Intrasac flow velocities predict sealing of type II endoleaks after endovascular abdominal aortic aneurysm repair

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Purpose: The purpose of this study was to determine whether intrasac spectral Doppler flow velocities can predict whether or not a type II endoleak will spontaneously seal and to relate intrasac flow to preoperative branch vessel anatomy.

Methods: Between October 1996 and June 2002, 265 patients with abdominal aortic aneurysms underwent endovascular repair. Patients with less than 24 months of follow-up and type I endoleaks were excluded. Type II endoleaks were confirmed with duplex scan and computed tomographic angiography. Two groups were identified: 14 patients with sealed endoleaks (<6 months) without intervention and 16 patients with persistent endoleaks greater than 6 months and without resolution. Spectral Doppler flow velocities were recorded from endoleaks within the aneurysm sac.

Results: The two groups were similar in age, demographics, and aneurysm morphology. The mean follow-up times were 29.9 ± 7.9 months for sealed endoleaks and 30.2 ± 8.6 months for persistent endoleaks (P = not significant). Spectral Doppler velocities were significantly lower in patients with sealed endoleaks compared with persistent endoleaks (75.5 ± 78.8 cm/s versus 138.2 ± 36.2 cm/s; P < .01). Patients with sealed endoleaks and low (<100 cm/s) intrasac Doppler velocities had significantly fewer patent inferior mesenteric arteries (43% versus 81%; P < .01), a smaller inferior mesenteric artery (5.6 ± 1.8 mm versus 7.2 ± 1.3 mm; P < .01), and fewer paired lumbar arteries (1.3 ± 0.8 versus 2.4 ± 0.6; P < .0001) compared with those with persistent endoleaks and high (>100 cm/s) intrasac flow velocities. Three patients with sealed endoleaks had Doppler velocities of 200 cm/s or greater. However, the diameter of the inferior mesenteric artery in these patients was 4 mm or less with no visualized lumbar arteries before surgery. Aneurysm diameter (<4.6 ± 5.6 mm) and volume (<0.9 ± 45.2 mL) decreased in patients with sealed endoleaks. Aneurysm diameter (1.8 ± 4.9 mm) and volume (18.5 ± 33.9 mL) increased slightly in patients with persistent endoleaks (P < .05). No ruptures or conversions occurred in any patient. Secondary interventions to treat type II endoleaks were unsuccessful in six of 16 patients (38%) with persistent endoleaks.

Conclusion: Intrasac Doppler velocities can be used to predict whether a type II endoleak will spontaneously seal. High-velocity type II endoleaks are related to preoperative large branch vessel diameter and number and are resistant to endovascular treatment. (J Vasc Surg 2003;37:8-15.)

Endovascular repair offers a less invasive treatment option for patients with infrarenal abdominal aortic aneurysms (AAAs); however, the long-term effectiveness of endovascular grafting is unknown. Despite a high technical success rate in the placement of endovascular grafts, persistent flow within the aneurysm sac (endoleaks) can be identified in 15% to 52% of patients after endovascular repair.1-6 Although the true significance of endoleaks is unknown, there is little debate that type I endoleaks associated with poor proximal or distal fixation are associated with continued risk of aneurysm rupture and need treatment. However, the significance of type II endoleaks on long-term outcome is unknown.

Type II endoleaks are the result of continued flow within the aneurysm sac from the inferior mesenteric artery (IMA) or lumbar arteries. Such endoleaks are often seen immediately after placement of an endovascular device and are more likely to persist in patients with a large-caliber IMA or with multiple patent lumbar arteries. To date, there is no consensus as to the relevance, treatment, or outcome of these endoleaks. It is not known whether treatments aimed at occluding these branch vessels to eliminate the type II endoleak reduces the subsequent risk of rupture. Although some investigators recommend an aggressive approach with transarterial or translumbar embolization, ligation, or surgical conversion, others recommend no treatment unless there is evidence of aneurysm enlargement or endograft migration.7-9 The purpose of this study was to determine whether the velocity of blood flow in the aneurysm sac as measured with intrasac spectral velocities can predict whether or not a type II endoleak will spontaneously seal without intervention and to relate flow velocities to preoperative branch vessel anatomy.

