Quality Improvement Guidelines for Angiography, Angioplasty, and Stent Placement for the Diagnosis and Treatment of Renal Artery Stenosis in Adults

Louis G. Martin, MD, John H. Rundback, MD, Michael J. Wallace, MD, John F. Cardella, MD, John F. Angle, MD, Sanjoy Kundu, MD, Donald L. Miller, MD, and Joan C. Wojak, MD

J Vasc Interv Radiol 2010; 21:421–430

Abbreviations: FMD = fibromuscular dysplasia, RAS = renal artery stenosis

PREAMBLE

The membership of the Society of Interventional Radiology (SIR) Standards of Practice Committee represents experts in a broad spectrum of interventional procedures from the private and academic sectors of medicine. Generally, Standards of Practice Committee members dedicate the vast majority of their professional time to performing interventional procedures; as such they represent a valid broad expert constituency of the subject matter under consideration for standards production.

Technical documents specifying the exact consensus and literature review methodologies as well as the institutional affiliations and professional credentials of the authors of this document are available upon request from SIR, 3975 Fair Ridge Dr., Suite 400 N., Fairfax, VA 22033.

METHODOLOGY

SIR produces its Standards of Practice documents using the following process. Standards documents of relevance and timeliness are conceptualized by the Standards of Practice Committee members. A recognized expert is identified to serve as the principal author for the standard. Additional authors may be assigned dependent upon the magnitude of the project.

An in-depth literature search is performed using electronic medical literature databases. Then a critical review of peer-reviewed articles is performed with regard to the study methodology, results, and conclusions. The qualitative weight of these articles is assembled into an evidence table, which is used to write the document such that it contains evidence-based data with respect to content, rates, and thresholds.

When the evidence of literature is weak, conflicting, or contradictory, consensus for the parameter is reached by a minimum of 12 Standards of Practice Committee members using a modified Delphi consensus method (Appendix A) (1,2). For purposes of these documents, consensus is defined as 80% Delphi participant agreement on a value or parameter.

The draft document is critically reviewed by the Revisions Subcommittee members of the Standards of Practice Committee by telephone conference calling or face-to-face meeting. The finalized draft from the Committee is sent to the SIR membership for further input/criticism during a 30-day comment period. These comments are discussed by the Subcommittee, and appropriate revisions made to create the finished standards document. Before its publication the document is endorsed by the SIR Executive Council.

INTRODUCTION

This guideline was revised collaboratively by the American College of Radiology and SIR. The present document is a revision of the SIR Quality Improvement Guideline entitled "Angiography, Angioplasty and Stent Placement for the Diagnosis and Treatment of Renal Artery Stenosis in Adults" published previously in the *Journal of Vascular and Interventional Radiology* (3). This collaborative document has also been published by the American College of Radiology in their Practice Guideline for the Performance of Angiography, Angioplasty, and Stenting for the

From the Department of Radiology (L.G.M.), Emory University Hospital, Atlanta, Georgia; Advanced Interventional Radiology Services (J.H.R.), Teaneck, New Jersey; Interventional Radiology, The University of Texas M.D. Anderson Cancer Center (M.J.W.), Houston, Texas; System Radiology (J.F.C.), Geisinger Health System, Danville, Pennsylvania; Department of Radiology (J.F.A.), University of Virginia Health System, Charlottesville, Virginia; Department of Medical Imaging (S.K.), Scarborough General Hospital, Toronto, Ontario, Canada; Department of Radiology and Radiologic Sciences (D.L.M.), Uniformed Services University of the Health Sciences; Department of Radiology (D.L.M.), National Naval Medical Center, Bethesda, Maryland; and Department of Radiology (J.C.W.), Our Lady of Lourdes Medical Center, Lafayette, Louisiana. Received November 1, 2009; final revision received December 6, 2009; accepted December 28, 2009. **Address correspondence to** L.G.M., c/o Debbie Katsarelis, 3975 Fair Ridge Dr., Suite 400 N., Fairfax, VA 22033; E-mail: lmart03@emory.edu

A previous version of this article appeared in *J Vasc Interv Radiol* 2002; 13:1069–1083; and *J Vasc Interv Radiol* 2003; 14(Suppl):S297–S310.

None of the authors have identified a conflict of interest.

DOI: 10.1016/j.jvir.2009.12.391

Diagnosis and Treatment of Renal Artery Stenosis in Adults (revised in 2009).

The SIR document herein has been adapted and abbreviated to meet the format and content requirements of SIR Quality Improvement document template and does not include sections pertaining to credentialing, documentation, or radiation safety. The content, language, and thresholds published in this document are equivalent to those in the American College of Radiology version.

Hypertension is a common problem, estimated to affect approximately 25%–30% of the adult population in the United States. It causes significant morbidity and mortality, with end-organ damage frequently affecting the kidneys and cardiovascular system. Although hypertension is most often "essential" or idiopathic in origin, renovascular disease is an important and potentially remediable cause of hypertension and progressive renal insufficiency.

Approximately 3%–5% of the hypertensive population has a renovascular etiology for their hypertension. Increasing age and coexisting atherosclerosis have significant effects on the prevalence of renovascular hypertension. The incidence of renovascular hypertension varied from 0% to 29% (with a weighted mean of 4%) among 8,899 patients in 12 studies (including their own) reviewed by Anderson et al (4).

Certain clinical scenarios may significantly increase the likelihood that renovascular disease is present (eg, abrupt onset of hypertension before the age of 30 years), but increasingly this is a disorder that is seen in older patients with complicating illnesses such as diabetes and systemic atherosclerosis that may render the diagnosis (and treatment) more difficult. This document reviews those circumstances that should prompt further evaluation for a possible renovascular cause of hypertension or chronic renal insufficiency. It also discusses the noninvasive imaging and the angiographic evaluation of such patients. Practice guidelines for the performance of renal artery angioplasty and stent placement are reviewed, as well as considerations of what constitutes a successful intervention.

These guidelines are written to be used in quality improvement programs to assess renal artery procedures. The most important processes of care are (i) patient selection, (ii) performing the procedure, and (iii) monitoring the patient. The outcome measures or indicators for these processes are indications, success rates, and complication rates. Outcome measures are assigned threshold levels.

DEFINITIONS

For the purpose of this guideline, the following definitions apply:

Cardiac disturbance syndrome is defined as recurrent "flash" pulmonary edema that is not believed to be secondary to impaired cardiac function, as sometimes seen in the setting of bilateral renal artery stenosis (RAS) or unilateral stenosis of the renal artery to a solitary kidney (5–7).

Hypertension is defined by the 1999 World Health Organization International Society of Hypertension Guidelines for the Management of Hypertension (8) as "systolic blood pressure of 140 mm Hg or greater and/or a diastolic blood pressure of 90 mm Hg or greater in subjects who are not taking antihypertensive medication" (8). The sixth report of The Joint National Committee on Prevention, Detection, Evaluation, and Treatment of high blood pressure (5) defined hypertension as "systolic blood pressure 140 mm Hg or greater, diastolic blood pressure 90 mm Hg or greater, or taking antihypertensive medication" (5).

