

ACR–SIR PRACTICE PARAMETER FOR THE PERFORMANCE OF ANGIOGRAPHY, ANGIOPLASTY, AND STENTING FOR THE DIAGNOSIS AND TREATMENT OF RENAL ARTERY STENOSIS IN ADULTS

The American College of Radiology, with more than 40,000 members, is the principal organization of radiologists, radiation oncologists, and clinical medical physicists in the United States. The College is a nonprofit professional society whose primary purposes are to advance the science of radiology, improve radiologic services to the patient, study the socioeconomic aspects of the practice of radiology, and encourage continuing education for radiologists, radiation oncologists, medical physicists, and persons practicing in allied professional fields.

The American College of Radiology will periodically define new practice parameters and technical standards for radiologic practice to help advance the science of radiology and to improve the quality of service to patients throughout the United States. Existing practice parameters and technical standards will be reviewed for revision or renewal, as appropriate, on their fifth anniversary or sooner, if indicated.

Each practice parameter and technical standard, representing a policy statement by the College, has undergone a thorough consensus process in which it has been subjected to extensive review and approval. The practice parameters and technical standards recognize that the safe and effective use of diagnostic and therapeutic radiology requires specific training, skills, and techniques, as described in each document. Reproduction or modification of the published practice parameter and technical standard by those entities not providing these services is not authorized.

PREAMBLE

This document is an educational tool designed to assist practitioners in providing appropriate radiologic care for patients. Practice Parameters and Technical Standards are not inflexible rules or requirements of practice and are not intended, nor should they be used, to establish a legal standard of care¹. For these reasons and those set forth below, the American College of Radiology and our collaborating medical specialty societies caution against the use of these documents 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 practitioner considering all the circumstances presented. Thus, an approach that differs from the guidance in this document, 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 this document when, in the reasonable judgment of the practitioner, such course of action is indicated by variables such as the condition of the patient, limitations of available resources, or advances in knowledge or technology after publication of this document. However, a practitioner who employs an approach substantially different from the guidance in this document may consider documenting in the patient record information sufficient to explain the approach taken.

The practice of medicine involves the science, and 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 the guidance in this document 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 purpose of this document is to assist practitioners in achieving this objective.

¹ *Iowa Medical Society and Iowa Society of Anesthesiologists v. Iowa Board of Nursing*, 831 N.W.2d 826 (Iowa 2013) Iowa Supreme Court refuses to find that the "ACR Technical Standard for Management of the Use of Radiation in Fluoroscopic Procedures (Revised 2008)" sets a national standard for who may perform fluoroscopic procedures in light of the standard's stated purpose that ACR standards are educational tools and not intended to establish a legal standard of care. See also, *Stanley v. McCarver*, 63 P.3d 1076 (Ariz. App. 2003) where in a concurring opinion the Court stated that "published standards or guidelines of specialty medical organizations are useful in determining the duty owed or the standard of care applicable in a given situation" even though ACR standards themselves do not establish the standard of care.

I. INTRODUCTION

This practice parameter was revised collaboratively by the American College of Radiology (ACR) and the Society of Interventional Radiology (SIR).

Hypertension (HTN) is a common problem, up to 45% of adults in the United States [1,2]. If poorly controlled,

HTN can lead to morbidity and mortality and result in significant end-organ damage, frequently affecting the kidneys and the cerebrovascular and cardiovascular systems. HTN is most often essential or idiopathic in origin. However, a subset of patients with diminished arterial perfusion to the kidney(s), and what is termed renovascular hypertension (RVH), have a potentially treatable and reversible cause for hypertension [3,4]. The incidence of RVH varies in the literature from 0% to 29%, with a weighted mean of about 4% in an analysis of 12 studies and 8,899 patients [5].

Renal artery stenosis (RAS) can be caused by different etiologies and may result in or lead to HTN, renal insufficiency, or no symptoms at all. The incidence of atherosclerotic RAS (ARAS) increases with age and the presence of associated cardiovascular risk factors [5]. For instance, the prevalence of ARAS in a 65-year-old patient with no cardiovascular disease risk factors is about 2%; whereas, in a similarly aged patient with cardiovascular disease, the prevalence of ARAS may be as high as 40% [6,7]. Many patients with severe HTN and/or chronic renal insufficiency (CRI) and ARAS do not necessarily have a component of RVH contributing to the HTN or CRI [8-10]. However, certain clinical scenarios significantly increase the likelihood that the ARAS is contributing to the HTN and/or CRI (eg, abrupt onset of labile or poorly controlled HTN in a patient older than 55 years of age, sudden worsening of stable HTN or CRI, and/or episodes of acute onset of congestive heart failure despite normal left ventricular heart function, which is known as a cardiac disturbance syndrome) [4,7,9]. However, trying to prospectively identify older patients with ARAS who have RVH as a contributing factor to HTN and/or CRI is challenging [9]. Therefore, performing an effective vascular assessment in a patient with an ARAS who has HTN and/or CRI requires an understanding of the pathophysiology of RVH, the most appropriate screening imaging and laboratory tests, and the indications for catheter-based diagnostic angiography and a renal artery intervention [4,8].

This document reviews the literature and circumstances that should prompt further evaluation of a patient with RAS as a potential cause for RVH or contributing factor to CRI (due to renal ischemia) and/or a cardiac disturbance syndrome. It also discusses both the noninvasive imaging and catheter-based angiographic evaluation of such patients and the criteria for determining whether an endovascular intervention has been successful. Practice parameters for the training and ongoing credentialing of practitioners performing catheter-based angiography and endovascular interventions are also presented.

For additional information on Definitions, see Appendix A, and for Methods, see Appendix B.

II. INDICATIONS FOR RENAL VASCULAR IMAGING OR ANGIOGRAPHY

Recent randomized trials have raised significant doubts about the clinical role of percutaneous transluminal renal angioplasty (PTRA) and/or renal artery stent therapy (RAST) for the treatment of ARAS in patients without clear pathophysiological evidence for RVH. Laboratory tests, noninvasive imaging, and invasive diagnostic evaluation may help to better define the physiological significance of a RAS and provide the needed information on how best to manage a patient with RAS [4,10-21]. Clinical features suggestive of RVH were first enumerated by the Cooperative Study of Renovascular Hypertension in 1972 [22] and have been recently updated [23-26]. The indications for evaluating a patient with HTN for the presence of RAS have historically included:

- Onset of HTN before the age of 30, especially in patients without a family history of HTN and where fibromuscular dysplasia or a vasculitis may be a consideration
- New onset of difficult to control or labile hypertension after the age of 55
- The presence of an abdominal bruit, particularly if it continues into diastole and the bruit lateralizes to one side of the abdomen
- Accelerated or resistant HTN as defined by failure to obtain adequate blood pressure control on 3 antihypertensive medications, including one diuretic
- Recurrent episodes of sudden onset of congestive heart failure, especially in patients with normal left ventricular function (eg, also known as a cardiac disturbance syndrome)
- Renal failure of uncertain cause, especially with a normal urinary sediment and less than 1 gm of urinary protein per day
- Coexisting, diffuse atherosclerotic vascular disease, especially in heavy smokers
- Acute renal failure that is precipitated or exacerbated by the use of an angiotensin-converting enzyme (ACE) inhibitor or angiotensin receptor blocker (ARB)

- Malignant or HTN that is very difficult to control with a unilateral small kidney
- HTN associated with medication intolerance

The Joint National Committee (JNC) 8 recommends treating hypertension to a goal of 150/90 mm Hg or less in those 60 years and older and with a goal of 140/90 mm Hg in individuals between the ages of 18 and 60 and in all patients with diabetes mellitus or chronic kidney disease (CKD), regardless of age [27], but makes no recommendations with regard to renal artery imaging for evaluation for RVH. The 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults [2] considers medical therapy to be the primary treatment of choice, with PTRAs and/or RAST reserved for patients with refractory HTN or progressive CRI without significant proteinuria and with a hemodynamically significant RAS [4,11,16,17,19,20,28,29].

"Any antihypertensive treatment regimen that effectively lowers blood pressure is associated with slowed progression of renal failure and improved cardiovascular survival" [30]. Prior to referral for RAS imaging, appropriate diligence is needed in reviewing the blood pressure history and what medication combinations have been utilized in an attempt to control HTN [27]. In particular, the history of ACE inhibitor usage and clinical response to ACE inhibitors are used in determining whether renal artery imaging is needed. The use of ACE inhibitors or ARBs in the setting of a significant RAS may cause a decrease in renal function [31,32].

Renal artery imaging should be performed to exclude stenosis as the etiology of unexplained new renal failure associated with initiating ACE inhibitors or ARBs [30]. Depending upon the clinical scenario, noninvasive evaluation may consist of imaging as outlined in the renal vascular hypertension ACR and Appropriate Use Criteria documents [2,33]. Diagnostic angiography remains the gold standard for identifying RAS [34]. Angiography may be indicated, in the appropriate clinical setting, following the discovery of a RAS by noninvasive imaging or in settings in which RVH or ischemic nephropathy (IN) is suspected clinically but noninvasive imaging is equivocal. Renal angiography provides a better quantification of the degree of stenosis and an opportunity to determine the physiologic significance of a stenosis.

A clinically and hemodynamically significant RAS occurs when the renal artery lumen is narrowed enough to reduce flow and perfusion to the affected kidney. Animal experiments have shown that a renal artery diameter stenosis of $\geq 50\%$ or a surface area reduction of $\geq 80\%$ is associated with an ipsilateral increase in renal renin secretion, a known contributor to RVH [14,35,36]. The effect of the RAS is affected by the length, irregularity, and multiplicity of the RAS as well as the vascular resistance in the distal renovascular bed [15,35].

"The physiologic significance of a stenosis depends on the resistance of the peripheral renal vasculature and the condition of the renal autoregulatory system" [30]. Doppler ultrasonography and nuclear renography may be useful in assessing the significance of a RAS, but the gold standard for measuring the physiologic significance of a stenosis is simultaneous measurement of the pressure in the aorta adjacent to the renal arteries and the pressure distal to the stenosis to determine a pressure gradient. These determinations are best performed using a guiding catheter positioned in the aorta adjacent to the renal artery ostium and a low-profile pressure-sensing wire or microcatheter distal to the stenosis. Note that a false elevation of the gradient could occur if a larger catheter were to be placed in the renal artery because the larger catheter might itself add to the flow restriction caused by the stenosis and hence falsely reduce the arterial pressure distal to the stenosis [11,14,16].