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Competition of interest: Drs Zarins and Arko are consultants for Medtronic.

Dr Fogarty owns shares of stock in Medtronic.


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METHODS

Patients. Between October 1996 and June 2002, 265 patients with infrarenal AAAs underwent endovascular repair with the AneuRx stent graft (n = 252; Medtronic AVE, Santa Rosa, Calif) or Talent stent graft (n = 13; Medtronic AVE). All patients were captured in a vascular database. Sixty-one patients (23%) had a type II endoleak at discharge. Of these, 30 patients had evidence of a type II endoleak identified before hospital discharge on both computed tomographic angiography (CTA) and duplex ultrasound scan (DUS). These patients had repeat assessment of their endoleaks with both imaging methods with complete agreement in follow-up. Endoleaks that disappeared within 6 months with no subsequent recurrence, and without treatment, were classified as sealed. Endoleaks that persisted longer than 6 months were classified as persistent type II endoleaks. There were 14 patients with sealed endoleaks and 16 patients with persistent endoleaks.

Imaging and aneurysm morphology. Preoperative and postoperative CTA and three-dimensional reconstructions were used to determine aneurysm morphology. Preoperative measurements included AAA diameter and volume. After surgery, changes in aneurysm diameter and volume were determined from serial measurements at discharge, at 1, 3, and 6 months, and yearly thereafter.

Before hospital discharge, patients underwent evaluation with CTA, DUS, and abdominal radiography to evaluate the position and integrity of the stent graft. Among the 265 patients, postoperative imaging surveillance was performed with CTA. Among the 265, 71 patients underwent both CTA and DUS with at least a 3-year follow-up before discharge and at 1, 3, and 6 months and yearly thereafter as a result of physician preference. Of these 71, 31 patients had no evidence of endoleak with CTA or DUS, five had type I endoleaks that were treated, four lacked sufficient imaging confirmation of endoleak, and one died within 30 days, leaving 30 patients to be analyzed for this study.

Endoleak assessment. The combination of DUS and CTA allowed accurate identification of the source of any endoleak and exclusion of patients with type I endoleaks. DUS was performed by a registered vascular technologist with a Sequoia 512 ultrasound system (Acuson, Mountain View, Calif) and a Sector V4 transducer. After the patients had fasted for 6 hours, transverse and sagittal imaging was performed to measure peak systolic diameters at the largest region of the proximal, mid, and distal segments of the abdominal aorta. Close attention was given to the stent device in grayscale and color Doppler scanning of the aneurysm sac to rule out endoleak. Duplex scan was used to determine presence or absence of flow in the aneurysm sac and branch vessels (Fig 1). Quantitative assessment of flow velocities and direction was performed within the aneurysm sac near the aneurysm wall with a constant angle of less than 60 degrees to the branch vessel (Figs 2 and 3). Spectral velocities were obtained at the entrance of the feeding vessel to the aneurysm sac only. No velocities were obtained within the aneurysm sac or in the outflow vessel. All scans were reviewed by a vascular surgeon and were technically satisfactory in each case in this series.

Spiral computed tomographic scan was performed with a Lightspeed QXi computed tomographic scanner (General Electric Medical Systems, Milwaukee, Wis). Precontrast studies were performed routinely. After preliminary timing of 15 mL of iodinated contrast bolus, 80 to 150 mL of nonionic iodinated contrast medium were injected at 4 mL/s. Computed tomographic scans were acquired at pitch 6.0 with a 2.5-mm nominal section thickness throughout the entire scan. All images were reconstructed at intervals equal to 50% of nominal section thickness and viewed interactively on a workstation.

Preoperative collateral vessel quantification. IMA patency was defined as visualization of the vessel in communication with aneurysm on preoperative CTA. The diameter was measured from outer wall to outer wall with electronic calipers on three-dimensional reconstructions. Three independent measurements were obtained and averaged. Similarly, all lumbar arteries visualized on the preoperative CTA from the renal arteries to the level of the aortic bifurcation were counted as paired lumbar arteries.

Statistical analysis. Results are expressed as the mean ± the standard deviation. Differences between groups were compared with the Student t test. Cox proportional hazards regression analysis was used to identify independent risk factors for type II endoleaks with elevated spectral velocities. Kaplan-Meier estimates were used to determine freedom from surgical conversion and aneurysm rupture. A P value of less than .05 were considered significant.

