

Renal artery stenting slows the rate of renal function decline

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Objective: The primary objective of this study was to analyze renal artery interventions performed at a tertiary medical center and to evaluate improvements in hypertension and renal excretory function.

Methods: A retrospective analysis was performed of patients treated at a tertiary medical center from January 2001 to December 2005. All patients treated with renal artery stenting by the Interventional Radiology or Endovascular Services were included. Descriptive and inferential analyses were performed.

Results: Forty patients with renal artery stenosis were evaluated for renal artery stenting, of these 22 were followed up with medical management. Twenty-six renal artery stents were placed in 18 patients (mean age, 70 ± 8 years), of whom 76% were treated for multidrug resistant hypertension, and 24% were treated for renal salvage. Mean follow-up was 15 months. Patients experienced a significant reduction in hypertension and in the number of antihypertensive agents, but this significance deteriorated at 6 months, when their blood pressure and number of medications returned to preprocedural values. Compared with a cohort that was followed up with medical management, the rate of renal function decline improved from -0.08 mg/dL per month to 0.00 mg/dL per month ($P < .05$) after intervention. Patients with baseline chronic renal insufficiency experienced the greatest benefit from renal artery stenting.

Conclusions: Renal artery stenting initially improves hypertension control, but the durability is lost after 6 months. Renal artery stenting dramatically slows the rate of renal function decline and could potentially delay a patient's requirement for hemodialysis. (*J Vasc Surg* 2007;45:726-32.)

Hypertension resulting from renovascular stenosis has been recognized for the past 70 years, but the appropriate treatment of this disease has been plagued by inconsistent results. Initial reports of nephrectomy for treatment of hypertension implicated the diseased kidney as the etiology of hypertension, but after nephrectomy, only 20% of patients remained normotensive at the 1-year follow-up.¹

Goldblatt² further focused attention on the kidney by demonstrating hypertension in a canine model after narrowing the renal artery.² Through a series of experiments, he suggested that a humoral mechanism was accounting for the hypertensive effects, and later, renin was localized to the juxtaglomerular cells of the kidney.³ After this period, operative revascularization was the mainstay of treatment, but endoluminal treatment was soon to follow when Grüntzig performed the first renal artery angioplasty.³

As endoluminal therapy progressed during the last 20 years, the treatment of renal atherosclerotic lesions shifted from angioplasty to angioplasty and stenting. Two random-

ized controlled trials compared angioplasty without stenting in the renal arteries with medical management, and both failed to show a clinically relevant effect on blood pressure and renal excretory function.^{4,5} The reintervention rate for assisted patency with angioplasty approaches 10% to 20% in the renal arteries, and this may contribute to the lack of impact on blood pressure and renal function. The initial surge of angioplasty and stenting of the renal arteries was thought to remedy the high restenosis rate and to potentially improve results.

Several series evaluate renal artery angioplasty with stenting, but to our knowledge no randomized controlled trials have compared renal artery stenting with best medical management. The results are difficult to interpret given the wide range of variability in outcomes. Several groups of providers treat the disease, including primary care physicians, internists, nephrologists, cardiologists, interventional radiologists, and vascular surgeons.

The purpose of this study was to examine the outcomes for renal artery stenting at a tertiary medical center and to evaluate patient selection for improvements in both hypertension and renal excretory function.

METHODS

We performed a retrospective review of patients that were presented at a Multidisciplinary Renovascular Conference, which included nephrologists, interventional radiologists, and vascular surgeons. Patients with atherosclerotic renal artery disease were included between January 2001 and June 2006. Patients presented at this conference were referred by nephrologists and internists for hypertension

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requiring multiple medications or worsening renal function with simultaneous duplex ultrasound evidence of renal artery stenosis. Renal ultrasound criteria reviewed were velocity ratio, kidney size (cm), resistive index, and peak systolic velocity (PSV).

The multidisciplinary team evaluated each patient for potential renal artery stenting, and as a result, a cohort of patients was followed up with medical management. The reasons patients were not offered stenting included an inadequate antihypertensive regimen, poor patient compliance, acute medical conditions, resistive index >0.80 , and lesions that were $<70\%$ stenosed. None of the criteria were exclusive. Inadequate antihypertensive regimen was defined by our nephrologist and most commonly consisted of a patient that was on multiple low-dose medications. Other patients were not taking angiotensin-converting enzyme inhibitors or angiotensin-receptor blockers. Resistive index was evaluated in the patient's overall presentation but was not used as absolute exclusion.

