Renovascular Hypertension

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Renovascular hypertension is one of the more common causes of secondary hypertension with reports of its frequency varying from < 1% in unselected patient populations to as much as 20% in patients referred to special centers [1 – 3]. By definition, renovascular hypertension refers to hypertension caused by renal hypoperfusion – a decrease in blood flow to the kidney [4]. Therefore, the presence of a solitary stenotic renal artery lesion does not establish the diagnosis of renovascular hypertension. It is absolutely necessary for renal ischemia to occur, and this happens when stenosis is > 75% of the luminal diameter [4 – 6]. This, in turn, sets off a cascade of events that leads to the stimulus that raises blood pressure; hence the diagnosis of renovascular hypertension. Most importantly, renovascular hypertension is an important diagnosis to make in that it the most common curable form of hypertension and is one of the few potentially reversible causes of chronic renal failure (CRF)[1]. This chapter describes pathophysiologic mechanisms in renovascular hypertension, clinical features of the disease, and staging of suspicion for disease to select appropriate diagnostic steps. New therapeutic options are reviewed, and the advantages and disadvantages of each are delineated.

Pathophysiology of Renovascular Hypertension

Renovascular hypertension in humans is the result of a stenotic lesion leading to ischemia that, in turn, initiates events that lead to hypertension (Figure 1) [4, 7]. The pathophysiologic state of ischemia serves as the stimulus for a release of renin. Resultant high levels of angiotensin II (Ang II) levels then readily increase renal vascular resistance causing a shift in the pressure-natriuresis curve [6, 8]. In this second phase of renovascular hypertension, volume is increased and quite often maintained despite markedly elevated blood pressure further accentuating the hypertension [6, 9]. The patient then exhibits the symptoms of sudden and significant hypertension, such as pounding headaches and palpitations.

Renal ischemia can continue for several years undetected. After years of renal ischemia, many patients may then enter a third phase of the disease. In these patients, regardless of whether the stenotic lesion is relieved or not, hypertension continues unabated. It is at this stage that widespread arteriosclerosis with concomitant glomerulosclerosis has already occurred in the contralateral, or non-stenotic, kidney [1, 9]. These pathophysiologic changes, specifically glomerulosclerosis along with medial hyperplasia of the blood vessels, are the result of the prolonged exposure to high blood pressures (and conse-

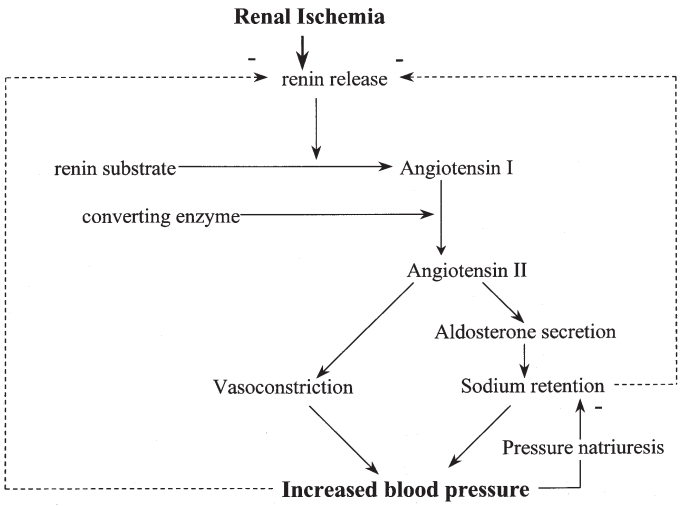

Figure 1.

quently increased glomerular pressures) in addition to the high levels of Ang II [1, 9]. The clinical relevance of this is that the sooner the lesion responsible for the renal ischemia is relieved, the greater chance of alleviating the hypertension and thereby preserving renal parenchymal tissue [1, 4].

