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Renovascular Disease: Open Surgical Treatment

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INTRODUCTION

Following Goldblatt's innovative work defining the causal relationship between renovascular disease and hypertension in 1934,¹ the surgical management of renovascular disease evolved through three eras: nephrectomy, open surgical correction, and finally renal artery angioplasty with or without stenting (Fig. 128.1). Each era was met with initial enthusiasm, which was soon tempered by the modest results of each treatment. Nephrectomy is now rarely indicated in the management of renovascular disease, and recent randomized clinical trials comparing renal artery stenting with best medical management have demonstrated no advantage of catheter-based intervention (see Ch. 129, Renovascular Disease: Endovascular Treatment). This chapter reviews patient selection for open repair of renovascular disease and then describes specific surgical approaches and their expected outcomes in contemporary practice.

INDICATIONS FOR SURGICAL REPAIR

The rationale for the treatment of renovascular disease by any method is to improve event-free survival. Our experience with

500 consecutive patients treated for atherosclerotic renovascular disease suggests that severe hypertension can be viewed as a key preoperative characteristic favoring clinical benefit, whereas renal function after operation can be considered the key clinical response.² An early incremental increase in excretory renal function has been the primary determinant of dialysis-free survival for all atherosclerotic patients.^{3,4}

Unfortunately, once renovascular disease is identified in combination with severe hypertension, discriminating predictors of blood pressure and renal function response are lacking. Lateralizing renin assays have predicted blood pressure benefit but not blood pressure cure.^{5,6} Moreover, renal function response in patients with ischemic nephropathy is uncertain after renal artery intervention.⁴ Studies have also described a significant correlation between parameters derived from segmental renal artery Doppler spectral analysis (i.e., resistive index) and response to intervention as well as progression to dialysis dependence.^{7,8} However, we have not been able to reproduce these results for open operative management of renal artery disease.⁹ Rather, in our clinical experience with more than 1000 patients with atherosclerotic renal artery disease submitted to open operative repair, factors favoring the recovery of renal function

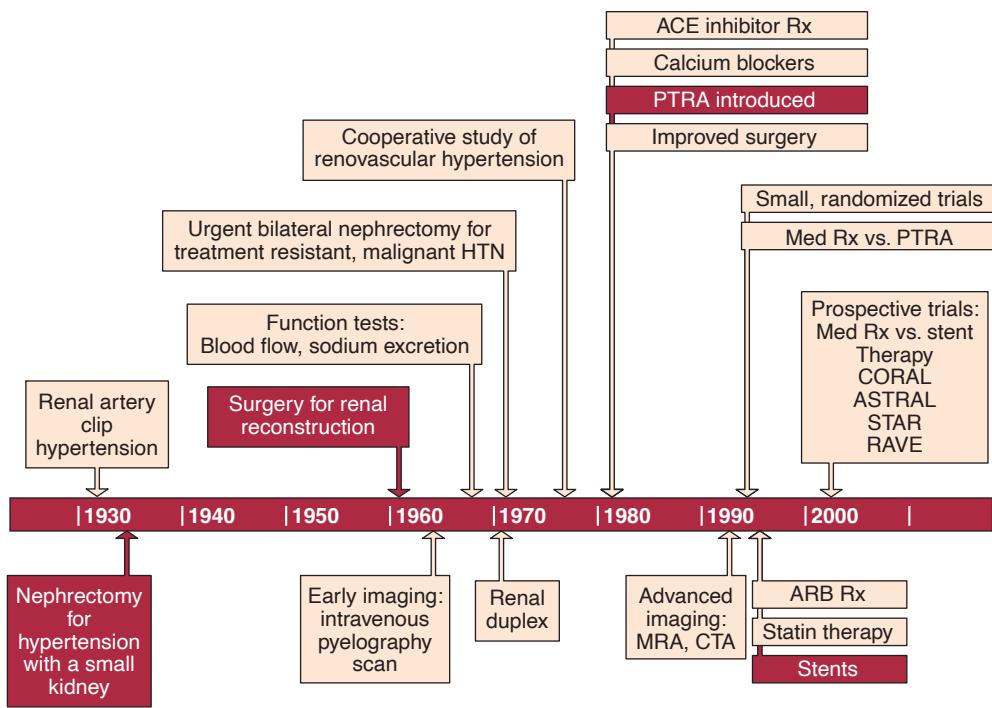


Figure 128.1 Time line showing evolution of the surgical management of renovascular disease through three eras. ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker; CTA, computed tomographic angiography; HTN, hypertension; MRA, magnetic resonance angiography; PTRA, percutaneous transluminal renal angioplasty; Rx, therapy.

after operation have included severe preoperative hypertension, bilateral or global atherosclerotic renovascular disease due to high-grade (>90%) stenosis or renal artery occlusion, and rapidly deteriorating renal function before surgery.^{4,6,10-13} When each of these favorable features was expressed in patients considered permanently dialysis dependent, 70% of those submitted to open operative repair were permanently removed from dialysis.^{4,13}

The quality of studies designed to define the natural history of the atherosclerotic renal artery has varied widely.^{10,14-23} Most commonly, authors have considered anatomic progression of atherosclerotic renovascular disease a certainty, one associated with an inevitable decline in kidney size and kidney function. This view has been cited to support intervention for renal artery disease whenever it is discovered, even in the absence of hypertension or renal insufficiency.

