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The pathophysiology of atherosclerotic renal artery stenosis (RAS) includes activation of the renin-angiotensin-aldosterone axis with resultant renovascular hypertension. Renal artery stenting has emerged as the primary revascularization strategy in most patients with hemodynamically significant atherosclerotic RAS. Despite the frequency with which hemodynamically significant RAS is observed and high rates of technical success of renal artery stenting, there remains considerable debate among experts regarding the role of medical therapy versus revascularization for renovascular hypertension. Modern, prospective, multicenter registries continue to demonstrate improvement in systolic and diastolic blood pressure with excellent safety profiles in patients with RAS. Modern randomized, controlled clinical trials of optimal medical therapy versus renal stenting particularly designed to demonstrate preservation in renal function after renal artery stenting have demonstrated limited benefit. However, these trials frequently excluded patients that may benefit from renal artery stenting. This document was developed to guide physicians in the modern practical application of renal stenting, to highlight the current limitations in the peer-reviewed literature, to suggest best-practices in the performance of renal stenting and to identify opportunities to advance the field.

Key words: renovascular hypertension; renal artery stenosis; stent

INTRODUCTION

The pathophysiology of renovascular hypertension because of stenosis of the renal arteries has been understood for over 50 years. Impairment of renal arterial blood flow results in activation of the renin-angiotensin-aldosterone axis with sequelae that include: vasoconstriction, sodium and water retention, aldosterone secretion, sympathetic nervous system activation, vascular

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2MRJ Massachusetts General Hospital, Boston, Massachusetts
3BHG University of South Carolina School of Medicine/ Greenville, Greenville, South Carolina
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5CJW The University of Queensland and the John Ochsner Heart & Vascular Institute, Ochsner Medical Center, New Orleans, Louisiana

MRJ: Non-compensated advisor: Abbott Vascular; Boston Scientific; Cordis Corporation; Covidien Vascular; Medtronic Vascular.

Conflict of interest: Nothing to report.

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Received 17 May 2014; Revision accepted 25 May 2014

DOI: 10.1002/ccd.25559

Published online 00 Month 2014 in Wiley Online Library (wileyonlinelibrary.com)
remodeling, and resultant hypertension (Fig. 1) [1]. The majority (>90%) of cases of renal artery stenosis (RAS) result from atherosclerosis. Despite the frequency with which hemodynamically significant RAS is found, particularly among patients with coronary artery disease, there remains considerable debate among experts regarding the role of medical therapy versus revascularization for renovascular hypertension.

Renal artery stent revascularization (renal artery stenting) has emerged as the primary revascularization strategy in most patients with hemodynamically significant atherosclerotic RAS [2]. Stent placement, with or without predilation with percutaneous transluminal angioplasty, has become the preferred endovascular technique [3]. Modern, prospective, multicenter registries continue to demonstrate improvement in systolic and diastolic blood pressure (SBP, DBP) with excellent safety profiles. However, because of their nonrandomized design, there has not been widespread acceptance of the benefits of renal artery stenting [4]. Modern randomized clinical trials of optimal medical therapy (OMT) versus renal stenting, particularly designed to demonstrate preservation in renal function have been plagued by serious methodological flaws in study design and execution [5]. The recently published CORAL trial, a prospective multicenter randomized controlled clinical trial which took a decade to complete likely excluded patients who may have gained benefit from renal artery stenting [6].

This document was developed to guide physicians in the modern practical application of renal stenting, to highlight the current limitations in the peer-reviewed literature, and to identify opportunities to advance the field.

ANATOMIC CONSIDERATIONS (DIAGNOSTIC TESTING)

The majority of RAS cases are because of atherosclerosis (Table I). Typical lesions involve the aorto-ostial junction or proximal segment of the renal artery. RAS because of fibromuscular dysplasia, vasculitis, or trauma are infrequently encountered, and are not covered in this manuscript.

