Current treatment of renal artery aneurysms may be too aggressive

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Objective: **Most studies recommend repair of renal artery aneurysms (RAAs) >2 cm in diameter in asymptomatic patients, but other studies have suggested that their natural history may be more benign. We hypothesized that rupture and death in patients with asymptomatic RAAs is low and that current recommendations for RAA treatment at 2 cm may be too aggressive.**

Methods: **Retrospective review of all RAAs treated at a tertiary care medical center from 2002 to 2012.**

Results: **Fifty-nine RAA were identified in 40 patients (mean age at diagnosis, 56 years; male:female ratio, 17:23); 31 were saccular, 8 were fusiform, and 5 were bilobed. Twenty-nine patients were asymptomatic; the remainder of patients presented with hematuria (n = 4), abdominal pain (n = 3), difficult-to-control hypertension (n = 3), or flank pain (n = 2). Aneurysm location included the main renal artery bifurcation (n = 35), main trunk (n = 7), primary branch (n = 6), pole artery (n = 6), and secondary branch (n = 1). Operative management of RAAs included vein patch (n = 6), prosthetic patch (n = 4), primary repair (n = 3), plication (n = 1), patch and implantation (n = 1), and ex vivo repair (n = 1). Eight asymptomatic RAAs were treated surgically (mean RAA diameter = 2.4 ± 0.1 cm, range, 2-3 cm), with the remaining 33 asymptomatic RAAs being managed conservatively (mean RAA diameter = 1.4 ± 0.1 cm, range, 0.6-2.6 cm). Mean hospital length of stay was 4 days, with no late postoperative complications and 0% mortality. Non-operated patients were followed for a mean of 36 ± 9 months, with no late acute complications and 0% mortality. Mean RAA growth rate of patients with multiple imaging studies was 0.60 ± 0.16 mm/y.**

Conclusions: **The rate of aneurysm rupture and death in our untreated RAA patients is zero, the growth rate is 0.60 ± 0.16 mm/y, and there were no adverse outcomes in asymptomatic RAAs >2 cm that were observed. We may currently be too aggressive in treating asymptomatic RAAs. (J Vasc Surg 2014;59:1356-61.)**

Renal artery aneurysms (RAAs) are uncommon, with an estimated incidence of 0.09%[1]; however, the increased use of cross-sectional abdominal imaging techniques such as computed tomography and magnetic resonance imaging has resulted in an increased frequency of incidentally discovered RAAs.[1-3] The current recommendation to repair all asymptomatic RAAs >2 cm in diameter has led to an increase in RAA procedures,[1-3] but there remains significant controversy surrounding these treatment criteria.[4,5]

Because of the rarity of RAA rupture, the natural history is not well defined, but the risk of rupture is thought to be <3% of diagnosed RAAs.[4] Both the rate of rupture and the growth rate of RAAs have yet to be precisely determined.[4] Furthermore, many authors dispute the concept that aneurysm size and risk of rupture are directly correlated, because studies have shown that aneurysms <2 cm rupture and aneurysms >2 cm may remain clinically silent for years.[1,6-8] Although there is consensus to repair RAAs in symptomatic patients and in women who are pregnant or of childbearing age because of the risk of rupture, the appropriate management of asymptomatic RAAs remains unclear.

The objectives of our study were to (1) examine our institution's experience with RAAs, with specific emphasis on the long-term outcome of asymptomatic RAAs, (2) measure the growth rate of asymptomatic RAAs, and (3) evaluate the contemporary validity of current treatment recommendations.

METHODS

Patients presenting to the University of California Los Angeles (UCLA) Division of Vascular Surgery and UCLA medical center with true RAAs between 2002 to 2012 were identified through the use of ICD-9 disease codes 442.1 (aneurysm of renal artery) and 442.89 (aneurysm of other specified artery). Patients presenting with both symptomatic and asymptomatic RAAs were included in this analysis. Symptomatic RAAs were defined through the use of previously published criteria, including: difficult-to-control hypertension, hematuria, flank pain, and/or abdominal pain.[1] Symptoms were attributed to the aneurysm if they resolved after surgery or if no other etiology was discovered.

Sources used to identify isolated RAA included: medical records, imaging studies, and pathology reports. Pseudoaneurysms, mycotic aneurysms, pararenal and juxtarenal aortic aneurysms, and aneurysms of the proximal renal artery

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as an extension from an aortic aneurysm were excluded from the study, leaving only "true" RAAs for the analysis.