RESULTS

There was no difference in gender, age, or comorbidities in patients with sealed or persistent endoleaks. The mean follow-up period was the same: 29.9 ± 7.9 months (range, 24 to 48 months) for sealed endoleaks and 30.2 ± 8.6 months (range, 24 to 49 months) for persistent endoleaks.

Preoperative aneurysm morphology. There was no significant difference between the two groups with regards to the proximal neck diameter or length (Table I). Patients with sealed endoleaks had larger aneurysms (63.4 ± 11.4 mm; range, 46 to 80 mm) than those with persistent endoleaks (55.6 ± 4.2 mm; range, 47 to 62 mm; P < .05).

Intrasac flow velocities and preoperative branch vessel anatomy. Intrasac Doppler flow velocities of type II endoleaks at the time of hospital discharge were different between the two groups. Patients with type II endoleaks that sealed in less than 6 months without intervention had significantly lower intrasac AAA flow velocities at the time of hospital discharge than patients with persistent type II endoleaks (75.5 ± 78.8 cm/s; range, 15 to 230 cm/s; versus 138.2 ± 36.2 cm/s; range, 100 to 200 cm/s; P < .01). These elevated intrasac AAA flow velocities were directly related to whether or not the IMA was patent (P < .01) and whether or not more than two paired lumbar
arteries were patent ($P < .0001$) on the preoperative CTA (Table II).

Three patients with sealed endoleaks had high flow velocities of 200 cm/s or greater. However, in these patients, the diameter of the IMA was less than 4 mm and no lumbar arteries were visualized before surgery. The other 11 patients all had intrasac flow velocities of less than 80 cm/s.

Intrasac flow velocity had decreased to zero in all patients with sealed endoleaks within 6 months, and lack of endoleak was confirmed with CTA in each instance. Patients with persistent endoleaks continued to have elevated intrasac flow velocities that remained greater than 100 cm/s during the follow-up period and did not change significantly over time (Fig 4).

**Secondary interventions.** Coil embolization of branch and collateral vessels was attempted in six of 16 patients (38%) with persistent type II endoleaks and high intrasac flow velocities in an effort to seal the type II endoleak. Four lumbar arteries and two IMAs were coil embolized via the hypogastric and superior mesenteric artery, respectively. The mean time to secondary transcatheter intervention was 16.6 months (range, 6 to 24 months). Although each procedure was technically successful in eliminating the endoleak angiographically at the time of the procedure, none of the type II endoleaks was completely obliterated with follow-up duplex scan imaging that was performed 3 months after intervention. Furthermore, in each instance, the velocities remained greater than 100 cm/s. In one patient with a persistent type II endoleak with high intrasac flow velocities (175 cm/s) and transarterial embolization of lumbar arteries, there was a significant increase in AAA diameter (58 mm to 72 mm) and volume (290 mL to 400 mL) over 26 months. The patient underwent laparoscopic clipping of the IMA and a lumbar vessel. Before clipping, the mean intrasac pressure was 60 mm Hg and nonpulsatile. After the clipping, the intrasac pressure decreased to 20 mm Hg. The endoleak resolved, and over the last 20 months, the diameter and volume of the aneurysm have remained stable.

**Postoperative morphology.** In patients with sealed type II endoleaks, the aneurysm diameter ($-4.6 \pm 5.6$ mm) and volume ($-0.9 \pm 45.2$ ml) decreased compared
with patients with persistent type II endoleaks where the aneurysm diameter (1.8 ± 4.9 mm) and volume (18.5 ± 33.9 ml) increased compared with the preoperative aneurysm sac dimensions (P < .05).

Clinical outcome. There were no aneurysm ruptures or surgical conversions in any of the patients with either sealed or persistent type II endoleaks at 36 months.