The pathophysiology of the patient with bilateral disease is a little different than that seen with a solitary lesion. In patients with bilateral disease, cardiac output is usually greater than in those with unilateral disease [4, 7, 10]. Therefore, the hypertension in the patient with bilateral renal artery disease is more dependent on volume. The most likely etiology for this difference is that stenosis does not develop symmetrically in the 2 kidneys. Presumably, most bilateral stenotic lesions are initially an undetected unilateral lesion. Thus, parenchymal disease will develop in the contralateral kidney during the years of prolonged hypertension before the development of a stenotic lesion on the unaffected side. The hypertensive kidney disease would then impair the pressure natriuresis by which the contralateral kidney (nonstenotic side) normally maintains the classic high-renin-normal volume pattern of unilateral renal artery stenosis. This volume retention would be further exacerbated when the second stenotic lesion develops in the contralateral kidney. During this phase of the renovascular disease, the stenotic kidneys then chronically continue to secrete excess renin with the resultant elevation in peripheral resistance and fluid volume [7].

Other factors have been proposed that may be interrelated with the primary renin-angiotensin mechanism of hypertension of renovascular hypertension. Recent studies have implicated increased sympathetic nervous system activity and enhanced vasopressin response as potentially important physiologic responses contributing to the hypertension of renovascular disease. Moreover, reduced vasodilatory prostaglandins have been seen in patients with renovascular hypertension [1, 6]. These prostaglandins are necessary to maintain renal blood flow (RBF). This finding may have important clinical applicability in that the use of nonsteroidal anti-inflammatory agents (NSAID; such as ibuprofen) may acutely decrease blood flow by further inhibiting prostaglandin production. This decrease in blood flow may lead to a further decrease

Table 1.

Characteristics	Essential Hypertension (%)	Renovascular Hypertension (%) Atheroma	Fibromuscular Dysplasia
Race (African-American)	29	7	10
Family history	67	58	41
Age at onset: < 20 years	12	2	16
> 50 years	7	39	13
Obesity	38	17	11
Abdominal bruit	7	41	57
Elevated renin profile	15	80	80
Smoking	42	88	17
Hypokalemia (K < 3.4 mEq/L)	7	14	71

in renal function and a subsequent increase in blood pressure.

Clinical Features

Variable clinical features of renovascular hypertension dominate in one of 2 patient groups (Table 1). The prevalence of renovascular hypertension is unknown in that results from all major studies, save one, were based predominantly on clinical assessments. The investigators did not use angiography to look for stenosis. Therefore, the prevalence rates are an underestimation of renal artery stenosis. However, renovascular hypertension prevalence is probably anywhere from 0.1 – 5% in the general hypertensive population and may be as high as 20% in those individuals with severe hypertension [1, 11 – 14]. Moreover, in Whites with more severe hypertension, there is a 6-fold greater prevalence than in the African-American population with similar blood pressure levels.

Lesions of Renovascular Hypertension

The 2 major causes of renovascular hypertension are atherosclerosis and fibromuscular dysplasia (FMD) [12, 15 – 18]. In those individuals with atherosclerosis, quite often the finding of a middle-aged, white male with other evidence of atherosclerotic disease (claudication, angina) raises the possibility of progressive renal artery atheromatous plaque formation leading to stenosis. In addition, these individuals may have a recurrent history of unexplained pulmonary edema and/or renal dysfunction prior to the hypertension becoming more prominent. A significant history of smoking increases the possibility that the hypertension seen is related to atheromatous renal artery disease; 88% of those with atheroma renal artery hypertension were smokers, while approximately 42% of those with essential hypertension had a similar history [18]. One laboratory test that may aid in suggesting renovascular hypertension in con-

junction with clinical clues is significant hypokalemia with nephrotic-range proteinuria (> 3.5 g/24hours).

The anatomic location of the atherosclerotic plaque has significant therapeutic implications. The atheromatous plaques occur most commonly in the proximal one-third of the renal artery [2, 13, 19]. Other less common areas for plaque formation are the distal region of the renal artery and the ostium of this same blood vessel. Ostium lesions form directly from aortic atheromatous lesions [18]. More importantly, these ostial lesions are less responsive to angioplasty therapy than proximal lesions (see *Therapy*) and quite often progress in a more aggressive fashion. Renal artery stenosis secondary to atherosclerotic disease has been studied both retrospectively with angiography and prospectively with renal duplex ultrasound. Rates of anatomic progression varied from 36 – 71% of patients. Progression to occlusion occurred in 8 – 16% of patients over a 3- to 5-year period [18, 20]. The rate of progression to total occlusion occurred more frequently when there was a higher grade stenosis on the initial angiogram. In the prospective study by Zierler et al. [21] renal arteries that were normal on first exam did not have any progression of disease, but the cumulative incidence of progression from < 60% renal artery stenosis to ≥ 60% was 23% ± 9% at 1 year and 42 ± 14% at 2 years. Those renal arteries that progressed to occlusion all had ≥ 60% stenosis at the initial visit with the cumulative incidence of progression to occlusion being 5% ± 3% at 1 year and 11% ± 6% at 2 years. The overall progression of renal artery stenosis in this study was about 20% per year.

