

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

Louis G. Martin, MD, John H. Rundback, MD, David Sacks, MD, John F. Cardella, MD, Chet R. Rees, MD, Alan H. Matsumoto, MD, Steven G. Meranze, MD, Marc S. Schwartzberg, MD, Mark I. Silverstein, MD, and Curtis A. Lewis, MD, MBA, for the Society of Interventional Radiology Standards of Practice Committee

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Abbreviation: FMD = fibromuscular dysplasia

## PREAMBLE

THE membership of the Society of Interventional Radiology (SIR) Standards of Practice Committee represents experts in a broad spectrum of interventional procedures from both 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, 10201 Lee Highway Suite 500, Fairfax, VA 22030.

## METHODOLOGY

SIR produces its Standards of Practice documents by 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 depending on the magnitude of the project.

An in-depth literature search is performed with use of electronic medical literature databases. Then, a critical review of peer-reviewed articles is performed with regards 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 by a Modified Delphi Consensus Method (Appendix 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 Standards of Practice Committee members in either a telephone conference call 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 Standards of Practice Committee, and appropriate revisions made to create the finished Standards Document. Before publication, the document is endorsed by the SIR Executive Council.

The National High Blood Pressure Education Program, coordinated by the National Heart, Lung, and Blood Institute of the National Institutes of Health, was established in 1972. Its mission is to increase the awareness, prevention, treatment, and control of hypertension. It is succeeding; from 1976 to 1991, the percentage of Americans with hypertension who are aware that they have high blood pressure increased from 51% to 73%. During the same period, the percentage treated for hypertension increased from 32% to 55% and the percentage of those patients whose blood pressure is controlled at less than

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A complete list of the members of the SIR Standards of Practice Committee is given at the end of this article. From the Department of Radiology (L.G.M., M.I.S.), Emory University Hospital; Manuel Maloof Imaging Center (C.A.L.), Atlanta, Georgia; SUNY Syracuse Health Sciences Center (J.F.C.), Syracuse; Department of Vascular and Interventional Radiology (J.H.R.), Columbia Presbyterian Medical Center, New York, New York; The Reading Hospital and Medical Center (D.S.), Reading, Pennsylvania; Department of Radiology (C.R.R.), Baylor University Medical Center, Dallas, Texas; Department of Radiology (A.H.M.), University of Virginia Health System, Charlottesville, Virginia; Vanderbilt University Hospital (S.G.M.), Nashville, Tennessee; Radiology Associates of Central Florida (M.A.S.), Leesburg, Florida. Address correspondence to SIR, 10201 Lee Highway, Suite 500, Fairfax, VA 22030.

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140/90 mm Hg increased from 10% to 29% (1,2). These changes have contributed to reductions in morbidity and mortality attributable to hypertension. Age-adjusted death rates from stroke have declined by nearly 60% and from coronary artery disease by 53%. Paradoxically, during this same time period, there has been an increase in the incidences of end-stage renal disease and congestive heart failure. In both, high blood pressure is antecedent in the majority of patients. Thus, the advances in medical antihypertensive therapy that have extended the life expectancy of the patient with hypertension have also changed the clinical profile of the patient with renovascular disease who presents for consideration of renal revascularization. A patient younger than 50 years of age with a history of recent discovery of hypertension, who would previously have presented for angiographic evaluation and treatment, is now adequately controlled medically and most often is not evaluated for a renovascular cause. Today, it is more common to be asked to treat a 70-year-old patient who has a 20-year history of treatment for hypertension, now poorly controlled, deteriorating renal function, and the diagnosis of renal artery stenosis suggested by noninvasive testing. How should we proceed with more definitive evaluation and treatment of this patient? What tests and treatments are appropriate? What is the relative risk of further evaluation and treatment compared with the benefit that is likely to be attained? This document will attempt to establish guidelines to answer these difficult questions. This document is directed toward endovascular renal revascularization; however, many of the issues pertain equally to surgical revascularization.

These guidelines are written for use in quality improvement programs to assess the diagnosis and treatment of renal artery stenosis. The most important processes of care are (a) patient selection, (b) performing the procedure, and (c) 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

**Hypertension:** Hypertension is defined by the 1999 World Health Or-

ganization-International Society of Hypertension Guidelines for the Management of Hypertension (3) 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." The sixth report of The Joint National Committee on prevention, detection, evaluation, and treatment of high blood pressure (2) defined hypertension as "systolic blood pressure 140 mm Hg or greater, diastolic blood pressure 90 mm Hg or greater, or taking antihypertensive medication."

**Renovascular Hypertension:** Hypertension secondary to renal artery stenosis, also known as renal vascular hypertension. It may be dependent on the renin-angiotensin system or a slow pressor response occurring as a result of globally reduced renal plasma flow and partially mediated by increased intravascular volume.

**Cure of Renovascular Hypertension:** Restoration of blood pressure to less than 140/90 mm Hg while the patient is taking no antihypertensive medications.

**Resistant Hypertension:** Hypertension should be considered resistant if the 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, with all three drugs prescribed in near maximal doses. In patients older than 60 years with isolated systolic hypertension, resistance is defined as failure of an adequate triple-drug regimen to reduce the SBP to less than 160 mm Hg (2).

**Stage 3 (Grade III) Hypertension:** Blood pressure equal to or above 180 mm Hg systolic and 110 mm Hg diastolic (2,3).

**Accelerated Hypertension:** Sudden worsening of previously controlled hypertension.

**Malignant Hypertension:** Sudden onset of severe hypertension with the coexistence of end-organ damage, which may include left ventricular hypertrophy, congestive heart failure, visual or neurological disturbance, and/or grade III-IV retinopathy.