DISCUSSION

The significance of type II endoleaks is unknown. The finding that systemic pressure can often be recorded in the aneurysm sacs of patients with type II endoleaks has caused some investigators to treat these leaks aggressively while others treat only when the aneurysm sac is increasing in size. Treatment options are varied and include transcatheter interventions with transarterial and translumbar coil embolizations and direct thrombin injection of the aneurysm sac, which have yielded mixed results.8–11 Although the best treatment option (or no treatment) for these patients is being studied and debated, there have been little data regarding the physiology or classification of type II endoleaks.

Most patients who undergo endovascular AAA repair have postoperative surveillance with CTA. This imaging study can usually determine the presence or absence of a type II endoleak and differentiate these from type I attachment site endoleaks.12 Type II endoleaks are often described qualitatively as being large or small depending on the volume of contrast seen in the aneurysm sac. Preoperative documentation of a large IMA or multiple patent lumbar arteries has been shown to be predictive evidence for the persistence of type II endoleaks after surgery.7,13 This may be due to high flow rates in these large branch vessels. Although CTA allows for excellent visualization of the AAA, aortic anatomy and tortuosity, and the stent graft, it offers little physiologic data. The use of DUS is helpful in this regard; it can be used to further characterize these type II endoleaks after surgery. Thus, the use of DUS both compliments and adds to the information obtained on CTA.

Fig 2. DUS and spectral velocities of type II endoleak after infrarenal AAA repair with endovascular graft. To and fro flow is seen within aneurysm during systole and diastole, respectively. For this study, spectral flow velocities were obtained adjacent to aneurysm wall (above-right image). This patient had persistent endoleak with postoperative velocities between 190 and 200 cm/s. It is important to notice position of where velocities were measured because velocities will decrease if flow is measured further away from wall (bottom-right image).
DUS has been widely used for morphologic surveillance of AAAs and also provides information on blood flow velocities and patterns. It is the procedure of choice for noninvasive imaging of the AAA and offers the advantages of wide availability, lower cost, no radiation exposure, noninvasiveness, and no nephrotoxicity compared with CTA, magnetic resonance angiography, and arteriography. DUS offers the additional advantage of being able to document flow velocity and direction in the aneurysm sac and branch vessels entering the aneurysm, thus providing dynamic flow information that is not possible with CTA.

Table I. Preoperative aneurysm morphologic measurements from CTA images

<table>
<thead>
<tr>
<th></th>
<th>Sealed endoleaks (n = 14)</th>
<th>Persistent endoleaks (n = 16)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Proximal neck diameter (mm)</td>
<td>21.7 ± 1.8 (19-25)</td>
<td>22.6 ± 1.6 (19-26)</td>
<td>NS</td>
</tr>
<tr>
<td>Proximal neck length (mm)</td>
<td>21.2 ± 9.4 (10-40)</td>
<td>22.6 ± 10.3 (10-46)</td>
<td>NS</td>
</tr>
<tr>
<td>AAA size (mm)</td>
<td>63.4 ± 11.4 (46-80)</td>
<td>55.6 ± 4.2 (47-62)</td>
<td>.04</td>
</tr>
</tbody>
</table>

Data are mean ± standard deviation (range). NS, Not significant.

Table II. Preoperative collateral vessel quantification and postoperative intrasac AAA Doppler flow velocities

<table>
<thead>
<tr>
<th></th>
<th>Sealed endoleaks (n = 14)</th>
<th>Persistent endoleaks (n = 16)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intrasac AAA Doppler flow velocities (cm/s)</td>
<td>75.5 ± 78.8 138.2 ± 36.2 &lt;.01</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IMA patent</td>
<td>43%</td>
<td>81%</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>IMA diameter (mm)</td>
<td>5.6 ± 1.8</td>
<td>7.2 ± 1.3</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Paired lumbar arteries</td>
<td>1.3 ± 0.8</td>
<td>2.4 ± 0.6</td>
<td>&lt;.0001</td>
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Fig 3. DUS of type II endoleak from IMA. Spectral flow velocities within aneurysm sac near wall are 19 cm/s. This endoleak resolved spontaneously and has not recurred on follow-up imaging.
We have previously shown that high-quality DUS is comparable with CTA for measuring aneurysm size and for identifying endoleaks after endovascular exclusion of AAA. The maximal transverse diameter as measured with duplex scanning (60.0 ± 9.8 mm) correlated closely (r = 0.93; P < .001) with CTA (58.8 ± 8.5 mm). In 92% of the scans, diameter measurements were within 5 mm of each other. Changes in aneurysm size throughout follow-up were −2.4 ± 5.8 mm on duplex scanning and −2.4 ± 4.7 mm on CTA without a significance difference. Both imaging methods were able to determine the presence or absence of endoleak with excellent correlation (P < .001). In comparison with CTA, diagnosis of endoleak with duplex scanning was associated with a sensitivity of 81%, a specificity of 95%, a positive predictive value of 94%, and a negative predictive value of 90%. Thus, the use of DUS is a valid technique in the surveillance of AAA after endovascular repair.