The diagnosis of hemodynamically significant RAS is critical to determining optimal therapy. A physical examination provides few specific clues to the presence of RAS except for the rare systolic/diastolic abdominal bruit radiating to the flank region. However, in patients with peripheral artery disease (PAD) or multi-vessel coronary artery disease (CAD), there is an increased association with hemodynamically significant RAS. In patients with significant CAD, the coexistent incidence of RAS observed at coronary angiography is approximately 20%, with the incidence rising in patients with higher burdens of extracoronary atherosclerosis [7,8].

In patients in whom there is a high clinical suspicion for RAS (Table II), and who are considered potential candidates for revascularization, a diagnostic evaluation for RAS should be undertaken [9]. A concise review of the diagnostic modalities can be found in the recently updated multisocietal guidelines [10].

Renal artery duplex ultrasonography (RADUS), which utilizes no radiation, is highly sensitive and specific, inexpensive, and can be repeated without risk or discomfort to the patient, remains an important diagnostic

### TABLE I. Common Causes of Renal Artery Stenosis

- Atherosclerotic renal artery stenosis
- Fibromuscular dysplasia
- Nephroangiosclerosis (Hypertensive injury)
- Diabetic nephropathy (small vessels)
- Renal thromboembolic disease
- Ateroembolic renal disease
- Aortorenal dissection
- Renal artery vasculitis
- Trauma
- Neurofibromatosis
- Thromboangiitis obliterans
- Scleroderma
- Extrinsic compression

### TABLE II. Clinical Clues Suggestive of Renal Artery Stenosis

- Onset of hypertension at <30 years of age or severe hypertension at >55 years of age
- Accelerated, resistant, or malignant hypertension
- Unexplained atrophic kidney or size discrepancy >1.5 cm between kidneys
- Sudden, unexplained pulmonary edema
- Unexplained renal dysfunction, including individuals starting renal replacement therapy
- Development of new azotemia or worsening renal function after administration of an ACE inhibitor or ARB agent
- Multivessel coronary artery disease or peripheral artery disease
- Unexplained congestive heart failure or refractory angina

Adapted from Ref. [10]
### TABLE III. Assessing Significance of Renal Artery Stenosis

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<th>Angiographic Stenosis Severity</th>
<th>Physiologic Testing</th>
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<td>&lt;50%</td>
<td>None</td>
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<td>50–70% with</td>
<td>Resting mean pressure gradient &gt; 10 mm Hg</td>
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<tr>
<td>50–70% with</td>
<td>Systolic hyperemic pressure gradient &gt; 20 mm Hg†</td>
<td>Significant</td>
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<tr>
<td>50–70% with</td>
<td>Renal Pd/Pa ≤ 0.8‡</td>
<td>Significant</td>
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<tr>
<td>&gt;70%</td>
<td>None</td>
<td>Significant</td>
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*Visual estimation.
*Translesional gradient measured with a nonobstructive catheter, ie ≤ 4 French or with an 0.014-in pressure wire (Pd/Pa).
*Hyperemia may be induced with intrarenal bolus of papaverine 30 mg or dopamine at 50 μg/kg [11,13,14].

Renal angiography is the gold standard for the invasive assessment of hemodynamically significant RAS. Angiographic stenosis severity can be simply categorized as: mild (<50%), moderate (50–70%), and severe (>70%). However, such assessments may not accurately define hemodynamically significant stenosis [10], and only hemodynamically significant RAS should be considered for renal stenting. Angiographic stenoses >70% are considered to be severe lesions and hemodynamically significant. Moderate angiographic stenoses between 50% and 70% may or may not be hemodynamically significant, and should have further confirmation of their hemodynamic severity prior to intervention. Expert consensus and experimental evidence have determined that hemodynamic severity is present when there exists a resting translesional mean pressure gradient of >10 mm Hg, a hyperemic peak systolic pressure gradient of >20 mm Hg or renal fractional flow reserve (FFR) ≤ 0.8 [11–14]. Angiographic stenoses <50% are mild, are not considered hemodynamically significant, and rarely warrant consideration for revascularization (Table III). The technical aspects of these measurements are discussed in Table III and the Technical Considerations section below.

