysms, DISP was repeated later in the follow-up, and MPIs remained consistently higher than in shrinking aneurysms (Table II).

Within this group of nine patients with expanding AAAs there were five patients who exhibited significant dilatation of the infrarenal neck (4-5 mm). In two other patients the stent graft was inadvertently deployed in a too distal position in the neck. In the remaining two patients no obvious explanation for AAA expansion was found.

**Unchanged AAAs.** DISP was performed in 10 patients (9 men/1 woman; age, 72 years [66-78 years]) with unchanged AAA diameter 18 months (1-94 months) after EVAR (Table I). The findings were diverse in terms of MPIs (30% [14%-63%]) and pulse pressure (6.50 mm Hg [1.00-12.00 mm Hg]).

Seven of these 10 patients underwent further contrast-enhanced CT scans at least 3 months after DISP and until any reintervention (Table III). Considering the total diameter changes in the same direction (ie, shrinking, expanding, or unchanged) for the entire follow-up, ie, both before and after DISP, four patients with MPIs between 21% and 30% exhibited AAA shrinkage after 10 to 19 months (diameter change, −17 to −6 mm). Two patients with MPI of 63% and 47% displayed aneurysm expansion (+7 mm over 17 months and +8 mm over 22 months, respectively). In these two patients a possible failure of the proximal seal was identified (migration and neck dilatation, respectively). The remaining patient with further CT follow-up had an MPI of 14% and showed no diameter change after DISP (−2 mm; Fig 1, *B*).

Three of the 10 patients with unchanged AAA diameter did not undergo further CT follow-up after DISP. At the time of DISP, one exhibited distal migration of the stent graft (10mm) and MPI of 57%. Another one had proximal sealing zone of the stent graft of 9 mm in an angulated neck, and the MPI was 54%. The third patient had an MPI of 19% 52 months after the EVAR.

### Aneurysms with endoleaks

**DISP in patients with type II endoleak.** Six patients (3 men/3 women; age, 79 years [69-87 years]) underwent DISP when a type II endoleak was present (Table IV and Fig 2). In one patient (patient 1, Table IV) the endoleak sealed spontaneously 1.5 months after DISP (MPI, 37%). Twenty-three months later DISP was repeated in this patient because the AAA had expanded 5 mm (measurement included in the Expanding AAAs without endoleaks group; MPI, 27%). Another patient with a lumbar endoleak (patient 2; Table IV) underwent DISP twice, first when the AAA diameter had shrunk (MPI, 22%) and later when it had expanded (MPI, 68%).

**DISP after embolization of type II endoleaks.** Four patients (3 men/1 woman; age, 76 years [71-81 years]) underwent DISP after embolization of type II endoleaks (after 3 months in one patient [patient 1; Table V] and after more than 2 years in the remaining three patients) (Table V). There were no detectable endoleaks in all four patients. In two patients (patients 1 and 3; Table V) DISP was performed before and after embolization. The remaining two patients underwent DISP only after endoleak embolization.

## DISCUSSION

EVAR aims at depressurization of the aneurysm sac and thereby to avoid its expansion, which can culminate in rupture and life-threatening hemorrhage. We have previously shown that AAA shrinkage after successful EVAR is associated with a significant reduction of the intra-aneurysm pressure and pulsatility.[1] The present data reinforce those findings. MPI of 19% in patients with shrinking AAAs in the absence of endoleaks is lower than that previously reported for residual intraoperative intra-aneurysm pressure both after clamping at the time of open repair of AAA[10-12] and after stent graft deployment.[11,13-15] Although no direct comparisons can be made between our results and these studies because of the different timing and measurement techniques used, this difference might indicate that the pressure reduction within the AAA takes place with time, explaining the previously reported delay of AAA shrinkage after successful EVAR.[16,17]