On the basis of a prospective, population-based study of progression of atherosclerotic renovascular disease in the elderly, we do not recommend “prophylactic” intervention for asymptomatic renovascular disease.²⁴ This longitudinal cohort study included 119 Cardiovascular Health Study participants, with 235 kidneys for study, and no kidney progressed to a renal artery occlusion during a mean follow-up of 8.5 years. Among participants with mild to moderate hypertension in the Cardiovascular Health Study, progression to hemodynamically significant renal artery stenosis was observed in only 4%, or an annualized rate of 0.5% per year.²⁴

When a patient has bilateral renal artery stenosis and severe hypertension, the decision for empiric open renal

reconstruction is based on the severity of the hypertension and the anatomy of the renal artery lesions. When disease consists of severe stenosis of one renal artery and only mild to moderate contralateral disease, it is treated as a unilateral lesion. If both renal artery lesions are moderately severe (60%–80% diameter-reducing stenosis), revascularization is undertaken only if associated hypertension is severe. In contrast, if both renal artery lesions are severe (>80% stenosis) and the patient has severe hypertension, bilateral simultaneous renal revascularization is performed, especially when excretory renal insufficiency is present. However, because the degree of azotemia usually parallels the severity of hypertension, a patient who presents with severe excretory renal insufficiency but only mild hypertension usually has concomitant renal parenchymal disease from another cause (e.g., diabetes). Characteristically, renovascular disease contributing to severe azotemia or dialysis dependence is associated with severe hypertension and severe bilateral stenosis or renal artery occlusion.^{3,4,10,13}

Open Operative Strategy

The presence of severe hypertension is considered a prerequisite for renal artery intervention, and functional studies are used to guide the management of unilateral lesions. Empiric renal artery repair is performed without functional studies when hypertension is severe, renal artery disease is bilateral, or the patient has ischemic nephropathy.^{3,6,25} Prophylactic renal artery repair in the absence of hypertension, whether as an isolated procedure or in combination with aortic reconstruction,

is not recommended. During surgical reconstruction, all hemodynamically significant renal artery disease is corrected in a single operation with the exception of disease requiring bilateral *ex vivo* reconstructions, which are staged. Nephrectomy is reserved for a nonfunctioning kidney (i.e., ≤10% function by renography) with unreconstructable renal artery disease, or a kidney acting as a pressor kidney.^{10,18,26} Direct aortorenal reconstructions are preferred to indirect methods because concomitant disease of the celiac axis is present in 40% to 50% of patients and bilateral repair is required in half.^{4,6} Failed renal artery repair is also associated with a significantly increased risk of dialysis dependence.²⁵ To minimize these failures, intraoperative duplex ultrasonography is used to evaluate the technical results of surgical repair before closure.²⁷

In our experience, percutaneous management does not improve hypertension and renal function as effectively as open operative repair.^{28–30} Consequently we continue to advocate open operative repair for select patients. These include children with hypoplastic lesions (see Ch. 132, Renovascular and Aortic Developmental Disorders),³¹ adults with dysplastic lesions other than medial fibroplasia, and patients with medial fibroplasia involving branch and segmental renal arteries complicated by a renal artery aneurysm.³² We also recommend open operative repair for atherosclerotic renovascular disease in good-risk patients, younger than 65 years of age, who demonstrate global renal ischemia manifested by critical renal artery stenosis or occlusion, especially when severe hypertension is associated with excretory renal insufficiency and rapidly deteriorating excretory function.^{3,4,13} Selection criteria for the repair of renal artery aneurysms are discussed in Chapter 131 (Renovascular Disease: Aneurysms and Arteriovenous Fistulae), but we have continued to consider open operative repair as opposed to coiling, stenting, or stent-grafting of these lesions.³²

Certain measures are used in almost all open renal artery operations. Although the data to support the use of mannitol is mixed, we favor using mannitol, and administer it intravenously in 12.5-g doses early in the operation during aortic and renal artery dissection. Repeated doses are administered before and after periods of renal ischemia, up to a total dose of 1 g/kg of patient body weight. Just before renal artery cross-clamping, 100 U/kg of heparin is given intravenously, and systemic anticoagulation is verified by activated clotting time. Unless it is required for hemostasis, protamine is not routinely administered for reversal of heparin at completion of the operation.

OPERATIVE EXPOSURE

See Chapter 56 for Abdominal Vascular Exposures.

Midline Exposure

A midline abdominal incision is made for operative repair of atherosclerotic renal artery disease when bilateral renal artery repair or combined aortic and renal repair is planned. The patient is positioned supine with the umbilicus at the level of the table break, with the operating table flexed 10 to 15 degrees. To obtain full exposure of the upper abdominal aorta and renal branches, it is important that the last 1 or 2 cm of the proximal incision be

made coursing to one side of the xiphoid, in order to release the costoxiphoid ligaments (Fig. 128.2A). Some type of fixed mechanical retraction is also advantageous, particularly when combined aortic and renal procedures are required. Otherwise, extended flank and subcostal incisions are reserved for correction of unilateral branch renal artery lesions or splanchnic–renal bypass.

When the midline xiphoid-to-pubis incision is used, the posterior peritoneum overlying the aorta is incised longitudinally and the duodenum mobilized at the ligament of Treitz (see Fig. 128.2B). The inferior mesenteric vein may need to be ligated to allow proximal exposure. During this maneuver, it is important to identify and spare the meandering mesenteric vessel that may be found at this level. Finally, the duodenum is reflected to the patient's right to expose the left renal vein. By extending the posterior peritoneal incision to the left along the inferior border of the pancreas, an avascular plane posterior to the pancreas can be entered (see Fig. 128.2B) to expose the entire left renal hilum. This exposure is of special importance when there are distal renal artery lesions to be managed (Fig. 128.3A). The left renal artery lies posterior to the left