A retrospective chart analysis was performed of inpatient and outpatient records. The patients who were not offered stenting were followed up as a comparison with those who did undergo stenting.

Initial screening of the renal vascular bed was performed with duplex ultrasound criteria, and if the study was noncontributory, magnetic resonance angiography (MRA) was performed as a confirmatory test. The definitions for reporting renal artery interventions were adapted from Rundback et al.⁶

- Renal duplex criteria for a $\geq 60\%$ stenosis at an angle of $\leq 60^\circ$ included a renal artery/aorta PSV ratio >3.5 , >180 cm/s renal artery PSV, or a resistive index difference of .15 between kidneys.
- MRA-detected renal artery stenosis were determined by a ratio of the narrowest portion of the vessel to reference vessel diameter.
- Hypertension was defined as a 6-month progression of systolic blood pressure (SBP) >140 mm Hg or a diastolic blood pressure (DBP) >90 mm Hg.
- Chronic renal insufficiency was defined as 6 months of an elevated serum creatinine level ≥ 1.5 mg/dL.
- Ostial lesions were defined as lesions ≤ 1 cm of the renal artery orifice.⁶

All renal artery stents in this cohort were considered a technical success if $<30\%$ residual stenosis after stent placement was confirmed with postdeployment angiogram. Anatomic placement of the stent with 1-mm to 2-mm projection into the aorta was the standard technique but was not consistently recorded.

Outcomes for hypertension were defined as cure (DBP <90 mm Hg, SBP <140 mm Hg, and no medications), improvement (DBP <90 mm Hg, SBP <140 mm Hg, the same number or reduced number of medications, or DBP reduction of 15 mm Hg on the same medications), and failure (neither a cure or improvement).⁶ Blood pressure and number of medications were recorded 1 month before the procedure, at the time of the procedure, and then at

each follow-up visit. To express the dynamic trend of patients' hypertensive control with fluctuating medication adjustments, we analyzed each variable of SBP, DBP, and number of medications at each time interval after intervention.

Outcomes of renal function were determined by plotting the inverse creatinine slope over time using breakpoint analysis.^{6,7} Inverse creatinine plots have been validated as a measure for estimating progression of renal failure.⁸ Plotting the inverse transforms the creatinine curve into a linear relationship, and after this transformation, a slope can be calculated by a least squares regression technique. Inverse creatinine plots were created 24 months before the intervention and then at each follow-up visit. Individual regression plots were performed to evaluate the response to treatment, and then mean values of slope were taken for group analysis. Clinical events included progression to hemodialysis, nonfatal myocardial infarction, and death.

Time sensitive values were recorded ± 2 weeks of the month and ± 2 months of the year for each time interval recorded. For the medically managed patients, the time analogous to intervention was the date at which they were discussed for potential intervention.

RESULTS

The Multidisciplinary Team evaluated 40 patients: 29 renal artery stents were placed in 18 patients, and 22 patients were followed up with medical management. Baseline characteristics were similar between the two groups (Table I). The renal artery stent group had a higher proportion of patients with baseline renal insufficiency and slightly higher systolic blood pressures.

Initial ultrasound evaluations were similar between the two groups (Table II). No significant differences existed in ultrasound measurements, and the difference between the percentage of patients with resistive index >0.8 was not significant. Nine patients in the medical treatment group underwent MRA, and the study failed to confirm the renal artery lesion to be $>60\%$ in five of the patients. Four patients in the renal artery stent group underwent MRA, and all four had confirmed stenoses.

All 29 renal artery stenting procedures were considered successful. No major procedure-related events (acute thrombosis, dissection, renal failure, rapid renal function decline, or hemorrhage) or procedure-related deaths occurred. Three patients experienced restenosis in the follow-up period, equating to a mean patency of 110 months (95% confidence interval [CI], 99 to 121 months; Fig 1). Two patients were treated with angioplasty, and one required angioplasty and stenting.

When we evaluated the impact on hypertension by standardized reporting guidelines, no patient experienced a cure. Only one patient with a renal artery stent met criteria for an improvement in hypertension, and the rest were categorized as failures.

