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Reduced pulsatile wall motion of abdominal aortic aneurysms after endovascular repair

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Purpose: **The reduced size of abdominal aortic aneurysms (AAAs) after endovascular repair suggests lowered intraaneurysmal pressure. In the presence of endoleaks, the size is not decreased. Although postoperative intraaneurysmal pressure is difficult to record, the pulsatile wall motion (PWM) of aneurysms can be measured noninvasively. The aim of this study was to assess the PWM of AAAs before and after endovascular repair and to relate the change in the PWM to aneurysmal size and presence of endoleaks.**
Methods: **Forty-seven patients underwent endovascular repair of an AAA. The aneurysm diameter and PWM were measured with the use of ultrasonic echo-tracking scans preoperatively; at 1, 3, and 6 months; and thereafter biannually. Fifteen aneurysms developed endoleaks, whereas 32 were completely excluded. The leaks were characterized with the use of computed tomographic scanning and angiography. Median follow-up was 12 months (interquartile range, 5 to 24 months).**
Results: **The preoperative PWM of the aneurysms was 1.0 mm (range, 0.8 to 1.3 mm). After complete endovascular exclusion, the PWM was 25% (range, 16% to 37%) of the preoperative value ($p < 0.001$), and aneurysm diameter decreased by 8 mm (range, 6 to 14 mm) ($p < 0.001$). After 18 months, no further diameter reduction occurred. In three patients without endoleaks but with enlarging aneurysms, the postoperative PWM showed less reduction ($p < 0.05$). Aneurysms with endoleaks showed no diameter decrease, and the postoperative PWM was 50% higher than that in the totally excluded cases ($p < 0.01$). In five patients with transient endoleaks, the PWM was reduced after leakage ceased ($p < 0.05$). Leaks of various sources displayed similar PWM.**
Conclusion: **The size and PWM of aneurysms are reduced after endovascular repair. The diameter reduction may cease after 1.5 years. Endoleaks are associated with higher PWM than expected. Pressure may be transmitted without evidence of leaks. (J Vasc Surg 1998;27:624-31.)**

Thousands of patients with abdominal aortic aneurysms (AAAs) have been treated with endovascular repair; midterm follow-up data are available.[1-11] Initially, the fate of the excluded aneurysmal sac was unknown, but recently, several investigators have reported decreasing aneurysm diameters after successful endovascular exclusion.[12-14]

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However, about one third of endovascularly treated AAAs maintain their size after stent grafting. It seems that even minor, asymptomatic collateral perfusion of the sac prevents aneurysm retraction.[13,14] Late rupture of the aneurysmal sac has been reported after both endovascular[15-17] and conventional[18] exclusion.

Endoleaks are best visualized with computed tomography (CT), which readily detects contrast accumulation outside the graft. Combined assessment with the use of CT and angiography usually can detect the source of extravasation. Contrast accumulation in the sac may be very discrete and requires special care to be mapped. Spiral CT scanning, including precontrast studies and angiography with superselective catheterization of the internal iliac artery branches, often is necessary to detect the run-off from the sac.

Endoleaks most often are not graft-related but instead caused by retrograde flow through lumbar

arteries or the inferior mesenteric artery. The graft-related leaks originate from the proximal or distal graft anchoring sites, from graft defects, or from the contralateral iliac occluder device used with aortouniiliac graft repair.

Postoperative pressure inside the excluded aneurysmal sac has, so far, been measured only experimentally in animals.[19,20] Intraoperative recordings suggest that the pressure drops by greater than 50%[21]; however, such recordings were carried out at a stage of the operation when retrograde blood flow from the contralateral internal iliac artery had not yet been eliminated.

It has been suggested that graft-related leaks transmit greater pressure into the sac than retrograde perfusion from the lumbar arteries.[22] Although endoleaks seem to inhibit aneurysm retraction, their hemodynamic significance is uncertain. Each endoleak must be the result of at least two patent access routes to the sac; otherwise, perfusion would not be maintained. The run-off often is difficult to map with contrast studies, and the risk of rupture is unknown. The few endovascularly treated patients with continued aneurysm expansion and massive endoleakage have mostly been subjected to repeat intervention.