**Renal Artery Stenosis:** 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 (ie, % renal artery stenosis =  $100 \times 1 - [\text{diameter of the narrowed lumen}/\text{the normal vessel diameter}]$ ). In the presence of an angiographically visible dissection at the treatment site, the residual lumen is measured from the widest opacified lumen, including intimal crack depth, knowing that the true lumen is difficult to measure accurately in this situation (4).

**Renal Revascularization:** Any procedure necessary to restore unobstructed arterial blood flow to the kidney.

**Ostial Renal Artery Stenosis:** 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 computed tomographic (CT) angiography (5).

**Truncal Renal Artery Stenosis:** Non-ostial renal artery stenosis occurring proximal to renal artery branching.

**Technically Successful Renal Revascularization:** 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 intimal dissections, knowing that the true lumen is difficult to measure accurately in this situation (4).

**Unstable Angina:** New-onset angina, angina at rest, or crescendo angina (6).

**Cardiac Disturbance Syndrome:** Recurrent "flash" pulmonary edema not believed to be secondary to impaired left ventricular systolic function or unstable angina frequently seen in the setting of bilateral renal artery stenoses or stenosis of the renal artery in a solitary kidney (2,6,7).

Although practicing physicians should strive to achieve perfect outcomes (eg, 100% success, 0% complications), all will fall short of this ideal to a variable extent. Therefore, indicator thresholds may be used to assess the efficacy of ongoing quality improvement programs. For the purposes of these guidelines, a threshold is a specific level of an indicator, which 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 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 placement treatment of renal artery stenosis, then values in excess of the defined threshold of 6% should trigger a review of policies and procedures within the department to determine the causes and implement changes to lower the incidence of 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. 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.

Complications can be stratified on the basis of outcome (Appendix 1). 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. The complication rates and thresholds that follow refer to major complications.

## INDICATIONS

### When Is It Appropriate to Treat a Renal Artery Stenosis?

Normal arteries do not have stenosis. Atherosclerotic stenoses are likely to be progressive (8–10). It is much easier to treat a vessel with a lesser degree of stenosis than one that has progressed to occlusion or near occlusion. Therefore, it would seem logical to seek and treat all renal artery steno-

ses that are incidentally discovered, including those without clinical significance. This would be reasonable if the stenosis could be completely eliminated in all cases, there was no associated morbidity, the technical result lasted indefinitely, and there was no financial or physical cost to the patient or society. Because none of these conditions can be met, the patient and the physicians providing care and counsel must weigh the risks and anticipated benefits of the revascularization before deciding on the course of treatment. When should a stenosis be treated?

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 narrowed by more than 50%. These numbers vary depending on characteristics of the stenosis, such as its length, irregularity, multiplicity, the resistance of the distal vascular bed, and the available collateral blood supply (11). Although mild stenoses are of no hemodynamic significance, most angiographers would agree that a stenosis that narrows the luminal diameter by 75% is almost certain to be of hemodynamic significance (12,13). 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 (14–16). What of those stenoses between 50% and 75%? Because of technical problems associated with the measurement of the renal artery, such as marginal blurring, difficulty selecting a reliable reference point, and the inability to obtain orthogonal views in the plane of the renal artery axis, it may be impossible to accurately measure the degree of stenosis within this range (17). In such cases, intraarterial pressure measurements can be used to supplement the data present on the radiographic image. There should be no abrupt drop in intravascular pressure across an arterial segment. A peak systolic pressure gradient of more than 10% or a mean pressure gradient of more than 5% ( $100 \times$  [blood pressure proximal to the stenosis – blood pressure distal to stenosis/blood pressure proximal to stenosis]) across the narrowed vascular lumen is generally accepted as an indication that a hemodynamically significant stenosis is present (18,19). This recommendation is clearly arbi-

trary. There is no consensus as to whether 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; difficulty in measuring the pressure without affecting it and physiologic variations that occur during measurement cause pressure gradient thresholds to be questionable. The Dutch iliac stent trial used a mean pressure gradient of 10 mm Hg, obtained either at rest or after vasodilation, to indicate the need for intervention or a technical failure of iliac angioplasty that would require stent placement (20). Gross et al (21) compared the use of mean and peak systolic gradients before and after induction of steady-state hyperemia by an infusion of increasing doses of nitroglycerine in the renal artery with stenosis and calculated the fractional flow reserve as the ratio of the mean distal intrarenal pressure measured with the wire to the mean arterial pressure measured with the guiding catheter. They concluded, "although vasodilatation accentuated the results in individual patients, such as those with stenoses of borderline hemodynamic significance, the routine use of a vasodilator did not improve their findings" (21). There is increasing evidence to suggest that accuracy can be increased by simultaneously measuring the aortic pressure via a guiding catheter in the aorta and the pressure distal to the renal artery stenosis by a pressure wire both with and without maximal pharmacological vasodilatation of the peripheral vascular bed (21–23). 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 the laboratory to evaluate stenoses that appear to be of borderline significance by criteria presently applied to linear measurements. Other tests that can lend support to the clinical significance of a renal artery stenosis of borderline hemodynamic significance include selective renal vein renin analysis, transcutaneous Doppler ultrasonography, and nuclear renography (13,24–28). There is recent evidence that patients likely to benefit from renal revascularization can be identified by a resistance index of less than 80 on a

meticulously performed Doppler ultrasound examination. Resistance index was calculated as  $(1 - [\text{end-diastolic velocity (cm/s)/maximal systolic velocity (cm/s)}]) \times 100$ . The index is dimensionless because both velocities are measured in centimeters per second (29). These "other tests" must be used cautiously; negative results should not prevent revascularization if clinical signs of renovascular hypertension are strong (30,31).

A hemodynamically significant renal artery stenosis 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, and quality of life factors. Therefore, in most cases, the clinical significance of a renal artery stenosis 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.