### Clinical Scenarios in Which Treatment of Significant RAS May Be Considered

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<th>Appropriate Care</th>
<th>Clinical Scenarios</th>
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<th>Appropriate Care</th>
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<tr>
<td>Cardiac Disturbance Syndromes (Flash Pulmonary Edema or acute coronary syndrome (ACS)) with severe hypertension</td>
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<tr>
<td>Resistant HTN (Uncontrolled hypertension with failure of maximally tolerated doses of at least three antihypertensive agents, one of which is a diuretic, or intolerance to medications)</td>
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<td>Ischemic nephropathy with chronic kidney disease (CKD) with eGFR &lt; 45 cc/min and global renal ischemia (unilateral significant RAS with a solitary kidney or bilateral significant RAS)</td>
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<td>without other explanation</td>
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<td>Unilateral RAS with CKD (eGFR &lt; 45 cc/min)</td>
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<td>Unilateral RAS with prior episodes of congestive heart failure (Stage C)</td>
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<td>Anatomically challenging or high risk lesion (early bifurcation, small vessel, severe concentric calcification, and severe aortic atheroma or mural thrombus)</td>
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<td>Unilateral, Solitary, or Bilateral RAS with controlled BP and normal renal function.</td>
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<td>Unilateral, solitary, or bilateral RAS with kidney size &lt;7 cm in pole-to-pole length</td>
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<td>Unilateral, Solitary, or Bilateral RAS with chronic end stage renal disease on hemodialysis &gt; 3 months.</td>
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<td>Unilateral, Solitary, or Bilateral renal artery chronic total occlusion</td>
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*Significant RAS is an angiographically moderate lesion (50–70%) with physiologic confirmation of severity or a >70% stenosis (see Table III).
A recent meta-analysis of six randomized controlled trials (RCTs), that evaluated the safety and efficacy of renal stenting to either treat hypertension or to delay progression of renal ischemia, showed no improvement in renal function (as measured by serum creatinine or reciprocal of the serum creatinine) or clinical outcomes with stenting, compared to OMT [16]. Despite these findings, many experts agree that the RCTs conducted to date had major flaws in design, patient selection, lesion severity, and sample size, thus limiting their clinical applicability [17,18]. For example, in ASTRAL, the largest of these trials, only 40% of patients had a stenosis between 50% and 70%, and the high-risk patients felt most likely to benefit from renal stenting (i.e. recurrent “flash” pulmonary edema) were excluded [17–19].

In the recently concluded Cardiovascular Outcomes in Renal Atherosclerotic Lesions (CORAL) Trial, patients with RAS and hypertension with SBP of greater than 155 mm Hg or higher while taking two or more antihypertensive drugs with angiographic RAS of at least 60% with evidence of a translesional gradient greater than 20 mm Hg or angiographic severity of greater than 80% (but less than 100% stenosis) were randomized to OMT versus renal artery stenting [6]. The primary endpoint was a composite of cardiovascular or renal death, stroke, myocardial infarction, hospitalization for congestive heart failure (CHF), progressive renal insufficiency, or the need for permanent renal replacement therapy. In 947 patients enrolled in the trial, there was no statistically significant difference observed in the primary endpoint (35.1% stent group vs 35.8% medical therapy group) with both groups demonstrating nearly a 15 mm Hg reduction in blood pressure over the course of the study. CORAL confirms that first line therapy for patients with RAS and hypertension is OMT. However, CORAL did not evaluate those who failed OMT, and many patients were not eligible for inclusion in the trial. Therefore, there are many patients commonly found in clinical practice whose management remains uncertain, and it is for those that this document is designed.

It is clear that anatomic findings of RAS in isolation do not necessarily result in any clinical syndrome, including renovascular hypertension chronic kidney disease. However, when considering renal artery stenting, and given the limitations of RCTs to date, we encourage clinicians to use the steps outlined in this document in conjunction with the recent multisocietal guidelines to guide their management strategy.