Charts of patients with true RAAs were reviewed for demographic information, clinical presentation, diagnostic technique, risk factors and comorbidities, perioperative data, operative data, pathology, and early and long-term outcomes. Aneurysm characteristics previously published in RAA studies that have been found to be significant were recorded, including location, morphology, maximum diameter, number of efferent branches, and calcification.

Aneurysms that were surgically repaired were classified as resection with primary repair, resection with patch, plication, ex vivo/complex repair, and nephrectomy (planned or unplanned). In patients followed by observation, serial images were analyzed for growth, intraluminal thrombus, or occlusion when available. The UCLA Institutional Review Board approved the database and study methods.

Statistics. All data were managed and retained in a Microsoft Excel (Version 14; Microsoft Corp, Redmond, Wash) database. Analysis was performed with the use of SPSS Statistics for Mac (Version 20.0; IBM Corp, Armonk, NY). Categorical variables are presented by frequency and percentage of study population; continuous variables are presented as mean ± standard error of mean (SEM), unless noted otherwise. Differences and level of significance between groups for categorical variables were determined with the use of the χ^2 test and Fisher exact test. Differences between continuous variables were analyzed with the use of the independent *t*-test or Mann-Whitney *U* test. Aneurysm growth rate was calculated for all patients with two or more imaging studies. If more than two imaging studies were available, a weighted average was used to calculate the overall change in aneurysm size per unit time. A value of $P < .05$ was considered significant.

RESULTS

Patient demographics and comorbidities. Between 2002 and 2012, 59 RAAs were identified in 40 patients at the UCLA Medical Center. Twenty-three women and 17 men had a mean age at diagnosis of 56 ± 2 years (range, 16-78), with comorbidities including hypertension, active or history of smoking, hypercholesterolemia, diabetes mellitus, and coronary artery disease (Table I). Two patients had Marfan syndrome (5%), no patients had documented chronic obstructive pulmonary disease, and no patients had documented impaired renal function at the time of diagnosis.

Twenty-nine patients (73%) were asymptomatic, whereas 11 patients (27%) presented with renal symptoms. Patients presented with hematuria, difficult-to-control hypertension, abdominal pain only, flank pain only, and both abdominal and flank pain (Table I). The mean systolic blood pressure at presentation was 136 ± 3 mm Hg SEM, and the mean diastolic blood pressure was 83 ± 2 mm Hg SEM. No patient presented with acute rupture. Computed tomography without contrast was the most frequently used imaging modality for diagnosis of RAA (n = 16; 40%); computed tomography angiography was the next most frequent (n = 11; 28%), followed by magnetic resonance angiography (n = 8; 20%), ultrasound (n = 3; 8%), catheter angiogram (n = 1; 3%), and unknown (n = 1; 3%). Six patients had concomitant extrarenal aneurysms, the most common site being the splenic artery.

Table I. Patient demographic, symptom, and risk factor information

	No.	*%*
Sex		
Female	23	58
Male	17	42
Age at diagnosis, years	56 ± 2	
Symptom		
Asymptomatic	29	73
Hematuria	4	10
Abdominal pain	3	8
Difficult-to-control hypertension	3	8
Flank pain	2	5
Comorbidities		
Hypertension	26	65
Diabetes mellitus	14	35
Smoking	8	20
Hypercholesterolemia	3	8
Coronary artery disease	3	8

Table II. Renal artery aneurysm (RAA) characteristics

	Nonsurgical (n = 43)	*Surgical (n = 16)*	*Total (n = 59)*	P *value*
Laterality				NS
Right	24	10	34	
Left	18	6	24	
Location				NS
Main bifurcation	26	9	35	
Main trunk	5	2	7	
Primary branch	4	2	6	
Pole artery	3	3	6	
Secondary branch	1	0	1	
Morphology				NS
Saccular	25	6	31	
Fusiform	7	1	8	
Bilobed	5	0	5	
Number of efferent branches				NS
None	2	0		
1	9	0		
2	17	3		
3	3	3		
Calcification				NS
Calcified	21	8	29	
Noncalcified	15	4	19	
Maximum diameter, cm	1.4 ± 0.1	2.1 ± 0.2	1.5 ± 0.1	<.001

NS, Not significant.