A paired analysis was performed from each point of follow-up back to the patients' preprocedural values (Table III) for SBP, DBP, and number of antihypertensive medications. No significant changes were found in blood pres-

Table I. Cohort demographics

Characteristic*	RAS n (%)	Medical treatment n (%)	P
Patient total	18	22	
Age, years	72 ± 9	67 ± 13	.518
Risk factors			
Hypertension	18 (100)	22 (100)	—
Hypercholesterolemia	14 (82)	16 (73)	.479 [†]
Diabetes	5 (29)	6 (27)	.883 [†]
Smoker			.249 [†]
Current	6 (35)	3 (14)	
Remote history	2 (12)	5 (23)	
Vascular disease:			
CAD	8 (47)	11 (50)	.855 [†]
CVD	5 (29)	6 (27)	.883 [†]
PVD	6 (35)	8 (36)	.945 [†]
Antihypertensive medications:			
β-blocker	14 (93)	16 (73)	.116 [†]
ACE	8 (53)	13 (59)	.729 [†]
ARB	6 (40)	9 (43)	.864 [†]
Diuretic	9 (60)	15 (68)	.609 [†]
Creatinine (mg/dL)	1.5	1.0	<.05 [†]
Chronic renal insufficiency	9 (52)	5 (22)	.091 [†]
SBP (mm Hg)	162 ± 17	142 ± 21	<.05
DBP (mm Hg)	75 ± 13	73 ± 13	.959
Number taken	3.5	4.0	.420 [‡]
Bilateral renal artery stenosis	11 (61)	12 (46)	.899 [†]
Previous stent	1 (5.9)	4 (18)	.363 [†]

RAS, Renal artery stenting; CAD, coronary artery disease; CVD, cerebrovascular disease; PVD, peripheral vascular disease; ACE, angiotensin converting enzyme inhibitor; ARB, angiotensin receptor blocker; SBP, systolic blood pressure; DBP, diastolic blood pressure.

*Continuous data are presented as mean ± standard deviation; categorical data as n (%).

[†]Pearson χ^2 test, Fischer exact where appropriate.

[‡]Mann-Whitney *U* test, and measure of central tendency reported as median.

Table II. Ultrasound measurements

Measurement (mean ± SD)	RAS (n = 18)	Medical treatment (n = 22)	P
Length (cm)	11.6 ± 0.8	10.0 ± 0.3	.627
AoPSV (cm/s)	86 ± 9	82 ± 20	.391
PSV (cm/s)	357 ± 39	276 ± 74	.288
PSV/AoPSV ratio	4.7	3.7	.478*
Resistive indices	0.75 ± 0.02	0.72 ± 0.04	.947
Resistive index >0.8 (%)	8 (44)	7 (32)	.480 [†]

RAS, Renal artery stent; AoPSV, aortic peak systolic velocity; PSV, peak systolic velocity.

*Mann-Whitney *U* test, and measure of central tendency reported as median.

[†]Pearson χ^2 test, Fischer exact where appropriate.

sure or medications within the medical management group. In the patients treated with renal artery stenting, the median number of medications initially dropped from 3.5 to 1.0 at the 1-month follow-up. At the 3-month and 6-month follow-up periods, the median number of medications in-

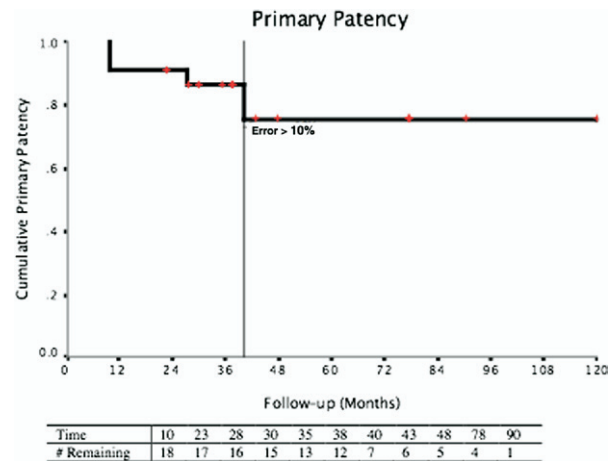


Fig 1. Kaplan-Meier Curve for primary patency among all patients treated with stenting. The vertical line represents the point at which error exceeds 10%.

creased to 3.0. At the 12-month follow-up visit, the number of antihypertensive medications was not significantly altered compared with preprocedural values. The SBP pressure was only significantly altered at the 3-month follow-up visit. Further analysis was performed comparing patients with unilateral and bilateral stenosis as well as stratifying patients with chronic renal insufficiency. The results were the same: the durability of the procedure was lost at 6 months.