Several standards have been proposed for determining the hemodynamic significance of RAS, and there is no consensus as to whether peak systolic or mean pressure gradient should be used, or whether the pressure should be measured during a resting or stimulated state [11]. A translesion systolic pressure gradient of 20 mm Hg is often considered to be hemodynamically significant and a level that activates the renin-angiotensin system [24]. Measurement of renin levels in human subjects using balloon inflation to create variable stenoses revealed that a 10% mean pressure gradient increases ipsilateral renal vein renin secretion. Mean pressure gradients are now a more widely accepted measure of a hemodynamically significant stenosis because mean pressures influence renal perfusion during both systole and diastole [15-17,37-40]. Other tests that can lend support to the clinical significance of a RAS of borderline hemodynamic significance include stimulated mean and/or systolic pressure gradients, intravascular ultrasound, optical coherence tomography (OCT), or selective renal vein renin sampling [15,17,37-40]. There is increasing consensus on the significance of a translesion pressure gradient, with recent

guidelines having agreed on the standards for revascularization. According to the recently published multisociety Appropriate Use Criteria for peripheral arterial intervention [8], >70% angiographic or intravascular ultrasound (IVUS) diameter stenosis or 50% to 69% stenosis with hemodynamic confirmation of a significant gradient represent indications for treatment. A RAS is considered to be hemodynamically significant if the resting or stimulated translesion systolic pressure gradient is >20 mm Hg or if the resting translesion mean pressure gradient is >10 mm Hg [11,16]. Standards in this document also reference use of fractional flow reserve (FFR) as a means of determining hemodynamic significance. FFR can be determined using dopamine or papaverine infusion and is considered significant when the FFR is <0.8 [41,42].

In addition to reviewing the indications for renal angiography, it is worth discussing potential prerequisites for performing angiography. Additional laboratory testing results that may be useful in determining whether or not to proceed to angiography include low urine protein levels (which predicts better outcomes with RAST) and high plasma renin levels (which have low sensitivity and high specificity for response to renal revascularization), and elevated brain natriuretic peptide (BNP) [18,20,43]. Angiotensin II, a potent vasoconstrictor that stimulates cellular hypertrophy and proliferation, also increases with elevated levels of plasma renin and results from its conversion from angiotensin I, and likely contributes to vascular and ventricular hypertrophy, accelerates atherosclerosis, and causes progressive glomerular sclerosis independent of its hemodynamic effect [44]. Therefore, whenever possible, an ACE inhibitor or ARB should be part of the treatment of HTN associated with CKD because these drugs have been shown to be organ-protective beyond their antihypertensive effect in certain renal disease categories [27].

III. SUCCES RATES FOR RENAL ARTERY INTERVENTION

Although a hemodynamically significant RAS may stimulate the renin-angiotensin system and result in systemic HTN or renal ischemia, there are other factors that may influence the clinical response to treating a RAS [30]. The etiology of the stenosis (eg, atherosclerosis, fibromuscular dysplasia, vasculitis, arterial/aortic dissections) and the age of the patient are important factors in determining clinical success. Additional factors that are important in older patients 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, any impairment in renal function or evidence of progressive nephron loss, and comorbid medical conditions [30]. Therefore, the clinical significance of a RAS and the likelihood that the clinical syndrome can be improved should guide the decision to revascularize a kidney rather than the morphologic or hemodynamic characteristics of the renal artery stenotic lesion alone [30]. "The majority of patients with hemodynamically significant RAS associated with HTN or reduced renal function can be managed medically without a risk of increased mortality or progression to end-stage renal disease" [30]. However, there are patient subpopulations in whom RAS may produce RVH, IN [43], or cardiac disturbance syndromes (eg, recurrent "flash" pulmonary edema not felt to be secondary to impaired left ventricular systolic function) and in whom an endovascular intervention may therefore be helpful. "Thus, the benefits of revascularization need to be individually determined based on the underlying clinical condition prompting intervention" [30].

Outcomes Following Renal Revascularization

III. SUCCES RATES FOR RENAL ARTERY INTERVENTION

A. Atherosclerotic RAS

1. The patient with HTN

"Only a small percentage of patients with ARAS are reported as cured following revascularization [10-12]. The clinical profile of the patient (with atherosclerosis, who) is most likely to be cured, has not been defined" [30]. There are findings that may help determine the outcomes of renal revascularization for ARAS, including the severity of the ARAS, if ARAS is unilateral or bilateral, diameter of the narrowed vessels, location of the narrowing, if there is involvement of branch points, patency of small arteries and arterioles distal to a RAS, renal mass available for revascularization (usually a measurement of kidney length or cortical thickness), function of the involved kidney as demonstrated by nuclear scintigraphy, presence of proteinuria [19,20,43], and intrinsic renal disease on the affected side (measured by duplex ultrasound determinations of resistive index) [45-48]. Randomized controlled trials (RCTs) [10,12,13,49-54] and multiple case series [55-58] report that renal revascularization in patients with ARAS results in only a

modest decrease in doses of medications or blood pressure. More recent studies have focused on the risk of cardiovascular events in patients with possible RVH and have failed to demonstrate an advantage for PTRA and/or RAST as compared with optimum medical therapy [10,12,13]. Whether the benefit of controlling blood pressure on less medication or a potential reduction in blood pressure on the same medications outweighs the risks of the procedure should still be considered on an individual patient basis [59-61]. Despite the findings of these RCTs, there may be patients with high blood pressure, refractory HTN, renal insufficiency, or severe bilateral ARAS who will have a positive clinical response to revascularization [43,57,62]. In the following sections, the clinical evidence regarding revascularization is discussed for specific indications.

2. The patient with resistant HTN (RHTN)

Although RHTN is uncommon, the incidence of ARAS by angiography in patients with RHTN is high (24.1%) [9]. True RHTN is defined as persistent hypertension in spite of adherence to maximally tolerated dosing of 3 or more antihypertensive medications, including a diuretic, and it represents only a small percentage of patients with HTN [63]. The available randomized clinical trials have often been cited for underrepresenting this population. In 2000, van Jaarsveld et al published one of the first RCTs focused on atherosclerotic RHTN. The study of 106 patients with RHTN with RAS found no difference between medical management and balloon angioplasty [12]. The trial has been cited for not including renal artery stents, but a meta-analysis of all of the RCTs also fails to demonstrate a benefit in patients with RHTN. There are more recent case-controlled series indicating that a carefully selected population of patients with RHTN and hemodynamically significant ARAS respond favorably to angioplasty and RAST [62,64-66]. Available large RCTs suggest that RHTN is not an indication for RAST [10]. However, the study populations were potentially biased, and the incongruity between these randomized trial studies and multiple case series leave questions on this indication for revascularization [28,67]. The clinical efficacy of treating RHTN, particularly in the setting of severe, bilateral ARAS, remains potentially unproven and should be reserved for a select patient group that clearly meets criteria for RHTN.

3. The patient with hypertensive crisis

The literature on renal revascularization in patients with a hypertensive crisis is limited [68]. The risks of stroke and access site complications are higher if blood pressure is not well controlled. There is general agreement that medical therapy is recommended for management of hypertensive crises.

III. SUCCES RATES FOR RENAL ARTERY INTERVENTION

B. HTN in the patient with fibromuscular dysplasia and RAS

There is strong evidence that when HTN is associated with hemodynamically significant renal artery fibromuscular dysplasia (FMD), patients may benefit from PTRA [69,70]. The mean cure rate in this population following renal revascularization is 44% to 46% [58,70,71]. Using logistic regression, Davidson et al found that younger age, milder severity, and shorter duration of HTN were statistically significant independent variables predicting a cure following PTRA in patients with FMD [72]. Classification of FMD has traditionally been on the basis of the histologic subtypes when surgery was a more frequent treatment option [73]. However, with the refinement in endovascular techniques [29,71] and a reduction in the need for surgery to treat FMD, histopathologic classification of FMD has become less practical. Therefore, a consensus statement from 2012 proposed a classification scheme based upon the imaging characteristics of the FMD, classifying lesions simply as being unifocal (<1 cm in length) and/or multifocal (>1 cm in length) [74]. Patients may have unifocal and multifocal lesions simultaneously in the same artery. Dissections and aneurysms, which are often seen in arteries of patients with FMD, are not considered to be imaging subtypes of the disease.

FMD most often involves the distal main and branch renal arteries. Fortunately, the technical and clinical response of FMD involving renal artery branches to PTRA is as good as in cases in which FMD is limited to the main renal artery [75,76]. The operator must understand that treatment should not be limited to main renal artery lesions because the best chance for a cure is achieved when all of the hemodynamically significant lesions are treated.

Renal artery FMD can be found by CT angiography (CTA) in 2.6% of potential kidney donors [77] and in 5.7% of patients undergoing angiography for ARAS [78]. There is also a strong association between renal FMD and carotid FMD [79], so a thorough screening, usually with CTA, is recommended whenever renal FMD is diagnosed [80]. FMD can also be found in 7.3% of first-degree or second-degree relatives, so consulting with the family is an important part of the evaluation process in patients with FMD.

III. SUCCES RATES FOR RENAL ARTERY INTERVENTION

C. Takayasu arteritis

Takayasu arteritis (TA) is a rare, large-vessel arteritis. TA primarily affects large vessels originating from the aorta, causing wall inflammation, fibrosis, and stenosis [81]. The reported incidence of TA in North American patients was found to be 2.6 per million per year [82]. Detection of RVH can be difficult to delineate because these patients can have bilateral subclavian artery stenosis that causes misleadingly low blood pressure measurements [83]. Glucocorticosteroids are the first-line agents and the gold standard in treatment for TA. After being prescribed glucocorticosteroids, most patients show improved quality of life. Prednisone can reverse stenotic lesions of the aorta and renal arteries and concomitantly reduce blood pressure [84]. Treatment of this disease entity can be challenging because it is often resistant to medical therapy [85]. Endovascular treatment with PTRAs may be offered for the treatment of RVH related to TA. A recent retrospective analysis demonstrated increased restenosis rate with stent placement compared with angioplasty alone [86]. Multiple retrospective analyses have confirmed these findings [87,88] and demonstrated better long-term patency of angioplasty compared with both surgery and stent placement [87]. Angioplasty alone should therefore be the mainstay of endovascular treatment of RVH in TA, with stent placement reserved for cases of clear angioplasty failure. Elevated erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) are indicators of the acute inflammatory stage of the disease. Care should be made not to perform angioplasty during the acute phase of the disease because it has been shown to have a higher risk of complications [89].

