2004 (Res. 26)
Amended 2006 (Res. 16g,17,34,35,36)
Effective 10/01/04

PRACTICE GUIDELINE FOR THE PERFORMANCE OFANGIOGRAPHY, ANGIOPLASTY, AND STENTING FOR THE DIAGNOSIS AND TREATMENT OF RENAL ARTERY STENOSIS IN ADULTS

PREAMBLE

These guidelines are an educational tool designed to assist practitioners in providing appropriate radiologic care for patients. They are not inflexible rules or requirements of practice and are not intended, nor should they be used, to establish a legal standard of care. For these reasons and those set forth below, the American College of Radiology cautions against the use of these guidelines in litigation in which the clinical decisions of a practitioner are called into question.

The ultimate judgment regarding the propriety of any specific procedure or course of action must be made by the physician or medical physicist in light of all the circumstances presented. Thus, an approach that differs from the guidelines, standing alone, does not necessarily imply that the approach was below the standard of care. To the contrary, a conscientious practitioner may responsibly adopt a course of action different from that set forth in the guidelines when, in the reasonable judgment of the practitioner, such course of action is indicated by the condition of the patient, limitations on available resources, or advances in knowledge or technology subsequent to publication of the guidelines. However, a practitioner who employs an approach substantially different from these guidelines is advised to document in the patient record information sufficient to explain the approach taken.

The practice of medicine involves not only the science, but also the art of dealing with the prevention, diagnosis, alleviation, and treatment of disease. The variety and complexity of human conditions make it impossible to always reach the most appropriate diagnosis or to predict with certainty a particular response to treatment. Therefore, it should be recognized that adherence to these guidelines will not assure an accurate diagnosis or a successful outcome. All that should be expected is that the practitioner will follow a reasonable course of action based on current knowledge, available resources, and the needs of the patient to deliver effective and safe medical care. The sole purpose of these guidelines is to assist practitioners in achieving this objective.

I. INTRODUCTION

This guideline was developed and written collaboratively by the Society of Interventional Radiology (SIR) and the American College of Radiology (ACR).

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 hypertensive Americans who are aware that they have high blood pressure has increased from 51% to 73%. During the same period, those treated for hypertension have increased from 32% to 55%, and those whose blood pressure is controlled below 140/90 mm Hg have 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 period there has been an increase in the incidence of end-stage renal disease and the incidence of 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 hypertensive patient have also changed the clinical profile of the patient with renovascular disease who presents for consideration of renal revascularization. The patient under the age of 50 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 who has a 20-year history of treatment for hypertension, now poorly controlled, deteriorating renal function, and a 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 to the benefit that is likely to be attained? This document will attempt to establish guidelines to answer these difficult questions. It is directed toward endovascular renal revascularization; however, many of the issues pertain equally to surgical revascularization.

II. DEFINITIONS

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

Hypertension – hypertension is defined by the 1999 World Health Organization's International Society of Hypertension Guidelines for the Management of Hypertension 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" [3]. The sixth report of The Joint National Committee on Prevention, Detection, Evaluation, and Treatment of high blood pressure defined hypertension as "systolic blood pressure 140 mm Hg or greater, diastolic blood pressure 90 mm Hg or greater, or taking antihypertensive medication" [2].

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

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

Resistant hypertension – 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. For patients above age 60 with isolated systolic hypertension, resistance is defined as failure of an adequate triple-drug regimen to reduce the SBP to below 160 mm Hg [2].

Stage 3 (grade 3) 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, i.e., % renal artery stenosis = 100 x 1 – (the narrowed lumen diameter / the normal vessel diameter).

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 CT angiography [5].

Truncal renal artery stenosis – nonostial 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 luminal 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 felt to be secondary to impaired left ventricular systolic function or unstable angina frequently seen in the setting of bilateral renal artery stenosis or single stenosis of the renal artery in the solitary kidney [2,6,7].

III. INDICATIONS/CONTRAINDICATIONS

A. When should catheter directed renal artery angiography be performed?

Approximately 3%-5% of the hypertensive population has a renovascular etiology. Increasing age and coexisting atherosclerosis have significant effects on the prevalence of renovascular hypertension. Anderson et al, reported a 10.2% prevalence of secondary forms of hypertension, including renovascular hypertension (3.1%) in 4,429 patients 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 (8.0%) [41]. 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, [41].

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 [33,42,43,44]. As listed by the Sixth Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure, they include: 1) onset of hypertension before the age of 30, especially without a family history, or recent onset of significant hypertension after the age of 55; 2) an abdominal bruit, particularly if it continues into diastole and is lateralized; 3) accelerated or resistant hypertension; 4) recurrent (flash) pulmonary edema; 5) renal failure of uncertain cause, especially with a normal urinary sediment; 6) coexisting, diffuse atherosclerotic vascular disease, especially in heavy smokers; and 7) 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 hypertension or reduced renal function.

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, computerized tomographic angiography, and magnetic resonance 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 in 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, under 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 only be performed to identify a renal artery stenosis that would be treated if found.

The Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure endorses the approach of minimal to no investigation for the cause of well-controlled hypertension, but states that "reversible causes of renal failure always should be sought and treated" [2]. Risk factors and clinical signs of ischemic nephropathy in the patient with renal insufficiency include: 1) unexplained azotemia, especially in the older patient, 2) recent onset or acceleration of renal impairment, 3) increasing number of extrarenal sites afflicted with atherosclerotic vascular disease, 4) accelerated reduction of glomerular filtration rate while on angiotensin converting enzyme inhibitors or angiotensin receptor blockers, 5) abdominal bruits, 6) high-grade retinopathy, and 7) unilateral loss of renal volume [10,44,50,51]. Patients with progressive renal failure that fit this profile should be evaluated for a correctable stenosis or occlusion of the renal artery. Statistical evaluations of anatomic and functional parameters predictive of success following 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 [52,53,54,55]. Reversible causes should always be sought in any patient who develops renal insufficiency.

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, since 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 to permit the use of an angiotensin-converting enzyme inhibitor in the treatment of hypertension is justified.

B. When is it appropriate to treat a renal artery stenosis?

Normal arteries are not stenotic. Atherosclerotic stenoses are likely to be progressive [8,9,10]. It is much easier to treat a vessel with a lesser degree of stenosis than one that has progressed to occlusion or near occlusion. It would seem logical therefore to seek and treat all renal artery stenoses 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. Since 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, then, 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 is narrowed by over 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 [11]. 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 [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 auto-regulatory system [14,15,16].

What, then, of those stenoses between 50% and 75%? Because of technical problems relating to 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, intra-arterial 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 greater than 10% or a *mean pressure gradient* greater than 5% [100 x (BP proximal to the stenosis - BP distal to stenosis / BP proximal to stenosis)] across the narrowed vascular lumen is generally accepted as indicating that a hemodynamically significant stenosis is present [18,19]. This recommendation is clearly arbitrary.

There is no consensus, however, as to 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. Difficulty in measuring the pressure without affecting it and physiologic variations that occur during its measurement make pressure gradient thresholds questionable. The Dutch iliac stent trial used a mean pressure gradient of 10 mm Hg obtained either at rest or after vasodilatation to indicate either the need for intervention or a technical failure of iliac angioplasty requiring stent placement [20].