Aneurysm characteristics. Characteristics of all RAAs that were evaluated are shown in Table II. The majority of aneurysms were saccular in shape (n = 31; 53%), rather than fusiform (n = 8; 14%) or bilobed (n = 5; 8%); the shape of the remaining aneurysms was not documented (n = 15; 25%). Aneurysms originated from the main renal

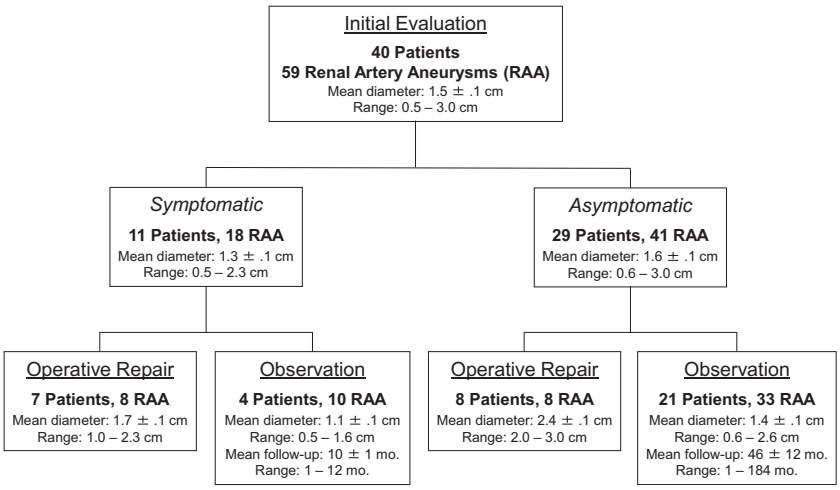

Fig 1. Treatment used for patients with renal artery aneurysms (*RAAs*).

artery bifurcation (59%), the main trunk (12%), a primary branch (10%), a pole artery (10%), a secondary branch (2%), and other/unknown (7%). Among operative aneurysms, 56% originated at the renal artery bifurcation, 19% from a pole artery, 13% from the main trunk, 13% from a primary branch, and none from a secondary branch. The location of operative aneurysms did not differ from the location of nonoperative aneurysms. The average number of efferent branches from the RAA was 1.8 ± 0.12 (range, 0-3), with no significant difference between operative and nonoperative aneurysms ($P = .15$). Half of all RAAs were unilateral (51%), and more than half of all RAAs were located on the right side (58%). There were fewer aneurysms with documented calcification (n = 19; 32%) than noncalcified aneurysms (n = 40; 68%).

The mean diameter of all RAAs was 1.5 ± 0.1 cm. Surgically treated RAAs were significantly larger than those observed (2.1 ± 0.2 cm vs 1.4 ± 0.1 cm; $P < .001$), and asymptomatic RAAs were significantly larger than symptomatic RAAs (1.6 ± 0.1 cm vs 1.3 ± 0.1 cm; $P = .041$). Eight patients (14%) had aneurysms that contained mural thrombus, six patients (10%) had aneurysms that were associated with ipsilateral fibromuscular dysplasia, and four patients (7%) had aneurysms that were associated with ipsilateral renal artery stenosis.

Treatment. Sixteen RAAs were repaired surgically, whereas 43 were followed by means of observation (Fig 1). Eight asymptomatic aneurysms (20%) were repaired and 33 asymptomatic aneurysms (80%) were not repaired. Asymptomatic RAAs that were repaired had a mean diameter of 2.4 ± 0.1 cm, with a range of 2.0 to 3.0 cm. No asymptomatic aneurysm <2.0 cm was repaired. Symptomatic aneurysms that were repaired had a mean diameter of 1.7 ± 0.1 cm, with a range of 1.0 to 2.3 cm. Ten symptomatic RAAs in four patients were not surgically repaired. Overall, symptomatic aneurysms that were not repaired had an average diameter of 1.1 ± 0.1 cm, with a range of 0.5 to 1.6 cm.

Technique. Six RAAs were repaired with vein patch (38%), four with prosthetic patch (25%), three with primary repair (19%), one with plication (6%), one with patch and implantation (6%), one with ex vivo repair (6%), and none required either planned or unplanned nephrectomy.

Outcomes and follow-up data. The median length of hospital stay for operative patients was 4 days. Perioperative complications occurred in three patients (20%). One patient had transient renal insufficiency, one patient had an incisional neuroma, and one patient had a self-limited cardiac arrhythmia. No patients had late postoperative complications, and the 30-day mortality rate was 0%. All patients presenting with symptomatic RAAs had resolution of symptoms after surgery. Of the 14 patients who had hypertension before operation, 1 patient was cured, 3 patients showed improvement in control of their hypertension (defined as a decrease ≥15 mm Hg or a decrease in number of antihypertensive medications), and 9 had no change. The number of antihypertensive medications taken was the same before and after surgery (mean = 2). The average blood pressures before and after surgery were not significantly different (138/86 before surgery, 138/84 after surgery).