In younger patients, especially in white females, FMD is the most likely etiology for renal artery stenosis [15, 17]. Rare in the black population, FMD should be considered when a young white female (< 20 years of age), without any family history of hypertension, develops moderate to severe hypertension. The lesion in FMD is classified into 4 types. The most common variant is that of medial fibroplasia, which constitutes 75% of all cases. It progresses less frequently than atheromatous lesions and produces the classic beaded appearance on angiograms. This appearance is the result of areas of medial thickening interspersed with aneurysmal dilations. Intimal, perimedial, and periarterial lesions are the more sharply localized fibrodysplastic lesions and progress to greater stenosis more rapidly [17]. Without intervention significant stenosis with concomitant hypertension can develop, and subsequent significant renal disease will ensue. FMD will progress in one-third of patients and may develop in contralateral vessels, but it rarely results in renal artery occlusion [3].

Diagnosis

Clinical Clues

Renovascular hypertension must be excluded as the etiology for hypertension in patients with hypertension. While extensive screening of all hypertensive patients is clearly impractical, certain clinical clues will help establish an index of suspicion and thereby justify further evaluation (Table 2). The indices of suspicion for renovascular hypertension are low, moderate, and high. The clinical index of suspicion serves as a sensible diagnostic guide for the patient with hypertension and suspected renovascular disease. Patients can then be examined and their history reviewed for potential signs and symptoms that may suggest underlying renovascular disease.

Table 2. Clinical Index of Suspicion: Guide for Selecting Patient Work-ups

Low (most should not be tested)
- borderline mild or moderate hypertension without any clinical evidence of vascular disease

Moderate (noninvasive tests recommended)
- diastolic blood pressure > 120 mm Hg
- hypertension refractory to standard therapy
- abrupt onset of hypertension; < 20 or > 50 years of age
- hypertension with an abdominal bruit
- diastolic blood pressures > 105 in a patient who smokes, has evidence of peripheral vascular disease, and/or unexplained azotemia
- rapid normalization of blood pressure with an ACE inhibitor in a patient with moderate hypertension who smokes and has evidence of peripheral vascular disease.

High (may consider proceeding directly to angiography)
- diastolic blood pressure > 120 mm Hg in a patient with either progressive renal disease and/or a patient with vascular disease that has not responded to aggressive antihypertensive treatment.
- accelerated or malignant hypertension
- hypertension with recent elevation of serum creatinine
- moderate or severe hypertension with incident detection of renal asymmetry

Most importantly, the patient with diabetes mellitus (DM), especially noninsulin-dependent diabetes (NIDDM, type II), should warrant a closer evaluation for possible renovascular disease. Many diabetic patients, while they will develop nodular glomerulosclerosis as a result of diabetic nephropathy, leading to hypertension, also have progressive and diffuse atherosclerotic vascular disease [14, 19, 22]. This is yet another manifestation of their DM, as their lipid profiles are quite often abnormal even in the face of normal renal function. The etiology for difficult blood pressure control and/or a more rapid decline than expected in renal function in these patients may suggest the presence of undetected renal artery stenosis. Therefore, the index of suspicion should be high in a diabetic patient with other manifestations of atherosclerotic disease.

The patient with well-controlled hypertension would fall into the low category. The prevalence of renovascular disease is likely to be <1%, with most of the positive test results turning out to be falsely positive. However, the patient who presents with a more complicated clinical picture, such as difficult-to-control blood pressure (diastolic blood pressures > 120 mm Hg) that appears to be refractory to standard therapy and/or abrupt onset of hypertension before the age of 20 or after the age of 50, should undergo aggressive evaluation.