Gross et al, compared the use of mean and peak systolic gradients before and following induction of steady-state hyperemia by an infusion of increasing doses of nitroglycerine into the stenotic renal artery 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 [21,22,23]. 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 his or her 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 [13,24,25,26,27,28].

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 follows: 1 – end-diastolic velocity [cm/second] ÷ maximal systolic velocity [cm/second] x 100. It is dimensionless because both velocities are measured in cm/sec [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, then, of the stenosis that is considered "pre-occlusive," i.e., 70%-90%? Multiple studies suggest that the term "pre-occlusive stenosis" is a misnomer; there is a significant risk, however, that stenoses will progress during short observational periods ranging from 6 to 36 months [8,10,32,33,34,35]. Although the viability of a kidney following 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 pre-occlusive stenoses is unclear. Although progression to occlusion is less than 10%, the rate of renal atrophy is greater than 25% and has been directly associated with the degree of stenosis [36,37]. Of those who had 60% or greater renal artery stenosis by duplex ultrasound, Caps et al, found that 16% lost greater than 1 cm of kidney length in 1 year, and 27% of the patients followed by Guzman et al, lost an average of 19 mm of renal length in a mean follow-up of 14 months. Watson et al, have shown 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 they treated [38].

Is there a role for prophylactic treatment of hemodynamically significant renal artery stenoses in patients with normal renal function who either are normotensive or have easily controlled hypertension? An electronic literature search of the National Library of Medicine found reference to only one such study. Chaikof et al, revascularized 43 kidneys in 32 patients (renal artery stenosis >70%; mean serum creatinine of 1.29 +/- 0.24 mg/dl; normotensive 22%, hypertension controlled with a single medication 78%) who were operated upon for infra-renal abdominal aortic aneurysm (62%) or aortic occlusive disease (38%). Blood pressure remained normal either with a single agent or without medication in 75%, and there was no statistically significant deterioration in renal function in the 96% of their patients who were available for late follow-up (median 64 months) [39]. Chaikof et al, justified their actions by consideration of 30% to 40% restenosis rates following percutaneous renal revascularization and the increased risks of reoperation 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 any adverse results.

The majority of patients with hemodynamically significant renal artery stenosis 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 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 on consideration of the patient's age, anticipated longevity, renal function, ability to withstand a procedural complication, 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.

IV. SUCCESS RATES

A. Benefits of Renal Revascularization

Renovascular hypertension (RVH), ischemic nephropathy (IN) and cardiac disturbance syndromes (i.e., recurrent "flash" pulmonary edema not felt to be 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 elderly, has been overlooked [59,60,61]. In 3,657 residents of East Boston, Massachusetts, age 65 and older, higher systolic pressure predicted linear increases in cardiovascular ($p < 0.0001$) and total ($p < 0.0007$) mortality. Higher diastolic pressure predicted increases in cardiovascular ($p = 0.006$) but not total ($p = 0.48$) mortality. SBP had a highly significant positive linear association with coronary deaths ($p=0.002$) [62]. Although renovascular hypertension and ischemic nephropathy or cardiac disturbance syndromes coexist in many patients, they are being discussed separately.

B. Clinical Success Following Renal Revascularization

1. Renovascular Hypertension

a. Cure of hypertension in the patient with atherosclerotic renal artery stenosis

Before 1995 most investigators used criteria for benefit established by the Cooperative Study of Renovascular Hyper-tension 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 greater than 20% of patients treated by percutaneous balloon angioplasty for atherosclerotic stenosis [67,68,69] seem unattainable today despite recent technological improvements, including the advent of stenting and expanded usage of platelet inhibitors. In a meta-analysis by Leertouwer et al, 54 of 544 (10%) patients with atherosclerotic renal artery stenosis treated by *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 (11%) treated by *renal artery stents* [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 [71,72,73]. Therefore the actual percentage of patients cured by percutaneous intervention was probably lower than 11%.

Barri et al, retrospectively analyzed the clinical characteristics of the patients in 63 consecutive renal revascularizations, 34 endovascular and 29 surgical, of which hypertension was cured in 21%. Univariate analyses were used to associate clinical variables with BP outcome. Categorical variables were assessed using chi-square tests, Fischer's Exact Test, and continuous variables with a two-sample Student's test. Duration of hypertension of less than 10 years, preintervention systolic BP of over 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 following renal revascularization is confounded by the coexistence of underlying essential hypertension in the majority of cases. It can be concluded; therefore, that cure of renovascular hyper-tension due to 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. Since only a low percentage of patients with atherosclerotic renal artery stenosis are reported as cured following revascularization, an effort should be made to define their clinical profile during future investigations.

b. Cure of hypertension in the patient with fibromuscular renal artery stenosis

The mean cure rate for renal revascularization for stenoses secondary to fibromuscular dysplasia (FMD) was 44% in a meta-analysis by Martin et al. 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% to 70% of the total 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 were as good as those involving only the main renal artery [76,77]. Using logistic regression, Davidson et al, found that younger age, milder hyper-tension, and shorter duration of hypertension were statistically significant independent variables predicting successful results from percutaneous transluminal renal angioplasty (PTRA) in FMD [78]. Schreiber et al, 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 [10].

Therefore *cure* of hypertension is a reasonable goal in a patient with the medial fibroplastic form of renal artery fibromuscular dysplasia. 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 (62% in the University Hospital Zurich Cooperative Study on Fibromuscular Hyperplasia) [79]. 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 due to the medial fibroplastic type of FMD is sufficiently high to recommend PTRA 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 upon on personal clinical experience.

c. 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] have reported that a decrease of blood pressure on lower doses of medication results from renal revascularization. Levels of statistical significance were reported by Leetouwer et al, [64] to have been reached in groups treated by both balloon angioplasty alone and stenting in the meta-analysis. Because of the low incidence of cure of renovascular hypertension and the ability to adequately control blood pressure medically 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. 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 SBP 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 on less medication outweighs the risks of the revascularization procedure must be considered on an individual case basis [82,83,84]. Whenever possible, an angiotensin-converting enzyme inhibitor or angiotensin blocker should be part of the antihypertensive treatment, since this drug has been shown to have renoprotective properties which are as important or more important than its antihypertensive effect and is the preferred medication 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 the stenotic renal artery. 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 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.

2. 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's 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 following 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, while 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 might contribute to progressive renal failure in 1.24% of the U.S. dialysis population or in 14% of the Caucasian patients with hypertensive nephrosclerosis [88]. Appel et al, found renal artery occlusive disease present in 22% of patients over 50 years of age who were entering renal replacement therapy, suggesting that screening for this risk factor should be performed in the older patient with unexplained deterioration of renal function [89]. 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 artery stenosis remain stable for prolonged periods if blood pressure is well controlled [2].

a. 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 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 due to 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.

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 have used 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 prior to the 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 it will be replaced in the future with a more direct determination of glomerular filtration [91,92,93].