In aneurysms that were not surgically repaired, serial imaging was performed at a mean of 17 ± 3 months between imaging studies. For all except two aneurysms, the imaging modality used at diagnosis was the same as that used for serial imaging. Two aneurysms were diagnosed by computed tomography angiography and followed serially by magnetic resonance imaging; therefore, for the calculation of growth rate, only magnetic resonance images were used to reduce variability because of difference in imaging modalities. The overall growth rate, calculated from 14 RAAs with two or more imaging studies available, was 0.60 ± 0.16 mm/y (Fig 2). Eleven of the 14 aneurysms were calcified and had a growth rate of 0.68 ± 0.19 mm/y compared with the 0.44 ± 0.24 mm/y growth rate of the three noncalcified aneurysms ($P = .567$). Nearly all of the 12 asymptomatic RAAs with imaging showed

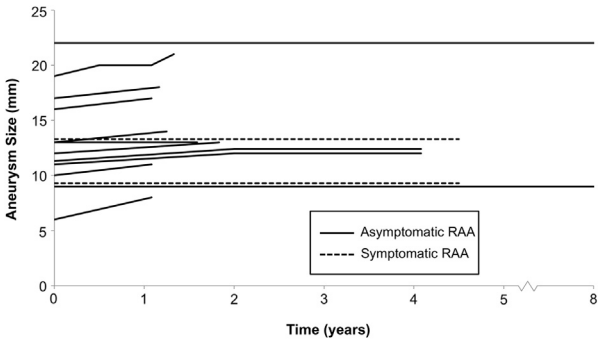

Fig 2. Aneurysm growth in patients treated nonsurgically. *RAA,* Renal artery aneurysm.

some growth, whereas neither of the two symptomatic aneurysms grew. There was no late acute complication for any aneurysm managed with observation, and the mean length of follow-up was 36 ± 9 months.

DISCUSSION

This retrospective study describes the clinical presentation, management, and long-term outcomes of RAAs at a large university medical center over a contemporary 10-year period from 2002 to 2012. It demonstrates that asymptomatic RAAs rarely rupture (in our experience, none ruptured) and have a very slow growth rate. The benign natural history in our series suggests that the current recommended treatment of asymptomatic RAAs, with repair at 2 cm, may be too aggressive.

The largest experience with RAA treatment, reported by Henke et al, studied 168 patients with 252 aneurysms over a time period of 35 years, from 1965 to 2000.[9] In this study, 55% of patients were asymptomatic, 19% had bilateral RAAs, and 79% were saccular. Three patients in this study presented with acute RAA rupture. Compared with the Henke et al study, our patient group had a higher incidence of asymptomatic presentation (73%), which is consistent with the rise in routine use of abdominal imaging in the past few decades. A more contemporary study by Morita et al looked at RAAs presenting from 1989 to 2009 and similarly showed an increased incidence of asymptomatically presenting RAAs of 70%[10]; more patients in our group presented with bilateral RAAs (49%) and fewer of our patients presented with saccular aneurysms (53%).

Treatment of patients in our study was based on current recommended guidelines but was modified by the individual clinician's expertise, taking into account patient presentation and comorbidities, as well as aneurysm characteristics such as calcification and size stability over time. Consistent with current guidelines, no asymptomatic aneurysm <2 cm in diameter was surgically repaired in this series. However, contrary to current recommendations, three asymptomatic aneurysms >2 cm were not surgically repaired. Two of these aneurysms were 2.1 cm, and one was 2.6 cm. Each of these aneurysms had been stable in size for at least 1 year and up to 5 years. The surgeon and patient decided together to continue serial imaging with consideration of surgical repair if size increased or if the aneurysm became symptomatic. Also, against the current recommendations, 10 symptomatic RAAs in four patients were not repaired. The patient with six RAAs who presented with difficult-to-control hypertension and fibromuscular dysplasia with multiple stenoses underwent balloon angioplasty of the renal arteries in an attempt to control the hypertension. In the patient with hematuria and a 1.2-cm calcified aneurysm, the surgeon concluded that because of the aneurysm calcification, it was probably an old aneurysm and could be followed with annual imaging. In the patient with two RAAs that were found during workup of microscopic hematuria, the surgeon thought that at maximum diameters of 1.1 cm and 1.2 cm in a patient with well-controlled hypertension, the aneurysms could safely be followed with annual imaging. In the patient with abdominal pain, well-controlled hypertension, and a 1.5-cm aneurysm, the surgeon thought that because of the aneurysm location, the patient was not a candidate for endovascular repair and would be at significant risk of losing a kidney if surgical repair were pursued. A computed tomography angiography in 1 year was recommended to observe for change in size. Despite the fact that the current recommendation was to surgically repair these aneurysms, none of these nonsurgically treated patients had development of progressive renal symptoms, and none of the aneurysms developed an acute complication. These experiences support the proposition that current recommendations for repair are too aggressive.

