Atherosclerotic Peripheral Vascular Disease Symposium II

Intervention for Renal Artery Disease

Krishna J. Rocha-Singh, MD, Chair; Andrew C. Eisenhauer, MD; Stephen C. Textor, MD, FAHA; Christopher J. Cooper, MD, FAHA; Walter A. Tan, MS, MD; Alan H. Matsumoto, MD; Kenneth Rosenfield, MD; for Writing Group 8

The primary goal of this American Heart Association renal intervention writing group was to discuss current controversies related to renal interventions and to recommend important areas of clinical research and advocacy initiatives in this peripheral arterial bed. The 4 areas covered in this section include (1) management of asymptomatic renal artery disease, (2) treatment of ischemic nephropathy, (3) prevention and treatment of athroembolism in renal artery interventions, and (4) treatment of renal in-stent restenosis (ISR).

Asymptomatic Renal Artery Disease: Indications for Treatment

Atherosclerotic renal artery disease is an often unrecognized contributor to refractory hypertension, renal insufficiency, and increased risk of cardiovascular death. Renal artery disease is associated with increased cardiovascular events (myocardial infarction, stroke, and death), and when associated with symptomatic coronary artery disease, it independently doubles the risk of death. Additionally, the presence of bilateral renal artery stenoses is associated with a reduced 4-year survival rate when compared with unilateral disease (47% versus 59%, P<0.001). Hypertension, renal insufficiency, and multisystem atherosclerosis are common entities, and the independent occurrence of these conditions is frequent. Thus, the physician must distinguish between association and causation in the evaluation of patients with atherosclerotic renal artery disease and critically appraise the potential for clinical improvement in selecting patients for renal artery intervention.

In contrast to other regional manifestations of atherosclerosis, it is impractical to classify patients with atherosclerotic renal artery disease into symptomatic or asymptomatic categories. Two of the cardinal manifestations of renal artery disease, hypertension and renal insufficiency, are frequently “silent” with regard to clinical manifestations until end-organ damage or uremia occurs. Thus, the majority of patients may be deemed asymptomatic. A more appropriate classification of patients with atherosclerotic renal artery disease may be to classify them in relation to potential clinical consequences. We propose the following classification scheme in patients with renal artery disease:

Grade I: Renal artery stenosis is present, but there are no clinical manifestations (normotensive with normal renal function).

Grade II: Renal artery stenosis is present, but patients have medically controlled hypertension and normal renal function.

Grade III: Renal artery stenosis is present, and patients have evidence of abnormal renal function, medically refractory hypertension, or evidence of volume overload.

This grading classification may be used to promote uniformity in trial design and reporting. A grade II or III categorization in a patient with a significant renal artery lesion (>60% diameter stenosis by quantitative analysis) provides information that may be used to effect patient management; however, the available data do not establish an imperative for revascularization of an atherosclerotic renal artery stenosis.

Cardiovascular risk factor modification should be instituted and optimized for all patients with atherosclerotic renal artery disease. In addition, patients should be monitored for the development of symptoms of end-organ disease resulting from hypertension (eg, angina, congestive heart failure, cerebrovascular ischemia) or deterioration in renal function. Surveillance of a renal artery stenosis >60% should be...
Factors favoring medical therapy and revascularization for renal artery stenosis

- Progressive decline in GFR during treatment of systemic hypertension
- Failure to achieve adequate blood pressure control with optimal medical therapy (medical failure)
- Rapid or recurrent decline in the GFR in association with a reduction in systemic pressure
- Decline in the GFR during therapy with ACE inhibitors or ARBs
- Recurrent congestive heart failure in a patient in whom the adequacy of left ventricular function does not explain a cause

Factors favoring medical therapy and surveillance of renal artery disease

- Controlled blood pressure with stable renal function (eg, stable renal insufficiency)
- Stable renal artery stenosis without progression on surveillance studies (eg, serial duplex ultrasound)
- Very advanced age and/or limited life expectancy
- Extensive comorbidities that make revascularization too risky
- High risk for or previous experience with atheroembolic disease
- Other concomitant renal parenchymal diseases that cause progressive renal dysfunction (eg, interstitial nephritis, diabetic nephropathy)

ACE indicates angiotensin-converting enzyme; ARBs, angiotensin receptor blockers.

advanced age and/or limited life expectancy. Extensive comorbidities that make revascularization too risky. High risk for or previous experience with atheroembolic disease. Other concomitant renal parenchymal diseases that cause progressive renal dysfunction (eg, interstitial nephritis, diabetic nephropathy).