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 [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, used the application of two-phase linear regression to identify and characterize changes in this slope using a micro-computer. 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]. The potential for error with this method 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.

b. Results of treatment in patients with ischemic nephropathy

Harden et al, found statistically significant benefit ($p<0.007$) as indicated by reduction in the rate of renal functional loss (from –4.3 at baseline to –0.55 $\times 10^{-6}$ L μmol^{-1}/ day^{-1} at last follow-up) in 32 patients treated by renal artery stenting despite a decrease in serum creatinine (by less than 20%) in only 34% of the patients and an increase in serum creatinine (by greater than 20%) in 28% of them [97]. In a study by Watson et al, stenting was performed in 33 patients with either bilateral renal artery stenosis or stenosis of the artery supplying a solitary functioning kidney, in all of whom the calculated slopes of the regression lines were negative before treatment. After treatment, these slopes became positive in 72%, indicating an improvement in renal function, and became less negative in 28%, signifying stabilization of renal function [38]. Similar results were found following revascularization by van Rooden et al, 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/1.73m^2 ($p< 0.01$) following surgical revascularization. Also, the rate of decline in renal function decreased from –2.7 to –0.66 mL/min/1.73m^2 for the year before surgery, which was better than the matched control of –0.84 mL/min/1.73m^2 [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 who found fault with the analysis and interpretation of the data by the authors [84]. The investigators in these trials found no statistical difference between the mean serum creatinine value at baseline and following treatment.

A 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 8 had a serum creatinine of 2 mg/dl and the serum creatinine level had risen to 7.0 mg/dl in 2 patients. Utilizing 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. Utilizing the mean change in 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 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 [38,97,98]. 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 [53,55,100, 101]. Using binary criteria, renal artery stenting resulted in 30% improved and 38% stabilized [64] and 26% improved and 48% stabilized [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 cysplatin 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: 1) normal appearance of the arterioles distal to the renal artery stenosis; 2) bilaterality of reconstructable disease; 3) a near normal volume of renal mass available for revascularization; 4) a test demonstrating function of the involved kidney; 5) renal biopsy demonstrating well preserved glomeruli and tubules with minimal arteriolar sclerosis; 6) severe and difficult to control hypertension; and 7) abrupt onset of renal insufficiency [52,54,86]. A Doppler ultrasound resistance index of 80 or greater [1 - (end-diastolic velocity/maximal systolic velocity) x 100] [29] and the absence of hypertension [86,103] are strong negative predictors of renal salvage after revascularization of any type.

3. 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 retention and vasoconstriction due to aldosterone and angiotensin, and it causes natriuresis due to improved glomerular filtration [6,105]. 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. Over 70% of 73 patients with cardiac disturbance syndromes, with this vascular profile, that were treated by percutaneous angioplasty and stenting 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 hypertensive control and renal function [6,7,106].

C. Technical Success Following Renal Revascularization

Although stents were initially used only to treat hemodynamically significant residual stenosis or a flow limiting dissection following balloon angioplasty, they have become the standard of care for ostial renal artery stenosis. A meta-analysis by Rees 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 of 1,417 arteries. There was 77% patency at a mean 7.9 months angiographic follow-up in 563 arteries that were stented [63]. Leertouwer et al, reported 26% restenosis in 236 arteries examined angiographically at a mean follow-up of 19 months [64]. This is not significantly better than 30% restenosis following balloon angioplasty in 515 patients reported by Rees, who points out that "the benefits of stents for long-term patency relative to PTA are mostly related to the markedly superior initial success rates rather than reduction of restenosis" [63].

Stents dilated to less than 6 mm, female sex, age greater 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 suggesting that these lesions may respond favorably to balloon angioplasty alone [65]. 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 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 below 5 mm 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.

V. COMPLICATIONS – Risks of Endovascular Revascularization

Publications reviewed for determining 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, which include 2,994 revascularizations (980 vessels stented) in 2,474 patients. 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 radiological-technical complications. Radiological-technical complications are defined by Beek et al, 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" – i.e., the need to use an additional stent to cover the renal artery ostium because the first stent was malpositioned [107].

Groin hematoma and puncture site trauma were the most common complications reported. 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, either surgical or endovascular, occurred in less than 1% of the cases. A surgical salvage operation was necessary in 2.5% of the cases reported by Martin et al, however, the incidence was below 1% in the later reported meta-analysis [64,77]. Symptomatic embolization occurred in 8% of the 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% respectively in meta-analyses by Martin et al and Leertouwer et al, [64,77]. A trend toward reduced complications was demonstrated in an earlier investigation by Martin et al, which found that the total complication rate fell 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, using the present technology and depending on patient selection the mean incidence of complications following endovascular renal revascularization is 14%. Most of these are not life threatening, nor do they 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%. Renal artery branch occlusions occur in less than 2% of cases. Cholesterol embolization resulting in decreased renal function or visceral or peripheral symptoms is expected in less than 3% of cases [64,77,107,108]. Although loss of life during 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.

VI. QUALIFICATIONS AND RESPONSIBILITIES OF PERSONNEL

A. The physician performing renal angioplasty/stenting must fully appreciate the benefits, alternatives, and risks of the procedure. He/she must have a thorough understanding of vascular anatomy (including congenital and developmental variants and common collateral pathways), angiographic equipment, radiation safety considerations, and physiologic monitoring equipment and have access to an adequate supply of catheters, guidewires, and personnel to perform the procedure safely.

Renal angioplasty/stenting procedures must be performed under the supervision of and interpreted by a physician who has the following qualifications pertinent to the scope of services to be provided and the specific privileges sought:

1. Certification in Radiology or Diagnostic Radiology by the American Board of Radiology, the American Osteopathic Board of Radiology, the Royal College of Physicians and Surgeons of Canada, or Le College des Medecins du Quebec and has performed the following procedures:

a. 100 peripheral diagnostic angiograms of which 10 should be renal arteriograms, and
b. 50 peripheral arterial interventions of which 10 should be renal PTA or stenting (bilateral may count as two cases), and
c. 10 peripheral arterial lysis procedures.

or

2. Successful completion of an Accreditation Council for Graduate Medical Education (ACGME) approved radiology residency training program or an American Osteopathic Association (AOA) approved residency program and/or interventional/vascular radiology fellowship, and must have a minimum of 6 months training on a service that is primarily responsible for the performance of percutaneous peripheral, visceral, and neurodiagnostic arteriography and vascular/interventional radiology. Documented formal training in the performance of invasive catheter angiographic procedures must be included. During this training, the physician should have performed the following procedures:
   a. 100 peripheral diagnostic angiograms of which 10 should be renal arteriograms, and
   b. 50 peripheral arterial interventions of which 10 should be renal PTA or stenting (bilateral may count as two cases), and
   c. 10 peripheral arterial lysis procedures.

or

3. Successful completion of an ACGME approved nonradiology residency or fellowship training and must have a minimum of 6 months of training on a service that is primarily responsible for the performance of percutaneous peripheral, visceral, or neurodiagnostic arteriography and vascular/interventional radiology. Documented formal training in the performance of invasive catheter arteriographic procedures must be included. During this training the physician should have performed the following procedures:
   a. 100 peripheral diagnostic angiograms of which 10 should be renal arteriograms, and
   b. 50 peripheral arterial interventions of which 10 should be renal PTA or stenting (bilateral may count as two cases), and
   c. 10 peripheral arterial lysis procedures.

or

4. Training by experience – physicians qualifying in this manner must meet the same training requirements as in 1 – 3 above by having performed the requisite number of procedures under the supervision of a physician who has previously met the training outlined above.

Note: For 1 – 4 above the physician must be primary operator for at least 50% of the above cases in all categories. These cases must be documented so that the director of the training program can certify that the physician is proficient in the performance of the procedures with acceptable success and complication rates within the quality assurance threshold rates laid out in this guideline [30].