Accelerated hypertension is defined as sudden worsening of previously controlled hypertension.

Malignant hypertension is defined as sudden onset of severe hypertension with the coexistence of end-organ damage, which may include left ventricular hypertrophy, congestive heart failure, visual or neurologic disturbance, and/or grade III/IV retinopathy.

Renovascular hypertension, also known as renal vascular hypertension, is defined as hypertension secondary to RAS.

Cure of renovascular hypertension is defined as restoration of blood pressure to below 140/90 mm Hg while taking no antihypertensive medications.

Resistant hypertension: hypertension should be considered resistant if the systolic blood pressure cannot be reduced to less than 140/90 mm Hg in patients who are adhering to an adequate and appropriate triple drug regimen that includes a diuretic agent, with all three drugs prescribed in near-maximal doses. For patients older than age 60 years with isolated systolic hypertension, resistance is defined as failure of an adequate triple drug regimen to reduce the systolic blood pressure to less than 160 mm Hg (5).

RAS is defined as narrowing of the renal artery lumen by 50% or greater, expressed in this document as a percentage of the diameter of a normal renal vessel, i.e., percent RAS is $100 \times (1 - [\text{narrowed lumen diameter} / \text{normal vessel diameter}])$.

Ostial RAS is defined as narrowing of the renal artery at its origin from the aorta, generally considered to be within its proximal 5 mm but may be extended to within 10 mm if confirmed by cross-sectional imaging (9).

Truncal RAS is defined as nonostial RAS occurring proximal to renal artery branching.

Renal revascularization is defined as any procedure that restores unobstructed arterial blood flow to the kidney.

Technically successful renal revascularization is defined by less than 30% residual stenosis measured at the narrowest point of the vascular lumen, and restoration of the pressure gradient to less than the selected threshold for intervention. In the presence of an angiographically visible dissection at the treatment site, the residual lumen is measured from the widest opacified lumen regardless of luminal dissections, knowing that the true lumen is difficult to measure accurately in this situation (10).

Unstable angina is defined as new-onset angina, angina at rest, or "crescendo" angina (7).

INDICATIONS/ CONTRAINDICATIONS

Diagnosis of Renovascular Hypertension

Clinical features suggestive of renovascular hypertension were enumerated by the Cooperative Study of Renovascular Hypertension in 1972 and have been expanded upon since that time (11–14). The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (5) states that "testing for identifiable causes [for hypertension] is not indicated generally unless [blood pressure] control is not achieved" (5). The report further states that "reversible causes of renal failure always should be sought and treated" (5).

Given this background, current indications for screening for RAS include:

a. Onset of hypertension before the age of 30 years, especially without a family

history, or recent onset of significant hypertension after the age of 55 years;
b. An abdominal bruit, particularly if it continues into diastole and is lateralized;
c. Accelerated or resistant hypertension;
d. Recurrent (ie, flash) pulmonary edema;
e. Renal failure of uncertain cause, especially with a normal urinary sediment and less than 1 g of protein per daily urinary output;
f. Coexisting, diffuse atherosclerotic vascular disease, especially in heavy
g. Acute renal failure precipitated by antihypertensive therapy, particularly angiotensin-converting enzyme inhibitors or angiotensin II receptor blockers (5).

American Heart Association guidelines additionally suggest that screening is appropriate for:

a. Malignant hypertension, defined as hypertension with end organ damage including left ventricular hypertrophy , congestive heart failure, visual or neurologic disturbance, or advanced retinopathy;
b. Hypertension with a unilateral small kidney;
c. Hypertension associated with medication intolerance; and
d. Unstable angina in the setting of suspected RAS (15).

In the proper clinical setting, these signs may prompt evaluation for arterial stenosis as the cause of hypertension or reduced renal function. Additional evaluations to determine anatomic and functional parameters predictive of success following renal revascularization include the status of the arterioles distal to the RAS, bilaterality of reconstructable disease, the amount of renal mass available for revascularization, function of the involved kidney as demonstrated by nuclear scintigraphy or other means, plasma renin levels (which have low sensitivity and high specificity for response to renal revascularization), the severity of the RAS, the presence of intrinsic renal disease on the affected side (measured by duplex determinations of resistive index or, more rarely, direct renal biopsy) (16–19), and possibly the measurement of pathophysiologically linked serum biomarkers, including brain natriuretic peptide (20).

Any antihypertensive treatment regimen that effectively lowers blood pressure is associated with slowed progression of renal failure and improved cardiovascular survival (21). In addition to its role as a potent vasoconstrictor, angiotensin II stimulates cellular hypertrophy and proliferation. Recent investigations indicate that high levels of angiotensin II are likely to contribute to vascular and ventricular hypertrophy, accelerate atherosclerosis, and cause progressive glomerular sclerosis independent of their hemodynamic effect (22). Whenever possible, an angiotensin-converting enzyme inhibitor should be part of the treatment of hypertension, as these drugs have been shown to be organ-protective beyond their antihypertensive effect in certain renal disease categories. Their use should not be limited by a correctable RAS (23–25). Renal artery imaging should be performed to exclude stenosis as the etiology of unexplained renal failure. Renal revascularization to permit the use of an angiotensin-converting enzyme inhibitor in the treatment of hypertension is justified.

Indications for Catheter-directed Diagnostic Angiography: Threshold of 95%

Appropriate indications for screening (as described previously) must be present, in addition to at least one of the following:

1. Noninvasive vascular imaging suggests that a significant RAS is present (greater than 50%);
2. Noninvasive imaging is not likely to have adequate sensitivity and specificity;
3. Onset of hypertension occurs in a patient under the age of 30 years; or older than age 30 years but with fibromuscular dysplasia (FMD) suspected as the etiology of RAS; or
4. There are appropriate indications for screening and a very high clinical suspicion of RAS, in which case noninvasive screening can be bypassed.

Treatment of RAS

Although a stenosis is the result of an abnormal process in the arterial wall, it is not usually of hemodynamic significance until the luminal cross-sectional area is reduced by 75% or the vessel diameter is narrowed by more than 50%. These numbers vary depending on characteristics of the stenosis such as its length, irregularity, and multiplicity; the resistance of the distal vascular bed; and the available collateral blood supply (26). Although mild stenoses are of no hemodynamic significance, most angiographers would agree that a stenosis that narrows the luminal diameter by 75% almost certainly is significant (27,28). The physiologic significance of lesser degrees of stenosis may depend on the resistance of the peripheral renal vasculature or the condition of the renal autoregulatory system (29–31).

Another method of determining the physiologic significance of a stenosis is to measure a pressure gradient across the lesion. However, there is no consensus whether an absolute systolic, peak systolic, or mean pressure should be used; whether the pressure should be measured during a resting or hyperemic state; or at what level the criterion for hemodynamic significance should be set. Whereas some authors have defined a significant pressure gradient as 10% of the systolic pressure, others have used a 10, 15, or 20 mm Hg systolic pressure gradient. Difficulty in measuring the pressure without affecting it, and the physiologic variations that occur during its measurement, make pressure gradient thresholds questionable.