What of the stenosis that is considered "preocclusive" (ie, 70%–90%)? Multiple studies suggest that the term "preocclusive stenosis" is a misnomer; however, there is a significant risk that stenoses will progress during short observational periods ranging from 6 to 36 months (8,10,32–35). Although the viability of a kidney after renal artery occlusion may be preserved if collateral circulation is adequate, its function is likely to be severely impaired and revascularization procedures more difficult and hazardous to perform. The benefit of prophylactic treatment of preocclusive stenoses is unclear. Although progression to occlusion is less than 10%, the rate of renal atrophy is more than 25% and has been directly associated with the degree of stenosis (36,37). In patients who had 60% or greater renal artery stenosis by duplex ultrasound, 16% of patients followed by Caps et al (36) lost more than 1 cm of kidney length in 1 year and 27% of patients followed by Guzman et al (37) lost an average of 19 mm of renal length in a mean follow-up of 14 months. Watson et al (38) showed that this loss can be stabilized by renal revascularization. At 19 months mean follow-up, reduction in kidney length in excess of 1 cm occurred

in only 7.8% of 33 patients who were treated (38). Is there a role for prophylactic treatment of hemodynamically significant renal artery stenoses in patients with normal renal function who are either normotensive or have easily controlled hypertension? An electronic literature search of the National Library of Medicine can find reference to only one such study. Chaikof et al (39) revascularized 43 kidneys in 32 patients (renal artery stenosis, >70%; mean serum creatinine,  $1.29 \text{ mg/dL} \pm 0.24$ ; normotensive, 22%; hypertension controlled with a single medication, 78%) who were treated for infrarenal abdominal aortic aneurysm (62%) or aortic occlusive disease (38%). Blood pressure remained normal with a single agent or without medication in 75% of patients and there was no statistically significant deterioration in renal function in the 96% of patients who were available for late follow-up (median, 64 mo) (39). Chaikof et al (39) justified their actions by consideration of 30%–40% restenosis rates after percutaneous renal revascularization and the increased risks of repeat operations, should future surgical revascularization become necessary. The authors of this document realize that there may be unusual circumstances that may justify "prophylactic renal percutaneous revascularization" in the absence of clinical indications, documented progression of arterial stenosis, or loss of renal mass; however, the interventionalist must understand that he or she is proceeding without scientific evidence of benefit and is accountable for the adverse results that may occur.

The majority of patients with hemodynamically significant renal artery stenosis associated with hypertension and reduced renal function can be managed medically without the 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 renal artery stenosis 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 renal artery stenosis and two kidneys (40). 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 upon

consideration of the patient's age, anticipated longevity, renal function, ability to withstand a procedural complication, condition of the contralateral kidney, and ease of performance of the procedure. Revascularization should be based on clinical symptoms and limited to hemodynamically significant stenoses.

### When Should Catheter-directed Renal Artery Angiography Be Performed?

Approximately 3%–5% of the population with hypertension has a renovascular etiology. Increasing age and coexisting atherosclerosis have significant effects on the prevalence of renovascular hypertension. Anderson et al (41) reported a 10.2% prevalence of secondary forms of hypertension, including renovascular hypertension (3.1%) in 4,429 patients who were referred by their physicians for a 1-day blood pressure study to investigate secondary causes of hypertension during an 18-year period. The concomitant presence of atherosclerosis significantly increased the prevalence of renovascular hypertension (9.5%) and renal insufficiency (8%) (41). The incidence of renovascular hypertension varied from 0% to 29% (weighted mean, 4%) among 8,899 patients in 12 studies (including their own) reviewed by Anderson et al (41). Clinical features suggestive of renovascular hypertension were enumerated by the Cooperative Study of Renovascular Hypertension in 1972 and have been expanded since that time (33,42–44). As listed by the Sixth Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure, they include: (a) onset of hypertension before 30 years of age, especially without a family history, or recent onset of significant hypertension after 55 years of age; (b) an abdominal bruit, particularly if it continues into diastole and is lateralized; (c) accelerated or resistant hypertension; (d) recurrent (flash) pulmonary edema; (e) renal failure of uncertain cause, especially with a normal urinary sediment; (f) coexisting, diffuse atherosclerotic vascular disease, especially in heavy smokers; and (g) acute renal failure precipitated by antihypertensive therapy, particularly ACE inhibitors or angiotensin II receptor blockers (2). In the proper clinical setting, these signs may initiate evaluation for arterial stenosis as the cause of hy-

**Table 1**  
**Indications for Catheter-directed Angiography for the Evaluation of Renal Artery Stenosis**

<p>Clinical signs of renovascular hypertension, ischemic nephropathy or a cardiac disturbance syndrome are present and at least one of the following:</p> <ol style="list-style-type: none"> <li>1. Noninvasive vascular imaging is suggestive that a renal artery stenosis of more than 50% is present,</li> <li>2. Progression of a hemodynamically significant renal artery stenosis is indicated by noninvasive vascular imaging,</li> <li>3. Noninvasive vascular imaging is technically inadequate, equivocal or cannot be obtained,</li> <li>4. Onset of hypertension occurs in a patient less than 30 years of age,</li> <li>5. Renal artery fibromuscular dysplasia is suspected as the etiology of renal artery stenosis,</li> <li>6. There is recent onset of hypertension in a patient 60 years of age or older,</li> <li>7. There is loss of renal mass or deterioration of renal function while hypertension is being controlled medically, especially when being treated with angiotensin converting enzyme inhibitors or angiotensin II receptor blockers.</li> </ol>
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Note.—The threshold for the presence of the indications for renal angiography is 95%.