**Table III.** CT follow-up after DISP in patients initially unchanged in diameter in the absence of endoleaks

| *Patient* | *ΔØ AAA before DISP (mm)* | *Follow-up before DISP (mo)* | *AAA pressure* | | *Follow-up after DISP (mo)* | *ΔØ AAA after DISP (mm)* |
|---|---|---|---|---|---|---|
| | | | *Systolic/diastolic (mean) (mm Hg)* | *MPI (%)* | | |
| 1 | +4 | 16 | 59/53 (56) | 63 | 17 | +3 (Expand) |
| 2 | 0 | 14 | 61/49 (55) | 47 | 22 | +8 (Expand) |
| 3 | +2 | 35 | 31/23 (27) | 30 | 13 | −6 (Shrink) |
| 4 | +3 | 14 | 33/30 (31) | 29 | 19 | −14 (Shrink) |
| 5 | +2 | 1 | 25/22 (23) | 21 | 14 | −17 (Shrink) |
| 6 | −3 | 53 | 28/20 (24) | 21 | 10 | −3 (Shrink) |
| 7 | −2 | 20 | 13/12 (13) | 14 | 16 | 0 (Unchanged) |

ΔØ, Diameter change; *AAA*, abdominal aneurysm repair; *DISP*, direct intra-aneurysm sac pressure measurement; *MPI*, mean pressure index.

**Table IV.** DISP in patients with type II endoleaks

| *Patient* | *Endoleak origin* | *Follow-up before DISP (mo)* | *ΔØ AAA (mm)* | *AAA pressure* | |
|---|---|---|---|---|---|
| | | | | *Systolic/diastolic (mean) (mm Hg)* | *MPI (%)* |
| 1 | Lumbar | 4.5 | −5 | 53/46 (49) | 37 |
| 2 | Lumbar | 23 | −5 | 24/18 (19) | 22 |
| | Lumbar | 34 | +11 | 71/58 (65) | 68 |
| 3 | Lumbar | 23 | +6 | 141/102 (122) | 92 |
| 4 | Lumbar | 22 | −1 | 71/62 (67) | 67 |
| 5 | Accessory renal | 15 | +2 | 96/74 (87) | 70 |
| 6 | Lumbar | 14 | +3 | 56/35 (45) | 37 |

Patient 2 underwent DISP twice with no reintervention in between. Initially the AAA shrank, and MPI was low. At the time of the second DISP the AAA had expanded, and MPI was higher.
*DISP*, Direct intra-aneurysm sac pressure measurement; ΔØ, diameter change; *AAA*, abdominal aortic aneurysm; *MPI*, mean pressure index.

**Table V.** DISP in patients who had previously undergone embolization of type II endoleaks

| *Patient* | *MPI before embolization (%)* | *AAA pressure* | | *Follow-up after embolization (mo)* | *ΔØ AAA after embolization (mm)* |
|---|---|---|---|---|---|
| | | *Systolic/diastolic (mean) (mm Hg)* | *MPI after embolization (%)* | | |
| 1 | 70 | 34/28 (30) | 31 | 21 | −5 |
| 2 | NA | 22/19 (20) | 13 | 46 | −15 |
| 3 | 67 | 25/18 (21) | 19 | 29 | +2 |
| 4 | NA | 53/40 (45) | 42 | 28 | +1 |

Patient 1 was previously described as patient 5 of DISP of type II endoleaks (Table IV). Patient 3 was previously described as patient 4 of DISP of type II endoleaks (Table IV).
*DISP*, Direct intra-aneurysm sac pressure measurement; *AAA*, abdominal aortic aneurysm; ΔØ, diameter change; *MPI*, mean pressure index; *NA,* not available.

Aneurysm expansion in the absence of endoleaks, a condition known as endotension,[9,18] has been attributed to blood flow within the AAA sac below the sensitivity of current imaging methods,[19] or pressure transmission through thrombus[4] or graft material.[20] However, this is, to the best of our knowledge, the first study in which expanding AAAs after EVAR in the absence of endoleaks are associated with significantly higher intra-aneurysm pressure and greater pulse pressures than shrinking aneurysms. This aneurysm pressurization also seems to persist over time, considering the consistently high MPIs in those patients with enlarging aneurysms undergoing repeated DISPs. In these situations the possible causes of AAA pressurization should be explored. In the majority of our patients it was most likely due to inadequate proximal seal, caused by neck dilatation or malpositioning of the stent graft.[21,22]

Patients with unchanged AAAs without endoleaks after EVAR appear to constitute a heterogeneous group in terms of intra-aneurysm pressure. In the two patients with intra-aneurysm pressure similar to the values of those with expanding aneurysms, the aneurysms subsequently expanded, whereas in four patients with lower pressures the aneurysms shrank (Fig 1). This corresponds to the expectations and might reflect a time lag for the pressure changes to be