An abdominal bruit is one clinical feature with clear discriminatory value in patients with renovascular hypertension. Heard in almost half of all patients with renovascular hypertension, an abdominal bruit is heard over the flank in only 9% of patients with essential hypertension. A bruit that is high-pitched with systolic and diastolic components and that radiates laterally strongly suggests functionally significant renal arterial stenosis. However, the absence of an abdominal bruit does not exclude renovascular hypertension.

Diagnostic Tests

The evaluation of hypertensive patients to identify a causative renal artery lesion depends on whether there is a low, moderate, or high level of suspicion for underlying renovascular hypertension [23]. Unfortunately, there are no perfect screening tests for its

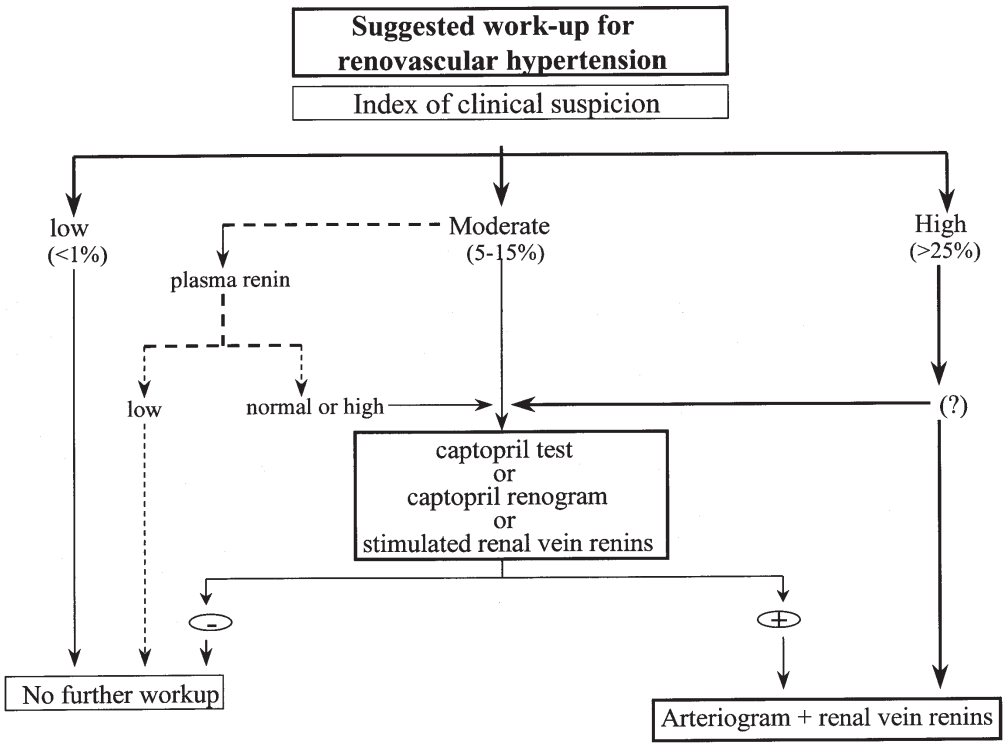

Figure 2.

detection. The major tests can be classified into 2 categories (Figure 2): physiologic and anatomic. Physiologic tests can be readily performed on an outpatient basis and do not indicate the involved kidney. These inexpensive tests are the measurement of peripheral plasma renal activity (PRA) and the captopril test and should be performed in those patients with a moderate level of suspicion for renovascular hypertension [23].

The second category includes tests that provide anatomic functional information about each kidney. These tests should ideally follow the noninvasive office visit tests. However, a noninvasive work-up should include a renal ultrasound to look for asymmetry in the patient with suspected disease followed by PRA and/or a captopril test.

PRA/captopril test. Approximately 75% of all patients with proven renovascular hypertension have markedly elevated PRA [24]. While this test can be normal in value, it is rarely, if ever, low in a patient with renovascular hypertension. Unfortunately, its predictive value in the absence of clinical clues for renovascular hypertension is low. It has a sensitivity of 57% and should not be used as a screening test for all hypertensives. In view of the limitations of the PRA test, alternative maneuvers have been used to enhance the sensitivity of the test. Of these, the response of PRA to captopril has been widely used.