Factors favoring medical therapy and revascularization for renal artery stenosis

- Progressive decline in GFR during treatment of systemic hypertension
- Failure to achieve adequate blood pressure control with optimal medical therapy (medical failure)
- Rapid or recurrent decline in the GFR in association with a reduction in systemic pressure
- Decline in the GFR during therapy with ACE inhibitors or ARBs
- Recurrent congestive heart failure in a patient in whom the adequacy of left ventricular function does not explain a cause

Factors favoring medical therapy and surveillance of renal artery disease

- Controlled blood pressure with stable renal function (eg, stable renal insufficiency)
- Stable renal artery stenosis without progression on surveillance studies (eg, serial duplex ultrasound)
- Very advanced age and/or limited life expectancy
- Extensive comorbidities that make revascularization too risky
- High risk for or previous experience with atheroembolic disease
- Other concomitant renal parenchymal diseases that cause progressive renal dysfunction (eg, interstitial nephritis, diabetic nephropathy)

ACE indicates angiotensin-converting enzyme; ARBs, angiotensin receptor blockers.

performed periodically with noninvasive imaging such as duplex ultrasound, magnetic resonance angiography, or computer-assisted tomographic angiography. Monitoring for extrarenal vascular disease (coronary artery disease, carotid artery disease, peripheral artery disease, and infrarenal aortic aneurysms) should also be considered in accordance with the patient’s clinical history and physical examination. Furthermore, given the increased prevalence of renal artery disease in patients with coronary or peripheral artery disease, it is reasonable to consider diagnostic nonselective screening abdominal aortography at the time of coronary or peripheral arteriography in patients with coronary artery or peripheral artery disease who are deemed to be at increased risk for renal artery stenosis and who are candidates for revascularization, as defined in the American College of Cardiology/American Heart Association peripheral arterial disease management guidelines document (Table). Nevertheless, the decision to revascularize any particular patient must be individualized to the particular circumstance because Level 1 evidence (based on randomized, controlled trials) supporting renal revascularization is not available. Therefore, if possible, eligible patients should be enrolled in the Cardiovascular Outcomes in Renal Atherosclerotic Lesions Trial (CORAL). This National Institutes of Health–sponsored trial will randomize >1000 hypertensive patients (systolic blood pressure ≥155 mm Hg) with severe atherosclerotic renal artery lesions (>60% diameter stenosis) to optimum medical therapy with or without renal artery stent implantation. The aim of this trial is to assess the effect of medical therapy alone compared with medical therapy plus renal artery stenting during a 5-year follow-up period on the incidence of composite cardiovascular and renal end points, including death, cerebrovascular accident, myocardial infarction, doubling of serum creatinine, hospitalization for congestive heart failure, and renal replacement therapy.

Ischemic Nephropathy: Selecting Patients for Treatment

High-grade renal artery stenosis may ultimately pose a threat to renal function in certain patients, but it is difficult to establish the precise level of risk for deterioration in function and the potential benefits of revascularization for an individual patient. Renal revascularization does not consistently produce improvement in renal function and on occasion may lead to adverse events, including atheroembolization. Indeed, the foundation of the CORAL trial underscores the fact that outcome data with current technology and medical therapy are inadequate to establish specific treatment guidelines for patients with renal insufficiency and renal artery disease. In the interim, clinicians are faced with treatment dilemmas and must make decisions about revascularization versus medical therapies on the basis of the balance of risks and benefits in each individual patient. Specifically, the extent of large- and small-vessel disease, the risk of progression of renal artery stenosis, the potential for the development of renal insufficiency related to other patient comorbidities, and the likely outcomes of the intervention must be considered in selecting a patient for revascularization.