To evaluate the impact on renal function, we plotted the inverse creatinine concentration vs time (Fig 2). When the 2-year period before intervention was evaluated, patients who were selected for renal artery stenting were experiencing a more rapid rate per month (−0.08 mg/dL) of decline than patients who were not stented (−0.03 mg/dL). After renal artery stenting, patients experienced a significant ($P < .05$) plateau in their renal function compared with their preprocedure slope; whereas, the medically followed up group continued at the same rate of decline during the follow-up period.

Patients treated with renal artery stenting were then stratified by resistive indices, unilateral or bilateral disease, and baseline chronic renal insufficiency. No significant difference was found in the per-month postintervention inverse creatinine slope for patients with a resistive index <0.8 (0.04 mg/dL) compared with a resistive index >0.8 (0.05 mg/dL). Patients with unilateral disease experienced a per-month postprocedural slope of 0.00 mg/dL, and those with bilateral disease had a slope of 0.02 mg/dL. This difference was not significant. The patients with baseline chronic renal insufficiency experienced the greatest benefit, with a per-month postprocedural slope of 0.03 mg/dL compared with −0.03 mg/dL for those patients without renal insufficiency ($P < .05$).

Within this cohort, no patient progressed to require hemodialysis during a mean follow-up period of 15 months. One patient had a stroke 2 years after renal artery stent placement. The most common event in the follow-up

	<i>Time (months)</i>							
<i>Variable</i>	<i>0</i>	<i>1</i>	<i>3</i>	<i>6</i>	<i>12</i>	<i>24</i>	<i>36</i>	<i>48</i>
Medical treatment								
SBP*	142 ± 21	151 ± 20	152 ± 12	132 ± 12	136 ± 13	146 ± 26	117 ± 12	137 ± 37
DBP*	73 ± 13	70 ± 12	73 ± 8	62 ± 16	69 ± 15	73 ± 14	66 ± 15	78 ± 28
Meds, n [†]	4	4	4	4	4	4	4	4
RAS								
SBP*	162 ± 17	151 ± 37	148 ± 21**	171 ± 23	152 ± 22	146 ± 10	167 ± 27	166 ± 30
DBP*	75 ± 13	74 ± 15	80 ± 15	78 ± 8	78 ± 8	76 ± 9	78 ± 13	80 ± 20
Meds, n [†]	3.5	1 [‡]	3 [‡]	3 [‡]	4	4	4	4

Measurements of declining renal function have typically been a 20% change in serum creatinine level at each follow-up visit, and reports for stenting have ranged from "no benefit" to "loss of durability" to "a significant improvement." This reporting method does not take into account the variability of the serum creatinine level with time and gives little information about rate of progression

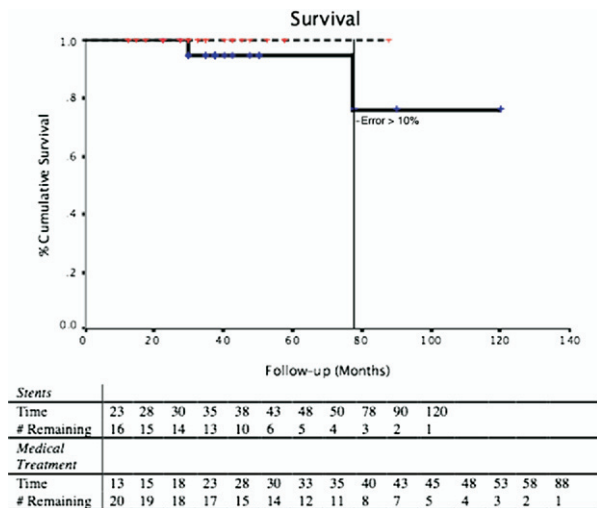


Fig 4. Kaplan-Meier survival curve. The *solid line* represents patients with renal artery stents, and the *hashed line* represents patients followed up with medical management. The *vertical line* represents the time at which error > 10%.