5. Substantiation in writing by the director of interventional radiology or the chief of the department, of the institution in which the physician will be providing these services, that the physician is familiar with all of the following:
   a. Indications and contraindications for the procedure.
   b. Periprocedural and intraprocedural assessment, monitoring, and management of the patient.
   c. Where applicable, pharmacology of moderate or "conscious" sedation medications and recognition and treatment of adverse reactions and complications.
   d. Fluoroscopic and radiographic equipment, mechanical injectors, rapid film changers, digital subtraction, and other electronic imaging systems.
   e. Where applicable, principles of radiation protection, the hazards of radiation exposure to both patients and radiologic personnel, and monitoring requirements.
   f. Where applicable, pharmacology of contrast agents and recognition and treatment of potential adverse reactions.
   g. Percutaneous needle and catheter introduction techniques.
   h. Technical aspects of performing the procedure, including the use of alternative catheter and guide-wire systems, selective angiographic methods, appropriate injection rates and volumes of contrast media, and filming sequences.
   i. Recognition of periprocedural complications and knowledge of treatment options for these complications (e.g., stenting, embolization, thrombolysis, suction embolectomy, surgery).
   j. Anatomy, physiology, and pathophysiology of peripheral and visceral arterial vasculature.
   k. Interpretation of peripheral and visceral arteriographic studies.

Maintenance of Competence

Physicians must perform a sufficient number of renal stenting procedures to maintain their skills, with acceptable success and complication rates as laid out in this guideline. Continued competence should depend on participation in a quality improvement program that monitors these rates.

Continuing Medical Education

The physician's continuing education should be in accordance with the ACR Practice Guideline for Continuing Medical Education (CME).

B. Qualified Medical Physicist

A Qualified Medical Physicist is an individual who is competent to practice independently in one or more of the subfields in medical physics. The American College of Radiology considers that certification in the appropriate subfield(s) and continuing education demonstrates the competence of the individual. The ACR recommends that the individual be certified in the appropriate subfield(s) by the American Board of Radiology (ABR) or for MRI, by the American Board of Medical Physics (ABMP) in magnetic resonance imaging physics.

The appropriate subfields of medical physics for this guideline are Diagnostic Radiological Physics and Radiological Physics.

The continuing education of a Qualified Medical Physicist should be in accordance with the ACR Practice Guideline for Continuing Medical Education (CME). 2006 (Res. 16g)

C. Radiologist Assistant

A radiologist assistant is an advanced level radiographer who is certified and registered as a radiologist assistant by the American Registry of Radiologic Technologists (ARRT) after having successfully completed an advanced academic program encompassing an ACR/ASRT (American Society of Radiologic Technologists) radiologist assistant curriculum and a radiologist-directed clinical preceptorship. Under radiologist supervision, the radiologist assistant may perform patient assessment, patient management and selected examinations as delineated in the Joint Policy Statement of the ACR and the ASRT titled "Radiologist Assistant: Roles and Responsibilities" and as allowed by state law. The radiologist assistant transmits to the supervising radiologists those observations that have a bearing on diagnosis. Performance of diagnostic interpretations remains outside the scope of practice of the radiologist assistant. 2006 (Res. 34)

D. Radiologic Technologist

1. The technologist, together with the physician and nursing personnel, should have responsibility for patient comfort and safety. The technologist should be able to prepare and position[1] the patient for the procedure and, together with the nurse, monitor the patient during the examination. The technologist should obtain the imaging data in a manner prescribed by the supervising physician. If intravenous contrast material is to be administered, qualifications for technologists performing intravenous injection should be in compliance with the current ACR policy statement[2] and existing operating procedures or manuals at the interventional radiology facility and/or imaging facility. The technologist should also perform the regular quality control testing of the equipment under supervision of the physicist.
2. Technologists should be certified by the American Registry of Radiologic Technologists or have an unrestricted state license with documented training and experience in the imaging modality used for the imaging-guided percutaneous procedure.

E. Nursing Services

Nursing services, when deemed appropriate by the performing physician, are an integral part of the team for pre- and post-procedure patient management and education and are recommended in monitoring the patient during the procedure.

[1]The American College of Radiology approves of the practice of certified and/or licensed radiologic technologists performing fluoroscopy only as a positioning or localizing procedure and then only if monitored by a supervising physician who is personally and immediately available, and the positioning or localizing procedure must have prior written approval by the medical director of the radiology department/service and there be written authority, policy and procedures for designating radiologic technologists who perform such procedures. 1987, 1997 (Res. 1-E)

[2] The American College of Radiology approves of the injection of contrast material and diagnostic levels of radiopharmaceutical by certified and/or licensed radiologic technologists and radiologic nurses under the direction of a radiologist or his or her physician designee who is personally and immediately available, if the practice is in compliance with institutional and state regulations. There must be prior written approval by the medical director of the radiology department/service of such individuals; such approval process having followed established policies and procedures, and the radiologic technologists and radiologic nurses who have been so approved maintain documentation of continuing medical education related to the materials being injected and to the procedures being performed. 1987, 1997 (Res. 1-H)

VII. SPECIFICATIONS OF THE EXAMINATION

Several technical requirements are necessary to ensure safe and successful renal angiography, angioplasty, or stenting. These include adequate arteriographic equipment and institutional facilities, physiologic monitoring equipment, and support personnel.

A. Angiographic Equipment and Facilities

The following are considered the minimum equipment requirements for performing renal procedures. In planning facilities for these procedures, equipment and facilities more advanced than those outlined below may be desired to produce higher quality studies with reduced risk and time of study. In general, the facility should include at a minimum:

1. A high-resolution image intensifier and television chain with standard angiographic filming capabilities (including serial film changers, if necessary). Digital subtraction angiographic systems with high spatial resolution are recommended, as they allow for reduced volumes of contrast material and reduced examination times. These digital acquisition systems are sufficient to offer an alternative to conventional film systems and are more flexible and therefore preferable for safe and accurate renal interventional procedures. In accordance with the ALARA principle, a radiation dose measurement package including pulsed fluoroscopy and last image hold capabilities are recommended. The use of cineradiography or small field mobile image intensifiers is inappropriate for the routine recording of noncoronary angiography, because these methods have an unacceptably high patient and operator radiation dose.

2. Adequate angiographic supplies such as catheters, guidewires, stents, balloons, needles, and introducer sheaths.

3. An angiographic injector capable of varying injection volumes and rates with appropriate safety mechanisms to prevent overinjection.

4. An angiography suite large enough to allow easy transfer of the patient from the bed to the table and to allow room for the procedure table, monitoring equipment, and other hardware such as intravenous pumps, respirators, anesthesia equipment, and oxygen tanks. Ideally, there should be adequate space for the operating team to work unencumbered on either side of the patient and for the circulation of other technical staff in the room without contaminating the sterile conditions.

5. An area within the institution appropriate for patient preparation prior to the procedure and for observation of patients after the procedure. At this location, there should be personnel to provide care as outlined in Patient Care Section below, and there should be immediate access to emergency resuscitation equipment.

B. Physiologic Monitoring and Resuscitation Equipment

1. Sufficient equipment should be present in the angiography suite to allow for monitoring the patient's heart rate, cardiac rhythm, and blood pressure. For facilities using moderate sedation, a pulse oximeter or an end-tidal carbon dioxide monitor should be available. (See the ACR Practice Guideline for Adult Sedation/ Analgesia.)