In the current study, a novel ultrasonic scan was used that is capable of detecting pulsatile arterial diameter changes of less than 8 μm (Figure 1).[23,24] The pulsatile wall motion (PWM) of the aneurysm sac was measured repeatedly before and after endovascular exclusion (Figure 2). It seems reasonable to assume that the PWM reflects the intraluminal systolic-diastolic pressure fluctuations. The postoperative PWM may therefore serve as an indicator of pressure inside the sac, although the PWM also may be influenced by factors such as aneurysmal wall stiffness and alterations in mean arterial pressure (MAP).

The aim of the current study was to assess the changing PWM in the excluded aneurysm sac and to relate these changes to the aneurysm diameter reduction and to the various forms of endoleaks.

PATIENTS AND METHODS

Seventy-five patients were treated with endovascular AAA repair at our institution between November 1993 and June 1997. Forty-one men and six women (median age, 72 years; range, 58 to 84 years) were included in this prospective study. Median follow-up was 12 months (range, 5 to 24 months). Patients from distant referral hospitals and patients in whom adiposity made ultrasonic evaluation technically impossible were excluded.

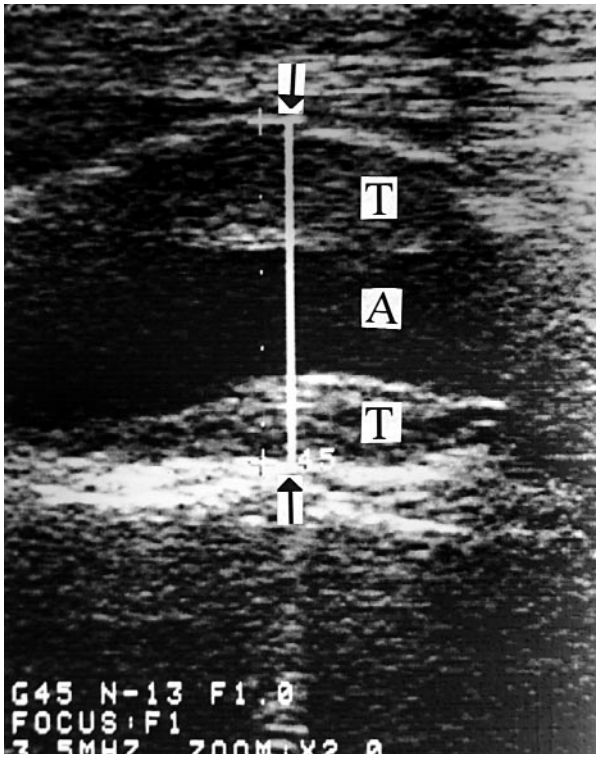

Fig. 1. The AAA lumen *(A)* was visualised in a longitudinal section on the real-time image of the ultrasound scanner. Intraluminal thrombus *(T)* is seen. Two electronic markers *(arrows),* each representing one tracking gate, were aligned with and locked to the luminal interface of the echo image of the anterior and posterior vessel walls at the site of the maximal aneurysm diameter.

A bifurcated graft was inserted in 15 patients (Chuter System[3]), and 32 patients received an aortouniiliac graft that was supplemented with a femorofemoral crossover bypass and a contralateral iliac occluder (Ivancev-Malmö System[11]). The graft material was Dacron (Cooley Very Soft) in all cases.

The maximum AAA diameter and PWM of the sac were assessed preoperatively and postoperatively at 1, 3, and 6 months and thereafter biannually with ultrasonic scanning (Diamove, Teltec, Lund, Sweden).[24] Based on CT scanning and angiography, patients were grouped depending on whether an endoleak was present.