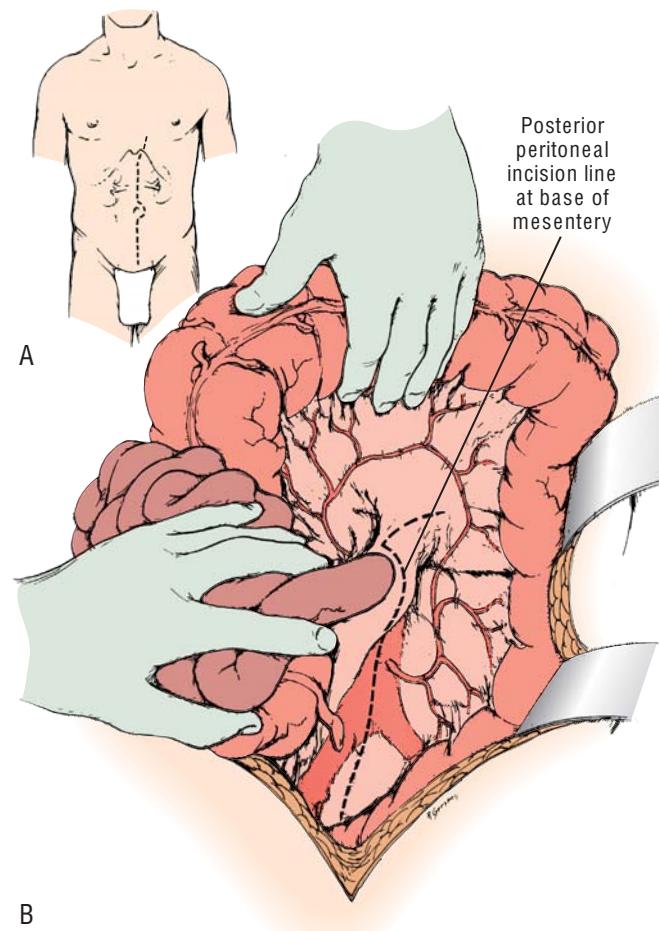


Figure 128.2 (A and B) Exposure of the aorta and left renal hilum through the base of the mesentery. Extension of the posterior peritoneal incision to the left, along the inferior border of the pancreas, provides entry to an avascular plane posterior to the pancreas. This allows excellent exposure of the entire left renal vein and hilum as well as of the proximal right renal artery. (From Benjamin ME, Dean RH. Techniques in renal artery reconstruction: part I. *Ann Vasc Surg*. 1996;10:306–314.)

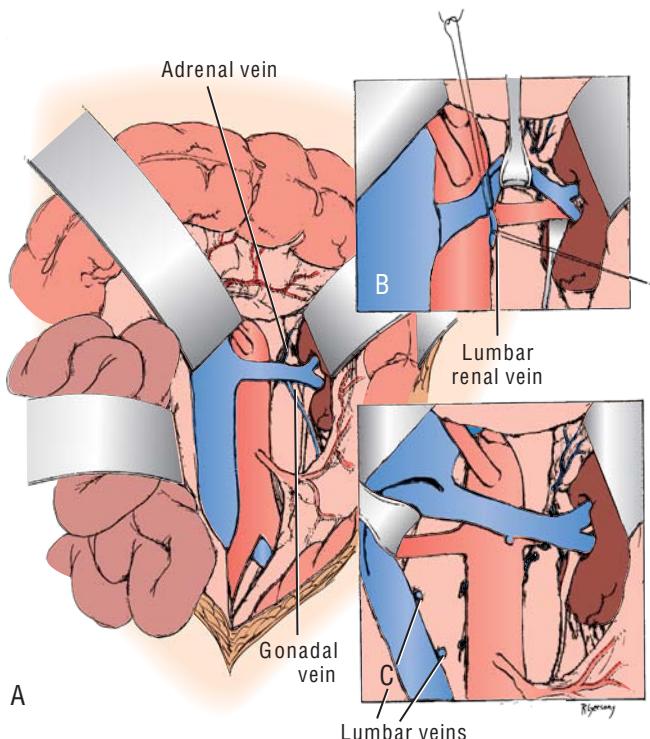


Figure 128.3 (A) Exposure of the proximal right renal artery through the base of the mesentery. (B) Mobilization of the left renal vein by ligation and division of the adrenal, gonadal, and lumbar-renal veins allows exposure of the entire left renal artery to the hilum. (C) On occasion, lumbar veins are ligated and divided to allow retraction of the vena cava to the right. Often, adequate exposure of disease of the proximal renal artery can be obtained without this maneuver. (From Benjamin ME, Dean RH. Techniques in renal artery reconstruction: part I. *Ann Vasc Surg*. 1996;10:306–314.)

renal vein. In some cases, the vein can be retracted cephalad to expose the artery; in other cases, caudal retraction of the vein provides better access. Usually, the gonadal and adrenal veins, which enter the left renal vein, must be ligated and divided to facilitate exposure of the distal artery. Frequently, a lumbar vein enters the posterior wall of the left renal vein; it can easily be injured unless special care is taken (see Fig. 128.3B). For full mobilization of the left renal vein, ligation and division of the lumbar branch is required. The proximal portion of the right renal artery can be exposed through the base of the mesentery by retracting the left renal vein cephalad and the vena cava to the patient's right (see Fig. 128.3C). However, mobilizing the duodenum and right colon medially best exposes the distal portion of the right renal artery; the right renal vein is mobilized and usually retracted cephalad to expose the artery.