The captopril test has been found to be the most reliable for distinguishing patients with renovascular hypertension [24]. For the test, the patient should maintain a normal salt in-

take, and, if possible, all antihypertensives (including diuretics) should be withdrawn 3 weeks before the test. This test involves measuring plasma renins in the seated patient after the administration of the angiotensin-converting enzyme (ACE) inhibitor captopril. The plasma renin response is classically greater in patients with renovascular hypertension than in those with essential hypertension. The criteria for distinguishing patients with renovascular hypertension are an absolute increase of 10 ng/mL/hour, a post-captopril level of > 12 ng/mL/hour, and/or a percentage increase of ≥ 150% in renins and/or ≥ 400% if the baseline PRA is < 3 ng/mL/hour. The sensitivity and specificity of this test is approximately 75% and 90%, respectively. However, this test is less reliable in patients with preexisting renal disease, thereby making it difficult to interpret in patients with renal insufficiency. In addition, it cannot distinguish between unilateral and bilateral disease, although it is positive in both of these diseases.

More involved tests to ascertain the presence of renal artery stenosis include renal vein renin determinations, renal scintigraphy, captopril renography and intravenous digital subtraction. Renal vein determinations measure renins through catheter placement in the patient's inferior vena cava. An increase of 25% is found in patients with renovascular hypertension and is usually 50% more in the ischemic kidney. No (or little) increase is found in the contralateral kidney. Renal scintigraphy, on the other hand, involves the use of iodohippurate sodium I^{131} that is selectively taken up by the kidney. However, this test has an unacceptably high rate of both false-positive and false-negative results and is relatively expensive.

The advantage of captopril renography is that it is based on the principle that glomerular filtration rate (GFR) and RBF of an ischemic kidney depend on the effects of angiotensin on the efferent glomerular arterioles and fall markedly with the administration of an ACE inhibitor [14]. The sensitivity and specificity of this test are relatively high (90 – 95%), but there is a low sensitivity (50 – 75%) in distinguishing between renovascular and renal parenchymal disease. Intravenous digital subtraction angiography was introduced with great fanfare. It involves visualization of the renal arteries without the potential complications of arteriography. However, this test suffers from the need for a large dye load, and it has poor resolution in obese patients. Most importantly, the most common atherosclerotic lesion, proximal stenosis, is often missed. Lastly, a promising and relatively noninvasive method of CO_2 injection has begun to be tested in patients with suspected renovascular hypertension. This method involves CO_2 injection and digital subtraction angiography to visualize the aorta and renal arteries. This procedure is currently in the phase of clinical validation and holds great promise for routine clinical use in patients with renovascular disease.

In facilities with an experienced technician in the use of duplex scanning, this should be the initial screening test [14, 16, 21]. However, in the patient with high suspicion and who is a candidate for angioplasty, the clinician should proceed directly to arterial angiography. This procedure provides an immediate answer as to whether there is renovascular disease and whether it is potentially curable [14, 19, 23]. Unlike the other studies, arteriography allows one to see the renal vascular architecture for potentially invasive therapy (i.e. surgical revision). Patients with a high index of suspicion should more than likely have a renal arteriogram, without the need for captopril studies. The predictive value of an arteriogram in patients with a high likelihood for disease is 32%, thereby justifying proceeding with this test first. Although

renal arteriography is almost always successful in diagnosing renovascular hypertension, it has relatively little value in determining surgical curability of renovascular hypertension.

Treatment

Once a physiologically and hemodynamically significant renal artery stenosis has been identified, the physician is still left with the question of how best to treat the lesion. Depending upon how one interprets the data, the literature can be used to support any of the 3 main treatment options: percutaneous transluminal angioplasty (PTA) with or without stent placement, operative intervention, or drug therapy. The character and known natural history of the lesion along with the patient's medical condition all influence the decision regarding treatment choice.

Quite often with the progression of renal artery stenosis there is an increase in serum creatinine, reflecting progressive renal dysfunction [4, 21]. Good blood pressure control through medical therapy does not seem to delay the progression of renal artery stenosis [18]. In comparing the value of drug therapy and operation, one study looked at an equal number of similar patients over a 7- to 14-year period [1]. In this study, 84% in the operation group survived and 93% were cured or showed significant improvement, whereas only 66% of those in the drug therapy group survived through the length of the follow-up period. Of those, 21% ultimately required operation for uncontrollable hypertension. Moreover, mortality in the medically treated group was significantly higher than that of the operation group. These differences applied to patients with both atherosclerotic and fibromuscular lesions of the renal artery.