Diamove is an electronic echo-tracking device interfaced with a B-mode real-time ultrasound scanner (EUB 240, Hitachi, Tokyo, Japan) and a 3.5 MHZ linear array transducer. It simultaneously tracks the inner surface of the anterior and posterior vessel walls (Figure 1). The difference between the

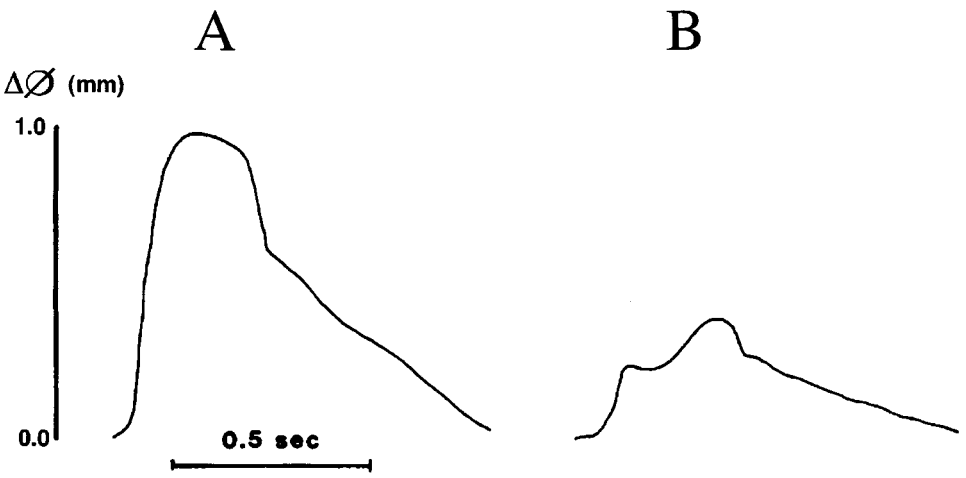

Fig. 2. Original tracings of the AAA diameter change during one heart cycle (i.e., the PWM) preoperatively *(A)* and after successful endovascular exclusion of the aneurysm *(B)*. The preoperative diameter curve shows a PWM of 1 mm, a rapid rate of diameter increase during early systole, and a dicrotic notch during early diastole. Postoperatively, there was a 75% reduction in PWM and a diminished rate of diameter increase during systole.

two echo registrations indicates the vessel diameter. The smallest detectable diameter change (i.e., PWM) is 7.8 μm. The repetition frequency of the echo-tracking loops is 870 Hz, rendering a time resolution of 1.2 milliseconds (Figure 2). To avoid tilting from the longitudinal axis of the aorta and to localize the site of its maximal diameter, the AAAs were visualized on the real-time image of the ultrasound scanner (Figure 1); this ensured that repeat measurements were performed at the same point of the aneurysm. The variability (CV) of the diameter measurements was 5% in healthy aortas and 3% in AAAs, with a diameter change of the AAA of at least ±2 mm considered significant.[24,25] The corresponding CVs regarding the PWM measurements in healthy aorta and AAA were 16% and 22%, respectively.[25,26]

Diamove also was used to measure the diameter and PWM of the endovascular graft inside the aneurysmal sac. The strain (S) of the grafts was defined as $S = (D_{syst} - D_{diast})/D_{diast}$, where D_{syst} and D_{diast} denote peak systolic and diastolic diameter, respectively.

All Diamove registrations were performed with the patient in the supine position after rest for at least 15 minutes. The brachial arterial pressure was recorded at each occasion according to the auscultatory method with a sphygmomanometer on the right upper arm. This was assumed to be equal to the intraarterial pressure of the infrarenal aorta.[26] The pulse pressure was calculated as the difference between the systolic and diastolic pressures. The MAP was defined as the sum of the diastolic pressure and one third of the pulse pressure.

Endoleaks, which cause contrast accumulation in the sac, were diagnosed with CT and angiography. Spiral CT scans were performed with 5 mm collimation and 5 mm table movement and reconstructed with 3 mm axial slice thickness. To distinguish leaks from calcifications and artifacts, precontrast scans always were included. In addition, multiplanar and three-dimensional reconstructions were performed. Whenever angiography was performed according to the follow-up protocol, the angiography catheter was used for intraarterial contrast injections as part of the CT study. During angiography, superselective catheterization of the internal iliac artery branches was performed whenever necessary.