Flank Exposure

When a branch renal artery repair is required, especially when *ex vivo* technique is used or when the supraceliac aorta is used as an inflow source for aortorenal bypass, an extended flank incision is useful. With the ipsilateral flank bumped, the incision extends from the opposite semilunar line into the flank, bisecting the abdominal wall between the costal margin and iliac

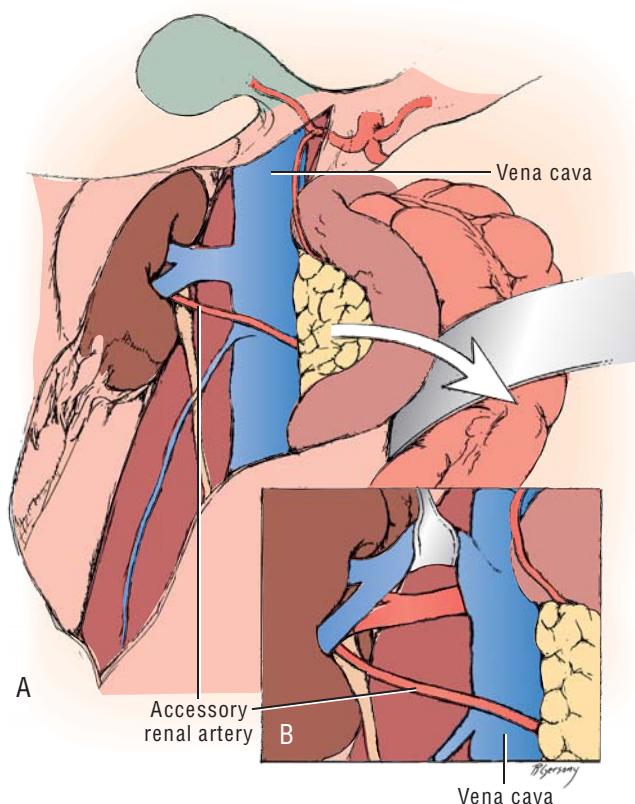


Figure 128.4 (A) Arteries encountered anterior to the vena cava should be considered accessory renal arteries and preserved. (B) The right renal vein is typically mobilized superiorly for exposure of the distal right renal artery. (From Benjamin ME, Dean RH. Techniques in renal artery reconstruction: part I. *Ann Vasc Surg*. 1996;10:306–314.)

crest. A left or right visceral mobilization allows access to the renal vasculature and the aortic crus. The crus can be divided, and an extra-pleural dissection of the descending thoracic aorta can provide access to the T9–10 thoracic aorta for proximal control and anastomosis.^{33,34}

Whether right or left branch renal artery exposure is required, the key is creation of the correct dissection plane between the mesentery anteriorly and Gerota's fascia posteriorly. The renal vein is identified first and mobilized from inferior vena cava (IVC) origin to renal hilum. On the right, small venous branches at the junction with the vena cava require ligation. The adrenal, gonadal, and lumbar branches on the left may be sacrificed to facilitate exposure.

Branch renal artery exposure on the right is achieved by colonic and duodenal mobilization. First, the hepatic flexure is mobilized at the peritoneal reflection (Fig. 128.4). With the right colon retracted medially and inferiorly, a Kocher maneuver mobilizes the duodenum and pancreatic head to expose the IVC and right renal vein (see Fig. 128.4A). Typically, the right renal artery is located just inferior to the accompanying vein, which can be retracted superiorly to provide the best exposure. Although accessory vessels may arise from the aorta or iliac vessels at any level, all arterial branches coursing anterior to the IVC should be considered accessory right renal branches and carefully preserved (see Fig. 128.4B).

DIRECT SURGICAL TECHNIQUES

A variety of open operative techniques have been used to correct renal artery disease, but three basic operations have been used most frequently: aortorenal bypass, renal artery thromboendarterectomy, and renal artery reimplantation. No single approach provides optimal repair for all types of renovascular lesions in all patients. Aortorenal bypass using saphenous vein is probably the most versatile technique; however, transaortic thromboendarterectomy is especially useful for ostial atherosclerosis involving multiple renal arteries, or in association with juxtarenal aortic procedures. On occasion, the renal artery will demonstrate sufficient redundancy to allow reimplantation; this is probably the simplest technique and one that is particularly appropriate to hypoplastic renal artery lesions in children (see Ch. 132, Renovascular and Aortic Developmental Disorders).^{30,31}

Aortorenal Bypass

Three types of materials are typically used for aortorenal bypass: autologous saphenous vein, autologous hypogastric artery, and synthetic graft. The decision as to which graft should be used depends on a number of factors. In most instances, the authors prefer the saphenous vein for older adults. However, if the vein is small (<4 mm in diameter) or sclerotic, the hypogastric artery or a synthetic graft may be preferable. In addition, vein enlargement can be anticipated in virtually all young adults. Although structural immaturity has been cited³¹ for enlargement and aneurysmal degeneration when saphenous vein is used for renal artery reconstruction in children, the normal renal blood flow in the young resembles that of an arteriovenous fistula with continuous forward flow. This fact alone may account for vein enlargement in the young patient with normal renovascular resistance. In lieu of vein, a 6-mm thin-wall polytetrafluoroethylene (PTFE) graft is satisfactory when the distal renal artery is of sufficient caliber (≥ 4 mm). Although we do not use them in our practice, polyethylene terephthalate (Dacron) grafts (6 mm) have also been used with success. Hypogastric artery autograft is preferred for aortorenal bypass in children when reimplantation is not possible.^{28,31,35,36}

End-to-side distal anastomoses are occasionally used to include polar renal arteries. In this instance, an end-to-side anastomosis between the polar renal artery and the graft is performed after the proximal aortic anastomosis. After the end-to-side anastomosis has been completed, the most distal main renal artery reconstruction is made in an end-to-end fashion (Fig. 128.5).