Many studies have shown a low morbidity and mortality for surgical repair of RAAs when performed by well-trained surgeons.[9,11-14] The Henke et al study showed excellent outcomes, with perioperative complications in only five patients, technical abnormalities requiring further intervention in seven patients, long-term complication of renal failure requiring hemodialysis in only one patient, and no deaths in patients undergoing surgery.[9] Henke et al also reported a significant decrease in blood pressure and the number of antihypertensive medications in patients who underwent surgical repair. Recently, endovascular repair of RAAs has emerged as an alternative to open surgical repair, allowing patients who previously would have been considered poor surgical candidates to undergo a less invasive treatment. Although most reports of endovascular repair of RAAs have been case studies or small series, good results have been obtained with endovascular treatment, whether by embolization or stenting, when cases have been carefully selected.[7,15-19] No patients in our study underwent endovascular repair, but we are increasingly considering endovascular repair as an alternative in high-risk patients and those who are reluctant to undergo a surgical procedure.

English et al reported a perioperative mortality rate of only 1.7% in 62 patients with 72 RAAs.[14] Our study reports no mortalities, low morbidity, and no unplanned nephrectomies; however, we did not find a significant

improvement in blood pressure after surgery in our series; only 29% of patients showed improvement in blood pressure, whereas the remainder of patients had no change.

Traditionally, indication for repair of an asymptomatic RAA is to prevent rupture. Studies have reported rupture rates of 0% to 14%, but most authors agree that the true rate is approximately 3%.[1,3,4,8,20-22] Three ruptures (1.2%) occurred in the Henke et al group, whereas Tham et al followed 83 RAAs for a mean of 4.3 years with no ruptures.[8,9] In another study, no ruptures were observed in 21 patients over an average of 3 years.[23] Our results, with no ruptures, add to this body of literature and confirm that rupture is an exceedingly rare occurrence.

Aneurysm size is used as a major factor to guide clinical decision-making; calcification of the arterial wall is an aneurysm characteristic that has also been used because calcification has been proposed to be a protective factor against rupture.[4,5] Harrow et al showed 15 of 18 ruptured aneurysms to be noncalcified, and Hidai et al showed that 14% of noncalcified aneurysms ruptured.[20,24] However, conclusive evidence that the presence of calcification, as well as the degree of calcification, affects the risk of aneurysm rupture is lacking.[4] In our study, we found no significant difference in the growth or rupture rates of calcified and noncalcified aneurysms.

The growth rate of asymptomatic RAAs has, to our knowledge, not previously been published; therefore, this study is the first to quantify the growth rate of RAAs. Our study found a growth rate of 0.60 ± 0.16 mm/y, but not all patients who were observed underwent annual imaging, and therefore the calculated growth rate was based on only 14 aneurysms. Furthermore, although three aneurysms >2 cm were followed nonsurgically, only one had multiple imaging studies. A larger sample with more frequent imaging is necessary to calculate a reliable growth rate. On the basis of our experience with aneurysm growth, it is not surprising that many other studies that have followed nonsurgical RAAs have observed a relatively stable size over years. On the basis of this calculated growth rate, >50% of the asymptomatic aneurysms in our study would not require surgical repair in the next 10 years if current guidelines for repair are used, and if the size threshold were increased, very few patients would require surgical repair during their expected lifespan.

Aneurysm morphology could not be identified in every case; however, RAAs frequently occurred at bifurcations and were saccular. These morphologic findings influence the type of repair and mandate surgical skill to avoid injury to branch vessels.

The high number of incidentally found asymptomatic RAAs suggests that the true incidence of RAAs may be higher than reported in most clinical series, but we were unable to determine risk factors to help screen patients who are likely to have RAAs. Because we had no ruptures, we were also unable to identify factors that are associated with aneurysm rupture.