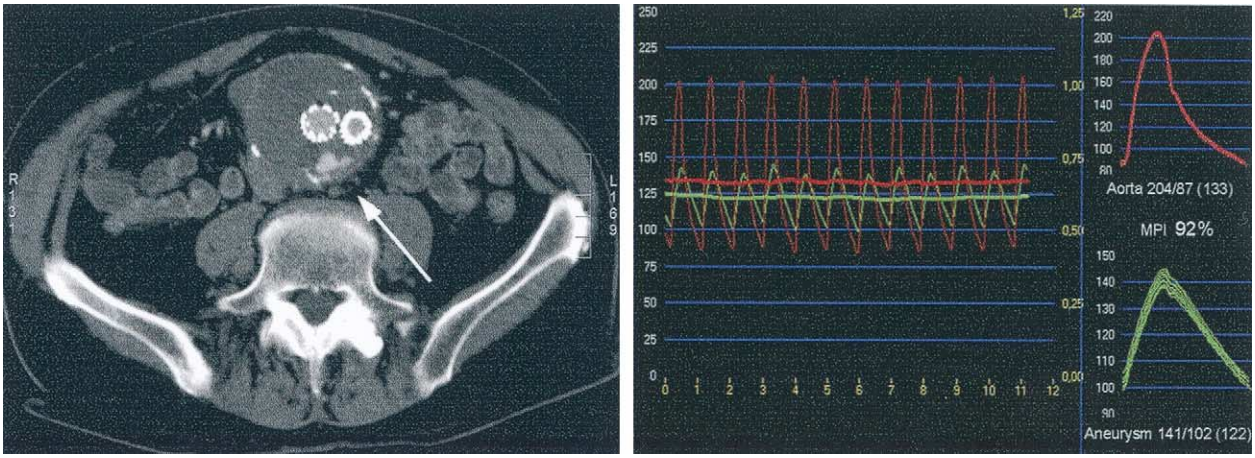


**Fig 2.** Computed tomography scan and recordings from direct intra-aneurysm sac pressure measurement in a patient with type II endoleak. A 6-mm abdominal aortic aneurysm diameter increase had occurred in the 23 months after endovascular aneurysm repair. Mean pressure index was 92%. *Red curves* represent the aortic pressure, and *green curves* represent the intra-aneurysm-sac pressure.

displayed by aneurysm diameter remodeling. After the continued follow-up, ie, considering the total diameter changes both before and after DISP, there were 15 patients with AAAs that shrank in diameter and 11 that expanded. The median MPI in expanding aneurysms (59%) continued to be significantly higher than the shrinking ones (20%; $P < .0001$; Fig 1, *C*). Pressure measurement seems, therefore, to be able to predict the degree of aneurysm exclusion, discriminating patients who will eventually exhibit aneurysm expansion from those expected to shrink or continue unchanged. Furthermore, it seems tempting to conclude that MPI above approximately 35% indicates a subsequent AAA expansion. However, this is still too small a series of patients to allow us any definitive conclusions. In addition, this does not imply that imaging follow-up can be replaced by pressure measurements. The information provided by DISP can only be interpreted when noninvasive imaging such as plain abdominal films and CT scans are taken into consideration. In patients without evidence of anatomic changes such as stent graft migration or deformation, aneurysm neck dilatation, and change of endoleak status, a low intra-aneurysm pressure provides further information to allow the safe continuation of routine imaging follow-up, which in our institution includes yearly CT scans and plain abdominal films. On the contrary, a high intra-aneurysm pressure requires an intensification of the imaging follow-up to facilitate any decision on possible reinterventions at earlier stage.

Type II endoleaks have been considered a diverse entity associated with varied outcomes regarding aneurysm size including sporadic rupture.[23-31] Type II endoleaks have been reported to be associated with systemic pressure when measured within the endoleak channel/nidus early in the follow-up.[2,3] In our series, type II endoleaks appear to be a varied and dynamic entity that can be responsible for different degrees of pressurization of the aneurysm sac even within the same patient at different time points. The difference with the previously mentioned studies[2,3] might be explained by the fact that type II endoleaks have a systemic perfusion pressure within the lumen/nidus. However, their low flow can condition the degree of pressure transmission and, thereby, the pressure level within the thrombus in which we performed our measurements. Successful type II endoleak embolization was associated with decrease of the intra-aneurysm pressure and decrease or no change of the aneurysm diameter. A remark should be made about the CT follow-up after embolization with glue and Lipiodol, because the radiopaque Lipiodol remnants made it difficult to detect persistent type II endoleaks with certainty.