Barring prohibitive medical risks, therefore, intervention for symptomatic renal artery stenosis is unquestionably justified, not only for blood pressure maintenance, but also for possible prevention of deterioration in renal function. An aggressive approach to renal revascularization is appropriate given the excellent outcomes of current surgical techniques with perioperative morbidity and mortality rates of 0 – 8% [12, 25]. Candidates for intervention include all patients with severe, difficult-to-control hypertension regardless of the nature or location of renal artery lesions (i.e. main stem vs. arterial branches, bilateral renal artery involvement), extrarenal vascular disease, or associated cardiovascular disease that may actually benefit from control of systemic hypertension. Age, duration of hypertension, type of lesion (fibromuscular vs.atherosclerotic), or the distribution and location of arterial lesions are not good determinants of successful outcomes following intervention. Clinically evident coronary artery disease, age > 70 and a serum creatinine > 3mg/dL, are co-morbid risk factors that may influence outcomes but should not necessarily preclude aggressive intervention on their own merits. The choice of PTA vs.surgical revascularization needs to be individualized.

Main renal artery FMD should be treated with PTA. When PTA is carried out by an experienced interventionalist, technical and clinical success rates with 2-year patency rates > 90% are usually reported. The results for nonorificial atherosclerotic lesions are similar, but these comprise only 15 – 20% of all atherosclerotic renal artery lesions, while the majority are orificial in that they are extensions of disease from the aorta. When reading the literature, one must look critically at the assumptions being made to define outcomes, differentiate between technical and clinical success, and note the length of follow-

up, especially with consideration of the natural history of the disease process in mind.

In the only prospective, randomized comparison of renal artery PTA with surgery, Weibull et al. [26] found a 2-year primary patency rate of 75% for renal PTA and 96% for operation. The secondary patency rate in the PTA group was 90% and in the surgical group 97%, yet nearly half of those patients with initial PTA failures underwent secondary surgical revascularization to achieve the quoted 90% secondary patency rate. On the basis of this study, we would find it difficult to recommend renal PTA as the initial treatment of choice in all-comers. Other studies have reported an 80% technical success rate with 68 – 90% clinical success and 5-year patencies as high as 88% following PTA in some series [1, 27]. In the literature, PTA results in 44 – 65% long-term improvement in blood pressure as compared to surgery, which is reported to yield sustained improvements of 90 – 93%. Renal function has been maintained or improved in 40 – 88% of patients following PTA and 70 – 94% of patients following surgical revascularization. In summary, PTA of ostial atherosclerotic lesions or fibrodysplastic lesions involving the renal artery branches yields outcomes inferior to that seen with surgical revascularization [28].

Whether or not these results can be improved upon by combining renal artery stent placement with PTA remains uncertain. The use of renal artery stents in combination with PTA as compared to surgical revascularization has not yet been studied in a prospective, randomized fashion. The published data thus far suggest that stenting of renal ostial stenoses has a higher initial success rate and improved intermediate term patency rate over PTA alone [29 – 31]. However, the benefits of PTA with stenting as compared to surgery are still unclear. Clinically, stents are indicated when significant elastic recoil exists in an artery undergoing PTA, dissection occurs following PTA, or restenosis develops following PTA. Placement of a stent in the renal artery will preclude certain surgical methods of revascularization. Proximally-placed stents will prevent the surgeon from performing aortorenal endarterectomy, should an operation become a consideration. Stents in the main portion of the body and especially those that encroach on the distal branch points will affect any subsequent surgical reconstruction that can be employed and potentially increase the difficulty of surgery. These factors must be taken into account if PTA with stenting is going to be offered as a therapeutic modality. Some of the complications related to PTA and/or stenting of the renal artery include arterial spasm, acute arterial occlusion, arterial perforation, embolization, and dye-related renal failure. Needless to say, good hydration prior to, during, and after the study is an important part of the management of these patients. With regard to blood pressure improvement or reversal and renal function, the results with the use of stents do not appear to be any different than those seen with successful PTA. The use of PTA with or without stents may be appropriate for initial therapy in those with amenable lesions as described above, but the primary patency and clinical benefits of these procedures in all other patients do not yet appear comparable to those seen with surgical revascularization.