There is emerging science regarding the best method for determining the hemodynamic relevance of RAS, including the routine use of low-profile pressure-sensing wires instead of catheters positioned across the stenosis and determinations of renal fractional flow reserve following the intraarterial administration of vasodilator medications (32). The accuracy of hemodynamic measurements can be increased by simultaneously measuring the aortic pressure via a guiding catheter in the aorta and the pressure distal to the RAS by a pressure wire (32–34). These techniques and devices are not available in every vascular laboratory and are not universally accepted. Therefore, it is the responsibility of each interventionalist to establish an objective test for hemodynamic significance for use in his or her laboratory to evaluate stenoses that appear to be of borderline significance by criteria presently applied to linear measurement. Other tests that can lend support to the clinical significance of an RAS of borderline hemodynamic significance include selective renal vein renin analysis, transcutaneous Doppler ultrasonography, and nuclear renography (28,35–38).

A hemodynamically significant RAS may stimulate the renin–angiotensin system, resulting in systemic hypertension;

however, other factors determine its clinical significance. These include the level of blood pressure control that can be attained medically, the patient's ability to tolerate and comply with the prescribed medical regimen, impairment in renal function, evidence of progressive nephron loss, comorbid medical conditions, and quality of life factors. Therefore, in most cases, the clinical significance of an RAS and the likelihood that the clinical syndrome can be improved should guide the decision to revascularize a kidney rather than its morphologic or hemodynamic characteristics.

The majority of patients with hemodynamically significant RAS associated with hypertension and reduced renal function can be managed medically without a risk of increased mortality or progression to end-stage renal disease. Renal mass and function must be followed very closely if medical treatment is the chosen option. This is especially true for those patients with bilateral RAS or stenosis of a solitary kidney, who have twice the risk of mortality and 1.5 times the risk of significant deterioration of renal function than patients with unilateral RAS and two kidneys (23). Patients should also be followed for changing or emerging clinical indicators that may prompt a reevaluation of the need for renal revascularization (eg, precipitant heart failure or loss of renal function).

In summary, the benefit of prophylactic treatment of very high grade stenoses to preserve renal mass is unproven. The decision to treat must be based on consideration of the patient's age, anticipated longevity, renal function, and ability to withstand a procedural complication, along with the condition of the contralateral kidney and the ease of performance of the procedure. Revascularization should be based on clinical symptoms and limited to hemodynamically significant stenoses.

Indications for Angioplasty or Stent Placement: Threshold of 95%

Hemodynamically significant RAS is defined as the following:

1. Greater than 50% diameter stenosis or greater than 75% reduction in cross sectional area; and
2. A systolic pressure gradient greater than 10% of systolic pressure or 10, 15, or 20 mm Hg.

Relative Contraindications for Renal Artery Stent Deployment: Threshold of 5%

1. A renal bifurcation lesion in which more than 50% of a kidney will be excluded by a stent;
2. The presence of sepsis; and
3. Renal artery diameter measuring 4 mm or less, unless a drug-eluting stent is used (39).

QUALITY IMPROVEMENT

Although practicing physicians should strive to achieve perfect outcomes (eg, 100% success, 0% complications), in practice all physicians will fall short of this ideal to a variable extent. Thus, indicator thresholds may be used to assess the effectiveness of ongoing quality improvement programs. For the purposes of these guidelines, a threshold is a specific level of an indicator that should prompt a review. "Procedure thresholds" or "overall thresholds" reference a group of indicators for a procedure (eg, major complications). Individual complications may also be associated with complication-specific thresholds. When measures such as indications or success rates fall below a minimum threshold or when complication rates exceed a maximum threshold, a review should be performed to determine causes and to implement changes, if necessary. For example, if the incidence of symptomatic cholesterol embolization of the kidney is one measure of the quality of renal angioplasty or stent implantation of the renal artery, then values in excess of the defined threshold should trigger a review of policies and procedures within the department to determine the causes and to implement changes to lower the incidence for the complication. Thresholds may vary from those listed here; for example, patient referral patterns and selection factors may dictate a different threshold value for a particular indicator at a particular institution. Thus, setting universal thresholds is very difficult and each department is urged to alter the thresholds as needed to higher or lower values, to meet its own quality improvement program needs.

Participation by the radiologist in patient follow-up is an integral part of renal angioplasty and stent implantation of the renal artery and will increase the success rate of the procedure. Close follow-up with monitoring and management of patients undergoing renal artery interventions is appropriate for the radiologist.

SUCCESS RATES AND THRESHOLDS

Benefits of Renal Revascularization

RAS may be asymptomatic or may produce renovascular hypertension, ischemic nephropathy, and cardiac disturbance syndromes (ie, recurrent flash pulmonary edema not felt to be secondary to impaired left ventricular systolic function or unstable angina in the setting of significant RAS. In addition, RAS produces pathophysiologic alterations that may be associated with a concordant increased risk of cardiovascular events, including myocardial infarction and stroke. Thus the benefits of revascularization need to be individually determined based on the underlying clinical condition prompting intervention.

Clinical Success Following Renal Revascularization

Cure of renovascular hypertension in the patient with atherosclerotic RAS.—Although a distinguishing advantage for revascularization compared with medical therapy alone is the potential for a hypertension cure, only a small percentage of patients with atherosclerotic RAS are reported as cured following revascularization (40). The clinical profile of the patient most likely to be cured has not been defined; an effort should be made to define this clinical profile during future investigations (41–50).

Cure of hypertension in the patient with fibromuscular RAS.—The mean cure rate for renal revascularization for stenoses secondary to FMD was 44% in a metaanalysis by Martin et al (51). No attempt was made to separate the results of treatment of the various types of FMD in this document. It seems reasonable to assume that the majority of those treated had the "medial fibroplastic" type of FMD, which is the most common variety. This type affects 60% –70% of patients with FMD, and most likely a higher percentage of the adult population (52).

Contrary to what one might predict, the technical and clinical results of angioplasty in those patients with FMD involving the renal artery branches were as good as those involving only the main renal artery (53,54). Using logistic regression,

Davidson et al (55) found that younger age, milder hypertension, and shorter duration of hypertension were statistically significant independent variables predicting successful results from percutaneous transluminal renal angioplasty in FMD. Schreiber et al (56) found progression of medial fibroplasia in 33% of 66 patients with FMD who were observed without intervention; however, no case progressed to occlusion and no patient developed renal failure.