pertension or reduced renal function (Table 1). Until recently, no test other than renal angiography has been sufficiently sensitive and specific to confidently exclude a renovascular cause when the appropriate clinical signs are present (31,45,46). Duplex ultrasound, nuclear renal imaging, CT angiography, and magnetic resonance (MR) angiography have recently evolved as useful screening techniques capable of excluding an endovascular or surgically correctable renal artery stenosis as a factor contributing to the patient's hypertension or impaired renal function (37,47,48). In such cases, a normal noninvasive test may have significant value in preventing further expensive and more invasive tests and counseling the patient concerning the prognosis of the disease process. Under optimal conditions noninvasive renal imaging may be of value in selecting those with renal artery stenosis most likely to benefit from a renal revascularization (26,27); however, when performed with less than optimal conditions, these tests may be misleading and delay or prevent necessary treatment (49). The same clinical conditions that govern the propriety of renal revascularization determine the appropriateness of a search for stenosis in its feeding arteries. In other words, why look for a lesion that will not be treated if found? Catheter-directed angiography should be performed only to identify a renal artery stenosis that would be treated if found (Tables 2,3).

The Joint National Committee on

Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (2) endorses the approach of minimal to no investigation for the cause of well-controlled hypertension, but states, "reversible causes of renal failure always should be sought and treated." Risk factors and clinical signs of ischemic nephropathy in patients with renal insufficiency include: (a) unexplained azotemia, especially in older patients; (b) recent onset or acceleration of renal impairment; (c) increasing number of extrarenal sites afflicted with atherosclerotic vascular disease; (d) accelerated reduction of glomerular filtration rate while on angiotensin converting enzyme inhibitors or angiotensin receptor blockers; (e) abdominal bruits; (f) high-grade retinopathy; and (g) unilateral loss of renal volume (10,44,50,51).

Patients with progressive renal failure who fit this profile should be evaluated for a correctable stenosis or occlusion of the renal artery. Statistical evaluation of anatomic and functional parameters predictive of successful renal revascularization include the status of the arterioles distal to the renal artery stenosis, bilaterality of reconstructible disease, the amount of renal mass available for revascularization, function of the involved kidney demonstrated by nuclear scintigraphy or other means, and renal biopsy demonstrating well-preserved glomeruli and tubules with minimal arteriolar sclerosis (52–55). Reversible causes should always be sought in any patient who develops renal insuf-

ficiency. Any antihypertensive treatment regimen that effectively lowers blood pressure will help slow progressive renal failure. 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 (56). Whenever possible, an angiotensin-converting enzyme inhibitor should be part of the treatment of hypertension because 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 renal artery stenosis (40,57,58). Renal artery imaging should be performed to exclude stenosis as the etiology of unexplained renal failure. Renal revascularization for the purpose of permitting the use of an angiotensin-converting enzyme inhibitor in the treatment of hypertension is justified.

Participation by the radiologist in patient follow-up is an integral part of the evaluation and treatment of renal artery stenosis and will increase the success rate of the procedure. Close follow-up, with monitoring and management of the evaluation and treatment, is appropriate for the radiologist. The threshold for the presence of the indications for renal angiography is 95%.

## SUCCESS RATES

### Benefits of Renal Revascularization

Renovascular hypertension, ischemic nephropathy, and cardiac disturbance syndromes, such as recurrent "flash" pulmonary edema not considered secondary to impaired left ventricular systolic function or unstable angina in the setting of significant renal artery stenosis, are the pathological conditions that are amenable to treatment by renal revascularization. Until recently, the significant morbidity associated with systolic hypertension, especially in the older patients has been overlooked (59–61). In 3,657 residents of East Boston, MA, aged 65 and older, higher systolic pressure predicted linear increases in cardiovascular ( $P < .0001$ ) and total ( $P < .0007$ ) mortality. Higher diastolic pres-

**Table 2**  
**Indications for Angioplasty of a Hemodynamically Significant Renal Artery Stenosis**

1. Hypertensive control
  - a. A reasonable likelihood of cure of renovascular hypertension.
    - i. Onset of hypertension before age 30,
    - ii. Recent onset of hypertension after age 60,
    - iii. Stenosis is caused by fibromuscular hyperplasia.
  - b. Hypertension is "refractory" to medical control with at least three medications of different classes including a diuretic.
  - c. Hypertension is "accelerated" (ie, there is sudden worsening of previously controlled hypertension).
  - d. Hypertension is "malignant" (ie, is associated with end-organ damage such as left ventricular hypertrophy, congestive heart failure, visual or neurological disturbance, grade III-IV retinopathy).
  - e. The patient is intolerant or noncompliant with antihypertensive medical treatment.
2. Renal salvage
  - a. Unexplained worsening of renal function.
  - b. Loss of renal mass, especially while under surveillance during medical antihypertensive treatment.
  - c. Impairment of renal function or acute renal failure secondary to antihypertensive medication, particularly with an angiotensin converting enzyme inhibitor.
  - d. Progression of a hemodynamically significant renal artery stenosis while under surveillance.
3. Cardiac Disturbance Syndrome
  - a. Recurrent "flash" pulmonary edema secondary to impaired left ventricular function.
  - b. Unstable angina.

Note.—For indications for angioplasty of a hemodynamically significant renal artery stenosis, the threshold is 95%.

**Table 3**  
**Indications and Relative Contraindications for Renal Artery Stent Deployment**

1. Indications
  - a. Failure to attain a satisfactory result by renal artery angioplasty as determined by:
    - i. More than 30% stenosis of the luminal diameter, measured from the outer margins of intimal fissures, after balloon angioplasty.
    - ii. Failure to eliminate a hemodynamically significant pressure gradient.
    - iii. Presence of a flow-limiting dissection of the renal artery.
  - b. Stenosis of the ostium of a renal artery that has a normal diameter of 5 mm or greater.
  - c. Restenosis of a lesion that was successfully treated with balloon angioplasty in the past.
2. Relative Contraindications
  - a. An inelastic stenosis that cannot be reduced to less than 50% with balloon angioplasty.
  - b. The presence of sepsis.
  - c. If the stent would preclude surgical salvage should restenosis occur, ie, isolation of branch arteries.
  - d. For stenosis of an artery normally measuring 4 mm or less in diameter.