III. SUCCES RATES FOR RENAL ARTERY INTERVENTION

D. Renal artery dissection

Spontaneous, isolated, renal artery dissection may be detected as part of a hypertensive or renal failure evaluation. It may also be first detected because of new flank pain or hematuria. It is often idiopathic but is frequently associated with HTN, FMD, connective tissue disease, and/or trauma. Acute dissection may cause new or accelerated HTN, renal failure, or flank pain. A case series of patients with acute symptomatic idiopathic renal artery dissection (no connective tissue disorders or other associated pathology) demonstrated clinical benefit to intervention, just as has conservative management with anticoagulation [90,91].

III. SUCCES RATES FOR RENAL ARTERY INTERVENTION

E. Atherosclerotic renal artery disease and IN

There is ongoing controversy concerning the degree of benefit that can be expected from revascularization of the patient with IN [30]. It is well recognized that there is progressive nephron loss with aging. The loss is accelerated by many disease states, including IN, in which, in addition to the loss of nephron tissue, there can be functional loss as a result of renal hypoperfusion and loss of renal autoregulation secondary to ARAS [30]. Measurement of estimated glomerular filtration rate (eGFR) remains the best measure of functional outcomes [30,92]. The slope of the linear relationship between the reciprocal of creatinine concentration (a surrogate for the calculation of eGFR) versus time can be used to predict the rate of decline in renal function [30,92]. If the slope of this curve can be altered with PTRAs or RAST, then the consequences of chronic renal failure (CRF) and renal replacement therapy may be delayed. Altering the progression along the slope of decline in renal function may indicate a benefit from intervention despite a lack of improvement in baseline serum creatinine [30].

Several case series of renal revascularization for IN have demonstrated statistically significant improvement in renal function at follow-up [12,21,43,93,94]. On the other hand, 3 prospective randomized renal revascularization studies demonstrated no improvement in renal function [10,12,13]. However, other markers, including baseline kidney size and resistive indices, were not included in these trials [95,96]. There are 3 indications that continue to be debated regarding renal revascularization for ischemia: acute renal failure, renal failure associated with prior renal artery manipulations, and renal angioplasty and/or stenting for preservation of renal mass. A study involving 1,052 patients with ARAS showed that patients with baseline proteinuria greater

than 300 mg/24 hours and CKD stages 3B/4/5 have increased risk of progression to renal replacement therapy after RAST for ARAS [19].

III. SUCCES RATES FOR RENAL ARTERY INTERVENTION

E. Atherosclerotic renal artery disease and IN

1. Acute IN

Although all the RCTs failed to demonstrate clinical benefit of revascularization for IN due to ARAS, these trials did enroll patients with CRI [10,12,13,43,49]. Renal revascularization can result in improvement of GFR in selected patients with acute IN [43,62,97]. Factors that are associated with a patient with acute IN to likely benefit from revascularization include:

- a. Normal angiographic appearance of the arteries distal to the RAS
- b. Bilateral severe RAS or RAS involving a single functioning kidney
- c. A near-normal volume of renal mass available for revascularization
- d. Renogram demonstrating adequate function of the involved kidney
- e. Renal biopsy demonstrating well-preserved glomeruli and tubules with minimal arteriolar sclerosis
- f. Severe, difficult to control HTN
- g. Abrupt onset of renal insufficiency [47-50,62,98,99]
- h. Renal artery FFR over 0.80 [100]
- i. Lack of increased baseline proteinuria [19,20,43]

Recent guidelines support renal artery revascularization in the setting of declining renal function in the patient with bilateral hemodynamically significant RAS, or significant RAS in a solitary functioning kidney. Revascularization may be appropriate in unilateral, severe RAS in the setting of declining renal function.

III. SUCCES RATES FOR RENAL ARTERY INTERVENTION

E. Atherosclerotic renal artery disease and IN

2. Renal failure associated with prior arterial interventions

None of the randomized trials of renal artery interventions for CRI address the management of patients with prior renal artery interventions. Acute renal failure in the setting of ARAS related to prior renal artery bypass, aortic endograft encroachment over the renal artery origins(s), or prior renal artery stent placement should be treated aggressively [101-103]. In these clinical scenarios, there is often a significant temporal relationship between serial imaging changes and deterioration in renal function that indicates a strong association between recurrent ARAS and renal failure. This recommendation for treatment is also based on the natural history of rapid progression to renal artery occlusion in previously treated renal arteries [104,105]. In one study, the only predictor for postcontrast acute kidney injury in patients undergoing RAST therapy was elevated baseline proteinuria [106].

III. SUCCES RATES FOR RENAL ARTERY INTERVENTION

E. Atherosclerotic renal artery disease and IN

3. Prophylactic treatment for renal mass preservation

There is no known benefit to prophylactic treatment to preserve renal mass [107].

III. SUCCES RATES FOR RENAL ARTERY INTERVENTION

F. Cardiac disturbance syndromes

“RAS may worsen angina or congestive heart failure in patients with coronary artery disease, left ventricular dysfunction, or cardiomyopathy as a result of complex pathophysiologic alterations” [30], such as changes in the renin-angiotensin axis that lead to volume overload and peripheral arterial vasoconstriction [108-111]. Renal revascularization may relieve these cardiac disturbance syndromes, particularly in patients with bilateral ARAS [30,62,110,112-114]. Over 70% of patients remain free of congestive heart failure and unstable angina at the 12-month mean follow-up after RAST [108,113]. In particular, there are multiple case series that suggest PTRAS in the setting of flash pulmonary edema may be beneficial [62,67,115-117]. Restoring unobstructed renal blood

flow has the additional potential benefit of allowing safe usage of ACE inhibitors without the risk of worsening renal failure [30].

Guidelines recommend renal artery revascularization with RAST of a hemodynamically significant ARAS in patients with sudden onset flash pulmonary edema. Renal artery stenting may also be of benefit in patients with recurrent heart failure uncontrolled on maximal medical therapy, or uncontrolled unstable angina in spite of maximal medical therapy [8].

III. SUCCES RATES FOR RENAL ARTERY INTERVENTION

G. Technical success and long-term patency of renal revascularization procedures

Intravascular stent placement is the standard of care for revascularization of ARAS [10,57]. Not all stent positions allow the opportunity for repeat intervention and assisted patency. The use of stents is relatively contraindicated if the stent traverses renal artery branches or if surgical revascularization is difficult or impossible in the event of restenosis. Stents dilated to less than 6 mm, female sex, age greater than 65 years, and smoking are statistically significant risk factors for developing in-stent restenosis (ISR) [118,119]. In the US Multicenter Renal Artery Stent Trial, the lowest-risk group was men with renal arteries 6 mm or greater, in whom there was a restenosis rate of 10.5%. There are very little data regarding stent use in nonstiaial RAS; however, 1 study suggests that these lesions may respond favorably to balloon angioplasty alone [120].

Technical success rates of PTRAs for renal FMD should approach 90% [70,71]. There is increasing emphasis on measures of technical success other than angiographic appearance for FMD. Pressure-wire manometry and intravascular ultrasound should be available and their use considered when treating FMD. Appropriate treatment of FMD includes dilatation of the entire diseased segment, even if it involves a branch point. The operator must be comfortable with the use of dual-wire access and kissing balloons. Renal artery stents have no role in the primary treatment of FMD. Stents may be indicated in PTRAs technical failures due to flow-limiting vessel dissections, but the remodeling capabilities of a post-PTRA renal artery with mild dissections should not be underestimated by the operator [29].

Long-term stent patency in most trials was assessed using periodic noninvasive monitoring. Follow-up of stents placed for ARAS should include regular duplex ultrasound, which, with appropriate baseline evaluation, provides a highly sensitive method to detect ISR [121,122]. CTA has limited use in follow-up after renal artery stent placement [123,124].

III. SUCCES RATES FOR RENAL ARTERY INTERVENTION

H. Restenosis

The evidence for the use of drug-eluting stents (DESs) to reduce the rate of ISR is limited. However, 1 small series involving 37 patients with 39 renal small or accessory renal arteries treated with a DES had a median ISR-free survival of 992 days, with only 11 of 37 (29.7%) developing an ISR [125]. It should also be noted that a repeat intervention for ISR has twice the restenosis rate of primary stent placement (20% vs 11%; $P = 0.003$) [105]. The methods for management of ISR are varied and have included percutaneous transluminal angioplasty (PTA), RAST, atherectomy, brachytherapy, cutting balloons, covered stents, and DES placement [103,125-129]. These case series are small and do not define the best therapeutic option for treating renal artery ISR [126-129].

III. SUCCES RATES FOR RENAL ARTERY INTERVENTION

I. Summary

There is growing consensus on the indications for renal intervention in patients with RAS and HTN and/or renal ischemia. There are several important subpopulations that will need further clinical investigation before global recommendations can be made regarding renal intervention, such as patients with hemodynamically significant ARAS (as determined by a minimum 10% mean translesion pressure gradient) and poorly controlled HTN, renal insufficiency due to IN, and/or cardiac disturbance syndromes. The response to medical therapy and technical success, long-term patency, and complication rates must also factor into the decision to proceed with revascularization.

IV. RISKS OF ENDOVASCULAR REVASCULARIZATION

In combination with improvements in imaging and interventional device technologies and operator experience, procedural complication rates related to the performance of PTRA and RAST have been decreasing over the years [130]. Complication rates related to these procedures have been previously reported in the 2010 SIR *Quality Improvement Guidelines for Angiography, Angioplasty, and Stent Placement for the Diagnosis and Treatment of Renal Artery Stenosis in Adults* [30]. Current medical therapy has also had an impact on renal artery stenting outcomes. The use of intensive lipid-lowering therapy in patients undergoing renal artery stenting has been shown to provide a significant benefit in renal protection during RAST for ARAS [131]. In addition, more recent renal artery stent trials have focused on primary and secondary outcome measures, including all-cause mortality, blood pressure control, and preservation of kidney function [10,13].

V. QUALIFICATIONS AND RESPONSIBILITIES OF PERSONNEL

A. Physician

Core Privileging: This procedure is considered part of or amenable to image-guided core privileging.