or decline. Harden et al¹³ first used the inverse creatinine curve as a way to evaluate renal function over time both before and after intervention. Watson et al⁷ used this method to demonstrate a dramatic improvement in renal function, with either stabilization or improvement in all surviving patients. Harden evaluated patients only with advanced renal dysfunction (3 patients were receiving hemodialysis) and found a high follow-up mortality (median survival, 22 months) and a 78% improvement in renal function. Watson evaluated all patients presenting with renal artery stenosis and found that 100% of patients experienced a favorable improvement in their renal function.

In the present study, no patient experienced immediate worsening of renal function, defined as a more negative inverse creatinine slope. During the mean 15-month follow-up, all patients benefited from renal artery stenting by slowing the rate of renal function decline. Although the magnitude of benefit for patients appears to be larger for patients with existing baseline renal insufficiency, the patients with a serum creatinine level of <1.5 mg/dL also responded favorably compared with their baseline. Some studies have suggested that patients with bilateral disease have an improved response. We identified a trend for improvement in patients with bilateral disease, although this was not significant.

Radermacher et al¹⁴ have reported that patients with resistive index >.80 do not experience improvement in blood pressure or renal function after renal artery angioplasty alone.¹⁴ These data have been used as a selection criteria for patients to undergo renal artery stenting, but in our results, the resistive index did not have a significant impact on the patients' improvement after stenting. The difference may be attributable to the inherent differences in angioplasty vs stenting. In Radermacher's evaluation, the

patients with a resistive index >.80 experienced a decline in their creatinine clearance during the follow-up period, but the creatinine clearance was never plotted for time period leading up to intervention. Although these patients were categorized as failures, the rate of their decline may have improved over time. Rocha-Singh et al¹⁵ also demonstrated an improvement or stabilization of renal function after renal artery stenting irrespective of resistive indices.

The resistive index is a measure of the underlying chronic parenchymal disease, but it does not seem to absolutely identify patients that may respond to endoluminal treatment of renal artery stenosis. In our evaluation, no patient progressed to hemodialysis, but by demonstrating an improvement in the rate of decline, we expect to delay hemodialysis therapy.

The outcome of blood pressure measurement is not significantly altered by renal artery stenting. One randomized controlled trial comparing renal artery angioplasty without stenting with best medical management was not able to show a clinically significant alteration in blood pressure management at 6 to 12 months postprocedure.⁵ There are no randomized trials for angioplasty with stenting. Several retrospective analyses exist with various results, but none are able to document a high cure rate.¹⁶⁻²² Nearly all patients require medical management after their procedure, and this is analogous with our results.

Patients typically have a dramatic blood pressure response immediately after renal artery stenting. They may initially wean off all their medications, but at each follow-up visit, their blood pressure continues to rise, requiring the resumption of antihypertensive therapy. Like previous retrospective studies, the durability of the stent for hypertensive control is lost. The fact that these patients do respond initially implicates the renal artery in their hypertensive management. The reason for failure was initially thought to be due to high restenosis rates with angioplasty alone, but now stenting is essentially able to approach a near 99% secondary patency rate.^{17,23} With stenting, hypertension control returns to baseline only after a short period, even when stratified for patients with unilateral or bilateral disease. Further investigation will be required to delineate the physiologic impact of dilating the renal artery and to determine why this response is lost over time. Using the indication of multi-drug hypertension as a sole indication for intervention should be cautioned.

This study is limited by a small cohort size and a retrospective analysis. The medically managed patients were not true controls but instead were selected comparisons that were identified by a conference of specialists making a comprehensive decision not to treat those patients. We attempted to create a weight-based formula for each antihypertensive medication, but the difference in dosing regimens, brands, and classes of medications made this task impossible. Given the small number of patients, we were unable to establish an impact on cardiac events or mortality after intervention.

CONCLUSIONS

This study confirms that renal artery stenting can be performed with a nearly negligible periprocedure complication rate, and 100% assisted patency can be obtained. In patients undergoing intervention for hypertension, the benefit is only transient, but renal function significantly improves with renal artery stenting.