Therefore, cure of hypertension is a reasonable goal in a patient with the medial fibroplastic form of renal artery FMD. It is logical to assume that the cure rate will be higher in patients with unilateral involvement (62% in the University Hospital Zurich Cooperative Study on Fibromuscular Hyperplasia [57]). Branch stenoses are not a contraindication to angioplasty. There are not enough data on endovascular revascularization of other forms of FMD to substantiate a recommendation. The rate of cure of renovascular hypertension resulting from the medial fibroplastic type of FMD is sufficiently high to recommend percutaneous transluminal renal angioplasty as a first line of treatment. Medical therapy should be reserved for older patients with FMD who have a prolonged history of hypertension. The success of treatment of other types of FMD is inconclusive, and treatment must be chosen based on personal clinical experience.

Benefits of renal revascularization other than "cure" of hypertension.—Three recent prospectively randomized controlled trials (58–60) and all of the studies included in three metaanalyses (44,46,51) have reported that renal revascularization results in a decrease of blood pressure with lower doses of medication. Blood pressure and antihypertensive medication dose reduction have been shown to be preserved to 24-month follow-up after intervention (61). Whether controlling blood pressure with less medication outweighs the risks of the revascularization procedure must be considered on an individual case basis (62–64). Whenever possible, an angiotensin-converting enzyme inhibitor or angiotensin blocker should be part of the antihypertensive treatment, as these drugs have been shown to have renoprotective properties that are as important as or more important than their antihypertensive effect and are the preferred medications in many cases of nonrenovascular hypertension (25). Therefore, although normotensive blood pressure levels can be maintained medically in cases of renovascular hypertension, it is not attained without some risk to the kidney with the stenotic renal artery, and if the clinician chooses to treat hypertension without knowing the status of renal artery patency he or she must be alert to signs of decreased renal function and loss of renal mass.

Ischemic nephropathy.—The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (5) recommends investigation of renal failure that occurs in patients being treated for hypertension. It suggests that surgical or endovascular revascularization may be necessary to preserve renal function, even though many patients with high-grade RAS remain in stable condition for prolonged periods if blood pressure is well controlled (65).

Criteria for benefit from revascularization.—There is a great deal of controversy concerning the degree of benefit that can be expected from revascularization of the patient with ischemic nephropathy. The main issue concerns measurement of the effect of the intervention. It is well recognized that there is progressive nephron loss with aging. This loss is manifest by a progressive decrease in the glomerular filtration rate and the size of the kidneys. The loss is accelerated by many disease states, including ischemic nephropathy, in which, in addition to the loss of nephron tissue, there can be functional loss resulting from hypoperfusion and loss of renal autoregulation secondary to RAS. The benefit of revascularization depends on recovering the functional loss, eliminating that portion of the accelerated cell death caused by ischemia, and returning the rate of decline of the glomerular filtration rate to that attributable to age and other coexisting disease processes other than ischemia. Delay in revascularization has been associated with a reduction in clinical benefit (48).

The slope of the linear relation between the reciprocal of creatinine concentration and time can be used to delineate the rate of change in renal function (66). Failure of progression along the slope of decrease in renal function may indicate a benefit from intervention even though there has been no improvement in baseline serum creatinine level. This method cannot compensate for the limitations in the use of creatinine values for assessing renal function, and users must be aware of the potential pitfalls in its use when there has been a change in muscle mass or diet (67). Measurements of glomerular filtration rate remain the recommended determinant of functional outcomes (68).

Results of treatment in patients with ischemic nephropathy.—No improvement in mean renal function was reported in three prospectively randomized studies of renal revascularization (14,58,60). These trials were criticized in a review by Sacks et al (63), who found fault with the analysis and interpretation of the data by the authors. The investigators in these trials found no statistical difference between continuous measures of the mean serum creatinine value at baseline and following treatment.

The problem with using the change in the mean creatinine level can be illustrated by the following example. Suppose an intervention was performed on 10 patients, each with a serum creatinine level of 3.0 mg/dL, and that at the time of final follow-up eight had serum creatinine levels of 2 mg/dL and the serum creatinine level had risen to 7.0 mg/dL in the other two patients. Using a 20% reduction of serum creatinine as a binary criterion for benefit, 80% would have benefited from the procedure and 20% would have failed to benefit; using the mean change in serum creatinine of the treated cohort measured as a continuous variable as the criterion for success, there would have been no benefit in this patient cohort. This oversimplification illustrates the problem with using a mean or average of a test that has a greater mathematical limit on the potential to improve than it does on the potential to fail. It also ignores benefit that can be derived by stabilizing the rate of nephron loss, as discussed previously.

Studies of renal revascularization that have analyzed the reciprocal slope of glomerular function have found statistically significant improvement in renal function in the population treated (69–71). Studies reporting binary results, using a less than 20% deterioration and/or a 20% lowering of the serum creatinine as a measure of functional stabilization or benefit, find a mean of 54% improved and 26% stabilized by surgical revascularization (16,17,72,73). Using binary criteria, two metaanalyses determined that renal artery stent implantation resulted in 30% improved and 38% stabilized (46) and 26% improved and 48% stabilized (44), although neither metaanalysis showed a significant decrease in overall serum creatinine values. A metaanalysis by the Agency for Healthcare Research and Quality (40) notes that

"improvements in kidney function were reported only among the angioplasty cohort studies and not in studies evaluating medical therapy alone" (40).

Endovascular revascularization can result in improvement of the glomerular filtration rate in selected patients with ischemic nephropathy. Signs that a patient with ischemic nephropathy is likely to benefit from revascularization include (i) normal appearance of the arterioles distal to the RAS; (ii) bilaterality of reconstructable disease; (iii) a near-normal volume of renal mass available for revascularization; (iv) a test demonstrating function of the involved kidney; (v) renal biopsy demonstrating well preserved glomeruli and tubules with minimal arteriolar sclerosis; (vi) severe, difficult-to-control hypertension; and (vii) abrupt onset of renal insufficiency (18,19,74).

Cardiac disturbance syndromes and prevention of cardiovascular events.—RAS may worsen angina or congestive heart failure in patients with coronary artery disease, left ventricular dysfunction, or cardiomyopathy as a result of complex pathophysiologic alterations, including change in the renin–angiotensin–aldosterone axis, resulting in a state of volume overload and peripheral vascular constriction (6,74–76). Renal revascularization may result in relief of these cardiac syndromes (7,75), particularly for patients with bilateral RAS.

More than 70% of patients may remain free of congestive heart failure and unstable angina at 12-month mean follow-up (6,7). Restoring unobstructed renal blood flow has the additional potential benefit of allowing safe usage of angiotensin-converting enzyme inhibitors without the risk of worsening renal failure and reducing coronary perfusion. The prevention of cardiovascular events and associated mortality is a possible salutary effect of renal revascularization that is currently undergoing investigation in large-scale trials (77,78).