The physician performing renal angioplasty/stenting must have a broad perspective on the benefits, alternatives, and risks of the procedure. The physician must have a thorough understanding of renovascular physiology, medical management of HTN and renal ischemia, vascular anatomy (including congenital and developmental variants and common collateral pathways), angiographic equipment, radiation safety considerations, and physiologic monitoring equipment. The physician must have access to and familiarity with an adequate supply of diagnostic catheters, guiding catheters, guide sheaths, intravascular pressure measurement devices and tools, angioplasty balloons and stents, and guidewires. The physician must also have awareness of the skills and numbers of ancillary personnel and medications needed to perform the procedure safely.

Renal angioplasty/stenting procedures must be performed under the supervision of and interpreted by a physician who meets the qualifications pertinent to the scope of services as stated in the ACR–SIR–SPR Practice Parameter for the Performance of Arteriography [132].

Maintenance of Competence

Physicians must perform a sufficient number of overall procedures applicable to the spectrum of core privileges to maintain their skills, with acceptable success and complication rates as previously referenced [30]. Continued competence should depend on participation in a quality improvement program that monitors these rates. Consideration should be given to the physician's lifetime practice experience.

CME

The physician's continuing education should be in accordance with the [ACR Practice Parameter for Continuing Medical Education \(CME\)](#) [133].

V. QUALIFICATIONS AND RESPONSIBILITIES OF PERSONNEL

B. Qualified Medical Physicist

For qualifications of the Qualified Medical Physicist, see the [ACR–AAPM Technical Standard for Diagnostic Medical Physics Performance Monitoring of Fluoroscopic Equipment](#) [134].

V. QUALIFICATIONS AND RESPONSIBILITIES OF PERSONNEL

C. Non-Physician Radiology Provider (NPRP)

NPRPs are all Non-Physician Providers (eg, RRA, RPA, RA, PA, NP, ...) who assist with or participate in portions of the practice of a radiologist-led team (Radiologists = diagnostic, interventional, neurointerventional radiologists, radiation oncologists, and nuclear medicine physicians). The term "NPRP" does not include radiology, CT, US, NM MRI technologists, or radiation therapists who have specific training for radiology related tasks (eg, acquisition of images, operation of imaging and therapeutic equipment) that are not typically performed by radiologists.

The term 'radiologist-led team' is defined as a team supervised by a radiologist (ie, diagnostic, interventional, neurointerventional radiologist, radiation oncologist, and nuclear medicine physician) and consists of additional healthcare providers including RRAs, PAs, NPs, and other personnel critical to the provision of the highest quality of healthcare to patients. (ACR Resolution 8, adopted 2020).

V. QUALIFICATIONS AND RESPONSIBILITIES OF PERSONNEL

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 position¹ the patient for the procedure and, together with the nurse, monitor the patient during the procedure. The technologist should obtain the imaging data in a manner prescribed by the supervising physician. If IV contrast material is to be administered, qualifications for technologists performing IV injection should be in compliance with the current ACR policy² and existing operating procedures or manuals at the 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 ARRT or have an unrestricted state license with documented training and experience in the imaging modality used for the imaging-guided percutaneous procedure.

¹The American College of Radiology approves of the practice of certified and/or licensed radiologic technologists performing fluoroscopy in a facility or department as a positioning or localizing procedure only, and then only if monitored by a supervising physician who is personally and immediately available*. There must be a written policy or process for the positioning or localizing procedure that is approved by the medical director of the facility or department/service and that includes written authority or policies and processes for designating radiologic technologists who may perform such procedures. (1987, 1997, 2007 - ACR Resolution 12-m)

*For the purposes of this parameter, "personally and immediately available" is defined in the manner of the "personal supervision" provision of CMS—a physician must be in attendance in the room during the performance of the procedure. (Program Memorandum Carriers, DHHS, HCFA, Transmittal B-01-28, April 19, 2001)

²See the ACR–SPR Practice Parameter for the Use of Intravascular Contrast Media.

V. QUALIFICATIONS AND RESPONSIBILITIES OF PERSONNEL

E. Nursing Services

Nursing services are an integral part of the team for preprocedure and postprocedure patient management and education and are recommended in monitoring the patient during and after the procedure.

V. QUALIFICATIONS AND RESPONSIBILITIES OF PERSONNEL

F. Other Licensed Independent Practitioners

Licensed independent practitioners may be involved in renal artery angioplasty and stenting procedures in accordance with their societal and local regulatory scope of practice under the supervision of the physician operator. Typically, they will be involved with patient preparation, patient monitoring, and patient education, and in some cases they may serve as "scrub" assistants.

VI. SPECIFICATIONS OF THE PROCEDURE

There are several technical requirements that are necessary in order to ensure safe and successful renal angiography, angioplasty, and stenting. These include adequate angiographic equipment and institutional facilities, physiologic monitoring equipment, and support personnel. These recommendations are adapted from the Cardiovascular Outcomes in Renal Atherosclerotic Lesions (CORAL) trial [10], the American Heart Association Intercouncil report on optimum resources for endovascular treatment [135] and previous published recommendations [136,137].

VI. SPECIFICATIONS OF THE PROCEDURE

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 in order to produce higher-quality studies with reduced risk and examination time. The facility should include the following, at a minimum:

A high-resolution image receptor (preferably with a 28-cm to 40-cm field of view [FOV]) and imaging chain with dose-reducing capabilities, such as pulsed fluoroscopy, dose reduction software, and last-image-hold capabilities, are recommended. Digital subtraction angiographic (DSA) systems with high spatial resolution are strongly recommended because they allow for reduced volumes of contrast material to be used, reduced examination times, and avoidance of complications related to the use of low radiopacity stents. In accordance with the “as low as reasonably achievable” (ALARA) principle, a radiation dose measurement package to provide operator and patient feedback is recommended.

1. Adequate angiographic supplies, such as catheters, guidewires, stents, balloons, needles, pressure transducers for measuring intravascular pressures, PTA balloons, vascular stents, and introducer sheaths. In particular, access to pressure wires and intravascular ultrasound is advisable in order to provide objective evidence of hemodynamic significance in cases of angiographically equivocal stenoses.
2. An angiographic injector capable of varying injection volumes and rates with appropriate safety mechanisms to prevent overinjection.
3. 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 IV 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 [136,137].
4. 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 the Patient Care section below, and there should be immediate access to emergency resuscitation equipment.

VI. SPECIFICATIONS OF THE PROCEDURE

B. Physiologic Monitoring and Resuscitation Equipment

1. 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–SIR Practice Parameter for Minimal and/or Moderate Sedation/Analgesia](#) [138]).
2. Appropriate emergency equipment and medications must be immediately available to treat adverse reactions associated with administered medications and/or procedural complications. The equipment should be monitored and medications inventoried for drug expiration dates on a regular basis. The equipment, medications, and other emergency support must also be appropriate for the range of ages or sizes in the patient population.
3. Equipment for invasive pressure monitoring.

VI. SPECIFICATIONS OF THE PROCEDURE

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 in the procedure should be assigned to periodically assess the patient's status. In cases in which moderate sedation is used or the patient is critically ill, an experienced licensed provider should be present whose sole responsibility is to monitor the patient's vital signs, sedation state, and level of comfort/pain. 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–SIR Practice Parameter for Minimal and/or Moderate Sedation/Analgesia](#) [138]).
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 renal interventional procedures. Therefore, appropriate personnel should be available to attend to the ongoing care and resuscitation of critically ill patients. Such personnel might include anesthesiologists; operating room (OR)–, intensive care unit– (ICU), and/or emergency department– (ED) 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.

VI. SPECIFICATIONS OF THE PROCEDURE

D. Acute Care 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.

VI. SPECIFICATIONS OF THE PROCEDURE

E. Patient Care

1. Preprocedure care

- a. The physician performing the procedure must have knowledge of the following:
 - i. Clinically significant history, including indications for the procedure
 - ii. Clinically significant physical or diagnostic examination, including knowledge and awareness of other clinical or medical conditions that may necessitate specific care, such as preprocedure antibiotics and other measures
 - iii. Assessment and documentation of patient's candidacy for conscious sedation.
 - iv. Possible alternative methods, such as surgical or medical treatments, to obtain the desired therapeutic result
- b. Informed consent must be in compliance with all state laws and the ACR–SIR–SPR Practice Parameter on Informed Consent for Image-Guided Procedures [139].

VI. SPECIFICATIONS OF THE PROCEDURE

E. Patient Care

2. Procedural care

- a. Adherence to the Joint Commission's Universal Protocol for Preventing Wrong Site, Wrong Procedure, Wrong Person Surgery™ is required for procedures in nonoperating room settings including bedside procedures. The organization should have processes and systems in place for reconciling differences in staff responses during the time-out.
- b. The physician performing fluoroscopy should have knowledge of exposure factors, fluoroscopic pulse rate,

magnification factor, and fluoroscopic dose rate and should consider additional parameters, such as collimation, FOV, distance from the patient to the image receptor, distance from the x-ray source to the patient, and last image-hold.

- c. 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:
 - i. Equipment needed for the procedure
 - ii. Patient monitoring, including conscious sedation

Protocols should be reviewed and updated periodically.

During the use of fluoroscopy, the physician should use exposure factors consistent with the ALARA radiation safety guidelines.

VI. SPECIFICATIONS OF THE PROCEDURE

E. Patient Care

3. Postprocedure care

- a. A procedure note should be entered 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 formal report is not likely to be available on the same day, a more detailed summary of the study should be entered 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. For further information see the [ACR–SIR–SPR Practice Parameter for the Reporting and Archiving of Interventional Radiology Procedures](#) [140].
- b. All patients should be on 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. Orthostasis and even hypotension can be encountered after renal artery revascularization, and antihypertensive medications should be managed proactively.
- c. During the initial postprocedure period, skilled nurses or other appropriately trained personnel should periodically monitor the puncture site and the status of the patient.
- d. The patient should be monitored for urinary output, cardiac symptoms, pain, changes in blood pressure and/or mental status, access site complications, and other indicators of systemic complications that may necessitate overnight care.
- e. 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.

VII. DOCUMENTATION

Documentation should be in accordance with the [ACR–SIR–SPR Practice Parameter for the Reporting and Archiving of Interventional Radiology Procedures](#) [140].