The etiology of renal disease is multifactorial, involving systemic lipid disorders, increasing age, segmental glomerulosclerosis, fibrosis, hypertensive nephropathy, endothelial dysfunction, and diabetic nephropathy. Renovascular disease is just one factor that when reversed with renal artery stenting has the potential to delay progressive loss of renal function over time. The ability to arrest this progression has not been established. Further randomized controlled trials will be needed to determine the impact on hemodialysis, cardiac events, and mortality.

AUTHOR CONTRIBUTIONS

Conception and design: ZA, BS, CA

Analysis and interpretation: ZA, BS, HC

Data collection: DC, VC

Writing the article: ZA, VC, DC

Critical revision of the article: BS, HC, CA

Final approval of the article: ZA, BS

Statistical analysis: ZA, DC, VC

Obtained funding: Not applicable

Overall responsibility: ZA

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DISCUSSION

Dr R. Eugene Zierler (Seattle, Wash). This report from the Madigan Army Medical Center adds to the growing body of evidence that documents the results of percutaneous interventions for atherosclerotic renal artery disease. Despite lingering concerns over the efficacy and durability of renal artery stenting, the favorable early technical results of catheter-based interventions, and the relatively high risks of open surgery in the atherosclerotic patient population, have made the less-invasive approach overwhelmingly preferred. In fact, renal artery bypass surgery in this setting has all but disappeared. So even though this 5½-year study included only

29 renal artery stents in 18 patients, I suspect that the corresponding number of open renal artery operations was even lower. Looking at outcomes in terms of hypertension and renal function, the main conclusion is that improvement in blood pressure control was transient; however, the rate of decline in renal function was significantly better than in a similar cohort of patients followed without intervention.

Since the indications for intervention in this study are listed as drug-resistant hypertension in 77% and renal salvage in 23%, this suggests that only a minority of the patients (that is, 23% or about

four) actually experienced any long-term benefit. Patient selection and timing are the key factors in maximizing benefit and minimizing both the risk and cost of intervention. The results of this study focus our attention on the level of renal function and rate of renal function decline. Other clinical features that may correlate with outcome include kidney length (renal atrophy), severity of renal artery stenosis, unilateral or bilateral renal artery disease, and renal resistive index or other measures of parenchymal blood flow. With that in mind, I have the following questions:

Number one, experience suggests that there is a "window of opportunity" for renal revascularization in preservation of renal function. Other than rate of decline in renal function, what factors would you take into account when selecting patients with renal insufficiency for renal artery intervention?

Number two, some hypertensive patients do appear to benefit from renal revascularization, although this was not shown in your study. Why do you think this patient group failed to respond to intervention? Are there any situations in which you would still recommend renal artery intervention for hypertension alone?

Number three, the manuscript mentions that nine of the patients followed without intervention underwent renal MRA, and a renal artery stenosis of $>60\%$ was not confirmed in five. Was the initial diagnosis of renal artery stenosis in these patients made by duplex ultrasound? If this is the case, can the authors comment on this apparent discrepancy between these two imaging methods?

I appreciate the opportunity to review and discuss this paper.

Dr Zachary Arthurs. Thank you, Dr Zierler. The first question is a question we are working through right now. Who are the patients that are responders and who should we treat? The rate of decline and the window of opportunity is something that we are now looking for and hopefully we can randomize these patients based on the indication for renal salvage. The point at which to intervene we do not know.

The factors that we look for that are appealing are the fact that we have now realized that the kidney obviously has humoral function. The impact of the renal angiotensin has well been defined with split renal function and its potential to damage a kidney with unilateral renal artery stenosis. The endothelium also has endocrine function impacting blood pressure control, and now the natriuretic peptides released from the heart are recognized as having an endocrine role. All these factors may play a role in renal artery hypertension. Maybe measuring natriuretic peptide could give the clinician a marker along with LVH that the patient's heart is attempting to respond to volume overload caused by a renal artery lesion. This is one of the measures that we would like to explore. We have not yet defined the percentage of renal function decline that should be treated with renal artery stenting. I think it is an excellent point.