In creating the distal anastomosis, the length of the arteriotomy should be at least three times the diameter of the renal artery so as to reduce the complication of late anastomotic stenosis. An anastomosis with 7-0 monofilament polypropylene as a continuous suture is created with loupe magnification. Just before completion of the renal artery anastomosis, the occluding clamps are temporarily removed to flush air and small debris. When renal artery bypass can be accomplished in less than 40 minutes, cold perfusion preservation is not required. The

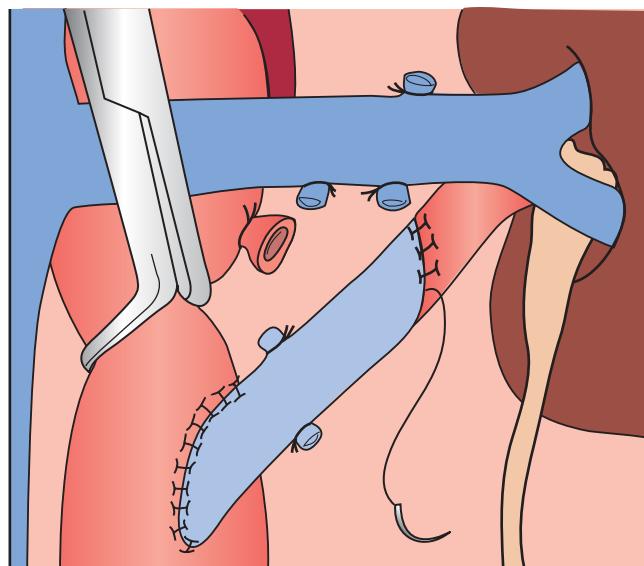


Figure 128.5 Technique for end-to-end aortorenal bypass grafting. The length of arteriotomy is at least three times the diameter of the artery so as to prevent recurrent anastomotic stenosis. For the anastomosis, 6-0 or 7-0 monofilament polypropylene sutures are used in continuous fashion with loupe magnification. If the apex sutures are placed too deeply or with excess advancement, stenosis can be created, posing a risk of late graft thrombosis. (From Benjamin ME, Dean RH. Techniques in renal artery reconstruction: part I. *Ann Vasc Surg*. 1996;10:306–314.)

aortic anastomosis is performed first, removing an ellipse of the anterolateral aortic wall. This is especially important when the aorta is relatively inflexible because of atherosclerotic involvement. A 4.0-mm aortic punch applied two or three times creates a satisfactory ellipse in most instances. In combined aortorenal reconstructions, the proximal anastomosis is performed first with a thin-wall 6-mm PTFE graft for renal artery bypass attached in end-to-side fashion to the larger polyester aortic graft. The distal aortic reconstruction is completed, and the distal renal anastomosis is created as the last step.

Thromboendarterectomy

In cases of ostial atherosclerosis of both renal artery origins, simultaneous bilateral endarterectomy may be the most suitable procedure. Endarterectomy may be either transaortic or transrenal.

For the majority of renal endarterectomies, the transaortic technique is used.³ This method is particularly applicable in patients with multiple renal arteries that demonstrate ostial disease. For optimal results, all visible and palpable renal artery atheromas should end within 1 cm of their aortic origin. Transaortic endarterectomy is performed through a longitudinal aortotomy with sleeve endarterectomy of the aorta and eversion endarterectomy of the renal arteries (Fig. 128.6). The aortotomy is closed with 5-0 polypropylene as a continuous suture. When combined aortic replacement is planned, the transaortic endarterectomy can be performed through the transected aorta. Alternatively, after endarterectomy the aortotomy can be closed primarily to the infrarenal aorta, and then clamp replaced infrarenally prior to aortic replacement. Whether a

longitudinal aortotomy is used or an endarterectomy is performed through the divided aorta, it is important to mobilize the renal arteries extensively to allow eversion of the artery into the aorta. This allows the distal endpoint to be completed under direct vision. When the aortic atheroma is divided flush with the adventitia by the use of scissors, tacking sutures are not usually required.

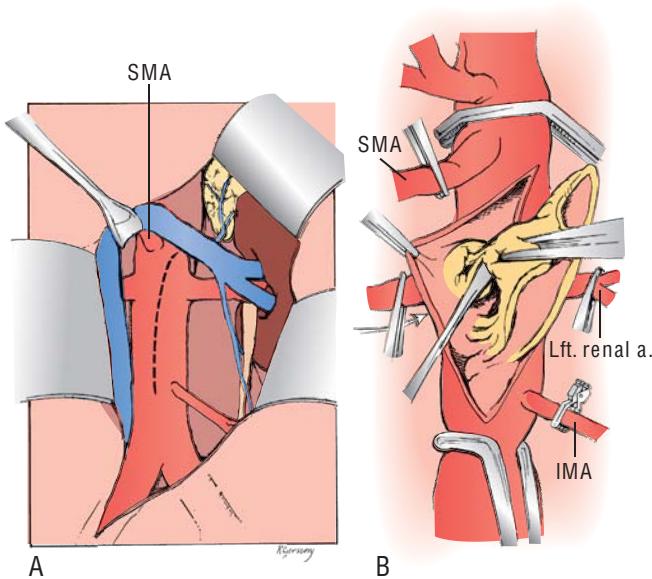
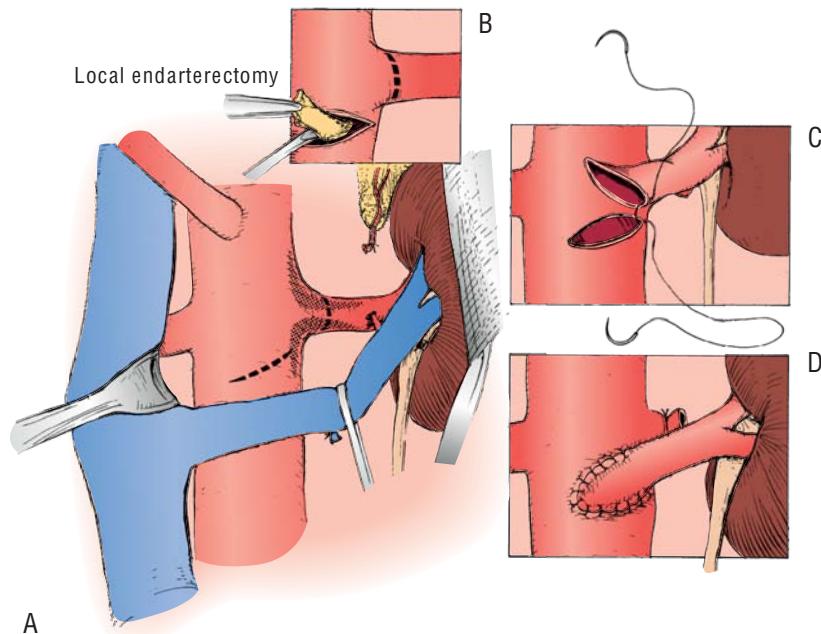