VIII. RADIATION SAFETY IN IMAGING

Radiologists, medical physicists, non-physician radiology providers, radiologic technologists, and all supervising physicians have a responsibility for safety in the workplace by keeping radiation exposure to staff, and to society as a whole, "as low as reasonably achievable" (ALARA) and to assure that radiation doses to individual patients are appropriate, taking into account the possible risk from radiation exposure and the diagnostic image quality necessary to achieve the clinical objective. All personnel who work with ionizing radiation must understand the key principles of occupational and public radiation protection (justification, optimization of protection, application of dose constraints and limits) and the principles of proper management of radiation dose to patients (justification, optimization including the use of dose reference levels). https://www-pub.iaea.org/MTCD/Publications/PDF/PUB1775_web.pdf

Nationally developed guidelines, such as the [ACR's Appropriateness Criteria](#)[®], should be used to help choose the most appropriate imaging procedures to prevent unnecessary radiation exposure.

Facilities should have and adhere to policies and procedures that require ionizing radiation examination protocols (radiography, fluoroscopy, interventional radiology, CT) to vary according to diagnostic requirements and patient body habitus to optimize the relationship between appropriate radiation dose and adequate image quality. Automated dose reduction technologies available on imaging equipment should be used, except when inappropriate for a specific exam. If such technology is not available, appropriate manual techniques should be used.

Additional information regarding patient radiation safety in imaging is available from the following websites – Image Gently[®] for children (www.imagegently.org) and Image Wisely[®] for adults (www.imagewisely.org). These advocacy and awareness campaigns provide free educational materials for all stakeholders involved in imaging (patients, technologists, referring providers, medical physicists, and radiologists).

Radiation exposures or other dose indices should be periodically measured by a Qualified Medical Physicist in accordance with the applicable ACR Technical Standards. Monitoring or regular review of dose indices from patient imaging should be performed by comparing the facility's dose information with national benchmarks, such as the ACR Dose Index Registry and relevant publications relying on its data, applicable ACR Practice Parameters, NCRP Report No. 172, Reference Levels and Achievable Doses in Medical and Dental Imaging: Recommendations for the United States or the Conference of Radiation Control Program Director's National Evaluation of X-ray Trends; 2006, 2009, amended 2013, revised 2023 (Res. 2d).

IX. QUALITY CONTROL AND IMPROVEMENT, SAFETY, INFECTION CONTROL, AND PATIENT EDUCATION

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 appearing under the heading *ACR Position Statement on Quality Control and Improvement, Safety, Infection Control and Patient Education* on the ACR website (<https://www.acr.org/Advocacy-and-Economics/ACR-Position-Statements/Quality-Control-and-Improvement>).

These practice parameters are to be used in quality improvement programs to assess the diagnosis and treatment of RAS. 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 [30].

Participation by the radiologist in patient follow-up is an integral part of the evaluation and treatment of RAS and will increase the success rate of the procedure [30].

Although practicing physicians should strive to achieve perfect outcomes (eg, 100% success, 0% complications), in practice all physicians will fall short of this ideal to a variable extent. Thus, indicator thresholds may be used to assess the efficacy of ongoing quality improvement programs. For the purposes of these (practice parameters), 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, for example, 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 RAS, then values in excess of the defined threshold (of 6%) should trigger a review of policies and procedures within the department to determine the causes and to implement changes to lower the incidence of the complication [30].

ACKNOWLEDGEMENTS

This practice parameter was revised according to the process described under the heading *The Process for Developing ACR Practice Parameters and Technical Standards* on the ACR website (<https://www.acr.org/Clinical-Resources/Practice-Parameters-and-Technical-Standards>) by the Committee on Practice Parameters – Interventional and Cardiovascular Radiology of the ACR Commission on Interventional and Cardiovascular Radiology in collaboration with the SIR.

Writing Committee – members represent their societies in the initial and final revision of this practice parameter

ACR

Alan H. Matsumoto, MD, FACR, Chair
Sidney Z. Brejt, MD
Drew M. Caplin, MD
Elizabeth A. Ignacio, MD
Claire Kaufman, MD
Stephen P. Reis MD MBA FSIR

SIR

Laura K. Findeiss, MD
Sanjay Misra, MD
Christopher S. Morris, MD, FACR, FSIR
Ellen A. Redstone, MD
Howard M. Richard, III, MD, FSIR
Alda L. Tam, MD, FSIR

Committee on Practice Parameters – Interventional and Cardiovascular Radiology

(ACR Committee responsible for sponsoring the draft through the process)

Drew M. Caplin, MD, Chair
Chaitanya Ahuja, MBBS
Douglas M. Coldwell, MD, PhD
Mandeep S. Dagli, MD
Kevin W. Dickey, MD, FACR
Meredith J. Englander, MD
C. Matthew Hawkins, MD

Margaret Hsin-Shung Lee, MD, FACR
Mary Lee Jensen, MD, FACR
Claire Kaufman, MD
Dennis Kay, MD, FACR
Kenneth F. Layton, MD, FACR
M. Victoria Marx, MD
Christopher D Yeisley, MD

Alan H. Matsumoto, MD, FACR, Chair, Commission on Interventional and Cardiovascular Radiology

David B. Larson, MD, MBA, Chair, Commission on Quality and Safety

Mary S. Newell, MD, FACR, Chair, Committee on Practice Parameters and Technical Standards

Comments Reconciliation Committee

C. Matthew Hawkins, MD, Chair
Elizabeth A. Ignacio, MD, Co-Chair
Sidney Z. Brejt, MD
Drew M. Caplin, MD
Harry R Cramer JR, MD
Richard Duszak Jr., MD, FACR
Laura K. Findeiss, MD
Antoinette S Gomes, MD, FACR
Klaus Dieter Hagspiel, MD
Claire Kaufman, MD
Amy L. Kotsenas, MD, FACR
David B. Larson, MD, MBA

Paul A Larson, MD, FACR
Alan H. Matsumoto, MD, FACR
Sanjay Misra, MD
Christopher S. Morris, MD, FACR, FSIR
Mary S. Newell, MD, FACR
Andrew Picel, MD
Ellen A. Redstone, MD
Stephen P. Reis MD MBA FSIR
Howard M. Richard, III, MD, FSIR
Lori Ann Sedrak, DO
Alda L. Tam, MD, FSIR
Matthew K Walsworth, MD

REFERENCES

1. Nwankwo T, Yoon SS, Burt V, Gu Q. Hypertension among adults in the United States: National Health and Nutrition Examination Survey, 2011-2012. Available at: <http://www.cdc.gov/nchs/data/databriefs/db133.htm>. Accessed February 20, 2014.
2. Whelton PK, Carey RM, Aronow WS, et al. 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *J Am Coll Cardiol* 2018;71:e127-e248.
3. Goldblatt H, Lynch J, Hanzal RF, Summerville WW. Studies on Experimental Hypertension : I. The Production of Persistent Elevation of Systolic Blood Pressure by Means of Renal Ischemia. *J Exp Med* 1934;59:347-79.
4. Textor SC. Renal Arterial Disease and Hypertension. *Med Clin North Am* 2017;101:65-79.
5. Labropoulos N, Ayuste B, Leon LR, Jr. Renovascular disease among patients referred for renal duplex ultrasonography. *J Vasc Surg* 2007;46:731-7.
6. de Mast Q, Beutler JJ. The prevalence of atherosclerotic renal artery stenosis in risk groups: a systematic literature review. *J Hypertens* 2009;27:1333-40.
7. Piecha G, Wiecek A, Januszewicz A. Epidemiology and optimal management in patients with renal artery stenosis. *J Nephrol* 2012;25:872-8.
8. Bailey SR, Beckman JA, Dao TD, et al. ACC/AHA/SCAI/SIR/SVM 2018 Appropriate Use Criteria for Peripheral Artery Intervention: A Report of the American College of Cardiology Appropriate Use Criteria Task Force, American Heart Association, Society for Cardiovascular Angiography and Interventions, Society of Interventional Radiology, and Society for Vascular Medicine. *J Am Coll Cardiol* 2019;73:214-37.
9. Benjamin MM, Fazel P, Filardo G, Choi JW, Stoler RC. Prevalence of and risk factors of renal artery stenosis in patients with resistant hypertension. *Am J Cardiol* 2014;113:687-90.
10. Cooper CJ, Murphy TP, Cutlip DE, et al. Stenting and medical therapy for atherosclerotic renal-artery stenosis. *N Engl J Med* 2014;370:13-22.
11. Mangiacapra F, Trana C, Sarno G, et al. Translesional pressure gradients to predict blood pressure response after renal artery stenting in patients with renovascular hypertension. *Circ Cardiovasc Interv* 2010;3:537-42.
12. 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;342:1007-14.
13. Wheatley K, Ives N, Gray R, et al. Revascularization versus medical therapy for renal-artery stenosis. *N Engl J Med* 2009;361:1953-62.
14. Gross CM, Kramer J, Weingartner O, et al. Determination of renal arterial stenosis severity: comparison of pressure gradient and vessel diameter. *Radiology* 2001;220:751-6.
15. Simon G. What is critical renal artery stenosis? Implications for treatment. *Am J Hypertens* 2000;13:1189-93.
16. De Bruyne B, Manoharan G, Pijls NH, et al. Assessment of renal artery stenosis severity by pressure gradient measurements. *J Am Coll Cardiol* 2006;48:1851-5.
17. 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;53:333-43.
18. Silva JA, Chan AW, White CJ, et al. Elevated brain natriuretic peptide predicts blood pressure response after stent revascularization in patients with renal artery stenosis. *Circulation* 2005;111:328-33.
19. Misra S, Khosla A, Allred J, Harmsen WS, Textor SC, McKusick MA. Mortality and Renal Replacement Therapy after Renal Artery Stent Placement for Atherosclerotic Renovascular Disease. *J Vasc Interv Radiol* 2016;27:1215-24.
20. Murphy TP, Cooper CJ, Pencina KM, et al. Relationship of Albuminuria and Renal Artery Stent Outcomes: Results From the CORAL Randomized Clinical Trial (Cardiovascular Outcomes With Renal Artery Lesions). *Hypertension* 2016;68:1145-52.
21. Harden PN, MacLeod MJ, Rodger RS, et al. Effect of renal-artery stenting on progression of renovascular renal failure. *Lancet* 1997;349:1133-6.
22. Maxwell MH, Bleifer KH, Franklin SS, Varady PD. Cooperative study of renovascular hypertension. Demographic analysis of the study. *JAMA* 1972;220:1195-204.
23. Albers FJ. Clinical characteristics of atherosclerotic renovascular disease. *Am J Kidney Dis* 1994;24:636-41.