Overall renal function is a direct reflection of the glomerular filtration rate (GFR) of the entire renal mass; however, removal of a healthy kidney in a patient with a normal GFR will not diminish the GFR by 50% but rather by ~35% to 40%, in part because of the compensatory hypertrophy of the remaining healthy kidney. Therefore, reduction in GFR below 50% of normal cannot be explained by unilateral disease alone. Thus, it has been presumed that revascularization of a unilateral renal artery stenosis will rarely produce clinically meaningful improvement in renal function; however, recent observations challenge this view and suggest that revascularization of unilateral renal artery stenosis sometimes can improve or stabilize renal function. Furthermore, percutaneous revascularization of a unilateral renal artery stenosis may improve the function of the kidney ipsilateral to the stenotic lesion and resolve the hyperfunctioning and proteinuria, presumably due to renal injury of the contralateral kidney. Nonetheless, predictable benefit from renal revascularization may be most pronounced in individuals with renovascular disease that affects the entire renal mass—that is, both kidneys or a solitary kidney.

The extent to which renal artery disease affects death rate and renal dysfunction is not well defined. Studies of Medicare beneficiaries starting renal replacement therapy (renal dialysis or transplantation) indicate that 5% to 10% of these individuals have identified renovascular disease. Both death rate and risk of renal dysfunction are higher in patients with bilateral renal artery stenoses or involvement of a solitary functioning kidney than in patients with 2 kidneys and unilateral disease. Of note, the risk of progression of an atherosclerotic renal artery lesion to occlusion is predicted by the level of arterial hypertension and initial severity of the lesion. Measurable progression of the severity of stenosis over 5 years approximates 50% in lesions that represent
>60% diameter obstruction at the time of initial detection. Clinical progression to renal dysfunction, reflected by a rise in serum creatinine, is less common, usually in the range of 10% to 15%. However, in the kidneys, large-vessel atherosclerotic disease is frequently superimposed on microvascular disease related to hypertension, aging, and diabetes mellitus. The presence of microvascular disease may explain why renal function fails to improve in many patients and why the severity of the main renal artery stenosis may not predict functional improvement after revascularization. Further studies to define the renal effects of vascular lesions and the potential for salvageability of kidney function are sorely needed.

Serum creatinine values (or estimated GFR) usually do not improve after renal stent therapy in patients with advanced chronic kidney disease (serum creatinine >3.0 mg/dL). Additionally, in patients with chronic renal insufficiency undergoing renal artery stenting, a rapid decline in GFR is seen in up to 20% of patients and may be secondary to procedure-related atheroembolization, vessel dissection, or contrast-induced nephropathy. Approximately 27% of patients with ischemic nephropathy may experience a meaningful improvement in GFR or a decline in the slope of the serum creatinine curve. In most series of renal stenting for renal insufficiency, the largest patient cohort experiences no appreciable change but rather stabilization in GFR. Still, these patients potentially may benefit from improved blood pressure control and by a reduction in the risk of developing volume overload. Therefore, renal revascularization may be beneficial to overall renal function in some patients with ischemic nephropathy, but this potential benefit may be counterbalanced by the risk of treatment failure, adverse events, and the development of ISR after renal stenting.

### Atheroembolization in Renal Interventions: Prevention and Management

Atheroembolization is a potential complication of all invasive cardiovascular and peripheral vascular procedures, including both endovascular and surgical interventions, and likely occurs more often than is appreciated or acknowledged. In patients undergoing renal intervention, atheroembolism may occur during the diagnostic angiogram, as a consequence of manipulation of catheters and guidewires within the abdominal aorta, or during an attempt to selectively catheterize the renal artery (type I embolization). It may also be caused by direct manipulation within the proximal main renal artery during balloon dilation and/or stenting, resulting from “active” disruption of the atherosclerotic plaque (type II embolization). The incidence of type I or type II atheroembolization during diagnostic or interventional procedures is not known. The diagnosis is suspected when subacute renal failure or peripheral evidence of embolization, such as blue toes and livedo reticularis, is evident. The diagnosis can be confirmed by a biopsy of the affected organ, although this is usually not necessary.