The concept of why did the hypertensive patients fail. I think that is what was most intriguing to me. Many of these patients are not taking any antihypertensive medications after their procedure, but progressively the medications are returned as their hypertension returns. This suggests that maybe there is another humoral or neural mechanism that allows an initial response but over time the kidney fails once again. The kidney may lose its auto feedback mechanisms, or it may be progression of underlying parenchymal disease. I do not have a real good answer to explain that initial transient response.

At this point I should recognize the fact that Dr Zierler, having dedicated so much time to the vascular laboratory, would have picked out the four discordances in MRA versus duplex and I did not look at those specifically. If there was a patient that was felt to maybe be poorly compliant, maybe could have gone up on their medications and the lesion was borderline on ultrasound, those were the patients that we would get a confirmatory study. If the MRA was either borderline or less than our ultrasound, we would use that to watch that patient and attempt medication control.

Dr James Watson (Seattle, Wash). You just answered one of my questions but I am curious as to what are the current indications for renal artery intervention at Madigan, and who will you intervene on when you see this patient in clinic?

Dr Arthurs. We are becoming extremely cautionary for intervening for hypertension as a sole indication and we are looking at evaluating a series of patients who are randomized based on their rate of renal function decline.

Dr Fred Weaver (Los Angeles, Calif). I may have missed it but did you provide any numbers with regard to immediate technical success of your stenting procedures?

Dr Arthurs. I left that out completely because there was 100% technical success at the time of the procedure and we had no periprocedural morbidity associated with regards to acute thrombosis, embolization, dissection or groin complications in this small series.

Dr Weaver. Do you have any follow-up data with regards to restenosis?

Dr Arthurs. At the 3 and 6-month mark, we have documented imaging for restenosis, and I do have a restenosis slide in the manuscript, but it is roughly the same across the cohort of 78% with 100% secondary patency.

Dr Weaver. The observation that the hypertensive improvement deteriorates after a year has been made by a number of authors, as you know. The University of Rochester reported a large series where at 5 years, the hypertension benefit deteriorated over the 5-year followup despite the fact that 100% of the renal artery stents were patent. With regards to renal function, do you use any renal protection when you do these procedures, either embolic protection or CO_2 , to minimize renal damage? As you know, with renal stenting, renal function actually deteriorates in a significant number of patients. Either the iodinated contrast and/or cholesterol embolization from the plaque is hypothesized as the culprit. What are your thoughts on renal protection?

Dr Arthurs. Periprocedural data were limited, but no protection devices were used. There were cases that required adjunctive measures such as lytic therapy.

Dr Wesley Moore (Los Angeles, Calif). As Dr Zierler indicated in his discussion, with the availability of stent balloon angioplasty, there was a rapid transition from direct surgical repair to this technique with the assumption that the two were equivalent. I think that we ought to take a hard look at whether or not they really are equivalent. I wonder if you had the opportunity to compare the data that you have presented with earlier surgical experience. My recollection is that the cure or improved rate using the same parameters that you have described as about 75% over long periods of time when direct surgical reconstruction of the renal artery was carried out. In view of your results and the results reported from UCSD this morning, I wonder if we ought to reconsider whether or not the two are equivalent and consider at the risk of being considered a surgical dinosaur maybe we ought to start looking again at direct renal surgical repair.

Dr Arthurs. Sir, I agree completely. It is hard for me to see how we transitioned to where we have. There is only one paper that I know of that has a comparison of medical management to operative revascularization, and that was in 1973. A large proportion of patients had fibromuscular dysplasia, only about 40 patients in each group, and they were able to show a benefit with regard to blood pressure and creatinine. They also had extremely long follow-up, 10 years. It suggested that there was a survival benefit as compared to medical management. What you sacrifice is a perioperative mortality rate associated with surgery, but that is the only study I know that is medical management compared to surgery.

Then we made the leap to angioplasty. To echo Dr Schneider's words at this conference, once you go to endoluminal therapy, the endoluminal problems are fixed with another endoluminal therapy. Once angioplasty was known to have a high restenosis rate, an acute thrombosis rate, and a learning curve, instead of going back to surgery we went on to stents. Now, we utilize primary stents primarily in the renal artery. The comparisons of angioplasty to surgery are really marginal and then we do not have any studies with renal artery stenting versus medical management or stenting versus surgery for that matter. It would take a well-designed randomized controlled trial to define these differences in outcome.