Note.—For indications for renal artery stent deployment, the threshold is 95%. For relative contraindications for renal artery stent deployment, the threshold is 5%.

sure predicted increases in cardiovascular ( $P = .006$ ) but not total ( $P = .48$ ) mortality. Systolic blood pressure had a highly significant positive linear association with coronary deaths ( $P = .002$ ) (62). Although renovascular hypertension and ischemic nephropathy or cardiac disturbance syndromes coexist in many patients, they are being discussed separately.

#### Technical Success following Renal Revascularization

Although initially used to treat only hemodynamically significant residual stenosis or a flow-limiting dissection after balloon angioplasty, stents have be-

come the standard of care for ostial renal artery stenosis. A meta-analysis by Rees reported 99% technical success after stent placement in 1,128 arteries compared with 55% of ostial and 70% of nonostial stenoses in 1,417 arteries treated with balloon angioplasty. There was 77% patency at a mean 7.9 months follow-up angiography in 563 arteries in which stents were placed (63). Leertouwer et al (64) reported 26% restenosis in 236 arteries examined angiographically at a mean follow-up of 19 months. This is not significantly better than 30% restenosis after balloon angioplasty in 515 patients reported by Rees (63), who claims, "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." Stents dilated to less than 6 mm, female sex, age older than 65 years, and smoking are statistically significant risk factors for restenosis. The lowest risk group was men with renal arteries 6 mm or greater, who had a restenosis rate of 10.5% in the U.S. Multicenter Renal Artery Stent Trial (63). There are very little data regarding stent use in nonostial renal artery stenosis; however, there are studies that suggest that these lesions may respond favorably to balloon angioplasty alone (65). Increased technical success and patency would be expected if the reference vessel was 6

mm or greater in diameter. 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 fibromuscular dysplasia. Renal artery stents are the preferred treatment for ostial stenosis in arteries whose reference diameter is 6 mm or greater. Their use in vessels less than 5 mm in diameter should be limited to technically failed balloon angioplasty. Their primary use in lesions where the normal diameter is 5 mm is left to the discretion of the interventionalist (Table 3). The threshold for technical success for percutaneous renal revascularization is 90%.

## RENOVASCULAR HYPERTENSION

### Cure of Hypertension in Patients with Atherosclerotic Renal Artery Stenosis

Before 1995, most investigators used the criteria for benefit established by the Cooperative Study of Renovascular Hypertension in 1972 that, ignoring the systolic blood pressure, defined cure as having a diastolic blood pressure of 90 mm Hg or less on no antihypertensive medication at follow-up (66). Now that systolic blood pressure is also considered, the early reports of "cure" of renovascular hypertension in more than 20% of patients treated by percutaneous balloon angioplasty for atherosclerotic stenoses (67–69) seem unattainable today despite recent technological improvements including the advent of stent placement and expanded usage of platelet inhibitors. In a meta-analysis by Leertouwer et al (64), 54 of 544 patients (10%) with atherosclerotic renal artery stenosis treated with balloon angioplasty alone were reported as "cured." Adjusting for some overlap of data in two meta-analyses, hypertension was reported as "cured" in 38 of 334 patients (11%) treated with renal artery stent placement (64,70). It must be noted that the studies included in these meta-analyses were not consistent in their definition of "cure." Most studies considered only the diastolic blood pressure; others included systolic blood pressures as high as 160 among those considered cured (71–73). Therefore, the actual per-

centage of patients cured by percutaneous intervention was probably lower than 11%. Barri et al (48) retrospectively analyzed the clinical characteristics of the patients in 63 consecutive renal revascularizations (34 endovascular and 29 surgical) in which hypertension was cured in 21%. Univariate analyses were used to associate clinical variables with blood pressure outcome. Categorical variables were assessed by  $\chi^2$  tests, Fisher exact test, and continuous variables with a two-sample Student *t* test. Duration of hypertension of less than 10 years, preintervention systolic blood pressure of more than 180 mm Hg, and male sex were collective variables that predicted cure with a sensitivity of 92%, specificity of 77%, positive predictive value of 52%, and negative predictive value of 97% (48). This small study is the only one to address the topic of prediction of cure of renovascular hypertension based on the clinical characteristics of an adult population.

The problem of establishing normal blood pressure on no antihypertensive medication after renal revascularization is confounded by the coexistence of underlying essential hypertension in the majority of cases. Therefore, it can be concluded that cure of renovascular hypertension caused by atherosclerotic renal artery stenosis is an unrealistic goal in most cases. The clinical profile of the patient most likely to be cured has not been defined; but cure is more likely in a young male with recent onset of hypertension that is poorly controlled medically. Because of the low percentage of patients with atherosclerotic renal artery stenosis who are reported as cured after revascularization, an effort should be made to define their clinical profile during future investigations.

### Cure of Hypertension in Patients with Fibromuscular Renal Artery Stenosis

The mean cure rate for renal revascularization of stenoses secondary to fibromuscular dysplasia (FMD) was 44% in a meta-analysis by Martin et al (74). No attempt was made to separate the results for treatment of the various types of FMD in this document (74). 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 (75). Contrary to what one might predict, the technical and clinical results of angioplasty in those patients with FMD involving the renal artery branches was as good as those involving only the main renal artery (76,77). By logistic regression, Davidson et al (78) 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 (10) found progression of medial fibroplasia in 33% of 66 patients with FMD who were observed without intervention; however, none progressed to occlusion or developed renal failure. Therefore, cure of hypertension is a reasonable goal in a patient with the medial fibroplastic form of renal artery FMD (Table 2). It is most likely to result in a younger patient with recent onset of hypertension. It is logical to assume that the cure rate will be higher in patients with unilateral involvement (eg, 62% in the University Hospital Zurich Cooperative Study on Fibromuscular Hyperplasia) (78). 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 caused by 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 decided based on personal clinical experience.