2. There should be ready access to emergency resuscitation equipment and drugs, to include the following: an emergency defibrillator, oxygen supply and appropriate tubing and delivery systems, suction equipment, tubes for endotracheal intubation, laryngoscope, ventilation bag-mask-valve apparatus, and central venous line sets. Drugs for treating cardiopulmonary arrest, contrast reaction, vasovagal reactions, narcotic or benzodiazepine overdose, bradycardia, and ventricular arrhythmias should also be readily available.

3. Equipment for invasive pressure monitoring.

C. Support Personnel

1. Radiologic technologists properly trained in the use of the diagnostic imaging equipment should assist in performing and imaging the procedure. They should demonstrate appropriate knowledge of patient positioning, arteriographic image recording, angiographic contrast injectors, adjunctive supplies, and the physiologic monitoring equipment. Certification as a vascular and interventional radiologic technologist is one measure of appropriate training. The technologists should be trained in basic cardiopulmonary resuscitation and in the function of the resuscitation equipment.

2. If the patient does not receive moderate sedation, one of the staff assisting the procedure should be assigned to periodically assess the patient's status. If the patient is to undergo moderate sedation, a nurse or other appropriately trained individual should monitor the patient as his/her primary responsibility. This person should maintain a record of the patient's vital signs, time and dose of medications given, and other pertinent information. Nursing personnel should

be qualified to administer moderate sedation. (See the ACR Practice Guideline for Adult Sedation/Analgesia.)

3. For unstable patients additional support may be necessary to ensure the safe performance of renal interventional procedures. The primary operator may be engaged in the details of the proper performance of the renal interventional procedures. Therefore, appropriate personnel should be available to attend to the ongoing care and resuscitation of the critically ill patients. Such personnel might include anesthesiologists; operating room, ICU, and/or ER trained nurses; or other physicians. The nurses may be radiology nurses and/or the same personnel responsible for monitoring and maintaining moderate sedation as discussed immediately above. Alternatively, the nurses may be supplied from other patient care units in the facility.

   All such additional personnel should work in concert with and under the overall supervision of the primary operator performing the renal interventional procedures, but within the scopes of service as defined by their professions, state regulations, and institutional guidelines.

   (For a more comprehensive description of the angiographic facility, see the Intersociety paper on optimum resources for endovascular treatment in Circulation 1994; 89:1481-1493.)

D. Surgical Support

Although surgical or other emergency treatment is needed infrequently for serious complications after renal interventional procedures, there should be prompt access to surgical and interventional equipment and specialists familiar with the management of patients with complications in the unlikely event of a life-threatening complication.

VIII. PATIENT CARE

The written or electronic request for an examination for diagnosis and treatment of renal artery stenosis should provide sufficient information to demonstrate the medical necessity of the examination and allow for the proper performance and interpretation of the examination.

Documentation that satisfies medical necessity includes 1) signs and symptoms and/or 2) relevant history (including known diagnoses). The provision of additional information regarding the specific reason for the examination or a provisional diagnosis would be helpful and may at times be needed to allow for the proper performance and interpretation of the examination.

The request for the examination must be originated by a physician or other appropriately licensed health care provider. The accompanying clinical information should be provided by a physician or other appropriately licensed health care provider familiar with the patient's clinical problem or question and consistent with the state scope of practice requirements. 2006 (Res. 35)

A. Preprocedure Care

The physician performing the procedure must have knowledge of the following:

1. Clinically significant history, including indications for the procedure.

2. Clinically significant physical or diagnostic examination, including knowledge and awareness of other clinical or medical conditions that may necessitate specific care, and certain diagnostic lab results.

3. Possible alternative methods, such as surgical or medical treatments, to obtain the desired therapeutic result.

Informed consent must be in compliance with all state laws and the ACR Practice Guideline on Informed Consent for Image Guided Procedures.

B. Procedural Care

1. Adherence to the JCAHO Universal Protocol for Preventing Wrong Site, Wrong Procedure, Wrong Person Surgery™ is required for procedures in non-operating room settings including bedside procedures. "Time out" must be conducted in the location where the procedure will be done, just before starting the procedure and must:
   - Involve the entire operative team.
   - Use active communication.
   - Be briefly documented, such as in a checklist, and
   - At the least, include:
     - Correct patient identity.
     - Correct side and site.
     - Agreement on the procedure to be done.
     - Correct patient position.
     - Availability of correct implants and any special equipment or special requirements.

   The organization should have processes and systems in place for reconciling differences in staff responses during the "time out."

2. The physician performing fluoroscopy should have knowledge of exposure factors, including kVp, mA, magnification factor, and dose rate,

and should consider additional parameters such as collimation, field of view, and last image hold.

3. Nursing personnel, technologists, and those directly involved in the care of patients undergoing renal interventional procedures should have protocols for use in standardizing care. These should include, but are not limited to, the following:
   a. Equipment needed for the procedure.
   b. Patient monitoring.

   Protocols should be reviewed and updated periodically.

During the use of fluoroscopy, the physician should use exposure factors consistent with the as-low-as-reasonably-achievable (ALARA) radiation safety guidelines.

C. Postprocedure Care

1. A procedure note should be written in the patient's chart summarizing the major findings of the study and any immediate complications. This note may be brief if a formal report will be available within a few hours. However, if the typed report is not likely to be on the chart the same day, a more detailed summary of the study should be written in the chart at the conclusion of the procedure. In all cases, pertinent findings should be communicated to the referring physician in a timely manner.

2. All patients should be at bed rest and observed in the initial postprocedure period. The length of this period of bed rest will depend on the patient's medical condition.

3. During the initial postprocedure period, skilled nurses or other appropriately trained personnel should periodically monitor the puncture site and the status of the patient.

4. The patient should be monitored for urinary output, cardiac symptoms, pain, and other indicators of systemic complications that may necessitate overnight care.

5. The operating physician or a qualified designee should evaluate the patient after the procedure, and these findings should be summarized in a progress note. If moderate sedation was administered prior to and during the procedure, recovery from the sedation must be documented. The physician or designee should be available for continuing care during hospitalization and after discharge. The designee may be another physician or a nurse.

IX. DOCUMENTATION

Reporting should be in accordance with the Practice Guideline for Reporting and Archiving of Interventional Radiology Procedures.

X. RADIATION SAFETY IN IMAGING

Radiologists, radiologic technologists, and all supervising physicians have a responsibility to minimize radiation dose to individual patients, to staff, and to society as a whole, while maintaining the necessary diagnostic image quality. This is the concept "As Low As Reasonably Achievable (ALARA)."

Facilities, in consultation with the medical physicist, should have in place and should adhere to policies and procedures, in accordance with ALARA, to vary examination protocols to take into account patient body habitus, such as height and/or weight, body mass index or lateral width. The dose reduction devices that are available on imaging equipment should be active or manual techniques should be used to moderate the exposure while maintaining the necessary diagnostic image quality. Patient radiation doses should be periodically measured by a medical physicist in accordance with the appropriate ACR Technical Standard. 2006 (Res. 17)

XI. QUALITY CONTROL AND IMPROVEMENT, SAFETY, INFECTION CONTROL, AND PATIENT EDUCATION CONCERNS

Policies and procedures related to quality, patient education, infection control and safety should be developed and implemented in accordance with the ACR Policy on Quality Control and Improvement, Safety, Infection Control, and Patient Education Concerns appearing elsewhere in the ACR Practice Guidelines and Technical Standards book.