Statistical values were calculated with the use of StatView software. Except when otherwise indicated, all data distribution is presented as median values and interquartile range because normal distribution could not be assumed. Wilcoxon's rank sum test was used for comparison of paired data, whereas the Mann-Whitney test was used for group comparisons unless more than two groups were compared; then, the Kruskal-Wallis test was applied. A value of $p < 0.05$ was considered statistically significant.

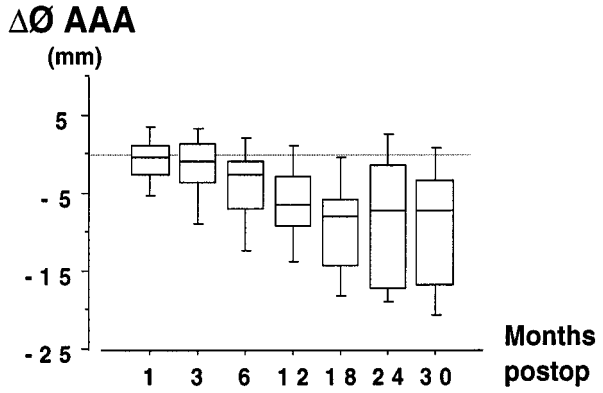

Fig. 3. The postoperative diameter change of the completely excluded aneurysmal sac. The diameter reduction seems to cease after 1.5 years at about 8 mm. Median and interquartile range (*within box*) and 10th and 90th percentile (*between bars*) values are given.

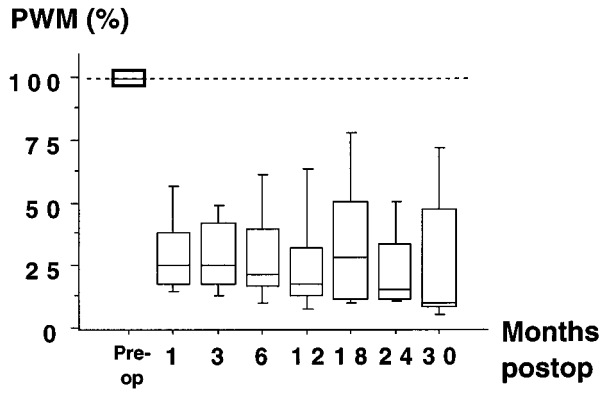

Fig. 4. The PWM of AAAs (percentage of preoperative values) before and after endovascular exclusion. A significant reduction was seen at 1 month ($p < 0.001$). The PWM was stable during the remaining follow-up period. Median and interquartile range (*within box*) and 10th and 90th percentile (*between bars*) values are given.

RESULTS

As shown with CT and angiography, 15 patients had contrast accumulation in the sac. Thirty-two patients had completely excluded AAAs. The median preoperative AAA diameter was 52 mm (range, 38 to 84 mm). The diameter was 50 mm in the group with subsequent leakage and 52 mm in the completely excluded cases (nonsignificant). The neck length was 25 mm (range, 9 to 72 mm), and the neck width was 23 mm (range, 15 to 29 mm). The proximal graft diameter ranged from 24 mm to 34 mm. Distally, the grafts were tapered to conform with the iliac diameter. The graft diameters did not differ significantly between the two groups.

The median MAP was 103 mm Hg preoperatively and 101 mm Hg at follow-up (nonsignificant). The pulse pressure was 65 mm Hg both preoperatively and postoperatively. There was no significant difference in blood pressure between the patients with totally excluded aneurysms and the patients with endoleaks.

Postoperatively, the diameter of the completely excluded aneurysmal sac gradually decreased, although the reduction seemed to cease after about 1.5 years (Figure 3). The aneurysm diameter then was 8 mm (range, 6 to 14 mm) less than the preoperative diameter ($p < 0.001$). In patients with persistent endoleaks, the aneurysm diameter at 18 months had increased 3 mm (range, 1 to 7 mm) compared with preoperative measurements (nonsignificant). The difference in aneurysm diameter at 18 months between the group with endoleaks and the group with completely excluded aneurysms was highly significant ($p < 0.001$). All the various types of endoleaks inhibited the postoperative reduction in aneurysm diameter in a similar manner.