### Benefits of Renal Revascularization Other Than "Cure" of Hypertension

All of the studies included in the above meta-analyses (64,70,74) and three recent prospectively randomized controlled trials (34,80,81) reported that a decrease of blood pressure on lower doses of medication results from renal revascularization. Levels of statistical significance were reported to have been reached in groups treated by both balloon angioplasty alone and by stent placement in the meta-analysis by Leertouwer et al (64). Because of the low

incidence of cure of renovascular hypertension and the ability to adequately control blood pressure with medicine in a large percentage of patients, many prominent hypertension specialists feel that patients with normal renal function have little to gain from renal revascularization and recommend it only for those patients who fail medical therapy (82,83). Hypertension should be considered resistant if "the blood pressure cannot be reduced to below 140/90 mm Hg in patients who are adhering to an adequate and appropriate triple-drug regimen that includes a diuretic, with all three drugs prescribed in near maximal doses" (Table 2). For older patients with isolated systolic hypertension, resistance is defined as "failure of an adequate triple-drug regimen to reduce the systolic blood pressure to below 160 mm Hg"; lower values of systolic blood pressure are recommended in certain patient populations, particularly those with diabetes or proteinuria (2). Additional diagnostic procedures may be indicated to seek causes of hypertension in patient groups whose blood pressures are responding poorly to drug therapy, those with well controlled hypertension whose blood pressures begin to increase, those with stage 3 hypertension and those with a sudden onset of hypertension (2).

Medical control of hypertension is not without risk. Whether controlling blood pressure with less medication outweighs the risks of the revascularization procedure must be considered on an individual case basis (82–84). Whenever possible, an angiotensin-converting enzyme inhibitor or angiotensin blocker should be part of the antihypertensive treatment, because these drugs have been shown to have renoprotective properties which are as important or more important than their antihypertensive effect and are the preferred medications in many cases of nonrenovascular hypertension (57). In the presence of a hemodynamically significant renal artery stenosis angiotensin II causes constriction of the efferent arterioles to maintain glomerular filtration pressure. By preventing the conversion of angiotensin I to angiotensin II, an angiotensin-converting enzyme inhibitor induces a rapid fall in the glomerular filtration rate. The blockade of the renin-angiotensin autoregulatory mechanism results in a fall of systemic blood pressure that may result in further loss of

function and perfusion of the kidney with renal artery stenosis. Ipsilateral ischemic loss of nephron function and mass may be masked because normal total functional levels can be maintained if the contralateral kidney is normally perfused. The total glomerular filtration rate may be further decreased if the contralateral kidney is absent or diseased, resulting in an elevation in the serum creatinine level. 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 renal artery stenosis. If the clinician chooses to treat hypertension without knowing the status of renal artery patency, he must be alert to signs of decreased renal function and loss of renal mass.

#### **Benefits of Medical Treatment Alone**

Because more than 90% of patients treated with renal angioplasty or stent placement for atherosclerotic stenosis require medication to remain normotensive, it is reasonable to consider medical control the first line therapy and reserve endovascular revascularization for those who are poorly controlled or unable to tolerate the medical regimen. The decision to treat hypertension medically carries with it the obligation to closely follow the patient for signs of impaired renal function and loss of renal mass. Renal size and function should be followed closely in those patients being treated medically for hypertension, especially those who have risk factors for renovascular hypertension or ischemic nephropathy. In this group, serial renal imaging should be performed with use of a standardized technique and serum creatinine levels should be obtained more frequently when medications or their doses are being altered. In patients with atherosclerotic renal artery stenosis, revascularization for the purpose of hypertension control should be reserved for those with "resistant hypertension" or those whose medical therapy is complicated by decreased renal function, loss of renal mass or recurrent episodes of pulmonary edema.

#### **ISCHEMIC NEPHROPATHY**

There is no international consensus document that agrees on a definition of

ischemic nephropathy. Of the two major international guidelines for the management of hypertension and the prevention of associated cardiovascular disorders, the Sixth Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (2) and the 1999 World Health Organization–International Society of Hypertension Guidelines for the Management of Hypertension (3), only the former mentions ischemic nephropathy. This has led to ambiguity concerning its meaning. Ischemic nephropathy has been defined by various authors as: kidney damage after stenosis or an obstructive lesion in the main kidney artery (85), critical main renal artery stenosis or occlusion with excretory renal insufficiency (86), clinically important reduction in GFR associated with a hemodynamically significant renal artery obstruction (87), and hypertension and a defect in renal function and/or loss of renal parenchyma caused by hemodynamic changes secondary to a renal artery stenosis (49). Some nephrologists think of ischemic nephropathy as an irreversible process of cellular death resulting in fibrosis and permanent nephron loss, whereas others expand this concept to include reversible ischemic nephron injury in the disease spectrum. Labels aside, all recognize the syndrome of renal hypoperfusion with impaired nephron function that may be recovered by renal revascularization. A review of atherosclerotic disease in 7,200 end-stage renal disease patients indicated that occlusive disease of the renal arteries may contribute to progressive renal failure in 1.24% of the United States dialysis population or in 14% of the caucasian patients with hypertensive nephrosclerosis (88). Appel et al (89) found renal artery occlusive disease in 22% of patients older than 50 years of age who were entering renal replacement therapy suggesting that screening for this risk factor should be performed in older patients who have unexplained deterioration of renal function. The Sixth Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure 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 renal ar-

tery stenosis remain stable for prolonged periods if blood pressure is well controlled (2).