Renal Artery Stenting Represents Appropriate Care

The strongest evidence supporting renal artery stenting for RAS is in patients with the presence of a cardiac disturbance syndrome or “flash” pulmonary edema [10,20]. We agree with the multisocietal guidelines which provide a Class I recommendation (Level of Evidence (LOE) B) in this subset of patients, in which a variety of physiologic mechanisms play a role [10]. Patients with severe bilateral RAS or stenosis to a solitary functioning kidney may lack adequate renal sodium handling capacity to generate “pressure...
natriuresis” to autoregulate BP, or they may demonstrate inappropriate peripheral vasoconstriction resulting in abrupt increases in afterload and resultant myocardial ischemia or heart failure. Each of these mechanisms, when managed with renal artery stenting, have resulted in clinical improvement in case series where such patients are carefully selected [21–23]. It must be noted that in CORAL, 20% of patients randomized to stent and 16% randomized to medical therapy had global renal ischemia, and there was no statistical difference in hospitalization for heart failure [6].

Patients with accelerated or resistant hypertension (failure of ≥3 maximally tolerated medications including the use of a diuretic), global renal ischemia (bilateral RAS or severe RAS in a solitary functioning kidney), or hypertension with medication intolerance also generally benefit from renal artery stenting after a trial of OMT [10,20]. Multiple, relatively small, prospective, and retrospective series have shown benefit from renal artery stenting minimizing recurrent symptoms and end organ injury. The multisocietal guidelines provide a Class II a (LOE B) recommendation in this subset of patients, though we feel more strongly that renal artery stenting in these patients is generally appropriate, particularly in light of more recent data that use the same definition of accelerated or resistant hypertension. De Bruyne and colleagues demonstrated a threshold severity for renal vein renin release determined by a ratio of translesional to aortic pressure (Pd/Pa) of <0.9 [13]. Patients with the highest baseline systolic blood pressures will have the greatest decrease in systolic pressure. There has been no correlation between blood pressure improvement after renal stenting and the variables of age, sex, race, severity of stenosis, number of vessels treated, baseline diastolic pressure, or baseline serum creatinine [24]. In a pooled analysis of 901 patients enrolled in five prospective investigational device exemption trials, systolic blood pressure >150 mm Hg (OR = 4.09, CI = 2.74-6.12, P < 0.0001) was positively associated with BP response following renal artery stent revascularization [25]. Multiple, prospective, multicenter nonrandomized trials have consistently demonstrated a significant improvement in blood pressure control following renal stenting in medically refractory patients [4].

For patients with progressive deterioration in renal function and global renal ischemia without another etiology for chronic kidney disease, clinical case series have demonstrated a significant reduction in the rate of loss of renal function in patients undergoing renal stenting [26–28]. Therefore, we agree with the multisocietal guidelines that patients with global renal ischemia and declining renal function may benefit from renal stenting and that such therapy represents appropriate care.

Renal Stenting May Represent Appropriate Care

There are a number of common clinical scenarios in which renal stenting for RAS remain controversial and where the data are inconclusive. In our opinion these scenarios often pose the greatest challenge to clinicians, and therefore require an individualized patient approach, particularly given the lack of conclusive evidence. The data for preservation of renal parenchymal function in patients at high risk for progressive ischemic nephropathy, particularly those with chronic kidney disease, suggests that revascularization may stabilize renal function [29–32]. However, as noted previously, generalizable results from RCTs designed to answer this question are limited [17,18]. Based on observational studies it appears that higher risk patients, such as those with global renal ischemia and eGFR <45 cc/min, including individuals with a solitary functioning kidney, may gain the greatest benefit from renal stenting [10]. The multisocietal guidelines offer a Class II b (LOE C) recommendation for revascularization in such patients, and as such, we agree that renal stenting may be appropriate in carefully selected patients.

Recent data suggest that RAS induces secretion of paracrine effectors that activate myocardial hypertrophic response genes and may have deleterious long-term impact upon the clinical course of heart failure. It remains speculative if renal artery stenting may reverse this cascade [33]. Renal stenting of patients with hemodynamically significant unilateral RAS with prior episodes of congestive heart failure (Stage C) without a primary cardiac etiology for such may represent appropriate care in selected patients.