Technical Success Following Renal Revascularization

Although stents were initially used to treat only hemodynamically significant residual stenosis or flow-limiting dissection following balloon angioplasty, they have become the standard of care for ostial RAS. A metaanalysis by Rees (79) reports 99% technical success following stent placement in 1,128 arteries, compared with 55% for ostial and 70% for nonostial stenoses treated by balloon angioplasty in 1,417 arteries. There was 77% patency at a mean 7.9 months angiographic follow-up in 563 arteries that were stent-implanted. Leertouwer et al (46) reported a 26% restenosis rate in 236 arteries examined angiographically at a mean follow-up of 19 months. This is not significantly better than the 30% restenosis rate following balloon angioplasty in 515 patients reported by Rees (79), who pointed out that "the benefits of stents for long-term patency relative to [percutaneous transluminal angioplasty] are mostly related to the markedly superior initial success rates rather than reduction of restenosis" (79).

Stents dilated to less than 6 mm, female sex, age greater than 65 years, and smoking are statistically significant risk factors for restenosis. In the US Multicenter Renal Artery Stent Trial (79) the lowest risk group was men with renal arteries 6 mm or greater, who had a restenosis rate of 10.5%. There are very few data regarding stent use in nonostial RAS; however, there are studies suggesting that these lesions may respond favorably to balloon angioplasty alone (80). Increased technical success and patency would be expected if the reference vessel diameter is 6 mm or greater.

The use of stents in ostial and nonostial locations is relatively contraindicated if they traverse renal artery branches or if restenosis would be likely to make surgical revascularization difficult or impossible. Renal artery stents have no established role in the primary treatment of FMD. They are the preferred treatment for ostial stenosis in arteries whose reference diameter is 6 mm or greater. Their use in vessels smaller than 5 mm should be limited to technically failed balloon angioplasty. Their primary use in lesions in which the normal diameter is 5 mm is left to the discretion of the interventionalist.

Technical Success of Percutaneous Renal Revascularization: Threshold of 90%

1. Defined by minimal thresholds of less than 30% residual stenosis or less than 10 mm Hg systolic pressure gradient.
2. Early bifurcation lesions are excluded from this analysis.

COMPLICATION RATES AND THRESHOLDS

The rates of complications and technical success of endovascular revascularization have shown some variability over time. Studies have shown improved success rates with diminished complications as experience with the procedure increases and as new technologies are introduced. Overall complication rates reported in the literature have ranged from 12% to 36%, with an estimated mean of approximately 14%.

Groin hematoma and puncture site trauma are the most common complications reported, with a rate of approximately 3%–5%. Major complications (and their incidence rates) include worsening of renal function (4%), occlusion of the renal artery (2%–3%), segmental infarction (1%–2%), requirement for surgical intervention for either nephrectomy or salvage (2%), and death (1%).

Two large series (81,82) and two metaanalyses (46,54) were reviewed for the present guideline. There was no overlap of data among these studies, which include 2,994 revascularizations (980 vessels treated with stents) in 2,474 patients. The total complication rate ranged from 12% (46) to 36% (82), with a mean complication rate of 14% excluding radiologic/technical complications (ie, "events that occur during catheterization or stent deployment that have no clinical consequences but lead to an increase in procedural time and/or cost") (82). Groin hematoma and puncture site trauma were the most common complications reported. The 30-day mortality rate was 1%, usually related to renal artery perforation, cholesterol embolization, acute renal failure, and arterial access puncture above the inguinal ligament. A surgical salvage operation was necessary in 1%–2.5% (46,54). Symptomatic embolization occurred in 1%–8% of the patients (46,82). Occlusion of the main renal artery was reported in 0.8%–2.5% and occlusion of a renal artery branch causing a segmental infarction in 1.1%–1.7% (46,54).

Cholesterol embolization resulting in decreased renal function or visceral or peripheral symptoms is expected in fewer than 3% of cases (46,54,81,82). A "no-touch" technique of positioning a guide catheter in the renal ostium with a second wire extending to the suprarenal aorta may potentially reduce cholesterol embolization, but the technique is unsubstantiated (83).

Specific Major Complications from Percutaneous Renal Revascularization

Complication	Reported Rate (%)	Threshold (%)
Mortality at 30 d	1	1
Secondary nephrectomy	<1	1
Surgical salvage operation	1	2
Symptomatic embolization	3	3
Main renal artery occlusion	2	2
Branch renal artery occlusion	2	2
Access site hematoma requiring surgery or transfusion or prolonging hospital stay	5	5
Acute renal failure	2	2
Worsening of chronic renal failure requiring an increase in level of care	2	5

A trend toward reduced complications was demonstrated in an earlier investigation by Martin et al (84) that found that the total complication rate decreased from 20% in the first 100 cases to 13% in the second 100 cases. The authors attributed the change to increased experience and improvement in technology and devices (84).

Complications are stratified on the basis of outcome. Major complications result in: admission to a hospital for therapy (for outpatient procedures), an unplanned increase in the level of care, prolonged hospitalization, permanent adverse sequelae, or death. Minor complications result in no sequelae; they may require nominal therapy or a short hospital stay for observation (generally overnight; see Appendix B.

Overall Threshold for Major Complications from Percutaneous Renal Revascularization

The overall threshold for major complications from percutaneous renal revascularization is 14%. Published rates for individual types of complications are highly dependent on patient selection and are based on series comprising several hundred patients, which is a larger volume than most individual practitioners are likely to treat. Generally the complication-specific thresholds should be set higher than the complication-specific reported rates listed earlier. It is also recognized that a single complication can cause a rate to cross above a complication-specific threshold when the complication occurs within a small patient series (eg, early in a quality improvement program). In this situation, an overall procedural threshold is more appropriate for use in a quality improvement program. In the (**Table**, all values are supported by the weight of literature evidence and panel consensus.

Acknowledgments: Louis G. Martin, MD, and John H. Rundback, MD, represented the SIR and participated in the authoring of the first draft of this document and served as topic leaders during the subsequent revisions of the draft. Sanjoy Kundu, MD, is chair of the SIR Standards of Practice Committee and Michael Wallace, MD, is the chair of the SIR Revisions Subcommittee. John F. Cardella, MD, is Councilor of the SIR Standards Division. All other authors are listed alphabetically. Other members of the SIR Revision Subcommittee who participated in the revision of this clinical practice guideline are as follows: Daniel B. Brown, MD, Horacio R. D'Agostino, MD, Sanjeeva P. Kalva, MD, Arshad Ahmed Khan, MD, Nasir H. Siddiqi, MD, LeAnn Stokes, MD, Anne C. Roberts, MD, Marc S. Schwartzberg, MD, and Darryl A. Zuckerman, MD.

APPENDIX A: CONSENSUS METHODOLOGY

Reported complication-specific rates in some cases reflect the aggregate of major and minor complications. Thresholds are derived from critical evaluation of the literature, evaluation of empirical data from Standards of Practice Committee members' practices, and, when available, the SIR HI-IQ System national database.

Consensus on statements in this document was obtained with use of a modified Delphi technique (1,2),

There were no issues on which the Committee was unable to reach consensus.