Once surgical revascularization has been decided upon, certain preoperative measures must be followed to optimize the surgical outcome. The patient's blood pressure must be adequately controlled to maintain near normal diastolic pressures. By the same token, the antihypertensive medications should be reduced to the minimum required while in hospital to maintain an adequate blood pressure. Importantly, the patient is appropriately hydrated in preparation for surgery and opti-

mized from a cardiopulmonary standpoint – in an intensive care unit (ICU) if necessary.

A variety of surgical techniques are available to the experienced vascular surgeon in order to achieve successful renal revascularization. The standard surgical approaches include aortorenal endarterectomy and aortorenal bypass. In the first approach, the aorta is opened transversely or transaortically depending upon the surgical requirements, and the diseased intima is endarterectomized to include its extension into the renal arterial orifices. This can be done for unilateral or bilateral disease and is becoming a desirable approach when concomitant aortic grafting is to be performed. Aortorenal bypass with either autologous or synthetic conduits can be performed with relative ease if the aorta itself is not too diseased to preclude safe application of a clamp or when aortic replacement with synthetic graft is also being performed. Using the diseased aorta itself as an inflow source when aortic replacement is not indicated has caused some surgeons to prefer the performance of nonanatomic bypasses. Other circumstances that might also suggest the use of alternative revascularization techniques include increased operative risk, anatomic considerations, bilateral disease, a desire to avoid the aorta secondary to scarring or disease, or the preference for a non-midline incision to avoid potential respiratory or gastrointestinal consequences associated with such an incision. The most commonly performed extra-anatomic bypasses are hepatorenal or splenorenal bypasses. Other less commonly used alternatives include iliorenal, superior mesenteric artery (SMA)-renal, and supraceliac aorto-renal bypasses. Each of these techniques has pros and cons too extensive to address here, but the nature of the disease, patient anatomy, and prior surgical history along with current medical condition will dictate the procedure to perform. Aside from iliorenal bypasses and possibly SMA – renal bypasses, which for technical reasons are less desirable approaches in most circumstances, the above-named techniques all should have similar outcomes when performed well.

One other technique, ex vivo reconstruction, is required when there is FMD and aneurysms or stenoses involving renal artery branches, renal artery dissection, congenital abnormalities of the branches requiring resection, and patients with prior grafts or surgery to the distal renal artery. This technique involves mobilization and removal of the kidney from its bed with either autotransplantation to another site (i.e. the iliac fossa) or replacement in its native bed.

Each of these techniques has its advocates. In appropriately selected patients and technically well-performed surgery, the outcomes for each are similar with one caveat. In combined aortic replacement and renal revascularization, there is increased morbidity and mortality as compared to the other “pure” renal revascularization techniques.

Finally, the introduction of newer antihypertensive agents may have some impact on the progression of disease. In short-term studies, dihydropyridine calcium channel blockers have been shown to greatly reduce blood pressure with minimal impairment of renal function in the patients with renovascular disease [32]. The long-term benefits of these agents are unknown. However, the use of ACE inhibitors is controversial. In patients with bilateral renal artery stenosis, treatment with ACE inhibitors can lead to sudden and dramatic increases in serum creatinine. The acute changes are by-and-large reversible, but cases of significant renal impairment have been reported.

In summary, an aggressive approach to revascularization should be pursued. Medical therapy alone in patients with physiologically significant renal artery stenosis will place

them at increased risk for disease progression and worsening renal function. Surgical revascularization still appears to be superior to percutaneous approaches in all but the straightforward main renal artery atherosclerotic and FMD patients. The outcomes and durability of primary PTA and stent placement in ostial atherosclerotic disease remain to be delineated in a large, randomized, prospective trial. Until then it can only be considered as an option to use in the setting of restenosis, poor immediate results, or technically difficult cases. Also, the potential impact that stent placement will have on future surgical revascularization options cannot be discounted. There is still room for improvement in technical designs of stents and their delivery systems that may impact on the viability and durability of this procedure as compared to surgery. The importance of a team approach including a nephrologist, interventional radiologist, and vascular surgeon all aware of the various issues addressed in this chapter and communicating with each other cannot be overemphasized in providing the patient with the highest quality care available.

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