The preoperative PWM was 1.0 mm (range, 0.8 to 1.3 mm). There was no significant difference in preoperative PWM between the patients with endoleaks and those with completely excluded sacs. Postoperatively, in completely excluded aneurysms, the PWM was reduced at 1 month to 0.3 mm (range, 0.2 to 0.4 mm) ($p < 0.001$); this corresponds to 25% (range, 16% to 37%) of the preoperative value. The PWM then remained stable throughout the observation period (Figure 4).

Fifteen patients had endoleaks at postoperative follow-up. There were 10 graft-related leaks originating from the proximal stent (n = 5), distal stent (n = 2), and occluder (n = 3). Five leaks were non-graft-related retrograde perfusions of the sac through collateral arteries. Most leaks were apparent on the first postoperative CT scan at 1 month. The endoleaks that did not appear until 6 months (n = 2) or 1 year (n = 1) postoperatively were proximal leaks associated with stent graft migration.

In the patients with endoleaks, the median PWM was 38% (range, 32% to 58%) of the preoperative value. This is significantly more than that in the completely excluded aneurysms ($p = 0.03$) (Figure 5). The PWM was 39% (range, 23% to 54%) in aneurysms with graft-related endoleaks and 30% (25% to 37%) in aneurysms with non-graft-related endoleaks ($p = 0.4$).

There were five patients in whom leakage ceased during follow-up and in whom the PWM was registered both during leakage and after it had stopped (Figure 6). In all five patients, the PWM was reduced after the leakage ceased ($p < 0.05$).

Among the 32 patients without endoleaks, there were three who had aneurysm enlargement at 6 months or later; the aneurysms enlarged by 3 mm, 3.6 mm, and 6.7 mm, respectively. Preoperatively, the PWM of the three patients was 0.8 mm, 0.9 mm, and 0.6 mm, which was not significantly different from the PWM of patients with subsequent aneurysm reduction ($p = 0.2$). Postoperatively, however, the PWM was 0.4 mm (50%), 0.7 mm (78%), and 0.6 mm (100%), respectively, which were higher values than the postoperative PWM of aneurysms with diameter reduction ($p = 0.05$).

During the study period, the diameter and PWM of the Dacron grafts did not change significantly. There was a weak correlation between the PWM of the graft and the PWM of the aneurysmal sac ($r^2 = 0.1$, $p < 0.01$), but no significant correlation was detected between the postoperative aneurysm diameter change and the PWM of the graft. The strain of the endovascular Dacron grafts was 0.014 (I.Q.R., 0.010 to 0.018); thus, the graft PWM was 1.4% of the graft diastolic diameter ($r^2 = 0.45$, $p < 0.0001$).

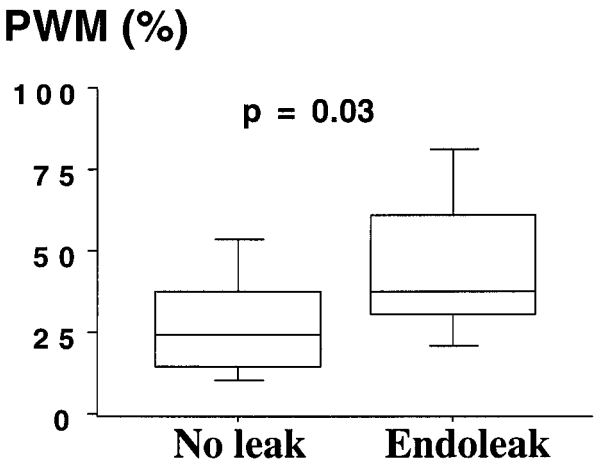

Fig. 5. The postoperative PWM of AAAs (percentage of preoperative values) was significantly higher in patients with endoleaks than in patients with completely excluded aneurysms. Median and interquartile range (*within box*) and 10th and 90th percentile (*between bars*) values are given.