### Criteria for Benefit from Renal Revascularization

There is a great deal of controversy concerning the degree of benefit that can be expected from revascularization of patients who have ischemic nephropathy (Table 2). 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 decline in the glomerular filtration rate and size of the kidneys. The loss is accelerated by many disease states including ischemic nephropathy where, in addition to the loss of nephron tissue, there can be functional loss resulting from hypoperfusion secondary to renal artery stenosis. 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 (90). Traditionally, benefit from revascularization was defined as a reduction of the serum creatinine by 20% of the baseline value and a 20% increase from baseline was considered as failure. Values in between were interpreted in a variety of ways by different investigators. Recently, most investigators use the change of mean or average serum creatinine as an indicator for benefit. Those who claim success when no change in the serum creatinine occurs justify it by contending that the procedure has "stabilized" or halted the progressive nephron loss that was occurring before revascularization. The burden of proof for this contention rests with the investigator; it is lacking in most reported series. It is important to realize that although measurement of serum creatinine has been used almost exclusively, this is an unreliable surrogate for glomerular filtration rate in the setting of advancing azotemia and hopefully will be replaced in the future with more direct determinations of glomerular filtration (91-93).

The slope of the linear relation between the reciprocal of creatinine con-

centration and time can be used to delineate the rate of change in renal function (94). Failure of progression along the slope of decline in renal function may indicate benefit from intervention even though there has been no improvement in baseline serum creatinine. Rowe et al (95) used the application of two-phase linear regression to identify and characterize changes in this slope with use of a microcomputer. Their method fits two intersecting lines to the data by computing a least-squares estimate of the position of the slope change and its 95% confidence limits, thus avoiding the potential bias of fixing the change at a preconceived time corresponding with an alteration in treatment. The program then produces a graphical output to aid interpretation (95). 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 (96). Utilizing this method, the potential for error is greatly increased by small numbers of data points. Although the calculations can be performed with as few as five data points, it is desirable to have as many as possible. Minor changes in this slope, perhaps indicating response to therapeutic intervention, can be difficult to identify and yet be of clinical importance. This method can be used to study the effect of intervention on an individual or a group.

### Results of Treatment in Patients with Ischemic Nephropathy

Harden et al (97) found statistically significant benefit ( $P < .007$ ) indicated by a reduction in the rate of renal functional loss from  $-4.3$  at baseline to  $-0.55 \times 10^{-6} \text{ L } \mu\text{mol}^{-1}/\text{d}^{-1}$  at last follow-up in 32 patients treated with renal artery stent placement despite a decrease in serum creatinine of less than 20% in only 34% of patients and an increase in serum creatinine of more than 20% in 28% of patients. In a study by Watson et al (38), stent placement was performed in 33 patients with bilateral renal artery stenosis or stenosis of the artery supplying a solitary functioning kidney in whom the calculated slopes of the regression lines were negative for all patients before treatment. After treatment, these slopes became positive in 72% of patients, indicating an improve-

ment in renal function, and became less negative in 28% of patients, signifying stabilization of renal function (38). Similar results were found after revascularization by van Rooden et al (98), who compared preoperative and postoperative slopes of estimated glomerular filtration rates with each other and with age- and sex-matched population controls. They found that the mean estimated glomerular filtration rate improved from 28.3 to 43.1 mL/min/1.73 m<sup>2</sup> ( $P < .01$ ) after surgical revascularization. Also, the rate of decline in renal function decreased from  $-2.7$  to  $-0.66$  mL/min/1.73 m<sup>2</sup> for the year before surgery, which was better than the matched control of  $-0.84$  mL/min/1.73 m<sup>2</sup> (98). There was no improvement in mean renal function reported in three prospectively randomized studies of renal revascularization (34,80,81). These trials were criticized in a review by Sacks et al (84), who found fault with the analysis and interpretation of the data by the authors. The investigators in these trials found no statistical difference between the mean serum creatinine value at baseline and after treatment. A problem with the use of 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 mg/dL and, at the time of final follow-up, the serum creatinine level was 2 mg/dL in eight patients and 7 mg/dL in two patients. Utilizing 20% reduction of serum creatinine as a binary criterion for benefit, 80% of patients would have benefited from the procedure and 20% of patients would have failed to benefit. Utilizing the mean change in the serum creatinine of the treated cohort as the criterion for success, there would have been no benefit in this patient cohort. This oversimplification illustrates the problem with the use of a mean or average of a test that has a greater mathematical limit on the potential to improve than it does to fail. It also ignores benefit that can be derived by stabilization of the rate of nephron loss. 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 (38,97,98). Studies reporting binary results, with a less than 20% deterioration and/or 20% decrease in the serum creatinine level as a measure of

**Table 4**  
**Specific Major Complications for Percutaneous Renal Revascularization**

Complication	Reported Rate (%)	Threshold (%)
30-day mortality	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, transfusion, or prolonged hospital stay	5	5
Acute renal failure	2	2
Worsening of chronic renal failure requiring an increase in the level of care	2	5

Note.—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 volume larger than most individual practitioners are likely to treat. Therefore, it is recommended that complication-specific thresholds usually should be set higher than the complication-specific reported rates listed in this table. It is also recognized that a single complication can cause a rate to cross above a complication-specific threshold when the complication occurs in a small volume of patients (eg, early in a quality-improvement program). In this situation, the overall procedure threshold is more appropriate for use in a quality-improvement program. All values were supported by the weight of literature evidence and panel consensus.

functional stabilization or benefit, find a mean of 54% of patients improved and 26% of patients stabilized by surgical revascularization (53,55,99–101). With use of binary criteria, renal artery stent placement resulted in improvement in 30% of patients and stabilization in 38% of patients (64) and improvement in 26% of patients and stabilization in 48% of patients (70), although neither showed a significant decrease in overall serum creatinine values in these meta-analyses.