Renal atheroembolism impairs renal function and may lead to end-stage renal disease and death. Preexisting renal insufficiency and long-standing hypertension are independent predictors of progression to end-stage renal disease in patients in whom renal atheroembolism has occurred. Atheroembolization can be subclinical and often may be subtle or occult. It may not become manifest for weeks after the inciting event.

Management of renal atheroembolism is largely palliative, and no therapies are available to mitigate its consequences. Because there are no effective treatments to preserve renal function once atheroembolism has occurred, steps should be taken to reduce the risk of atheroembolism during renal interventions. These preventive measures include appropriate case selection of patients, the use of proper and cautious techniques, and the potential application of embolic protection devices. Operator training and technical expertise are important factors in minimizing the risk of atheroembolization. The interventionalist should also carefully examine preprocedural noninvasive studies, such as magnetic resonance angiography or computer-assisted tomographic angiography. These studies provide valuable information about the diameter of the aorta and the iliac artery that will provide access during the procedure, vessel tortuosity, the amount of calcification and plaque burden within the aorta and affected renal artery, the number and size of renal arteries, the orientation of the renal artery origins, and the course of the renal arteries relative to the abdominal aorta. By reviewing the noninvasive study, the operator can then select the appropriate access site and catheter shape for direct renal artery catheterization; minimize the number of catheter exchanges; define the proper angulation of the image intensifier to allow profiling of the affected renal artery origin, which allows for a reduction in the number of contrast angiograms; and minimize contact with the aortic wall. Scraping of the wall with catheters and other devices is more likely to disrupt friable or loose plaque and cause atheroembolization. In an effort to minimize the risk of atheroembolization during renal artery catheterization, use of the so-called “no-touch technique” has been described. With this technique, a 0.035-inch J-tip guidewire is positioned simultaneously alongside a 0.014-inch steerable guidewire through the guide catheter. The guide catheter is maneuvered to a position adjacent to the affected renal ostium. The J-wire is then retracted so that its tapered, softer transition point is positioned closer to the tip of the guide catheter, which allows the guide catheter to assume its preset shape and approximate the ostium of the renal artery. The presence of the J-wire, in theory, minimizes direct contact of the guide catheter with the aortic wall, thereby reducing the potential for atheroembolization. From this position, the 0.014-inch wire is directed through the renal lesion and distally in the renal artery. The J-wire is then removed, which allows the tip of the guide catheter to fully engage the renal ostium in a less traumatic fashion. Although this technique may reduce the risk for atheroembolism during renal artery interventions, it is not known whether it is less traumatic and safer than other meticulous renal artery catheterization techniques.

Atheroembolization into the renal vascular bed may occur during any stage of a renal intervention. Hiromoto et al used an ex vivo model of renal angioplasty and stenting in human aorto–renal artery atherosclerotic surgical specimens to document the extent of atheroembolization during a hypothetical renal stent procedure. They measured the quantity and size of atheroembolic debris particles, noting that debris emboliza-
tion occurred during all phases of renal stenting but was most marked during balloon predilation before stent placement and during stent deployment. Therefore, the use of distal occlusion balloons or filters may reduce the incidence of distal atheroemboli and minimize the deleterious effects on renal function. In a retrospective study, Henry et al. reported a high frequency of debris retrieval using both a temporary occlusive balloon device system (GuardWire, Medtronic, Minneapolis, Minn) and another filter system (EPI Filter, Boston Scientific Corp, Natick, Mass) during renal artery stenting. Similar results have been reported by others. Although these initial results are encouraging, the devices used in these renal applications initially were designed for use in coronary saphenous vein grafts or carotid stenting, and their utility in the kidneys has not been determined. Appropriately sized devices with profiles, transitions, and “landing zones” that allow them to be applied in the renovascular bed must take into consideration the size and orientation of the renal arteries and their bifurcation patterns, while allowing sufficient length to accommodate the use of guide catheter, balloon, and stent systems.