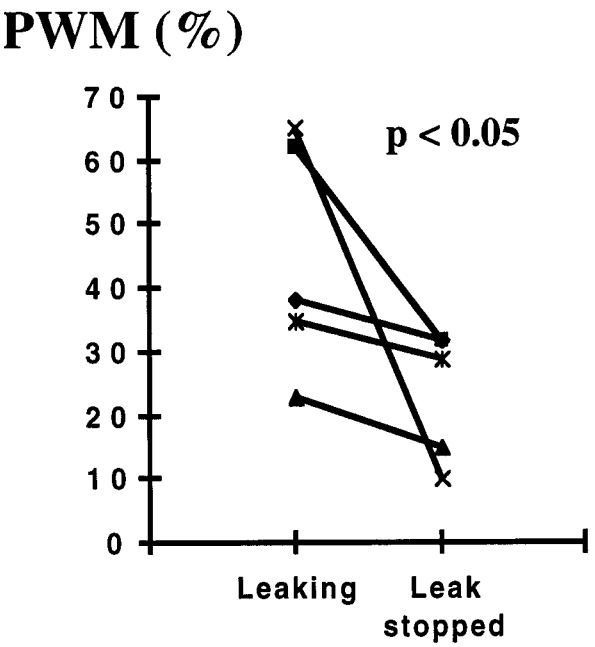

Fig. 6. The PWM was significantly reduced in cases in which leakage ceased during the observation period.

DISCUSSION

In the current study, complete endovascular exclusion of AAAs was associated with an immediate 70% reduction in the PWM and a continuous diameter decrease. The grafts were stable throughout follow-up without postoperative changes in diameter or PWM.

In cases with endoleaks, the aneurysm diameter was not reduced, and the postoperative PWM was about 50% higher than that in completely excluded aneurysms. However, there was no difference between graft-related and non-graft-related endoleaks.

Diameter reduction in the sac. The diameter of most aneurysms will decrease after endovascular repair.[12-14] This was confirmed by the results of the current study. However, the long follow-up in our study suggests that the process of aneurysm reduction may slow down during the second postoperative year (Figure 3); the median aneurysm diameter at that time was 8 mm less than the preoperative diameter, but the aneurysms were still 43 mm wide.

Because the PWM was reduced immediately after endovascular repair but aneurysm diameter reduction was a continuous and slow process, the changing aneurysm size seems to be the result of remodeling of the thrombus rather than of elastic recoil of the aneurysmal sac after the exclusion from systemic pressure. The reason why most of the treated aneurysms remained greater than 4 cm is unknown. The interrupted decrease in AAA size may be due to

reduced tissue resorption inside the sac, changing osmotic pressure, and cellular ingrowth, making the thrombus less prone to further remodeling. It is reassuring that the PWM remained stable during the entire follow-up period (Figure 4) because it may suggest that no major hemodynamic changes caused the interrupted process of diameter reduction.

Mechanisms of the PWM reduction. The current study shows that the PWM is reduced postoperatively.

The PWM reflects a beat-to-beat variation of the aneurysm diameter and hence of the aneurysm volume. Assuming that the aneurysm is a sphere with a diastolic diameter (D) of 5 cm, its diastolic volume is 65.5 ml: $\text{volume}_{(\text{sphere})} = 4\pi(D/2)^3/3$.

The preoperative PWM of 1 mm implies that the volume of such a sphere will be 4 ml larger in systole than in diastole.

Postoperatively, the PWM will be affected by the inserted graft. The graft volume is 15.7 ml given the graft is a cylinder that is 2 cm wide and 5 cm long. The strain of the Dacron graft was 1.4%, which implies that the PWM of such a graft cylinder is 0.3 mm and its volume increases by 0.5 ml during systole. The systolic expansion of the graft will then be transmitted to the sac. A spheric sac of 5 cm must increase its diameter by 0.2 mm to increase its volume by 0.5 ml (see the above equation). This calculated PWM of 0.2 mm corresponds well with the measured PWM of 0.3 mm in the excluded aneurysms.

PWM and endoleaks. Endoleaks have been reported in 0% to 40% of aneurysms after endovascular grafting. Aneurysm rupture may occur when endoleaks are present.[15-17] Several studies have shown that aneurysm diameter does not decrease postoperatively in the presence of an endoleak as opposed to when the aneurysm is completely excluded.[13,14] This phenomenon is supported by the results of the current study. Furthermore, this study demonstrates that endoleaks affect the PWM. The mechanisms of endoleakage, however, are complex and not fully understood. Endoleaks seem to allow additional blood to enter the sac during systole, thereby increasing the PWM of the sac.