In patients with challenging or anatomically difficult or high-risk renal lesions (i.e. early bifurcation, small (< 3.0 cm) diameter vessels, in vessels with severe concentric calcification, in patients with diffuse aortic atherosclerosis or mural thrombus, and those in which RAS is seen in conjunction with a renal artery aneurysm or juxtarenal abdominal aortic aneurysm), the risk-to-benefit ratio will depend on individual patient circumstances and individual operator skill, making these cases indeterminate for appropriate use.

Renal Stenting Rarely Represents Appropriate Care

While there are scenarios in which revascularization for RAS remains controversial, it is clear that hemodynamically mild to moderate stenoses (e.g. peak to peak translesional gradient < 20 mm Hg, mean translesional
gradient <10 mm Hg or renal FFR >0.8) do not merit revascularization. Given the inaccuracy of invasive angiography in determining the physiologic significance of moderate RAS, it would be rarely appropriate for an intervention to be performed on an angiographically moderate 50% to 70% diameter stenosis without hemodynamic confirmation of the severity of the lesion [11–14,34]. Patients with long standing ischemic nephropathy, such as those requiring chronic hemodialysis for greater than three months or in those with marked renal atrophy (< 7 cm pole to pole), are not likely to benefit from revascularization [35,36]. Similarly, chronic total occlusions of renal arteries do not warrant revascularization.

TECHNICAL CONSIDERATIONS (PERFORMANCE OF RENAL STENTING)

Preparation for Angiography
Renal angiography is justified when there is an appropriate clinical indication for renal artery revascularization and, in most clinical scenarios, the presence of RAS has been confirmed by a noninvasive evaluation or when noninvasive imaging is nondiagnostic, confirmed by arteriography. Performance of renal angiography in a patient without an indication for revascularization is not advised, as this practice may lead to inappropriate revascularization and unwarranted complications. Prior to the procedure, all noninvasive studies should be reviewed for the presence of aortic atherosclerosis, accessory renal arteries, location of the RAS, angulation of the renal arteries, the presence of fibromuscular dysplasia, and pole-to-pole kidney size. Additional information that may alter the procedural approach include the presence of an abdominal aortic aneurysm, accessibility renal arteries, location of the RAS, angulation of the renal arteries, the presence of fibromuscular dysplasia, and pole-to-pole kidney size. Additional information that may alter the procedural approach include the presence of an abdominal aortic aneurysm with or without mural thrombus, aortic calcification, and iliac artery atherosclerotic disease. This information will influence the choice of access (radial, brachial, or femoral) for both diagnostic angiography and revascularization.

Performance of Renal Angiography
The classic approach of performing abdominal aortography, followed by selective renal angiography, is safe and effective. The use of the radial artery for vascular access may be considered to reduce the risk of procedural access site complications. However, when performing renal stenting to preserve renal function in patients with chronic kidney disease, every attempt should be made to minimize iodinated contrast load. In these cases, one may perform limited abdominal aortography with dilute contrast or carbon dioxide (CO2) with a focus towards accessing the renal arteries. Alternatively, when appropriate, one can directly perform selective renal arteriography using aortic and renal artery calcification as a guide. We strongly recommend digital subtraction angiography and contrast-sparing techniques, particularly when performing renal stenting for the preservation of kidney function. Selective renal angiography should be performed with visualization of the entire kidney. Lack of perfusion to a particular segment may indicate the presence of an infarcted segment, an accessory renal artery, or renal mass or cyst. The differentiation of each may be complemented by data obtained on preprocedure noninvasive imaging.

Translesional Pressure Gradient Assessment
Translesional pressure gradients should be routinely assessed as a component of the invasive evaluation of moderate (50–70%) RAS. Several investigators have demonstrated that a hyperemic systolic gradient of approximately 20 mm Hg induced by the administration of intrarenal papaverine (30 mg intra-arterial bolus) or dopamine (50 μg/kg intra-arterial bolus) represents the greatest single predictor of blood pressure reduction after renal stenting [11,14]. It must be noted that adenosine is a vasoconstrictor in the renal artery and will not induce renal hyperemia. The most important reason for performing hemodynamic assessment is to discriminate hemodynamically significant stenoses from insignificant moderate angiographic stenoses. In general, the translesional pressure gradient (Pd/Pa ratio) <0.8, a resting mean gradient of ≥10 mm Hg, or a ≥20 mm Hg hyperemic systolic gradient are considered significant [11,12,14].