No established treatment exists to “cure” the consequences of atheroembolization once these occur. Cholesterol and atheroembolic debris lodged in the terminal vessels (arterials and capillaries) can cause local inflammation, scarring, and disruption of the local vascular architecture. Statins may have a protective effect on the inflammatory reaction of vessels to atheroemboli. In a prospective evaluation of predictors of outcomes in patients with atheroembolic renal disease, patients undergoing statin therapy experienced a lower risk of developing end-stage renal disease. Administration of N-acetylcysteine (Mucomyst, Apothecon Inc) and sodium bicarbonate and volume expansion before administration of contrast for a renal intervention are recommended to reduce the risk for developing contrast-induced nephrotoxicity. It is not known whether these therapies reduce the risk of renal insufficiency resulting from atheroembolism.

**Treatment of Renal ISR**

The principal cause of restenosis within renal stents is neointimal hyperplasia in response to arterial injury. The initial response to vascular wall injury is initiated within minutes, with the deposition of platelet- and fibrin-enriched thrombus. Subsequent migration of smooth muscle cells and endothelial progenitor cells leads to cellular proliferation and matrix deposition. In combination with predisposing genetic factors, the excessive response to arterial injury ultimately leads to ISR. ISR can occur as a focal stenosis at the area of an articulation or gap between stents, or as a focal or multifocal stenosis at the proximal or distal margins of the stent, or as a focal or diffuse stenosis within the central portion of the stent. Restenosis within stainless steel renal artery stents, as defined by duplex ultrasound criteria or angiography, occurs in ~17% to 22% of patients within the first 12 months after stent implantation. Factors that influence the development of renal ISR can be divided into 3 broad categories: patient related, vessel related, and device/technical related. A number of factors have been shown to be predictive of renal ISR, including vessels <6 mm in diameter, female sex, continued smoking, longer stent length, and the presence of gold coating. Inadequate stent-to-vessel wall apposition, which may occur in as many as 33% of cases as demonstrated by intravascular ultrasound, is also associated with higher rates of renal ISR. The occurrence of renal ISR can be minimized by maximizing stent diameter with the use of devices (eg, intravascular ultrasound) that optimize stent expansion to ≥6 mm.

Several options are available for the treatment of renal ISR. Balloon angioplasty is an effective treatment of renal ISR, with patency rates at 6 to 11 months of 75% to 79%. Placement of a second balloon-expandable bare-metal stent has also been used as an effective treatment for renal ISR. Newer technologies have been applied to treatment of renal ISR, but none have been shown to be superior in limited case series reports. Use of a polytetrafluoroethylene-covered balloon expandable stent (iCast Covered Stent, Atrium Medical Corp, Hudson, NH, or Graft Master Stent, Abbott Vascular Devices, Santa Clara, Calif) is another potential method to treat renal ISR, although neither device is approved for this indication, nor is there evidence to support their use. Cryoplasty (PolarCath, Boston Scientific Corp) involves the use of a balloon catheter system in which liquid nitrous oxide is converted to a gaseous state and cools the outer surface of a balloon to ~10°C. Interstitial fluid within the tissues adjacent to the balloon freezes and expands, which causes microfractures in the plaque. In theory, cryoplasty induces smooth muscle cell apoptosis and alters the elastic fibers, which reduces elastic recoil. Nonetheless, evidence is insufficient to support its routine use in renal ISR. Brachytherapy has been used to minimize or treat renal ISR. Iridium-192 (a γ-radiation emitter) and β-radiation–emitting systems demonstrated good initial success rates, with subsequent restenosis rates of ~20% in small case series; however, there are currently no commercially available brachytherapy devices for treatment of renal ISR. Drug-eluting stents reduce ISR at 12 months in coronary arteries. The efficacy of a sirolimus drug-eluting stent in the renal circulation has been studied in a small feasibility trial, the GREAT trial (Palmaz Genesis Peripheral Stainless Steel Balloon Expandable Stent in Renal Artery Treatment), in which 6-month follow-up data demonstrated a decrease in renal ISR from 14% in the bare-stent group to 6.7% in the drug-eluting stent cohort on the basis of follow-up angiography.