APPENDIX B: SOCIETY OF INTERVENTIONAL RADIOLOGY STANDARDS OF PRACTICE COMMITTEE CLASSIFICATION OF COMPLICATIONS BY OUTCOME

Minor Complications

A. No therapy, no consequence.
B. Nominal therapy, no consequence; includes overnight admission for observation only.

Major Complications

C. Require therapy, minor hospitalization (<48 hours).
D. Require major therapy, unplanned increase in level of care, prolonged hospitalization (>48 hours).
E. Permanent adverse sequelae.
F. Death.

References

1. Fink A, Kosecoff J, Chassin M, Brook RH. Consensus methods: characteristics and guidelines for use. Am J Public Health 1984; 74:979–983.
2. Leape LL, Hilborne LH, Park RE, et al. The appropriateness of use of coronary artery bypass graft surgery in New York State. JAMA 1993; 269:753–760.
3. Martin LG, Rundback JH, Sacks D, et al, for the SIR Standards of Practice Committee. Quality improvement guidelines for angiography, angioplasty, and stent placement in the diagnosis and treatment of renal artery stenosis in adults. J Vasc Interv Radiol 2002; 13:1069–1083.
4. Anderson GH Jr, Blakeman N, Streeten DH. The effect of age on prevalence of secondary forms of hypertension in 4429 consecutively referred patients. J Hypertens 1994; 12:609–615.
5. The sixth report of the Joint National Committee on prevention, detection, evaluation, and treatment of high blood pressure. Arch Intern Med 1997; 157:2413–2446.
6. 1999 World Health Organization-International Society of Hypertension Guidelines for the Management of Hypertension. Guidelines Subcommittee. J Hypertens 1999; 17:151–183.
7. Kaatee R, Beek FJ, Verschuyl EJ, et al. Atherosclerotic renal artery stenosis: ostial or truncal? Radiology 1996; 199: 637–640.
8. Sacks D, Marinelli DL, Martin LG, Spies JB. Reporting standards for clinical evaluation of new peripheral arterial revascularization devices. Technology As-

sessment Committee. J Vasc Interv Radiol 1997; 8:137–149.
9. Khosla S, White CJ, Collins TJ, Jenkins JS, Shaw D, Ramee SR. Effects of renal artery stent implantation in patients with renovascular hypertension presenting with unstable angina or congestive heart failure. Am J Cardiol 1997; 80:363–366.
10. Bloch MJ, Trost DW, Pickering TG, Sos TA, August P. Prevention of recurrent pulmonary edema in patients with bilateral renovascular disease through renal artery stent placement. Am J Hypertens 1999; 12:1–7.
11. Albers FJ. Clinical characteristics of atherosclerotic renovascular disease. Am J Kidney Dis 1994; 24:636–641.
12. Krijnen P, van Jaarsveld BC, Steyerberg EW, Man in't Veld AJ, Schalekamp MA, Habbema JD. A clinical prediction rule for renal artery stenosis. Ann Intern Med 1998; 129:705–711.
13. Simon N, Franklin SS, Bleifer KH, Maxwell MH. Clinical characteristics of renovascular hypertension. JAMA 1972; 220:1209–1218.
14. van Jaarsveld B, Krijnen P, Bartelink A, et al. The Dutch Renal Artery Stenosis Intervention Cooperative (DRASTIC) study: rationale, design and inclusion data. J Hypertens 1998; 16(Suppl):S21–S27.
15. Hirsch AT, Haskal ZJ, Hertzer NR, et al. ACC/AHA 2005 guidelines for the management of patients with peripheral arterial disease (lower extremity, renal, mesenteric, and abdominal aortic): executive summary a collaborative report from the American Association for Vascular Surgery/Society for Vascular Surgery, Society for Cardiovascular Angiography and Interventions, Society for Vascular Medicine and Biology, Society of Interventional Radiology, and the ACC/AHA Task Force on Practice Guidelines (Writing Committee to Develop Guidelines for the Management of Patients With Peripheral Arterial Disease) endorsed by the American Association of Cardiovascular and Pulmonary Rehabilitation; National Heart, Lung, and Blood Institute; Society for Vascular Nursing; TransAtlantic Inter-Society Consensus; and Vascular Disease Foundation. J Am Coll Cardiol 2006; 47:1239–1312.
16. Dean RH, Englund R, Dupont WD, et al. Retrieval of renal function by revascularization. Study of preoperative outcome predictors. Ann Surg 1985; 202:367–375.
17. Hallett JW Jr, Fowl R, O'Brien PC, et al. Renovascular operations in patients with chronic renal insufficiency: do the benefits justify the risks? J Vasc Surg 1987; 5:622–627.
18. Martin LG, Casarella WJ, Gaylord GM. Azotemia caused by renal artery stenosis: treatment by percutaneous angioplasty. AJR Am J Roentgenol 1988; 150:839–844.
19. Novick AC. Atherosclerotic ischemic nephropathy: epidemiology and clinical considerations. Urol Clin North Am 1994; 21:195–200.
20. Silva JA, Chan AW, White CJ, et al. Elevated brain natriuretic peptide predicts blood pressure response after stent revascularization in patients with renal artery stenosis. Circulation 2005; 111:328–333.
21. Dahlof B, Sever PS, Poulter NR, et al. Prevention of cardiovascular events with an antihypertensive regimen of amlodipine adding perindopril as required versus atenolol adding bendroflumethiazide as required, in the Anglo-Scandinavian Cardiac Outcomes Trial-Blood Pressure Lowering Arm (ASCOT-BPLA): a multicentre randomised controlled trial. Lancet 2005; 366:895–906.
22. Conlon PJ, Athirakul K, Kovalik E, et al. Survival in renal vascular disease. J Am Soc Nephrol 1998; 9:252–256.
23. Chabova V, Schirger A, Stanson AW, McKusick MA, Textor SC. Outcomes of atherosclerotic renal artery stenosis managed without revascularization. Mayo Clin Proc 2000; 75:437–444.
24. Conlon PJ, O'Riordan E, Kalra PA. New insights into the epidemiologic and clinical manifestations of atherosclerotic renovascular disease. Am J Kidney Dis 2000; 35:573–587.
25. Moore MA, Epstein M, Agodoa L, Dworkin LD. Current strategies for management of hypertensive renal disease. Arch Intern Med 1999; 159:23–28.
26. Kohler TR. Hemodynamics of arterial occlusive disease. In: Strandness DE Jr, van Breda A, eds. Vascular diseases: surgical and interventional therapy, 1st ed. New York: Churchill Livingstone, 1994; 65–71.
27. Imanishi M, Akabane S, Takamiya M, et al. Critical degree of renal arterial stenosis that causes hypertension in dogs. Angiology 1992; 43:833–842.
28. Simon G. What is critical renal artery stenosis? Implications for treatment. Am J Hypertens 2000; 13:1189–1193.
29. Haimovici H, Zinicola N. Experimental renal-artery stenosis diagnostic significance of arterial hemodynamics. J Cardiovasc Surg (Torino) 1962; 3:259–262.
30. May AG, Van De Berg L, Deweese JA, Rob CG. Critical arterial stenosis. Surgery 1963; 54:250–259.
31. Pemsel HK, Thermann M. The haemodynamic effects of renal artery stenosis. Rofo 1978; 129:189–192.
32. Gross CM, Kramer J, Weingartner O, et al. Determination of renal arterial stenosis severity: comparison of pressure gradient and vessel diameter. Radiology 2001; 220:751–756.
33. De Bruyne B, Pijls NH, Heyndrickx GR, Hodeige D, Kirkeeide R, Gould KL. Pressure-derived fractional flow reserve to assess serial epicardial stenoses: theoretical basis and animal validation. Circulation 2000; 101:1840–1847.
34. Pijls NH, Van Gelder B, Van der Voort P, et al. Fractional flow reserve: a useful index to evaluate the influence of an epicardial coronary stenosis on myocardial blood flow. Circulation 1995; 92:3183–3193.
35. Harward TR, Poindexter B, Huber TS, Carlton LM, Flynn TC, Seeger JM. Selection of patients for renal artery repair using captopril testing. Am J Surg 1995; 170:183–187.
36. Johansson M, Jensen G, Aurell M, et al. Evaluation of duplex ultrasound and captopril renography for detection of renovascular hypertension. Kidney Int 2000; 58:774–782.
37. Radermacher J, Chavan A, Schaffer J, et al. Detection of significant renal artery stenosis with color Doppler sonography: combining extrarenal and intrarenal approaches to minimize technical failure. Clin Nephrol 2000; 53:333–343.
38. Taylor AT Jr, Fletcher JW, Nally JV Jr, et al. Procedure guideline for diagnosis of renovascular hypertension. Society of Nuclear Medicine. J Nucl Med 1998; 39:1297–1302.
39. Misra S, Thatipelli MR, Howe PW, et al. Preliminary study of the use of drug-eluting stents in atherosclerotic renal artery stenoses 4 mm in diameter or smaller. J Vasc Interv Radiol 2008; 19:833–839.
40. Balk EM, Raman G. Comparative effectiveness of management strategies for renal artery stenosis: 2007 update. Comparative Effectiveness Review No. 5 update. Rockville, MD: Agency for Healthcare Research and Quality, 2007 (Prepared by Tufts–New England Medical Center under contract 290–02–0022). Ann Intern Med 2006; 145:901–912.
41. Barri YM, Davidson RA, Senler S, et al. Prediction of cure of hypertension in atherosclerotic renal artery stenosis. South Med J 1996; 89:679–683.
42. Dorros G, Jaff M, Mathiak L, et al. Four-year follow-up of Palmaz-Schatz stent revascularization as treatment for atherosclerotic renal artery stenosis. Circulation 1998; 98:642–647.
43. Greminger P, Steiner A, Schneider E, et al. Cure and improvement of renovascular hypertension after percutaneous transluminal angioplasty of renal artery stenosis. Nephron 1989; 51:362–366.