In conclusion, high intra-aneurysm pressure is found to be associated with AAA enlargement and low pressure with shrinkage. A time lag might be needed for these pressure changes to be expressed by aneurysm remodeling and thereby identified by imaging methods. DISP is a new and promising tool for EVAR evaluation. It provides information allowing for an early detection of future failures and optimization of the follow-up after EVAR, especially when combined with noninvasive imaging techniques. It is, therefore, our current policy to seek treatable causes of AAA repressurization after EVAR even if the aneurysm diameter is unchanged. In contrast, if the pressure is low and the diameter is unchanged, the conservative imaging follow-up program is continued. However, a definitive pressure threshold for intervention needs to be defined by further studies.

## REFERENCES

1. Sonesson B, Dias N, Malina M, Olofsson P, Griffin D, Lindblad B, et al. Intra-aneurysm pressure measurements in successfully excluded abdominal aortic aneurysm after endovascular repair. J Vasc Surg 2003;37:733-8.

2. Baum RA, Carpenter JP, Cope C, Golden MA, Velazquez OC, Neschis DG, et al. Aneurysm sac pressure measurements after endovascular repair of abdominal aortic aneurysms. J Vasc Surg 2001;33:32-41.
3. Velazquez OC, Baum RA, Carpenter JP, Golden MA, Cohn M, Pyeron A, et al. Relationship between preoperative patency of the inferior mesenteric artery and subsequent occurrence of type II endoleak in patients undergoing endovascular repair of abdominal aortic aneurysms. J Vasc Surg 2000;32:777-88.
4. Schurink GW, van Baalen JM, Visser MJ, van Bockel JH. Thrombus within an aortic aneurysm does not reduce pressure on the aneurysmal wall. J Vasc Surg 2000;31:501-6.
5. Lin PH, Bush RL, Katzman JB, Zemel G, Puente OA, Katzen BT, et al. Delayed aortic aneurysm enlargement due to endotension after endovascular abdominal aortic aneurysm repair. J Vasc Surg 2003;38:840-2.
6. White GH, May J, Petrasek P, Waugh R, Sthephen M, Harris J. Endotension: an explanation for continued AAA growth after successful endoluminal repair. J Endovasc Surg 1999;6:308-15.
7. Veith FJ, Baum RA, Ohki T, Amor M, Adiseshiah M, Blankensteijn JD, et al. Nature and significance of endoleaks and endotension: summary of opinions expressed at an international conference. J Vasc Surg 2002;35: 1029-35.
8. Malina M, Länne T, Ivancev K, Lindblad B, Brunkwall J. Reduced pulsatile wall motion of abdominal aortic aneurysms after endovascular repair. J Vasc Surg 1998;27:624-31.
9. 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;35:1048-60.
10. Hans SS, Jareunpoon O, Huang R, Hans B, Bove P, Zelenock GB. Relationship of residual intraluminal to intrathrombotic pressure in a closed aneurysmal sac. J Vasc Surg 2003;37:949-53.
11. Vallabhaneni SR, Gilling-Smith GL, How TV, Brennan JA, Gould DA, McWilliams RG, et al. Aortic side branch perfusion alone does not account for high intra-sac pressure after endovascular repair (EVAR) in the absence of graft-related endoleak. Eur J Vasc Endovasc Surg 2003; 25:354-9.
12. Gawenda M, Brunkwall J. Endotension in the closed aneurysm sac during aortic and iliac cross clamping: implications of pressure and flow measurements. J Endovasc Ther 2003;10:760-5.
13. Gawenda M, Heckenkamp J, Zaehringer M, Brunkwall J. Intra-aneurysm sac pressure: the holy grail of endoluminal grafting of AAA. Eur J Vasc Endovasc Surg 2002;24:139-45.
14. Chuter T, Ivancev K, Malina M, Resch T, Brunkwall J, Lindblad B, et al. Aneurysm pressure following endovascular exclusion. Eur J Vasc Endovasc Surg 1997;13:85-7.