XII. QUALITY IMPROVEMENT

These guidelines are to be used in quality improvement (QI) programs to assess the diagnosis and treatment of renal artery stenosis. The most important processes of care are 1) patient selection, 2) performance of the procedure, and 3) monitoring the patient. The outcome measures or indicators for these processes are indications, success rates, and complication rates. Outcome measures are assigned threshold levels.

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 both the evaluation and treatment, is appropriate for the radiologist.

While practicing physicians should strive to achieve perfect outcomes (e.g., 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 efficacy 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 refer to a group of indicators for a procedure, e.g., 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 stenting of renal artery stenosis, then values in excess of the defined threshold 6% should trigger a review of policies and procedures within the department to determine the causes and to 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. 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.

A. Indications for Catheter-Directed Angiography for Evaluating Renal Artery Stenosis (Threshold – 95%)

Clinical signs of renovascular hypertension, ischemic nephropathy, or a cardiac disturbance syndrome are present, and at least one of the following:

1. Noninvasive vascular imaging suggests that a renal artery stenosis great than 50% is present.

2. Progression to a hemodynamically significant renal artery stenosis is indicated by noninvasive vascular imaging.

3. Noninvasive vascular imaging is technically inadequate, equivocal, or cannot be obtained.

4. Onset of hypertension occurs in a patient under the age of 30.

5. Renal artery fibromuscular dysplasia is suspected as the etiology of renal artery stenosis.

6. There is recent onset of hypertension in a patient age 60 or older.

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.

B. Indications for Angioplasty of a Hemodynamically Significant Renal Artery Stenosis (Threshold – 95%)

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,” i.e., there is sudden worsening of previously controlled hypertension.
   d. Hypertension is “malignant,” i.e., is associated with end-organ damage such as left ventricular hypertrophy, congestive heart failure or visual or neurological disturbance, grade III-IV retinopathy.
   e. The patient is intolerant of or not complying 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.

C. Indications for Renal Artery Stent Deployment (Threshold – 95%)

1. Failure at attain a satisfactory result by renal artery angioplasty as determined by:
   a. Greater than 30% stenosis of the luminal diameter, measured from the outer margins of intimal fissures, following balloon angioplasty.
   b. Failure to eliminate a hemodynamically significant pressure gradient.
   c. Presence of a flow limiting dissection of the renal artery.

2. Stenosis of the ostium of a renal artery whose normal diameter is 5 mm or greater.

3. Restenosis of a lesion that was successfully treated by balloon angioplasty in the past.

D. Relative Contraindications for Renal Artery Stent Deployment (Threshold – 5%)

1. An inelastic stenosis that cannot be reduced to less than 50% by balloon angioplasty.

2. The presence of sepsis.

3. If the stent would preclude surgical salvage should restenosis occur, i.e., isolation of branch arteries.

4. For stenosis of an artery measuring 4 mm or less.

SUCCESS:

Threshold for Technical Success for Percutaneous Renal Revascularization - 90%

Complications can be 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 A. The complication rates and thresholds below refer to *major* complications.

Specific Major Complications from Percutaneous Renal Revascularization

	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 prolonging hospital stay	5%	5%
Acute renal failure	2%	2%
Worsening of chronic renal failure requiring an increase in the level of care	2%	5%

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. Generally the complication-specific thresholds should be set higher than the complication-specific reported rates listed above. 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 volume (e.g., early in a quality improvement program). In this situation, the overall procedure threshold is more appropriate for use in a quality-improvement program.

In the above table, all values were supported by the weight of literature evidence and panel consensus.

Overall threshold for major complications of percutaneous renal revascularization - 10%

ACKNOWLEDGEMENTS

This guideline was developed according to the process described in the ACR Practice Guidelines and Technical Standards book by the Guidelines and Standards Committee of the Cardiovascular and Interventional Radiology Commission with the collaboration of the Society of Interventional Radiology (SIR).

Principal Drafter: Louis G. Martin, MD

ACR Guidelines and Standards Committee
Curtis A. Lewis, MD, Chair
Curtis Bakal, MD
Daniel B. Brown, MD
John F. Cardella, MD
Kyran Dowling, MD

Clement J. Grassi, MD
Patrick C. Malloy, MD
Louis G. Martin, MD
Calvin D. Neithamer, Jr., MD
Reed A. Omary, MD
David Sacks, MD
Richard B. Towbin, MD
Anthony C. Venbrux, MD

Matthew A. Mauro, MD, Chair, Commission
Anne C. Roberts, MD, Chair, CSC Subcommittee