44. Isles CG, Robertson S, Hill D. Management of renovascular disease: a review of renal artery stenting in ten studies. Q J Med 1999; 92:159–167.
45. Kuhn FP, Kutkuhn B, Torsello G, Modder U. Renal artery stenosis: preliminary results of treatment with the Strecker stent. Radiology 1991; 180: 367–372.
46. Leertouwer TC, Gussenhoven EJ, Bosch JL, et al. Stent placement for renal arterial stenosis: where do we stand? A meta-analysis. Radiology 2000; 216:78–85.
47. Martin EC, Mattern RF, Baer L, Fankuchen EI, Casarella WJ. Renal angioplasty for hypertension: predictive factors for long-term success. AJR Am J Roentgenol 1981; 137:921–924.
48. Maxwell MH, Bleifer KH, Franklin SS, Varady PD. Cooperative study of renovascular hypertension: demographic analysis of the study. JAMA 1972; 220: 1195–1204.
49. Tegtmeyer CJ, Kellum CD, Ayers C. Percutaneous transluminal angioplasty of the renal artery: results and long-term follow-up. Radiology 1984; 153: 77–84.
50. van de Ven PJ, Beutler JJ, Kaatee R, et al. Transluminal vascular stent for ostial atherosclerotic renal artery stenosis. Lancet 1995; 346:672–674.
51. Martin LG, Rees CR, O'Bryant T. Percutaneous angioplasty of the renal arteries. In: Strandness DE Jr, van Breda A, ed. Vascular diseases: surgical and interventional therapy, 1st ed. New York: Churchill Livingstone, 1994; 721–742.
52. Harrison EG Jr, McCormack LJ. Pathologic classification of renal arterial disease in renovascular hypertension. Mayo Clin Proc 1971; 46:161–167.
53. Cluzel P, Raynaud A, Beyssen B, Pagny JY, Gaux JC. Stenoses of renal branch arteries in fibromuscular dysplasia: results of percutaneous transluminal angioplasty. Radiology 1994; 193:227–232.
54. Martin LG. Renal revascularization using percutaneous balloon angioplasty for fibromuscular dysplasia and atherosclerotic disease. In: Calligaro KD, Doughtery MJ, ed. Modern management of renovascular hypertension and renal salvage. Baltimore, MD: Williams and Wilkins, 1996; 125–144.
55. Davidson RA, Barri Y, Wilcox CS. Predictors of cure of hypertension in fibromuscular renovascular disease. Am J Kidney Dis 1996; 28:334–338.
56. Schreiber MJ, Pohl MA, Novick AC. The natural history of atherosclerotic and fibrous renal artery disease. Urol Clin North Am 1984; 11:383–392.
57. Luscher TF, Keller HM, Imhof HG, et al. Fibromuscular hyperplasia: extension of the disease and therapeutic outcome. Results of the University Hospital Zurich Cooperative Study on Fibromuscular Hyperplasia. Nephron 1986; 44(Suppl 1):109–114.
58. Plouin PF, Chatellier G, Darne B, Raynaud A.Blood pressure outcome of angioplasty in atherosclerotic renal artery stenosis: a randomized trial. Essai Multicentrique Medicaments vs Angioplastie (EMMA) Study Group. Hypertension 1998; 31:823–829.
59. van Jaarsveld BC, Krijnen P, Pieterman H, et al. The effect of balloon angioplasty on hypertension in atherosclerotic renal-artery stenosis. Dutch Renal Artery Stenosis Intervention Cooperative Study Group. N Engl J Med 2000; 342:1007–1014.
60. Webster J, Marshall F, Abdalla M, et al. Randomised comparison of percutaneous angioplasty vs continued medical therapy for hypertensive patients with atheromatous renal artery stenosis. Scottish and Newcastle Renal Artery Stenosis Collaborative Group. J Hum Hypertens 1998; 12:329–335.
61. Rocha-Singh K, Jaff MR, Rosenfield K. Evaluation of the safety and effectiveness of renal artery stenting after unsuccessful balloon angioplasty: the ASPIRE-2 study. J Am Coll Cardiol 2005; 46:776–783.
62. Ritz E, Mann JF. Renal angioplasty for lowering blood pressure. N Engl J Med 2000; 342:1042–1043.
63. Sacks D, Rundback JH, Martin LG. Renal angioplasty/stent placement and hypertension in the year 2000. J Vasc Interv Radiol 2000; 11:949–953.
64. Textor SC. Revascularization in atherosclerotic renal artery disease. Kidney Int 1998; 53:799–811.
65. Chobanian AV, Bakris GL, Black HR, et al. The seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: the JNC 7 report. JAMA 2003; 289:2560–2572.
66. Mitch WE, Walser M, Buffington GA, Lemann J Jr. A simple method of estimating progression of chronic renal failure. Lancet 1976; 2:1326–1328.
67. Mitch WE. The influence of the diet on the progression of renal insufficiency. Annu Rev Med 1984; 35:249–264.
68. Rundback JH, Sacks D, Kent KC, et al. Guidelines for the reporting of renal artery revascularization in clinical trials. American Heart Association. Circulation 2002; 106:1572–1585.
69. Harden PN, MacLeod MJ, Rodger RS, et al. Effect of renal-artery stenting on progression of renovascular renal failure. Lancet 1997; 349:1133–1136.
70. van Rooden CJ, van Bockel JH, De Backer GG, Hermans J, Chang PC. Long-term outcome of surgical revascularization in ischemic nephropathy: normalization of average decline in renal function. J Vasc Surg 1999; 29:1037–1049.
71. Watson PS, Hadjipetrou P, Cox SV, Piemonte TC, Eisenhauer AC. Effect of renal artery stenting on renal function and size in patients with atherosclerotic renovascular disease. Circulation 2000; 102:1671–1677.
72. Cambria RP, Brewster DC, L'Italien GJ, et al. Renal artery reconstruction for the preservation of renal function. J Vasc Surg 1996; 24:371–380.
73. van Damme H, Jeusette F, Pans A, et al. The impact of renal revascularisation on renal dysfunction. Eur J Vasc Endovasc Surg 1995; 10:330–337.
74. Dean RH, Tribble RW, Hansen KJ, O'Neil E, Craven TE, Redding JF II. Evolution of renal insufficiency in ischemic nephropathy. Ann Surg 1991; 213: 446–455.
75. Jaff MR. Management of atherosclerotic renal artery stenosis: interventional versus medical therapy. Curr Interv Cardiol Rep 2001; 3:93–99.
76. Messina LM, Zelenock GB, Yao KA, Stanley JC. Renal revascularization for recurrent pulmonary edema in patients with poorly controlled hypertension and renal insufficiency: a distinct subgroup of patients with arteriosclerotic renal artery occlusive disease. J Vasc Surg 1992; 15:73–80.
77. Cooper CJ, Murphy TP, Matsumoto A, et al. Stent revascularization for the prevention of cardiovascular and renal events among patients with renal artery stenosis and systolic hypertension: rationale and design of the CORAL trial. Am Heart J 2006; 152:59–66.
78. Mistry S, Ives N, Harding J, et al. Angioplasty and stent for renal artery lesions (ASTRAL trial): rationale, methods and results so far. J Hum Hypertens 2007; 21:511–515.
79. Rees CR. Stents for atherosclerotic renovascular disease. J Vasc Interv Radiol 1999; 10:689–705.
80. Baumgartner I, von Aesch K, Do DD, Triller J, Birrer M, Mahler F. Stent placement in ostial and nonostial atherosclerotic renal arterial stenoses: a prospective follow-up study. Radiology 2000; 216:498–505.
81. Bakker J, Goffette PP, Henry M, et al. The Erasme study: a multicenter study on the safety and technical results of the Palmaz stent used for the treatment of atherosclerotic ostial renal artery stenosis. Cardiovasc Intervent Radiol 1999; 22:468–474.
82. Beek FJ, Kaatee R, Beutler JJ, van der Ven PJ, Mali WP. Complications during renal artery stent placement for atherosclerotic ostial stenosis. Cardiovasc Intervent Radiol 1997; 20:184–190.
83. Feldman RL, Wargovich TJ, Bittl JA. No-touch technique for reducing aortic