Intravascular Ultrasound Assessment
Intravascular ultrasound (IVUS) may provide information regarding minimal luminal area, plaque burden, reference vessel diameter, presence of calcification, and postintervention characteristics like stent apposition. However, IVUS has not been demonstrated to improve outcomes in patients undergoing renal stenting, and therefore cannot be recommended for routine use [14]. At the operator’s discretion, IVUS may be used to improve anatomic assessment of individual lesions and to facilitate stenting with the optimal stent diameter.

Renal Artery Stenting Technique
We recommend the use of a guide catheter with a curve and caliber appropriate to the intervention to be performed. Operators should minimize trauma, or scraping of the peri-renal aorta, by either using the “no-touch” technique or a telescoping technique with a 4F diagnostic catheter [37,38]. Currently, there are three 0.014” platform FDA approved balloon expandable catheterization and Cardiovascular Interventions DOI 10.1002/ccd. Published on behalf of The Society for Cardiovascular Angiography and Interventions (SCAI).
Complications

Renal artery stenting is a safe procedure with a major complication rate of ≤ 2%. The most common complications are related to femoral access (hematoma, pseudoaneurysm, arteriovenous fistula, or localized deep venous thrombosis). Less common complications including retroperitoneal hemorrhage, renal artery perforation, arterial and aortic dissection, atheromatous embolization, renal infarction, and death have all been reported. In general, radial artery access, conservative balloon sizing for predilatation or direct stenting, sizing the stent 1:1 to the reference vessel diameter, and attention to patient complaints of peri-procedural pain will minimize serious complications.

Follow-up

There are no standard guidelines for routine follow-up after renal artery stenting. In general, most operators use RADUS for follow-up assessment [41]. The recent appropriate use (AU) guidelines for peripheral vascular ultrasound suggest a baseline RADUS one-month following renal stenting. Following the one-month assessment, annual RADUS in asymptomatic patients is appropriate [42]. The recurrence of uncontrolled hypertension or progressive deterioration in renal function without other explanation is an appropriate indication for repeat RADUS to evaluate for the presence of renal artery in-stent restenosis [43].

CONCLUSION

Rigorously conducted clinical trials are critical to our understanding of the optimal treatment of our complex RAS patients. The CORAL trial and others have added to our understanding of the pathophysiology of RAS and the role of renal artery stenting [6]. However, as is commonly found in multicenter randomized trials, variable inclusion and exclusion criteria, outdated technology and technique, and an enrollment bias may limit the generalizability of results.

Planning novel, clinically relevant trial designs requires an appreciation for the unanswered questions in the field and the likelihood of enrollment over a reasonable time frame. Our “May Represent Appropriate Care” category of renal stenting offers an opportunity to shed light on these patient characteristics, and given the absence of currently available data, “clinical equipoise” certainly exists. Enrolling patients with ischemic nephropathy, unilateral RAS and hypertension, and/or congestive heart failure with hemodynamically significant RAS using core lab adjudicated metrics (i.e. angiography, translesional pressure gradients with and without hyperemia, novel intravascular imaging techniques) would be challenging, but the outcomes may impact patient care.

Improving technology for hemodynamic and imaging assessment of RAS will hopefully yield significant...
improvements in safety and efficacy of renal stenting. Smaller studies continue to explore the role of adjunctive pharmacology and EPD in renal artery revascularization. Nonetheless, given the paucity of scientifically valid data guiding our clinical decision making, the practitioner is required to use individual patient characteristics and best medical evidence (as referenced herein) to yield optimal and appropriate patient outcomes.

REFERENCES

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†Significant relationship.

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