In the absence of significant elevation of the estimated glomerular filtration rate, valid individual or population-based improvement in renal function can be demonstrated by stabilization of the serum creatinine or cystatin C value (102). Improvement of the rate of decline of the reciprocal slope of serum creatinine versus time after a medical, endovascular or surgical intervention on the kidney can demonstrate this, provided that there are sufficient data points for analysis and there was no other concurrent alteration in therapy. 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: (a) normal appearance of the

arterioles distal to the renal artery stenosis; (b) bilaterality of reconstructible disease; (c) a near normal volume of renal mass available for revascularization; (d) a test demonstrating function of the involved kidney; (e) renal biopsy demonstrating well preserved glomeruli and tubules with minimal arteriolar sclerosis; (f) severe and difficult to control hypertension; and (g) abrupt onset of renal insufficiency (52,54,86). A Doppler ultrasound resistance index of 80 or greater ( $1 - [\text{end-diastolic velocity} / \text{maximal systolic velocity}] \times 100$ ) (29) and the absence of hypertension (86,103) are strong negative predictors of renal salvage after revascularization of any type.

### CARDIAC DISTURBANCE SYNDROMES

Renal artery stenosis may worsen angina or congestive heart failure in patients with coronary artery disease, left ventricular dysfunction, or cardiomyopathy due to alterations in the renin-angiotensin-aldosterone axis resulting in a state of volume overload and peripheral vascular constriction (7,104,105). Renal revascularization may result in relief of these cardiac syndromes due to normalization of excess renin production, which reduces sodium and water reten-

sion and vasoconstriction caused by aldosterone and angiotensin and causes natriuresis because of improved glomerular filtration (6,105) (Table 2). Restoring unobstructed renal blood flow has the additional benefit of allowing safe usage of angiotensin-converting enzyme inhibitors without the risk of worsening renal failure and reducing coronary perfusion. Bilateral renal artery stenosis or stenosis of a solitary functioning kidney are frequently present in a patient with a cardiac syndrome who is likely to receive benefit from percutaneous renal revascularization. More than 70% of 73 patients with cardiac disturbance syndromes with this vascular profile who were treated with percutaneous angioplasty and stent placement were free of congestive heart failure and unstable angina at 12-month mean follow-up (6,7). Additional benefits in this patient group also frequently include improvement of hypertension control and renal function (6,7,106).

## COMPLICATIONS

### Risks of Endovascular Revascularization

Publications reviewed for the determination of the risks involved with endovascular revascularization included two large series (107,108) and two meta-analyses (64,77). There was no overlap of data among these studies that include 2,994 revascularization procedures in 2,474 patients, with stents placed in 980 vessels. The total complication rate varied from a high of 36% (107) to a low of 12% (64); the weighted mean complication rate was 14%, excluding complications classified as radiologic/technical complications. Radiologic/technical complications are defined by Beek et al (107) as "events that occur during catheterization or stent deployment that have no clinical consequences but lead to an increase in procedural time and/or cost," such as the need to use an additional stent to cover the renal artery ostium because the first stent was malpositioned. Groin hematoma and puncture site trauma were the most commonly reported complications. Thirty-day mortality was 1%, usually related to renal artery perforation, cholesterol embolization, acute renal failure, and arterial access puncture above the inguinal ligament. Secondary nephrectomy, ei-

ther surgical or endovascular, was performed in less than 1% of the cases. A surgical salvage operation was necessary in 2.5% of the cases reported by Martin et al (77); however, the incidence is less than 1% in the later reported meta-analysis (64). Symptomatic embolization occurred in 8% of patients treated by Beek et al (107), but in less than 1% in a meta-analysis by Leertouwer et al (64). Occlusion of the main renal artery was reported in 2.5% and 0.8% and occlusion of a renal artery branch causing a segmental infarction in 1.7% and 1.1% of patients, respectively, in meta-analyses by Martin et al (77) and Leertouwer et al (64). A trend toward reduced complications was demonstrated in an earlier investigation by Martin et al (77), which found that the total complication rate decreased from 20% in the first hundred cases to 13% in the second hundred cases. The authors attributed the change to increased experience and improvement in technology and devices (109). In summary, with use of the present technology and depending on patient selection, the mean incidence of complications after endovascular renal revascularization is 14%. Most of these are not life threatening and do not result in renal functional loss. The combined incidence of 30-day mortality, occlusion of the main renal artery, loss of a kidney, renal artery perforation, and the need for surgical salvage is expected to be less than 4% (Table 4). Renal artery branch occlusions occur in less than 2% of cases. Cholesterol embolization resulting in decreased renal function, visceral symptoms, or peripheral symptoms is expected to be less than 3% (64,77,107,108). Although loss of life during percutaneous renal revascularization is rare, there is significant risk of a serious complication that may result in loss of renal function or require treatment that is likely to increase the duration and cost of patient care. The anticipated benefit from renal revascularization must outweigh the risks involved. Each case must be evaluated individually. The overall threshold for major complications of percutaneous renal revascularization is 10%.

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#### APPENDIX 1: SOCIETY OF INTERVENTIONAL RADIOLOGY STANDARDS OF PRACTICE COMMITTEE CLASSIFICATION OF COMPLICATIONS BY OUTCOME

##### Minor Complications

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

##### Major Complications

C. Require therapy, minor hospitalization (<48 h),  
D. Require major therapy, unplanned increase in level of care, prolonged hospitalization (>48 h),  
E. Have permanent adverse sequelae, or  
F. Result in death.

#### APPENDIX 2: 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 (110,111).

The Committee was unable to reach consensus on the following: none.

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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 judgement 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.