REFERENCES

1. Burt VL, Cutler JA, Higgins M, et al. Trends in the prevalence, awareness, treatment, and control of hypertension in the adult U.S. population: data from the health examination surveys, 1960 to 1991. *Hypertension* 1995;26:60-69.
2. The sixth report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (published erratum appears in Arch Intern Med 1998 [Mar 23]; 158:573). *Arch Intern Med* 1997;157:2413-2446.
3. 1999 World Health Organization's International Society of Hypertension Guidelines for the Management of Hypertension. Guidelines subcommittee. *J Hypertens* 1999;17:151-183.
4. Sacks D, Marinelli DL, Martin LG, et al. Reporting standards for clinical evaluation of new peripheral arterial revascularization devices. Technology assessment committee. *J Vasc Interv Radiol* 1997;8:137-149.
5. Kaatee R, Beek FJ, Verschuyl EJ, et al. Atherosclerotic renal artery stenosis: ostial or truncal? *Radiology* 1996;199:637-640.
6. Khosla S, White CJ, Collins TJ, et al. 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.
7. Bloch MJ, Trost DW, Pickering TG, et al. Prevention of recurrent pulmonary edema in patients with bilateral renovascular disease through renal artery stent placement. *Am J Hypertens* 1999;12:1-7.
8. Caps MT, Perissinotto C, Zierler RE, et al. Prospective study of atherosclerotic disease progression in the renal artery. *Circulation* 1998;98:2866-2872.
9. Crowley JJ, Santos RM, Peter RH, et al. Progression of renal artery stenosis in patients undergoing cardiac catheterization. *Am Heart J* 1998;136:913-918.
10. Schreiber MJ, Pohl MA, Novick AC. The natural history of atherosclerotic and fibrous renal artery disease. *Urol Clin North Am* 1984;11:383-392.
11. Kohler TR. Hemodynamics of arterial occlusive disease; In: Strandness DE Jr, van Breda A, eds. *Vascular Diseases: Surgical and Interventional Therapy*. 1st edition. New York, NY: Churchill Livingstone; 1994:65-71.
12. Imanishi M, Akabane S, Takamiya M, et al. Critical degree of renal arterial stenosis that causes hypertension in dogs. *Angiology* 1992;43:833-842.
13. Simon G. What is critical renal artery stenosis? Implications for treatment. *Am J Hypertens* 2000;13:1189-1193.
14. May AG, van de Berg L, De Weese JA, et al. Critical arterial stenosis. *Surgery* 1963;54:250-259.
15. Haimovici H, Zinicola N. Experimental renal artery stenosis diagnostic significance of arterial hemodynamics. *J Cardiovascular Surg* 1962;3:259-262.
16. Pemsel HK, Thermann M. The haemodynamic effects of renal artery stenosis (author's translation). *ROFO Fortschr Geb Rontgenstr Nuklearmed* 1978;129:189-192.
17. Paul JF, Cherrak I, Jaulent MC, et al. Interobserver variability in the interpretation of renal digital subtraction angiography. *AJR* 1999;173:1285-1288.
18. Archie JP, Jr. Analysis and comparison of pressure gradients and ratios for predicting iliac stenosis. *Ann Vasc Surg* 1994;8:271-280.
19. Udoff EJ, Barth KH, Harrington DP, et al. Hemodynamic significance of iliac artery stenosis: pressure measurements during angiography. *Radiology* 1979;132:289-293.
20. Tetteroo E, Haaring C, van der Graaf Y, et al. Intra-arterial pressure gradients after randomized angioplasty or stenting of iliac artery lesions. Dutch Iliac Stent Trial Study Group. *Cardiovasc Intervent Radiol* 1996;19:411-417.
21. 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.
22. 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.
23. DeBruyne B, Pijls NH, Heyndrickx GR, et al. Pressure-derived fractional flow reserve to assess serial epicardial stenosis: theoretical basis and animal validation. *Circulation* 2000;101:1840-1847.
24. 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.
25. 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.
26. 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.
27. Harward TR, Poindexter B, Huber TS, et al. Selection of patients for renal artery repair using captopril testing. *Am J Surg* 1995;170:183-187.
28. Setaro JF, Chen CC, Hoffer PB, et al. Captopril renography in the diagnosis of renal artery stenosis and the prediction of improvement with revascularization: the Yale Vascular Center experience. *Am J Hypertens* 1991;4:698S-6705S.
29. Radermacher J, Chavan A, Bleck J, et al. Use of Doppler ultrasonography to predict the outcome of therapy for renal-artery stenosis. *N Engl J Med* 2001;344:410-417.
30. van Jaarsveld BC, Krijnen P, Derkx FH, et al. The place of renal scintigraphy in the diagnosis of renal artery stenosis: fifteen years of clinical experience. *Arch Intern Med* 1997;157:1226-1234.
31. Martin LG, Cork RD, Wells JO. Renal vein renin analysis: limitations of its use in predicting benefit from percutaneous angioplasty. *Cardiovasc Intervent Radiol* 1993;16:76-80.
32. Zierler RE, Bergelin RO, Davidson RC, et al. A prospective study of disease progression in patients with atherosclerotic renal artery stenosis. *Am J Hypertens* 1996;9:1055-1061.
33. 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 Suppl* 1998;16:S21-S27.
34. 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.
35. Tollefson DF, Ernst CB. Natural history of atherosclerotic renal artery stenosis associated with aortic disease. *J Vasc Surg* 1991;14:327-331.
36. Caps MT, Zierler RE, Polissar NL, et al. Risk of atrophy in kidneys with atherosclerotic renal artery stenosis. *Kidney Int* 1998;53:735-742.
37. Guzman RP, Zierler RE, Isaacson JA, et al. Renal atrophy and arterial stenosis: a prospective study with duplex ultrasound. *Hypertension* 1994;23:346-350.
38. Watson PS, Hadjipetrou P, Cox SV, et al. Effect of renal artery stenting on renal function and size in patients with atherosclerotic renovascular disease. *Circulation* 2000;102:1671-1677.
39. Chaikof EL, Smith RB 3rd, Salam AA, et al. Empirical reconstruction of the renal artery: long-term outcome. *J Vasc Surg* 1996;24:406-414.
40. Chabova V, Schirger A, Stanson AW, et al. Outcomes of atherosclerotic renal artery stenosis managed without revascularization. *Mayo Clin Proc* 2000;75:437-444.
41. Anderson GH Jr., Blakeman N, Streeten DH. The effect of age on prevalence of secondary forms of hypertension in 4,429 consecutively referred patients. *J Hypertens* 1994;12:609-615.
42. Simon N, Franklin SS, Bleifer KH, et al. Clinical characteristics of renovascular hypertension. *JAMA* 1972;220:1209-1218.
43. Krijnen P, van Jaarsveld BC, Steyerberg EW, et al. A clinical prediction rule for renal artery stenosis. *Ann Intern Med* 1998;129:705-711.
44. Albers FJ. Clinical characteristics of atherosclerotic renovascular disease. *Am J Kidney Dis* 1994;24:636-641.
45. Roubidoux MA, Dunnick NR, Klotman PE, et al. Renal vein renins: inability to predict response to revascularization in patients with hypertension. *Radiology* 1991;178:819-822.
46. Mann SJ, Pickering TG. Detection of renovascular hypertension: state of the art. *Ann Intern Med* 1992;117:845-853.
47. Bongers V, Bakker J, Beutler JJ, et al. Assessment of renal artery stenosis: comparison of captopril renography and gadolinium-enhanced breath-hold MR angiography. *Clin Radiol* 2000;55:346-353.
48. 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.
49. Wilcox CS. Ischemic nephropathy: noninvasive testing. *Semin Nephrol* 1996;16:43-52.
50. O'Neil EA, Hansen KJ, Canzanello VJ, et al. Prevalence of ischemic nephropathy in patients with renal insufficiency. *Am Surg* 1992;58:485-490.
51. Hall KA, Wong RW, Hunter GC, et al. Contrast-induced nephrotoxicity: the effects of vasodilator therapy. *J Surg Res* 1992;53:317-320.
52. Novick AC. Atherosclerotic ischemic nephropathy: epidemiology and clinical considerations. *Urol Clin North Am* 1994;21:195-200.
53. 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.
54. Martin LG, Casarella WJ, Gaylord GM. Azotemia caused by renal artery stenosis: treatment by percutaneous angioplasty. *AJR* 1988;150:839-844.
55. 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.
56. Conlon PJ, Athirakul K, Kovalik E, et al. Survival in renal vascular disease. *J Am Soc Nephrol* 1998;9:252-256.
57. Moore MA, Epstein M, Agodoa L, et al. Current strategies for management of hypertensive renal disease. *Arch Intern Med* 1999;159:23-28.
58. 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.