Figure 128.6 Exposure for a longitudinal transaortic endarterectomy is made by the standard transperitoneal approach. The duodenum is mobilized at the ligament of Treitz in standard fashion, or for more complete exposure, the ascending colon and small bowel are mobilized. (A) The dotted line shows the location of the aortotomy. (B) The plaque is transected sharply, and with eversion of the renal arteries, the atherosclerotic plaque is removed from each renal ostium. The aortotomy is typically closed with a running 4-0 or 5-0 polypropylene suture. IMA, inferior mesenteric artery; SMA, superior mesenteric artery. (From Benjamin ME, Dean RH. Techniques in renal artery reconstruction: part I. *Ann Vasc Surg*. 1996;10:306–314.)



As is the case for thromboendarterectomy at all sites, the procedure is contraindicated by the presence of preaneurysmal degeneration of the aorta and transmural calcification. Transmural calcification can be subtle and may be missed unless careful attention is given to gentle palpation of the aorta. Aortic atheroma complicated by transmural calcification resembles fine-grade sandpaper on palpation of the outer surface of the aorta. Endarterectomy in this setting is characterized by numerous sites of punctate bleeding after blood flow is restored.

Reimplantation

After the renal artery has been dissected from the surrounding retroperitoneal tissue, the vessel may be somewhat redundant. When the renal artery stenosis is orificial and there is sufficient vessel length, the renal artery can be transected and reimplanted onto the aorta at a slightly lower level. The renal artery must be spatulated and a portion of the aortic wall removed, as in renal artery bypass (Fig. 128.7). This technique has particular application to children with orificial lesions, avoiding the need for graft material^[31]; however, it is suitable for selected atherosclerotic lesions as well.^[37] Unlike prosthetic bypass performed during combined aortic replacement in adults, anastomosis from the renal artery to the graft is usually performed immediately after the proximal aortic anastomosis, followed by distal aortic reconstruction.

INDIRECT SURGICAL TECHNIQUES

Splanchnic–Renal Bypass

Splanchnic–renal bypass and other indirect revascularization procedures have received increased attention as alternative methods for renal revascularization.^[38] The authors do not believe that these procedures demonstrate durability equivalent

Figure 128.7 When the renal artery is redundant, the vessel can be reimplemented at a lower level (A). Local endarterectomy (B) allows placement of the monofilament suture in the aortic wall (C). The native renal artery is then ligated, proximally spatulated, and reimplemented (D). (From Benjamin ME, Dean RH. Techniques in renal artery reconstruction: part II. *Ann Vasc Surg*. 1996;10:409–414.)

to that of direct aortorenal reconstructions, but they are useful in a highly select subgroup of high-risk patients.^{3,39}

Hepatorenal Bypass

A right subcostal incision is used to perform hepatorenal bypass.³⁸ The lesser omentum is incised to expose the hepatic artery both proximal and distal to the gastroduodenal artery (Fig. 128.8). Next, the descending duodenum is mobilized by a Kocher maneuver, the IVC is identified, the right renal vein is identified, and the right renal artery is exposed either immediately cephalad or caudad to the renal vein.

A greater saphenous vein graft is usually used to construct the bypass. The hepatic artery anastomosis of the vein graft can be placed at the site of the amputated stump of the gastroduodenal artery (see Fig. 128.8A). However, this vessel may serve as an important collateral for intestinal perfusion. Therefore the proximal anastomosis is usually made to the common hepatic artery, routing the graft through the foramen of Winslow. The renal artery is then transected and brought anterior to the vena cava for anastomosis end to end to the graft (see Fig. 128.8B).

Splenorenal Bypass

Splenorenal bypass can be performed through a midline or a left subcostal incision.^{38,39} The posterior pancreas is mobilized by reflecting the inferior border cephalad. A retropancreatic plane is developed and the splenic artery mobilized from the left gastroepiploic artery to the level of its branches. The left renal artery is exposed cephalad to the left renal vein after division of the adrenal vein. After the splenic artery has been mobilized, it may be divided distally, spatulated, and anastomosed end-to-end to the transected renal artery (Fig. 128.9).

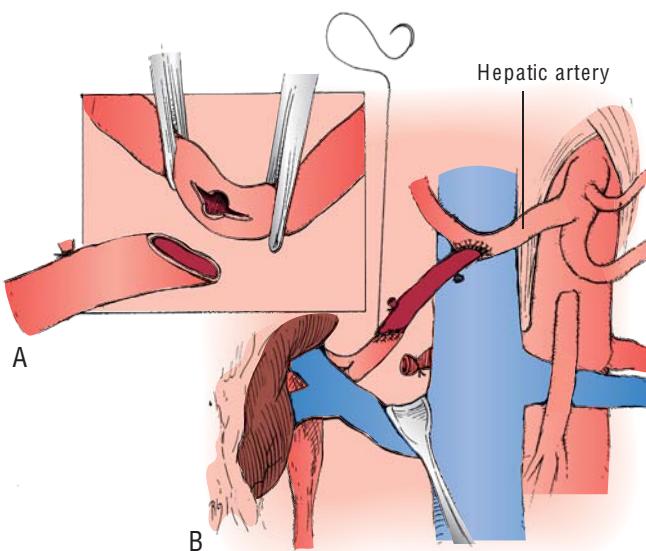


Figure 128.8 The reconstruction is completed with a saphenous vein interposition graft between the side of the hepatic artery (A) and the distal end of the transected right renal artery anterior to the vena cava (B). (From Benjamin ME, Dean RH. Techniques in renal artery reconstruction: part II. *Ann Vasc Surg*. 1996;10:409–414.)