If there is an endoleak with poor run-off from the sac, there will be systolic flow through that endoleak until the MAP in the sac equals the systemic MAP. Then, blood will merely oscillate in the endoleak channel. This oscillating blood may increase the PWM but will not be detected by contrast enhanced imaging. Pressure may therefore be transmitted without radiologic evidence of an endoleak. This may explain why there were three cases of enlarging aneurysms without accumulation of contrast in the sac. The PWM of these cases was higher than expected, which may support this hypothesis. In animal experiments, it has previously been shown that pressure may be transmitted into the sac in the absence of contrast accumulation outside the graft.[20]

The various endoleaks presented with a similar PWM. This may indicate that the type of leakage or perfusion is not decisive for its hemodynamic significance. The number of patients with the various kinds of leakage was, however, small, and a Type I error cannot be excluded. Although in clinical praxis it is tempting to neglect minimal persistent perfusion through small lumbar arteries, there is no evidence that this kind of leakage would be less stressful to the aneurysmal wall than leakage past graft anchoring stents. Furthermore, the classification of endoleakage is uncertain. In all kinds of leaks, at least two routes into the aneurysmal sac must remain patent for circulation to occur. Although the main route of contrast inflow into the sac can be mapped quite easily, it is technically difficult to visualize all the routes, particularly the outflow. Often, angiography and contrast enhanced CT are unable to fully classify the various routes to a perfused sac.

Other factors influencing the PWM. Although the PWM of aneurysms seems to correlate with the pulse pressure inside the sac, several other factors must be considered.

After successful endovascular grafting, most of the pressure gradient and concomitant wall stress are transferred from the aneurysm wall to the graft wall. This probably is one of the reasons why the size of excluded aneurysms decreases with time. The blood pressure will have an impact on the graft rather than on the aneurysm wall, and the PWM of the graft will determine the postoperative PWM of the sac. The graft PWM is influenced by the stiffness of the graft fabric and by the graft diameter. Similarly, the stiffness of the aortic wall and the diameter of the aneurysmal sac will influence the PWM of aneurysms; so will the systemic blood pressure, because the pressure-diameter curve is nonlinear.[23,27]

These factors may vary among individuals and with time. They may account for the overlap of PWM data from different patient groups (Figure 5) and make the assessment of individual patients a difficult task. However, several reasons make it unlikely that the results of the current study were biased. The reduction in the PWM does not seem to be due to an immediate postoperative increase in wall stiff-

ness; on the contrary, the reduced pressure inside the excluded sac[21] is expected to provide reduced distention of the aneurysmal sac and thereby reduced aneurysm stiffness.

Furthermore, the graft diameters were not significantly different in patients with and without endoleaks even though various graft diameters had to be used within each group. Regarding graft fabric, there was no dilation and no change of strain during follow-up. Finally, there was no significant difference in the systemic blood pressure between the two groups of patients or between the preoperative and postoperative investigations. Therefore, blood pressure did not confound the PWM measurements.

A potential weakness of the present study is that it could not be blinded. The Diamove operators visualized the aneurysms (Figure 1) and knew instantly whether the patients had been treated. This same problem applies to CT scans. However, the postoperative changes in both aneurysm diameter and PWM were too large to be affected by this potential bias. Recent studies have shown that Diamove variability allows detection of diameter changes of at least 2 mm,[24,25] whereas the previous reports were based on CT scans in which the cutoff point was set at 3 mm.[14]

Conclusions. The PWM of untreated abdominal aortic aneurysms was about 1 mm. The PWM was reduced by 75% after successful endovascular exclusion of the sac. The PWM was 50% greater in patients with endoleaks than in patients with totally excluded aneurysms. Pressure may be transmitted into the sac despite absence of contrast accumulation outside the graft.

The decrease in AAA diameter in successfully excluded aneurysms may cease after 1.5 years.

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