24. Gerhard-Herman MD, Gornik HL, Barrett C, et al. 2016 AHA/ACC Guideline on the Management of Patients With Lower Extremity Peripheral Artery Disease: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *Circulation* 2017;135:e726-e79.
25. Krijnen P, van Jaarsveld BC, Steyerberg EW, Man in 't Veld AJ, Schalekamp MA, Habbema JD. A clinical prediction rule for renal artery stenosis. *Ann Intern Med* 1998;129:705-11.
26. Simon N, Franklin SS, Bleifer KH, Maxwell MH. Clinical characteristics of renovascular hypertension. *JAMA* 1972;220:1209-18.
27. James PA, Oparil S, Carter BL, et al. 2014 evidence-based guideline for the management of high blood pressure in adults: report from the panel members appointed to the Eighth Joint National Committee (JNC 8). *JAMA* 2014;311:507-20.
28. Mann SJ, Sos TA. The cardiovascular outcomes in renal atherosclerotic lesion study and the future of renal artery stenting (editorial). *J Clin Hyperten* 2014;14:163-65.
29. Meuse MA, Turba UC, Sabri SS, et al. Treatment of renal artery fibromuscular dysplasia. *Tech Vasc Interv Radiol* 2010;13:126-33.
30. Martin LG, Rundback JH, Wallace MJ, et al. Quality improvement guidelines for angiography, angioplasty, and stent placement for the diagnosis and treatment of renal artery stenosis in adults. *J Vasc Interv Radiol* 2010;21:421-30; quiz 230.
31. Chrysochou C, Foley RN, Young JF, Khavandi K, Cheung CM, Kalra PA. Dispelling the myth: the use of renin-angiotensin blockade in atheromatous renovascular disease. *Nephrol Dial Transplant* 2012;27:1403-9.
32. Hackam DG, Duong-Hua ML, Mamdani M, et al. Angiotensin inhibition in renovascular disease: a population-based cohort study. *American Heart Journal* 2008;156:549-55.
33. American College of Radiology. ACR Appropriateness Criteria® - renovascular hypertension. Accessed October 1, 2020.
34. Tullus K. Renal artery stenosis: is angiography still the gold standard in 2011? *Pediatr Nephrol* 2011;26:833-7.
35. Imanishi M, Akabane S, Takamiya M, et al. Critical degree of renal arterial stenosis that causes hypertension in dogs. *Angiology* 1992;43:833-42.
36. Kohler TR. Hemodynamics of arterial occlusive disease. In: Strandness DE Jr, van Breda A., ed. *Vascular diseases: surgical and interventional therapy*. 1st ed. New York, NY: Churchill Livingstone; 1994:65-71.
37. Harward TR, Poindexter B, Huber TS, Carlton LM, Flynn TC, Seeger JM. Selection of patients for renal artery repair using captopril testing. *Am J Surg* 1995;170:183-7.
38. Johansson M, Jensen G, Aurell M, et al. Evaluation of duplex ultrasound and captopril renography for detection of renovascular hypertension. *Kidney Int* 2000;58:774-82.
39. 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;39:1297-302.
40. Leesar MA, Varma J, Shapira A, et al. Prediction of hypertension improvement after stenting of renal artery stenosis: comparative accuracy of translesional pressure gradients, intravascular ultrasound, and angiography. *J Am Coll Cardiol* 2009;53:2363-71.
41. De Bruyne B, Pijls NH, Heyndrickx GR, Hodeige D, Kirkeeide R, Gould KL. Pressure-derived fractional flow reserve to assess serial epicardial stenoses: theoretical basis and animal validation. *Circulation* 2000;101:1840-7.
42. 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;92:3183-93.
43. Takahashi EA, Harmsen WS, Misra S. Impact of Renal Function Trajectory on Renal Replacement Therapy and Mortality Risk after Renal Artery Revascularization. *J Vasc Interv Radiol* 2020;31:592-97.
44. Conlon PJ, Athirakul K, Kovalik E, et al. Survival in renal vascular disease. *J Am Soc Nephrol* 1998;9:252-6.
45. Dean RH, Englund R, Dupont WD, et al. Retrieval of renal function by revascularization. Study of preoperative outcome predictors. *Ann Surg* 1985;202:367-75.
46. 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;5:622-7.
47. Martin LG, Casarella WJ, Gaylord GM. Azotemia caused by renal artery stenosis: treatment by percutaneous angioplasty. *AJR Am J Roentgenol* 1988;150:839-44.
48. Novick AC. Atherosclerotic ischemic nephropathy. Epidemiology and clinical considerations. *Urol Clin North Am* 1994;21:195-200.

49. Bax L, Woittiez AJ, Kouwenberg HJ, et al. Stent placement in patients with atherosclerotic renal artery stenosis and impaired renal function: a randomized trial. *Ann Intern Med* 2009;150:840-8, W150-1.
50. Webster J, Marshall F, Abdalla M, et al. Randomised comparison of percutaneous angioplasty vs continued medical therapy for hypertensive patients with atheromatous renal artery stenosis. Scottish and Newcastle Renal Artery Stenosis Collaborative Group. *J Hum Hypertens* 1998;12:329-35.
51. Plouin PF, Chatellier G, Darne B, Raynaud A. Blood pressure outcome of angioplasty in atherosclerotic renal artery stenosis: a randomized trial. Essai Multicentrique Medicaments vs Angioplastie (EMMA) Study Group. *Hypertension* 1998;31:823-9.
52. Rossi GP, Seccia TM, Miotto D, et al. The Medical and Endovascular Treatment of Atherosclerotic Renal Artery Stenosis (METRAS) study: rationale and study design. *J Hum Hypertens* 2012;26:507-16.
53. Scarpioni R, Michieletti E, Cristinelli L, et al. Atherosclerotic renovascular disease: medical therapy versus medical therapy plus renal artery stenting in preventing renal failure progression: the rationale and study design of a prospective, multicenter and randomized trial (NITER). *J Nephrol* 2005;18:423-8.
54. Schwarzwald U, Hauk M, Zeller T. RADAR - A randomised, multi-centre, prospective study comparing best medical treatment versus best medical treatment plus renal artery stenting in patients with haemodynamically relevant atherosclerotic renal artery stenosis. *Trials* 2009;10:60.
55. Isles CG, Robertson S, Hill D. Management of renovascular disease: a review of renal artery stenting in ten studies. *QJM* 1999;92:159-67.
56. 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.
57. Weinberg I, Keyes MJ, Giri J, et al. Blood pressure response to renal artery stenting in 901 patients from five prospective multicenter FDA-approved trials. *Catheter Cardiovasc Interv* 2014;83:603-9.
58. Martin LG, Rees CR, O'Bryant T. Percutaneous angioplasty of the renal arteries. In: Strandness DE Jr, van Breda A., ed. *Vascular diseases: surgical and interventional therapy*. 1st ed. New York, NY: Churchill Livingstone; 1994:721-42.
59. Ritz E, Mann JF. Renal angioplasty for lowering blood pressure. *N Engl J Med* 2000;342:1042-3.
60. Sacks D, Rundback JH, Martin LG. Renal angioplasty/stent placement and hypertension in the year 2000. *J Vasc Interv Radiol* 2000;11:949-53.
61. Textor SC. Revascularization in atherosclerotic renal artery disease. *Kidney Int* 1998;53:799-811.
62. Ritchie J, Green D, Chrysochou C, Chalmers N, Foley RN, Kalra PA. High-risk clinical presentations in atherosclerotic renovascular disease: prognosis and response to renal artery revascularization. *Am J Kidney Dis* 2014;63:186-97.
63. Hayek SS, Abdou MH, Demoss BD, et al. Prevalence of resistant hypertension and eligibility for catheter-based renal denervation in hypertensive outpatients. *Am J Hypertens* 2013;26:1452-8.
64. Jaff MR, Bates M, Sullivan T, et al. Significant reduction in systolic blood pressure following renal artery stenting in patients with uncontrolled hypertension: results from the HERCULES trial. *Catheter Cardiovasc Interv* 2012;80:343-50.
65. Kumar N, Calhoun DA, Dudenbostel T. Management of patients with resistant hypertension: current treatment options. *Integr Blood Press Control* 2013;6:139-51.
66. Protasiewicz M, Kadziela J, Poczatek K, et al. Renal artery stenosis in patients with resistant hypertension. *Am J Cardiol* 2013;112:1417-20.
67. Textor SC. Renovascular hypertension: is there still a role for stent revascularization? *Curr Opin Nephrol Hypertens* 2013;22:525-30.
68. Summaria F, Patrizi R, Romagnoli E, Mustilli M, Pagnanelli A. Simultaneous transradial coronary and renal in stent restenosis treatment in diabetic patient with NSTEMI complicated by hypertensive emergency. *Med Arh* 2012;66:344-7.
69. Smit JV, Wierema TK, Kroon AA, de Leeuw PW. Blood pressure and renal function before and after percutaneous transluminal renal angioplasty in fibromuscular dysplasia: a cohort study. *J Hypertens* 2013;31:1183-8.
70. Trinquart L, Mounier-Vehier C, Sapoval M, Gagnon N, Plouin PF. Efficacy of revascularization for renal artery stenosis caused by fibromuscular dysplasia: a systematic review and meta-analysis. *Hypertension* 2010;56:525-32.
71. Olin JW, Gornik HL, Bacharach JM, et al. Fibromuscular dysplasia: state of the science and critical unanswered questions: a scientific statement from the American Heart Association. *Circulation*

2014;129:1048-78.