15. Treharne GD, Loftus IM, Thompson MM, Lennard N, Smith J, Fishwick G, et al. Quality control during endovascular aneurysm repair: monitoring aneurysmal sac pressure and superficial femoral artery flow velocity. J Endovasc Surg 1999;6:239-45.
16. Malina M, Ivancev K, Chuter TA, Lindh M, Länne T, Lindblad B, et al. Changing aneurysmal morphology after endovascular grafting: relation to leakage or persistent perfusion. J Endovasc Surg 1997;4:23-30.
17. Prinssen M, Blankensteijn JD. The sac shrinking process after EAR does not start immediately in most patients. Eur J Vasc Endovasc Surg 2002;23:426-30.
18. Gilling-Smith G, Brennan J, Harris P, Bakran A, Gould D, McWilliams R. Endotension after endovascular aneurysm repair: definition, classification, and strategies for surveillance and intervention. J Endovasc Surg 1999;6:305-7.
19. Schurink GW, Aarts NJ, Wilde J, van Baalen JM, Chuter TA, Schultze Kool LJ, et al. Endoleakage after stent-graft treatment of abdominal aneurysm: implications on pressure and imaging—an in vitro study. J Vasc Surg 1998;28:234-41.
20. Gawenda M, Jaschke G, Winter S, Wassmer G, Brunkwall J. Endotension as a result of pressure transmission through the graft following endovascular aneurysm repair: an in vitro study. Eur J Vasc Endovasc Surg 2003;26:501-5.
21. Makaroun MS, Deaton DH. Is proximal aortic neck dilatation after endovascular aneurysm exclusion a cause for concern? J Vasc Surg 2001;33(suppl):S39-45.
22. Wever JJ, de Nie AJ, Blankensteijn JD, Broeders IA, Mali WP, Eikelboom BC. Dilatation of the proximal neck of infrarenal aortic aneurysms after endovascular AAA repair. Eur J Vasc Endovasc Surg 2000; 19:197-201.
23. Resch T, Ivancev K, Lindh M, Nyman U, Brunkwall J, Malina M, et al. Persistent collateral perfusion of abdominal aortic aneurysm after endovascular repair does not lead to progressive change in aneurysm diameter. J Vasc Surg 1998;28:242-9.
24. Rhee RY, Eskandari MK, Zajko AB, Makaroun MS. Long-term fate of the aneurysmal sac after endoluminal exclusion of abdominal aortic aneurysms. J Vasc Surg 2000;32:689-96.
25. White RA, Donayre C, Walot I, Stewart M. Abdominal aortic aneurysm rupture following endoluminal graft deployment: report of a predictable event. J Endovasc Ther 2000;7:257-62.
26. Arko FR, Rubin GD, Johnson BL, Hill BB, Fogarty TJ, Zarins CK. Type-II endoleaks following endovascular AAA repair: preoperative predictors and long-term effects. J Endovasc Ther 2001;8:503-10.
27. Hinchliffe RJ, Singh-Ranger R, Davidson IR, Hopkinson BR. Rupture of an abdominal aortic aneurysm secondary to type II endoleak. Eur J Vasc Endovasc Surg 2001;22:563-5.
28. White RA, Walot I, Donayre CE, Woody J, Kopchok GE. Failed AAA endograft exclusion due to type II endoleak: explant analysis. J Endovasc Ther 2001;8:254-61.
29. Parry DJ, Kessel DO, Robertson I, Denton L, Patel JV, Berridge DC, et al. Type II endoleaks: predictable, preventable, and sometimes treatable? J Vasc Surg 2002;36:105-10.
30. Dattilo JB, Brewster DC, Fan CM, Geller SC, Cambria RP, Lamuraglia GM, et al. Clinical failures of endovascular abdominal aortic aneurysm repair: incidence, causes, and management. J Vasc Surg 2002;35:1137-44.
31. Fransen GA, Vallabhaneni SR , Van Marrewijk CJ, Laheij RJ, Harris PL, Buth J. Rupture of infra-renal aortic aneurysm after endovascular repair: a series from EUROSTAR Registry. Eur J Vasc Endovasc Surg 2003; 26:487-93.

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