One of the main limitations of this study is that it only included a small number of patients from a single tertiary care medical center. Although 59 aneurysms were identified in 40 patients, imaging modalities that were used in the diagnosis, management, and observation of these aneurysms varied by the preferences of referring physicians and the consulting vascular surgeon. To better define the growth rate, risk of rupture and long-term outcomes of asymptomatic RAAs, a larger study with observation of the natural history of aneurysms >2 cm will be required.

CONCLUSIONS

This contemporary series reveals that asymptomatic aneurysms rarely rupture and have a slow growth rate. There were no adverse outcomes in asymptomatic aneurysms >2 cm that were observed, despite current recommendations suggesting surgical repair. Although our numbers are small, these results suggest that the current recommendations for the surgical repair of asymptomatic RAAs may be too aggressive, and thus, further investigation is warranted.

AUTHOR CONTRIBUTIONS

Conception and design: JK, PL
Analysis and interpretation: JK, MHL, AP, EL, BD, PL
Data collection: JK, AP, EL, PL
Writing the article: JK, MHL, PL
Critical revision of the article: JK, MHL, BD, PL
Final approval of the article: JK, MHL, BD, PL
Statistical analysis: JK, MHL
Obtained funding: Not applicable
Overall responsibility: PL

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DISCUSSION

Dr Fred A. Weaver (*Los Angeles, Calif*). Suffice it to say that the natural history and risk of rupture of a renal artery aneurysm is uncertain. In the largest series in the literature from the University of Michigan, out of 252 renal artery aneurysms, only three ruptures were recorded. Other series have reported the rate of rupture to be 0% to 14%, but most report rupture rates less than 5%. Traditionally, indications for repair have focused on the prevention of rupture. This has resulted in the recommendation of repair for renal artery aneurysms >2 cm based really on very little data. In today's presentation, Dr Lawrence and his colleagues at UCLA provide additional information regarding the risk of rupture of a renal artery aneurysm with a focus on the size of the aneurysm as an indication for repair. Over a 10-year period at UCLA, 40 patients with 59 renal artery aneurysms were treated. The majority of patients were asymptomatic, with only 27% displaying symptoms known to be associated with the aneurysm. Sixteen aneurysms were repaired, all with diameters greater than 2 cm. Most of the patients who underwent repair were asymptomatic. At a mean length of follow-up of 3 years, in patients in whom the aneurysm was not repaired, no ruptures occurred. Their conclusion: the criteria of repair for a renal artery aneurysm greater than 2 cm may be too aggressive.

To begin the discussion of this thought-provoking paper, I have three questions. First, one of the concerns I have with this recommendation is that not all renal artery aneurysms are the same. A 2-cm renal artery aneurysm of the main renal artery may not be the same as a 2-cm aneurysm of a branch vessel. In your paper, you make no distinction in the size of the aneurysm with regards to the renal branch involved. Do you think the rupture risk is equivalent between the two? Should the location of the aneurysm as well as the size be considered when contemplating repair? Second, how would you factor age into your recommendation? There is evidence to suggest particularly in the premenopausal female that risk of rupture is greater. That certainly has been our experience at USC. Should the threshold for repair be dependent on other factors including age, sex, presence of hypertension and menopause status? Finally, if 2 cm is too small, then what would be your threshold for repair based on size? Should it be 2.5 cm, 3 cm? In general, I agree with your recommendation with regard to size and indication for operation, but as with most things in medicine, it is difficult to draw distinct lines, and individual patient factors are probably more important than size alone.

Ms Jill Q. Klausner. Dr Weaver brings up three excellent points. Because our series included no ruptured aneurysms, it is not possible for us to make conclusions regarding the effect of location, age, gender, menopause status, or presence of hypertension on rupture risk. We found no statistically significant difference in the location of operative versus nonoperative aneurysms, suggesting that location is not currently being used as the basis for treatment decisions. Only one patient in our series was a female of child-bearing age, and her aneurysm was surgically repaired based on established guidelines to repair renal aneurysms of women of child-bearing age. The largest renal aneurysm series, from the University of Michigan, has shown that repair of renal artery aneurysms is associated with decreased blood pressure, but our smaller series did not find a similar effect. We agree that size greater than 2 cm should not be the only indication for repair. We also recognize the complexity of this issue and the multiple factors that should enter into a decision about renal artery aneurysm repair, and we are currently conducting a large multi-institutional trial to determine specific risk factors for aneurysm growth and rupture, as well as a specific threshold for repair, based on size, if such a recommendation is warranted.