Alternatively, a segment of saphenous vein may be used as a bypass.

Ex Vivo Reconstruction

Because of the widespread use of percutaneous techniques for main renal artery diseases, a significant proportion of open renal artery reconstructions may require branch exposure and branch reconstruction. These procedures are complex and culminate in prolonged renal ischemia. Available data suggest that when more than 40 minutes of warm renal ischemia is required for renal revascularization, measures to protect renal function should be instituted.^{40–43}

Several pharmacologic therapies have been promoted to provide protection during renal ischemia; however, no therapy to date has surpassed hypothermia for protection when renal

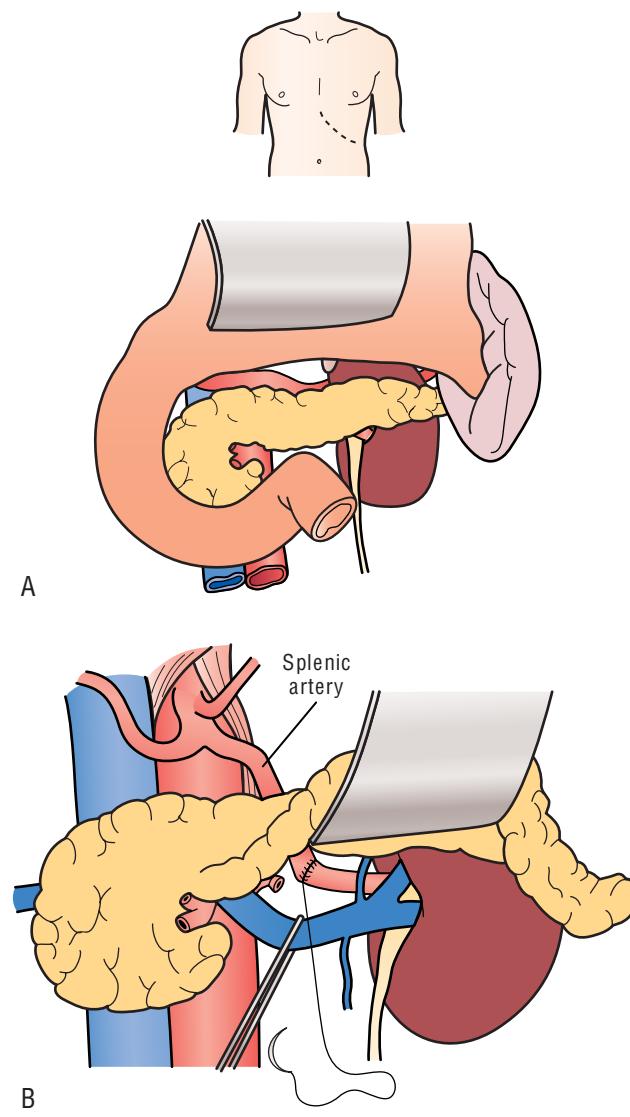


Figure 128.9 Exposure of the left renal hilum in preparation for splenorenal bypass (A). The pancreas has been mobilized along its inferior margin and retracted superiorly (B). The transected splenic artery may be anastomosed end to end to the transected left renal artery. A splenectomy is not routinely performed. (From Benjamin ME, Dean RH. Techniques in renal artery reconstruction: part II. *Ann Vasc Surg*. 1996;10:409–414.)

ischemia exceeds 1 hour.^{42,44–47} Surface cooling and hypothermic perfusion have been proposed, but the advantages of each are not well defined. Renal tolerance to ischemia is, in part, related to the duration of ischemia, the adequacy of collateral circulation, and the method of vascular control. Unprotected warm renal ischemia is best tolerated when only the renal artery is controlled.⁴² In a similar manner, intermittent renal perfusion is associated with greater dysfunction than is isolated arterial control.⁹

Numerous methods for renal cooling and hypothermia during branch renal artery repair have been described. Our preference has been intermittent hypothermic perfusion and topical ice slush. Otherwise, several steps are common to each branch renal artery reconstruction. To promote renocortical perfusion, small doses of mannitol are administered intravenously throughout renal artery exposure and reperfusion.^{47–49} Prior to division of the renal artery, heparin (100 U/kg) is administered intravenously and monitored, as described earlier.

Hypothermia appears to be more important than the composition of the perfusate; however, our preference is a perfusate with an intracellular composition of electrolytes. This composition theoretically limits ion exchange and intracellular volume shifts that contribute to organelle dysfunction associated with decreased activity of membrane-bound sodium–potassium adenosine triphosphatase.^{42,50,51} Regardless of whether the renal vein is divided and reattached, branch renal artery repairs with use of cold perfusion preservation are made in an orthotopic fashion, with the kidney returned to the renal fossa rather than autotransplanted into the pelvis.

Several exposures for hypothermic branch renal artery reconstructions are available. When isolated branch renal repair is performed with orthotopic replacement, an extended

flank incision is made from the midline to the posterior axillary line, as described earlier. This method is the authors' preferred approach for *ex vivo* reconstruction. The ureter is mobilized to the pelvic brim along with a large amount of periureteric soft tissue. A Silastic sling is placed around the ureter to control ureteric collaterals and to prevent subsequent renal rewarming.