72. Davidson RA, Barri Y, Wilcox CS. Predictors of cure of hypertension in fibromuscular renovascular disease. *Am J Kidney Dis* 1996;28:334-8.
73. Harrison EG, Jr., McCormack LJ. Pathologic classification of renal arterial disease in renovascular hypertension. *Mayo Clin Proc* 1971;46:161-7.
74. Persu A, Touze E, Mousseaux E, Barral X, Joffre F, Plouin PF. Diagnosis and management of fibromuscular dysplasia: an expert consensus. *Eur J Clin Invest* 2012;42:338-47.
75. Cluzel P, Raynaud A, Beyssen B, Pagny JY, Gaux JC. Stenoses of renal branch arteries in fibromuscular dysplasia: results of percutaneous transluminal angioplasty. *Radiology* 1994;193:227-32.
76. Martin LG. Renal revascularization using percutaneous balloon angioplasty for fibromuscular dysplasia and atherosclerotic disease. In: Calligaro KD D, M J, ed. *Modern management of renovascular hypertension and renal salvage*. Philadelphia, Pa: Williams and Wilkins; 1996:125-44.
77. McKenzie GA, Oderich GS, Kawashima A, Misra S. Renal artery fibromuscular dysplasia in 2,640 renal donor subjects: a CT angiography analysis. *J Vasc Interv Radiol* 2013;24:1477-80.
78. Hendricks NJ, Matsumoto AH, Angle JF, et al. Is fibromuscular dysplasia underdiagnosed? A comparison of the prevalence of FMD seen in CORAL trial participants versus a single institution population of renal donor candidates. *Vasc Med* 2014;19:363-7.
79. Olin JW, Froehlich J, Gu X, et al. The United States Registry for Fibromuscular Dysplasia: results in the first 447 patients. *Circulation* 2012;125:3182-90.
80. Narula N, Kadian-Dodov D, Olin JW. Fibromuscular Dysplasia: Contemporary Concepts and Future Directions. *Prog Cardiovasc Dis* 2018;60:580-85.
81. Johnston SL, Lock RJ, Gompels MM. Takayasu arteritis: a review. *J Clin Pathol* 2002;55:481-6.
82. Hall S, Barr W, Lie JT, Stanson AW, Kazmier FJ, Hunder GG. Takayasu arteritis. A study of 32 North American patients. *Medicine (Baltimore)* 1985;64:89-99.
83. Delles C, Weidner S, Schobel HP, Rupprecht HD. Renal-artery stenosis in a patient with Takayasu's arteritis. *Nephrol Dial Transplant* 2002;17:1339-41.
84. Qi Y, Yang L, Zhang H, et al. The presentation and management of hypertension in a large cohort of Takayasu arteritis. *Clin Rheumatol* 2018;37:2781-88.
85. Chaudhry MA, Latif F. Takayasu's arteritis and its role in causing renal artery stenosis. *Am J Med Sci* 2013;346:314-8.
86. Park HS, Do YS, Park KB, et al. Long term results of endovascular treatment in renal arterial stenosis from Takayasu arteritis: angioplasty versus stent placement. *Eur J Radiol* 2013;82:1913-8.
87. Kinjo H, Kafa A. The results of treatment in renal artery stenosis due to Takayasu disease: comparison between surgery, angioplasty, and stenting. A monocentric retrospective study. *G Chir* 2015;36:161-7.
88. Peng M, Ji W, Jiang X, et al. Selective stent placement versus balloon angioplasty for renovascular hypertension caused by Takayasu arteritis: Two-year results. *Int J Cardiol* 2016;205:117-23.
89. Saadoun D, Lambert M, Mirault T, et al. Retrospective analysis of surgery versus endovascular intervention in Takayasu arteritis: a multicenter experience. *Circulation* 2012;125:813-9.
90. Pellerin O, Garcon P, Beyssen B, et al. Spontaneous renal artery dissection: long-term outcomes after endovascular stent placement. *J Vasc Interv Radiol* 2009;20:1024-30.
91. Stawicki SP, Rosenfeld JC, Weger N, Fields EL, Balshi JD. Spontaneous renal artery dissection: three cases and clinical algorithms. *J Hum Hypertens* 2006;20:710-8.
92. Mitch WE, Walser M, Buffington GA, Lemann J, Jr. A simple method of estimating progression of chronic renal failure. *Lancet* 1976;2:1326-8.
93. van Rooden CJ, van Bockel JH, De Backer GG, Hermans J, Chang PC. Long-term outcome of surgical revascularization in ischemic nephropathy: normalization of average decline in renal function. *J Vasc Surg* 1999;29:1037-49.
94. Watson PS, Hadjipetrou P, Cox SV, Piemonte TC, Eisenhauer AC. Effect of renal artery stenting on renal function and size in patients with atherosclerotic renovascular disease. *Circulation* 2000;102:1671-7.
95. Alfke H, Radermacher J. Renal artery stenting is no longer indicated after ASTRAL: pros and cons. *Cardiovasc Intervent Radiol* 2010;33:883-6.
96. Bommart S, Cliche A, Therasse E, et al. Renal artery revascularization: predictive value of kidney length and volume weighted by resistive index. *AJR Am J Roentgenol* 2010;194:1365-72.
97. Valluri A, Severn A, Chakraverty S. Do patients undergoing renal revascularization outside of the ASTRAL

- trial show any benefit? Results of a single-centre observational study. *Nephrol Dial Transplant* 2012;27:734-8.
98. Dean RH, Tribble RW, Hansen KJ, O'Neil E, Craven TE, Redding JF, 2nd. Evolution of renal insufficiency in ischemic nephropathy. *Ann Surg* 1991;213:446-55; discussion 55-6.
 99. Muray S, Martin M, Amoedo ML, et al. Rapid decline in renal function reflects reversibility and predicts the outcome after angioplasty in renal artery stenosis. *Am J Kidney Dis* 2002;39:60-6.
 100. Manoharan G, Pijls NH, Lameire N, et al. Assessment of renal flow and flow reserve in humans. *J Am Coll Cardiol* 2006;47:620-5.
 101. Gal-Oz A, Wolf YG, Rosen G, Sharon H, Schwartz IF, Chernin G. When the chimney is blocked: malignant renovascular hypertension after endovascular repair of abdominal aortic aneurysm. *BMC Nephrol* 2013;14:71.
 102. Saad A, Herrmann SM, Crane J, et al. Stent revascularization restores cortical blood flow and reverses tissue hypoxia in atherosclerotic renal artery stenosis but fails to reverse inflammatory pathways or glomerular filtration rate. *Circ Cardiovasc Interv* 2013;6:428-35.
 103. Simone TA, Brooke BS, Goodney PP, et al. Clinical effectiveness of secondary interventions for restenosis after renal artery stenting. *J Vasc Surg* 2013;58:687-94.
 104. Baril DT, Rhee RY, Kim J, Makaroun MS, Chaer RA, Marone LK. Duplex criteria for determination of in-stent stenosis after angioplasty and stenting of the superficial femoral artery. *J Vasc Surg* 2009;49:133-8; discussion 39.
 105. Stone PA, Campbell JE, Aburahma AF, et al. Ten-year experience with renal artery in-stent stenosis. *J Vasc Surg* 2011;53:1026-31.
 106. Takahashi EA, Kallmes DF, Fleming CJ, et al. Predictors and Outcomes of Postcontrast Acute Kidney Injury after Endovascular Renal Artery Intervention. *J Vasc Interv Radiol* 2017;28:1687-92.
 107. Modrall JG, Timaran CH, Rosero EB, et al. Longitudinal changes in kidney parenchymal volume associated with renal artery stenting. *J Vasc Surg* 2012;55:774-80; discussion 80.
 108. Bloch MJ, Trost DW, Pickering TG, Sos TA, August P. Prevention of recurrent pulmonary edema in patients with bilateral renovascular disease through renal artery stent placement. *Am J Hypertens* 1999;12:1-7.
 109. Jaff MR. Management of Atherosclerotic Renal Artery Stenosis: Interventional Versus Medical Therapy. *Curr Interv Cardiol Rep* 2001;3:93-99.
 110. Messina LM, Zelenock GB, Yao KA, Stanley JC. Renal revascularization for recurrent pulmonary edema in patients with poorly controlled hypertension and renal insufficiency: a distinct subgroup of patients with arteriosclerotic renal artery occlusive disease. *J Vasc Surg* 1992;15:73-80; discussion 80-2.
 111. Rundback JH, Sacks D, Kent KC, et al. Guidelines for the reporting of renal artery revascularization in clinical trials. *J Vasc Interv Radiol* 2003;14:S477-92.
 112. Chrysochou C, Schmitt M, Siddals K, Hudson J, Fitchet A, Kalra PA. Reverse cardiac remodelling and renal functional improvement following bilateral renal artery stenting for flash pulmonary oedema. *Nephrol Dial Transplant* 2013;28:479-83.
 113. Khosla S, White CJ, Collins TJ, Jenkins JS, Shaw D, Ramee SR. Effects of renal artery stent implantation in patients with renovascular hypertension presenting with unstable angina or congestive heart failure. *Am J Cardiol* 1997;80:363-6.
 114. Wright JR, Shurrab AE, Cooper A, Kalra PR, Foley RN, Kalra PA. Progression of cardiac dysfunction in patients with atherosclerotic renovascular disease. *QJM* 2009;102:695-704.
 115. McMahan CJ, Hennessy M, Boyle G, Feely J, Meaney JF. Prevalence of renal artery stenosis in flash pulmonary oedema: determination using gadolinium-enhanced MRA. *Eur J Intern Med* 2010;21:424-8.
 116. Messerli FH, Bangalore S, Makani H, et al. Flash pulmonary oedema and bilateral renal artery stenosis: the Pickering syndrome. *Eur Heart J* 2011;32:2231-5.
 117. van den Berg DT, Deinum J, Postma CT, van der Wilt GJ, Rixsen NP. The efficacy of renal angioplasty in patients with renal artery stenosis and flash oedema or congestive heart failure: a systematic review. *Eur J Heart Fail* 2012;14:773-81.
 118. Rocha-Singh KJ, Novack V, Pencina M, et al. Objective performance goals of safety and blood pressure efficacy for clinical trials of renal artery bare metal stents in hypertensive patients with atherosclerotic renal artery stenosis. *Catheter Cardiovasc Interv* 2011;78:779-89.
 119. Zanolli L, Rastelli S, Marcantoni C, et al. Renal artery diameter, renal function and resistant hypertension in patients with low-to-moderate renal artery stenosis. *J Hypertens* 2012;30:600-7.