wall trauma during renal artery stenting. Cathet Cardiovasc Interv 1999; 46:245–248.

84. Martin LG, Casarella WJ, Alspaugh JP, Chuang VP. Renal artery angioplasty: increased technical success and decreased complications in the second 100 patients. Radiology 1986; 159:631–634.

SIR DISCLAIMER

The clinical practice guidelines of the Society of Interventional Radiology attempt to define practice principles that generally should assist in producing high quality medical care. These guidelines are voluntary and are not rules. A physician may deviate from these guidelines, as necessitated by the individual patient and available resources. These practice guidelines should not be deemed inclusive of all proper methods of care or exclusive of other methods of care that are reasonably directed towards the same result. Other sources of information may be used in conjunction with these principles to produce a process leading to high quality medical care. The ultimate judgment regarding the conduct of any specific procedure or course of management must be made by the physician, who should consider all circumstances relevant to the individual clinical situation. Adherence to the SIR Quality Improvement Program will not assure a successful outcome in every situation. It is prudent to document the rationale for any deviation from the suggested practice guidelines in the department policies and procedure manual or in the patient's medical record.

CME TEST QUESTIONS

Examination available at http://directory.sirweb.org/jvircme.

The CME questions in this issue are derived from Martin et al, "Quality Improvement Guidelines for Angiography, Angioplasty, and Stent Placement for the Diagnosis and Treatment of Renal Artery Stenosis in Adults"

1. According to these guidelines, which of the following is an indication for screening for renal artery stenosis?
   a. Rapid onset of proteinuria
   b. Recurrent flash pulmonary edema
   c. Acute renal failure related to nephrotoxic antibiotic therapy
   d. Onset of hypertension before the age of 60

2. Which of the following would be classified as malignant hypertension?
   a. Hypertension despite triple drug antihypertensive therapy
   b. Sudden onset of severe hypertension associated with Grade I retinopathy
   c. Hypertension associated with a unilateral kidney
   d. Sudden onset of severe hypertension associated with congestive heart failure

3. Which of the following conditions is considered a relative contraindication for renal artery stent deployment?
   a. Any lesion involving a renal artery bifurcation
   b. A renal artery stenosis involving the main renal artery ostium
   c. A hemodynamically significant renal artery stenosis related to fibromuscular dysplasia
   d. Renal artery diameter measuring 4 mm or less, unless a drug-eluting stent is used

4. Signs that a patient with ischemic nephropathy is more likely to benefit from revascularization include which of the following?
   a. Shrunken kidneys associated with renal artery stenosis
   b. Poor visualization of renal arterioles distal to the renal artery stenosis
   c. Renal stenosis in the setting of a renal biopsy showing well-preserved glomeruli and tubules with minimal arteriolar sclerosis
   d. Unilateral reconstructable disease