Use of cutting balloons, lasers, and atherectomy devices for renal ISR has been reported, but no significant cases series or trials of these devices in the renovascular system are available. In addition, there are no data on the use of statins, antiplatelet agents, drug-coated balloons, or oral antiproliferative agents and their effects on reducing renal ISR, although there are some encouraging preliminary data in the coronary literature. Use of warfarin has not been effective in reducing the incidence of renal ISR.

**Summary and Recommendations**

The treatment of atherosclerotic renal artery disease is evolving and remains controversial. Antihypertensive drugs are often effective in lowering blood pressure in patients with
renal artery stenosis, but some patients have persistent hyper-
tension despite treatment with multiple antihypertensive
agents or develop worsening renal function. Renal artery
stening is widely available and frequently used to treat
patients with renal artery stenosis and poorly controlled
hypertension and/or renal insufficiency. Nevertheless, it is
still not known whether percutaneous revascularization adds
incremental value to optimal medical therapy to prevent the
adverse consequences of renal artery disease. Accordingly,
the present writing group recommends that physicians enroll
hypertensive patients with atherosclerotic renal artery steno-
sis into the CORAL trial to acquire outcomes and selection
data necessary to answer some of the issues raised in this
document.

In patients with declining renal function due to ischemic
nephropathy, when obstructive renal artery disease affects
the entire renal mass, renal artery stenting can be expected to
either improve or stabilize renal function in the majority of
patients and reduce the risk of developing volume overload;
however, this potential benefit must be weighed against the
potential risk of worsening renal function due to procedure-
related atheroembolization or contrast-induced nephropathy,
other adverse events, and ISR. Therefore, additional research
in this area is recommended.

Proper catheter techniques, including paying close atten-
tion to the atherosclerotic burden of the perirenal aorta, may
reduce the risk of atheroembolism. Renal distal protection
devices have not established their utility in reducing the
possible decline in renal function observed in some patients
after renal stenting. The efficacy of renal distal protection
devices should be tested in randomized clinical trials.

ISR may be reduced by use of the shortest possible stent,
dilated to its maximum but safe diameter (preferably to
≥6 mm) to effect good stent-to-wall approximation. Once
renal ISR develops, balloon angioplasty appears to be as
effective as any other intervention. Brachytherapy may pro-
vide some benefit, but it is cumbersome and difficult to apply
in this vascular bed, and limited data on its use are available.
Newer technology, devices, and medications are promising,
but no data support their routine use for the treatment of renal
ISR, and further investigation is recommended.

Disclosures

Potential conflicts of interest for members of the writing groups
for all sections of these conference proceedings are provided in
a disclosure table included with the Executive Summary,
which is available online at http://circ.ahajournals.org/cgi/
reprint/118/25/2811.