120. Baumgartner I, von Aesch K, Do DD, Triller J, Birrer M, Mahler F. Stent placement in ostial and nonostial atherosclerotic renal arterial stenoses: a prospective follow-up study. *Radiology* 2000;216:498-505.
121. Parenti GC, Palmarini D, Bilzoni M, Campioni P, Mannella P, Ginevra A. Role of color-Doppler sonography in the follow-up of renal artery stenting. *Radiol Med* 2008;113:242-8.
122. Sharafuddin MJ, Raboi CA, Abu-Yousef M, Lawton WJ, Gordon JA. Renal artery stenosis: duplex US after angioplasty and stent placement. *Radiology* 2001;220:168-73.
123. Behar JV, Nelson RC, Zidar JP, DeLong DM, Smith TP. Thin-section multidetector CT angiography of renal artery stents. *AJR Am J Roentgenol* 2002;178:1155-9.
124. Steinwender C, Schutzenberger W, Fellner F, et al. 64-Detector CT angiography in renal artery stent evaluation: prospective comparison with selective catheter angiography. *Radiology* 2009;252:299-305.
125. Jundt MC, Takahashi EA, Harmsen WS, Misra S. Restenosis Rates After Drug-Eluting Stent Treatment for Stenotic Small-Diameter Renal Arteries. *Cardiovasc Intervent Radiol* 2019;42:1293-301.
126. Jahraus CD, St Clair W, Gurley J, Meigooni AS. Endovascular brachytherapy for the treatment of renal artery in-stent restenosis using a beta-emitting source: a report of five patients. *South Med J* 2003;96:1165-8.
127. Patel PM, Eisenberg J, Islam MA, Maree AO, Rosenfield KA. Percutaneous revascularization of persistent renal artery in-stent restenosis. *Vasc Med* 2009;14:259-64.
128. Stoeteknuel-Friedli S, Do DD, von Briel C, Triller J, Mahler F, Baumgartner I. Endovascular brachytherapy for prevention of recurrent renal in-stent restenosis. *J Endovasc Ther* 2002;9:350-3.
129. Zeller T, Sixt S, Rastan A, et al. Treatment of reoccurring instent restenosis following reintervention after stent-supported renal artery angioplasty. *Catheter Cardiovasc Interv* 2007;70:296-300.
130. Martin LG, Casarella WJ, Alspaugh JP, Chuang VP. Renal artery angioplasty: increased technical success and decreased complications in the second 100 patients. *Radiology* 1986;159:631-4.
131. Peng M, Dong H, Jiang X, et al. A randomized unblinded trial to compare effects of intensive versus conventional lipid-lowering therapy in patients undergoing renal artery stenting. *J Cardiol* 2019;74:443-50.
132. American College of Radiology. ACR–SIR–SPR practice parameter for performance of arteriography. Available at: <https://www.acr.org/-/media/ACR/Files/Practice-Parameters/Arteriog.pdf?la=en>. Accessed January 31, 2019.
133. American College of Radiology. ACR practice parameter for continuing medical education (CME). Available at: <https://www.acr.org/-/media/ACR/Files/Practice-Parameters/CME.pdf?la=en>. Accessed January 31, 2019.
134. American College of Radiology. ACR–AAPM technical standard for diagnostic medical physics performance monitoring of fluoroscopic equipment. Available at: <https://www.acr.org/-/media/ACR/Files/Practice-Parameters/Fluoro-Equip.pdf?la=en>. Accessed January 31, 2019.
135. Cardella JF, Casarella WJ, DeWeese JA, et al. Optimal resources for the examination and endovascular treatment of the peripheral and visceral vascular systems. AHA Intercouncil Report on Peripheral and Visceral Angiographic and Interventional Laboratories. *J Vasc Interv Radiol* 2003;14:S517-30.
136. Baerlocher MO, Kennedy SA, Ward TJ, et al. Society of Interventional Radiology Position Statement: Staffing Guidelines for the Interventional Radiology Suite. *J Vasc Interv Radiol* 2016;27:618-22.
137. Baerlocher MO, Kennedy SA, Ward TJ, et al. Society of Interventional Radiology: Resource and Environment Recommended Standards for IR. *J Vasc Interv Radiol* 2017;28:513-16.
138. American College of Radiology. ACR–SIR practice parameter for sedation/analgesia. Available at: <https://www.acr.org/-/media/ACR/Files/Practice-Parameters/Sed-Analgesia.pdf?la=en>. Accessed January 31, 2019.
139. American College of Radiology. ACR–SIR–SPR practice parameter on informed consent for image-guided procedures. Available at: <https://www.acr.org/-/media/ACR/Files/Practice-Parameters/InformedConsent-ImagGuided.pdf?la=en>. Accessed January 31, 2019.
140. American College of Radiology. ACR–SIR–SPR practice parameter for the reporting and archiving of interventional radiology procedures. Available at: <https://www.acr.org/-/media/ACR/Files/Practice-Parameters/Reporting-Archiv.pdf?la=en>. Accessed January 31, 2019.
141. 1999 World Health Organization-International Society of Hypertension Guidelines for the Management of Hypertension. Guidelines Subcommittee. *J Hypertens* 1999;17:151-83.
142. The sixth report of the Joint National Committee on prevention, detection, evaluation, and treatment of high blood pressure. *Arch Intern Med* 1997;157:2413-46.
143. Whelton PK, Carey RM, Aronow WS, et al. 2017

ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults: Executive Summary: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *J Am Coll Cardiol* 2018;71:2199-269.

144. Kaatee R, Beek FJ, Verschuyt EJ, et al. Atherosclerotic renal artery stenosis: ostial or truncal? *Radiology* 1996;199:637-40.
145. Wang L, Li NF, Zhou KM, et al. [Etiology analysis of 628 patients with refractory hypertension]. *Zhonghua Xin Xue Guan Bing Za Zhi* 2009;37:138-41.
146. Sacks D, Marinelli DL, Martin LG, Spies JB. Reporting standards for clinical evaluation of new peripheral arterial revascularization devices. Technology Assessment Committee. *J Vasc Interv Radiol* 1997;8:137-49.

*Practice parameters and technical standards are published annually with an effective date of October 1 in the year in which amended, revised, or approved by the ACR Council. For practice parameters and technical standards published before 1999, the effective date was January 1 following the year in which the practice parameter or technical standard was amended, revised, or approved by the ACR Council.

Development Chronology for this Practice Parameter

2004 (Resolution 26)

Amended 2006 (Resolution 16g, 17, 34, 35, 36)

Amended 2007 (Resolution 12m, 38)

Revised 2009 (Resolution 26)

Amended 2014 (Resolution 39)

Revised 2015 (Resolution 22)

Amended 2019 (Resolution 23)

Amended 2020 (Resolution 8)

Revised 2021 (Resolution 6)

Amended 2023 (Resolution 2c, 2d)

APPENDIX A

DEFINITIONS

For the purpose of this practice parameter, the following definitions apply:

Hypertension: HTN is defined by the 2003 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" [30,141]. In 2014, the eighth [27] report of The Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure defined HTN as "systolic blood pressure 140 mm Hg or greater, diastolic blood pressure 90 mm Hg or greater, or taking antihypertensive medication" [142]. However, 2018 recommendations by the American College of Cardiology and the American Heart Association now define stage 1 HTN as a systolic blood pressure of 130 to 139 mm Hg or a diastolic blood pressure of 80 to 89 mm Hg [143]. Any of these definitions would be appropriate.

Accelerated hypertension: Sudden worsening of previously controlled HTN, which may indicate the development of a secondary cause of HTN [30].

Resistant hypertension: HTN should be considered resistant if the systolic blood pressure (SBP) 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 3 drugs prescribed in near maximal doses. For patients older than age 60 with isolated systolic HTN, resistance is defined as failure of an adequate triple-drug regimen to reduce the SBP to below 160 mm Hg [30].

Cardiac disturbance syndrome: Recurrent "flash" pulmonary edema, not felt to be secondary to impaired cardiac function. This can be seen in the setting of bilateral RAS or unilateral stenosis of the renal artery in the setting of

a solitary kidney [30,108,113,142].

Hypertensive crisis: According to AHA guidelines, "Hypertensive crises can present as hypertensive urgency or as a hypertensive emergency."

Hypertensive urgency: SBP of 180 mm Hg or greater, or diastolic blood pressure of 100 mm Hg or greater. There may be associated headache, shortness of breath, nosebleeds, or anxiety.

Hypertensive emergency: Hypertensive urgency plus the coexistence of end-organ damage, which may include retinal hemorrhage, stroke, angina, myocardial infarction, aortic dissection, or pulmonary edema.

Malignant hypertension: HTN with end-organ damage, including left ventricular hypertrophy (LVH), congestive heart failure (CHF), visual or neurologic disturbance, or advanced retinopathy [30].

Renal artery stenosis (RAS): Anatomic narrowing of the renal artery lumen diameter by 50% or greater, expressed in this practice parameter as a percentage of the diameter of a normal renal vessel, namely, % RAS = $100 \times (1 - (\text{the narrowed lumen diameter} / \text{the normal vessel diameter}))$ [30].

Ostial RAS: Anatomic narrowing within the proximal 5 mm of the artery. Lesions within 10 mm of the aorta may also be considered ostial, when atheromatous plaque increases the distance between the extra-aortic renal artery and the aortic lumen on cross-sectional imaging [30,144].

Truncal RAS: Nonostial RAS occurring proximal to renal artery branching [30].

Renovascular hypertension: RVH is secondary HTN due to activation of the renin-angiotensin system by a hemodynamically significant RAS [30,145].

Ischemic nephropathy (IN): Renal vascular compromise leading to decreased estimated glomerular filtration rate (eGFR) without evidence of a medical cause. There may or may not be evidence of decreasing renal parenchyma. Renal revascularization: Any procedure that restores unobstructed arterial blood flow to the kidney [30].

Technically successful endovascular renal revascularization: Less than 30% residual stenosis measured at the narrowest point of the vascular lumen and pressure gradient 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 [30,146].

Clinical Success in the Endovascular Treatment of Renal Vascular Hypertension or Ischemia:

Cure: Restoration of blood pressure below 140/90 mm Hg and no longer taking antihypertensive medications. For renal insufficiency, a cure would be return of eGFR to normal baseline levels [30].

Partial response: Reduced blood pressure by 10 mm Hg systolic or diastolic on the same medications, or a comparable blood pressure on a reduced number or dose of medications after renal intervention. For renal insufficiency, improvement, or stabilization of eGFR is a partial response [57].

APPENDIX B

METHODS

A literature search was performed for English-language articles published through January 2018 with the following keywords: renovascular hypertension, renal artery stent (RAS), renal artery denervation, drug-eluting stents (DES), paclitaxel or renal artery angioplasty complications. Randomized trials in adult populations, except those relating to congenital or inherited disorders, were selected for review. In developing a consensus document, the authors also reviewed case series, case reports, and expert opinion titles for relevance in answering the following questions: indications for renal artery imaging and renal angiography, indications for

percutaneous renal artery intervention, procedure techniques, patient management, outcomes of renal artery
Revised 2021 (Resolution 6)
angioplasty and stenting, qualifications for operators, and facilities required to safely perform these procedures.