59. Kostis JB, Davis BR, Cutler J, et al. Prevention of heart failure by antihypertensive drug treatment in older persons with isolated systolic hypertension. SHEP Cooperative Research Group. *JAMA* 1997;278:212-216.
60. Petrovitch H, Vogt TM, Berge KG. Isolated systolic hypertension: lowering the risk of stroke in older patients. SHEP Cooperative Research Group. *Geriatrics* 1992;47:30-32, 35-38.
61. Prevention of stroke by antihypertensive drug treatment in older persons with isolated systolic hypertension. Final results of the Systolic Hypertension in the Elderly Program (SHEP). SHEP Cooperative Research Group. *JAMA* 1991;265:3255-3264.
62. Glynn RJ, Field TS, Rosner B, et al. Evidence for a positive linear relation between blood pressure and mortality in elderly people. *Lancet* 1995:345:825-829.
63. Rees CR. Stents for atherosclerotic renovascular disease [published erratum appears in J Vasc Interv Radiol 1999; 10:1118]. *J Vasc Interv Radiol* 1999;10:689-705.
64. 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.
65. Baumgartner I, von Aesch K, Do DD, et al. Stent placement in ostial and nonostial atherosclerotic renal arterial stenosis: a prospective follow-up study. *Radiology* 2000;216:498-505.
66. Maxwell MH, Bleifer KH, Franklin SS, et al. Cooperative study of renovascular hypertension: demographic analysis of the study. *JAMA* 1972;220:1195-1204.
67. Tegtmeyer CJ, Kellum CD, Ayers C. Percutaneous transluminal angioplasty of the renal artery: results and long-term follow-up. *Radiology* 1984;153:77-84.
68. Martin EC, Mattern RF, Baer L, et al. Renal angioplasty for hypertension: predictive factors for long-term success. *AJR* 1981;137:921-924.
69. Greminger P, Steiner A, Schneider E, et al. Cure and improvement of renovascular hypertension after percutaneous transluminal angioplasty of renal artery stenosis. *Nephrom* 1989;51:362-366.
70. Isles CG, Robertson S, Hill D. Management of renovascular disease: a review of renal artery stenting in ten studies. *QJM* 1999;92:159-167.
71. Kuhn FP, Kutkuhn B, Torsello G, et al. Renal artery stenosis: preliminary results of treatment with the Strecker stent. *Radiology* 1991;180:367-372.
72. van de Ven PJ, Beutler JJ, Kaatee R, et al. Transluminal vascular stent for ostial atherosclerotic renal artery stenosis. *Lancet* 1995;346:672-674.
73. 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.
74. Martin LG, Rees CR, O'Bryant T. Percutaneous angioplasty of the renal arteries. In: Strandness DE Jr., van Breda A, eds. *Vascular Diseases: Surgical and Interventional Therapy*. 1st edition. New York, NY: Churchill Livingston, Inc; 1994:721-742.
75. Harrison EG Jr., McCormack LJ. Pathologic classification of renal arterial disease in renovascular hypertension. *Mayo Clin Proc* 1971;46:161-167.
76. Cluzel P, Raynaud A, Beyssen B, et al. Stenoses of renal branch arteries in fibromuscular dysplasia: results of percutaneous transluminal angioplasty. *Radiology* 1994;193:227-232.
77. Martin LG. Renal revascularization using percutaneous balloon angioplasty for fibromuscular dysplasia and atherosclerotic disease. In: Calligaro KD, Doughtery MJ, eds. *Modern Management of Renovascular Hypertension and Renal Salvage*. Philadelphia, Pa: Williams and Wilkins; 1996:125-144.
78. Davidson RA, Barri Y, Wilcox CS. Predictors of cure of hypertension in fibromuscular renovascular disease. *Am J Kidney* Dis 1996;28:334-338.
79. 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;44:109-114.
80. Plouin PF, Chatellier G, Darne B, et al. 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.
81. Webster J, Marshall F, Abdalla M, et al. Randomized 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.
82. Textor SC. Revascularization in atherosclerotic renal artery disease. *Kidney Int* 1998;53:799-811.
83. Ritz E, Mann JF. Renal angioplasty for lowering blood pressure. *N Engl J Med* 2000;342:1042-1043.
84. Sacks D, Rundback JH, Martin LG. Renal angioplasty/stent placement and hypertension in the year 2000. *J Vasc Interv Radiol* 2000;11:949-953.
85. Zuccala A, Zucchelli P, Campieri C, et al. 3 R study: renal outcome in renal ischemia: revascularization or medical treatment. *J Nephrol* 2000;13:106-109.
86. Dean RH, Tribble RW, Hansen KJ, et al. Evolution of renal insufficiency in ischemic nephropathy. *Ann Surg* 1991;213:446-455; discussion 455-456.
87. Butterly DW, Schwab SJ. Renal artery stenosis: the case for conservative management. *Mayo Clin Proc* 2000;75:435-436.

88. Textor SC. Atherosclerotic renovascular disease as a cause of end-stage renal disease: cost consideration. *Blood Purif* 1996;14:305-314.
89. Appel RG, Bleyer AJ, Reavis S, et al. Renovascular disease in older patients beginning renal replacement therapy. *Kidney Int* 1995;48:171-176.
90. Chaikof EL, Smith RB 3rd, Salam AA, et al. Ischemic nephropathy and concomitant aortic disease: a ten-year experience. *J Vasc Surg* 1994;19:135-146; discussion 146-148.
91. Vinge E, Lindergard B, Nilsson-Ehle P, et al. Relationships among serum cystatin C, serum creatinine, lean tissue mass and glomerular filtration rate in healthy adults. *Scand J Clin Lab Invest* 1999;59:587-592.
92. Rahn KH, Heidenreich S, Bruchner D. How to assess glomerular function and damage in humans. *J Hypertens* 1999;17:309-317.
93. Spinler SA, Nawarskas JJ, Boyce EG, et al. Predictive performance of ten equations for estimating creatinine clearance in cardiac patients. Iohexol Cooperative Study Group. *Ann Pharmacother* 1998;32:1275-1283.
94. Mitch WE, Walser M, Buffington GA, et al. A simple method of estimating progression of chronic renal failure. *Lancet* 1976;2:1326-1328.
95. Rowe PA, Richardson RE, Burton PR, et al. Analysis of reciprocal creatinine plots by two-phrase linear regression. *Am J Nephrol* 1989;9:38-43.
96. Mitch WE. The influence of the diet on the progression of renal insufficiency. *Annual Rev Med* 1984;35:249-264.
97. Harden PN, MacLeod MJ, Rodger RS, et al. Effect of renal-artery stenting on progression of renovascular renal failure. *Lancet* 1997;349:1133-1136.
98. van Rooden CJ, van Bockel JH, DeBacker GG, et al. Long-term outcome of surgical revascularization in ischemic nephropathy: normalization of average decline in renal function. *J Vasc Surg* 1999;29:1037-1049.
99. Novick AC, Pohl MA, Schreiber M, et al. Revascularization for preservation of renal function in patients with atherosclerotic renovascular disease. *J Urol* 1983;129:907-912.
100. van Damme H, Jeusette F, Pans A, et al. The impact of renal revascularization on renal dysfunction. *Eur J Vasc Endovasc Surg* 1995;10:330-337.
101. 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; discussion 380-382.
102. Randers E, Erlandsen EJ, Pedersen OL, et al. Serum cystatin C as an endogenous parameter of the renal function in patients with normal to moderately impaired kidney function. *Clin Nephrol* 2000;54:203-209.
103. Deitch JS, Hansen KJ, Regan JD, et al. Infected renal artery pseudoaneurysm and mycotic aortic aneurysm after percutaneous transluminal renal artery angioplasty and stent placement in a patient with a solitary kidney. *J Vasc Surg* 1998;28:340-344.
104. Jaff MR. Management of atherosclerotic renal artery stenosis: interventional versus medical therapy. *Curr Interv Cardiol Rep* 2001;3:93-99.
105. Messina LM, Zelenock GB, Yao KA, et al. 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; discussion 80-82.
106. Bush RL, Martin LG, Lin PH, et al. Endovascular revascularization of renal artery stenosis in the solitary functioning kidney. *Ann Vasc Surg* 2001;15:60-66.
107. Beek FJ, Kaatee R, Beutler JJ, et al. Complications during renal artery stent placement for atherosclerotic ostial stenosis. *Cardiovasc Intervent Radiol* 1997;20:184-190.
108. 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.
109. Martin LG, Casarella WJ, Alspaugh JP, et al. Renal artery angioplasty: increased technical success and decreased complications in the second 100 patients. *Radiology* 1986;159:631-634.

APPENDIX A

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.