References

2. Garovic VD, Teotro SC. Renovascular hypertension and ischemic ne-
3. Conlon P, Little MA, Pieper K, Mark DB. Severity of renal vascular
disease predicts mortality in patients undergoing coronary angiography.
Rosenfeld KA, Rundback JH, Linas SL. Indications for renal arte-
riography at the time of coronary angiography: a science advisory from
the American Heart Association Committee on Diagnostic and Interven-
tional Cardiac Catheterization, Council on Clinical Cardiology, and the
Councils on Cardiovascular Radiology and Intervention and on Kidney in
5. Hirsch AT, Haskal ZJ, Hertzler NR, Bakal CW, Creager MA, Halperin JL,
Hiratzka LF, Murphy WR, Olin JW, Poschet JB, Rosenfield KA, Sacks
D, Stanley JC, Taylor LM Jr, White CL, White I, White RA, Antman EM,
Smith SC Jr, Adams CD, Anderson JL, Faxon DP, Foster V, Gibbons RJ,
Hunt SA, Jacobs AK, Nishimura R, Ornato JP, Page RL, Riegel B.
ACC/AHA 2005 practice guidelines for the management of patients with
peripheral arterial disease (lower extremity, renal, mesenteric,
and abdominal aortic): a collaborative report from the American Association
for Vascular Surgery/Society for Vascular Surgery, Society for Cardio-
vascularAngiography and Intervention, Society for Vascular Medicine
and Biology, Society of Interventional Radiology, and the ACC/AHA Task
Force on Practice Guidelines (Writing Committee to Develop Guidelines
for the Management of Patients with Peripheral Arterial Disease).
Circulation. 2006;113:e463–e654.
6. Cooper CJ, Murphy TP, Matsumoto A, Steffes M, Cohen DJ, Jaff M,
Kuntz R, Jamerson K, Reid D, Rosenfeld K, Rundback J, D’Agostino R,
Henrich W, Dworkin L. Stent revascularization for the prevention of
cardiovascular and renal events among patients with renal artery stenosis
and systolic hypertension: rationale and design of the CORAL trial.
7. Zeller T, Frank U, Müller C, Bürgelin K, Sinn L, Bestehorn HP,
Cook-Brunns N, Neumann FJ. Predictors of improved renal function after
percutaneous stent-supported angioplasty of severe atherosclerotic ostial
8. Muray S, Martín M, Amoedo ML, García C, Jornet AR, Vera M, Oliveras
A, Gómez X, Craver L, Real MI, García L, Botey A, Montanyà X,
Fernández E. Rapid decline in renal function reflects reversibility and
predicts the outcome after angioplasty in renal artery stenosis. Am J
renal function outcome after renal angioplasty in patients with unilateral
function preservation after renal artery stenting in patients with pro-
gressive ischemic nephropathy. Cathet Cardiovasc Interv. 2002;57:
135–141.
11. Kalra PA, Guo H, Kausz AT, Gilbertson DT, Liu J, Chen SC, Ishani A,
Collins AJ, Foley RN. Atherosclerotic renovascular disease in United
States patients aged 67 years or older: risk factors, revascularization,
Multicenter Palmaz stent renal artery stenosis revascularization registry
report: four-year follow-up of 1,058 successful patients. Catheter Car-
13. Teotro SC. Ischemic nephropathy: where are we now? J Am Soc
14. Cheung CM, Wright JR, Shurrab AE, Mamorta H, Foley RN,
O’Donoghue DJ, Waldek S, Kalra PA. Epidemiology of renal dysfunction
and patient outcome in atherosclerotic renal artery occlusion. J Am
15. Paulsen D, Klöw NE, Rogstad B, Leivelst T, Bier B, Fachwald P.
Preservation of renal function by percutaneous transluminal angi-
oplasty in ischemic renal disease. Nephrol Dial Transplant. 1999;14:
1454–1461.
SJ, Lau J. Effectiveness of management strategies for renal artery steno-
17. Lim ST, Rosenfeld K. Renal artery stent placement: indications and
18. Thadhanri RL, Camargo CA Jr, Xavier RJ, Fang LS, Bazari H.
Atheroembolic renal failure after invasive procedures: natural history based
Mulhirt F, Matoera R. Predictors of renal and patient outcomes in
21. Kolluri R, Goldstein JA, Rocha-Singh KJ. Percutaneous vascular inter-
Atheroembolism during renal artery angioplasty: an ex vivo study. J Vasc


**Key Words**: AHA Conference Proceedings, renal artery intervention, peripheral arterial disease, atheroembolism, ischemic nephropathy.
Atherosclerotic Peripheral Vascular Disease Symposium II: Intervention for Renal Artery Disease
Krishna J. Rocha-Singh, Andrew C. Eisenhauer, Stephen C. Textor, Christopher J. Cooper, Walter A. Tan, Alan H. Matsumoto, Kenneth Rosenfield and for Writing Group 8

_Circulation_. 2008;118:2873-2878
doi: 10.1161/CIRCULATIONAHA.108.191178

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2008 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/118/25/2873

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in _Circulation_ can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to _Circulation_ is online at:
http://circ.ahajournals.org//subscriptions/