Because of the uncertainty and consequences of type II endoleaks, some physicians choose to treat endoleaks as soon as they are seen on follow-up CTA. This strategy is often not always effective in obliterating the endoleak. Because it is not clear that the presence of a type II endoleak exposes the patient to a significant risk of aneurysm rupture, it is possible that some patients are undergoing unnecessary interventions for type II endoleaks that may obliterate spontaneously.

We evaluated two groups of patients with type II endoleaks, those that sealed within 6 months and those with persistent type II endoleaks. DUS of these two different types of endoleaks showed two different flow characteristics. Those with persistent endoleaks had significantly higher intrasac flow velocities than those with sealed endoleaks. This increased flow velocity corresponded with an increased number of patent preoperative collateral vessels. Knowledge of whether a type II endoleak has low or high intrasac flow velocities can be used to select patients for treatment because patients with low intrasac flow velocities are likely to have complete thrombosis of the aneurysm sac and disappearance of the endoleak without the need for treatment. The sluggish flow within the aneurysm sac may facilitate thrombosis of the endoleak. This watchful waiting of low velocity type II endoleaks during the initial 6 months after stent graft placement can decrease the number of percutaneous interventions performed and decrease the morbidity and mortality associated with added hospitalizations and procedures. Furthermore, in this group of patients with sealed endoleaks, within 6 months, there was a decrease in both aneurysm volume and diameter.

The presence of high-velocity type II endoleaks appears to be related to the presence of multiple patent preoperative collateral vessels. One could argue that these collateral branch vessels should be treated before surgery before stent graft placement. However, this is not our strategy because the overall significance of high velocity type II endoleaks is still unknown. In this small group of patients, there has been little change in aneurysm size and no aneurysm ruptures or conversions. Although there was an overall minor increase in the mean diameter (1 mm) and volume in patients with persistent endoleaks, only two patients (13%) had an actual increase in size and one (6%) had a decrease. This is in contrast to patients with low-velocity type II endoleaks, which sealed with subsequent shrinkage of the aneurysm sac. Interestingly, patients with sealed endoleaks had larger aneurysms than those with persistent endoleaks.
What effect this increased size has on type II endoleaks is unknown. However, the increased size and thrombus within the sac may have provided more resistance, further allowing the type II endoleak to seal. This will require further study in the future.

Six patients underwent transarterial coil embolization during our early endograft experience. These were performed to obliterate all extrastent flow. Although it appears resistant to transarterial coil embolization. Whether they are resistant to other therapies, including translumbar embolization or thrombin injection of the AAA sac, is unknown from this study. Future studies looking at these therapies should attempt to classify type II endoleaks into low and high velocities to determine whether these interventions are more effective than transarterial embolization.

The long-term outcome for patients with persistent high flow type II endoleaks has thus far been no different from patients without endoleaks or sealed endoleaks. Freedom from surgical conversion and aneurysm rupture was 100% at 3 years. Thus, we treat type II endoleaks conservatively. Patients with type II endoleaks with a stable or decreasing aneurysm are routinely followed on a biannual basis. However, patients with a type II endoleak and an increase in aneurysm size are treated. Treatment includes ensuring secure proximal and distal stent graft fixation from just at the level of the renal arteries to the hypogastric arteries bilaterally and no evidence of type I endoleak. If there is continued increase in aneurysm size, treatment with translumbar coil embolization or thrombin injection of the aneurysm sac can be attempted, although the long-term success of this approach is not clear. However, laparoscopic clipping of these vessels was feasible and was successfully used in one patient. This method of treatment may become more useful in the future but is often technically challenging.

In conclusion, intrasac flow Doppler velocities can be used to predict whether type II endoleaks will spontaneously seal. Patients with low-velocity endoleaks (<80 cm/s) are likely to have thrombosis without treatment. Those with high velocity (>100 cm/s) are related to preoperative large branch vessel diameter and number. These appear resistant to treatment with transarterial embolization. Categorization of type II endoleaks into high-velocity and low-velocity endoleaks with DUS can be used to test different therapeutic interventions for type II endoleaks in the future. In this group of patients, type II endoleaks are not associated with aneurysm rupture or surgical conversion. Longer follow-up is needed to confirm these observations.

REFERENCES

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DISCUSSION

Dr George H. Meier III (Norfolk, Va). We have had a long-standing interest in endoleak detection with ultrasound, and I am sure you have seen our papers on flow spectrum. And obviously, when you talk about duplex, there are a lot of other parameters you can get. Clearly, some of those parameters that we are concerned about are resistive indices, the presence of diastolic flow, and also volume flows. And the first question I would ask is, are there any data, do you have any data, on those specific criteria? Since we did not find peak flow velocities predictive.
The second issue is, what happens to these waveforms over time? We have also put our opinion out there in the literature on what that means. We feel that flow spectrum often change before these endoleaks occlude, and I was wondering if you found any evidence of that in your series?

**Dr Frank R. Arko.** With regard to the flow velocities over time, patients with the sealed endoleaks decreased to zero without evidence of flow within the aneurysm sac.

In the patients with persistent type II endoleaks, the flow velocities remained relatively unchanged from their immediate postoperative state in that they continued to range in the 100 to 200 cm/s range. In the patients with transarterial embolization, there was some decrease in the flow velocities, but never below 100 cm/s.

Regarding resistive indices, we did not look at these so I do not have any data to share. It is important to note that in patients with persistent type II endoleaks there was both to-and-fro flow during systole and diastole.

**Dr John J. Ricotta (Stony Brook, NY).** I just wanted a clarification. Did you redo the Dopplers after your transarterial embolization, and did the flow velocities change? I do not understand, maybe you could speculate on it, since your hypothesis is that flow velocity predicts the sealing, and presumably you change the flow velocity with the transarterial embolization, can you speculate on why that did not effectively seal these endoleaks?

**Dr Arko.** After patients underwent transarterial embolization, they did not immediately have color duplex imaging performed as this was a retrospective study. CTA and duplex were performed between 3 and 6 months later with an endoleak present on both studies. The velocities did decrease, but again, they remained in the elevated range from 100 to 200 cm/s. I suspect that several of these patients had multiple lumbar arteries and not all of them were embolized. So I suspect that the endoleaks persisted as a result of continued flow in the lumbar that were not embolized.

**Dr David C. Brewster (Boston, Mass).** I just want to make sure that we understand the clinical implications of your findings. Is it fair to say that your data would suggest that type II endoleaks are not really clinically relevant and therefore the intervention of persistent endoleaks would not be indicated?

**Dr Arko.** I am not sure that I would go so far as to say that all type II endoleaks are clinically insignificant. However, I think our data suggest that management of type II endoleaks with coil embolizations within 3 months is an aggressive strategy, especially when there is no increase in aneurysm size.

Patients with type II endoleaks that have an increase in aneurysm size should be treated. However, coil embolization of these endoleaks via a transarterial route has not been very successful. A translumbar approach may be better, and we have had some success with thrombin injection of the aneurysm sac. One patient who had elevated velocities underwent transarterial embolization that failed, and the aneurysm continued to increase in size. There was secure proximal and distal fixation of the stent graft. The patient underwent laparoscopic ligation of the IMA and one lumbar artery. That patient’s aneurysm initially increased from 55 mm to 72 mm during that time period prior to laparoscopic ligation. Intracranial pressures during the procedure were 60 mm Hg and nonpulsatile. Following the clipping, the pressures decreased down to 20 mm Hg and there has been no further increase in aneurysm size and no endoleak.

So, I think in patients who have an increase in aneurysm size, they should be treated. But otherwise, I think if there is no increase in size, or there is a decrease, I think they can be safely followed. That has been our experience.