Gerota's fascia is opened with a cruciate incision; the kidney is completely mobilized, and the renal vessels are divided. Hypothermia may be accomplished by topical ice slush placement, as illustrated in Figure 128.10. To perform hypothermic perfusion, the kidney is placed in a plastic sling and the renal artery perfused with a chilled renal preservative solution. Continuous perfusion during the period of total renal ischemia is possible with perfusion pump systems, and this may be superior for prolonged renal preservation.⁵² However, simple intermittent flushing with a chilled preservative solution provides equal protection during the shorter periods (2–3 hours) required for *ex vivo* dissection and branch renal artery reconstruction. The chilled (5–0°C) solution is hung at a height of at least 2 m. Immediately after its removal from the renal fossa, the kidney is flushed with 300 to 500 mL of solution until the venous effluent is clear and all segments of the kidney have blanched. As each anastomosis is completed, the kidney is perfused with an additional 100 to 200 mL of chilled solution. In addition to maintaining satisfactory hypothermia, periodic perfusion demonstrates suture-line leaks that are repaired before reimplantation. With this technique, renal core temperatures are maintained at 10°C or below throughout the period of reconstruction.

Even though it is an accepted method after *ex vivo* reconstruction, autotransplantation to the iliac fossa is unnecessary

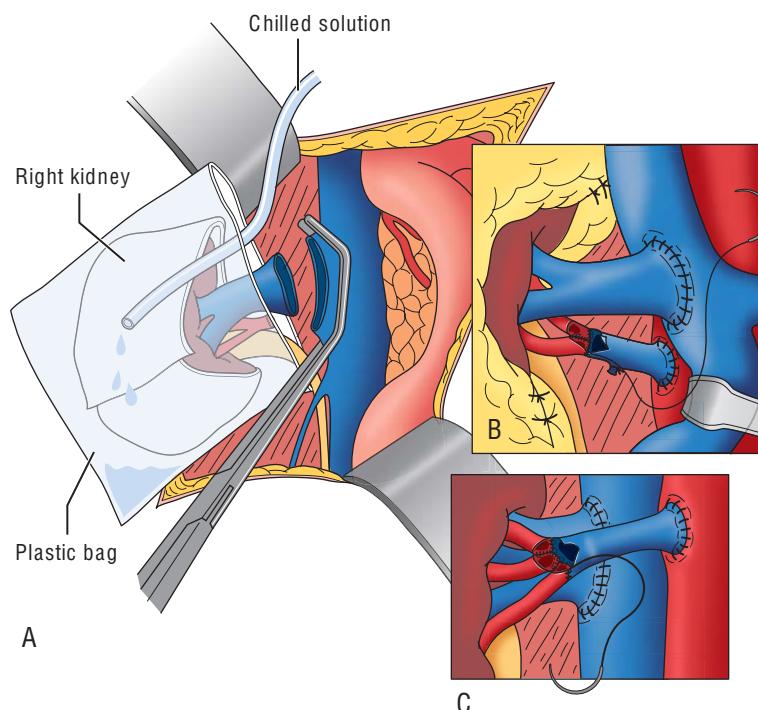


Figure 128.10 An ellipse of the vena cava containing the renal vein origin is excised by placement of a large partially occluding clamp (A). After *ex vivo* branch repair, the renal vein can be reattached without risk of anastomotic stricture (B). The kidney is returned to its native bed after *ex vivo* repair. Gerota's fascia is reapproximated to provide stability to the repaired kidney. Arterial reconstruction can be accomplished by end-to-end anastomoses after syndactylizing the distal branch or combined with end-to-side anastomoses (C). (From Benjamin ME, Dean RH. Techniques in renal artery reconstruction: part II. *Ann Vasc Surg*. 1996;10:409–414.)

for most *ex vivo* reconstructions. This technique was adopted from renal transplantation. Reduction in the magnitude of the operative exposure, manual palpation of the transplanted kidney, and ease of removal when treatment of rejection fails are all practical reasons to place the transplanted kidney into the recipient's iliac fossa. However, none of these advantages applies to the patient requiring autogenous *ex vivo* reconstruction. Because many *ex vivo* procedures are performed in relatively young patients, the durability of the operation must be measured in terms of decades. For this reason, attachment of the kidney to the iliac arterial system within or below sites that are susceptible to subsequent atherosclerosis subjects the repaired vessels to disease that may threaten their long-term patency. Subsequent management of peripheral vascular disease may be complicated by the presence of the autotransplanted kidney. Finally, if the kidney is replaced in the renal fossa and the renal artery graft is properly attached to the aorta at a proximal infrarenal site, the result should equal those of the standard aortorenal bypass and thus carry a high probability of technical success and long-term durability.

INTRAOPERATIVE ASSESSMENT

Provided that the best method of reconstruction is chosen for renal artery repair, the short course and high blood-flow rates characteristic of direct renal reconstructions favor their patency. Consequently, flawless technical repair plays a dominant role in determining postoperative success.^{53–60} The negative impact of technical errors unrecognized at operation is implied by the fact that we have observed no late thromboses of renovascular reconstructions free of disease after 1 year.⁶¹

Intraoperative Duplex Sonography

The risks and inherent limitations of completion angiography are not shared with intraoperative duplex sonography.²⁷ Because the ultrasound probe can be placed immediately adjacent to the vascular repair, high carrying frequencies may be used to provide excellent B-scan detail sensitive to 1.0-mm defects. Once defects are imaged, they can be viewed in a multitude of projections during conditions of uninterrupted pulsatile blood flow. In addition to excellent anatomic detail, important hemodynamic information is obtained from the spectral analysis of the Doppler-shifted signal proximal and distal to the imaged defect.²⁷ The freedom from static projections, absence of potentially nephrotoxic contrast material, and hemodynamic data provided by Doppler spectral analysis make duplex sonography a useful intraoperative method to assess both renovascular and mesenteric repairs. For these advantages of intraoperative duplex sonography to be realized, close cooperation between the vascular surgeon and the vascular technologist is required for accurate intraoperative assessment (see Ch. 22, Vascular Laboratory: Arterial Duplex Scanning).

Currently, we use a 5/7.0-MHz compact linear array probe with Doppler color flow designed specifically for intraoperative assessment. The probe is placed within a sterile sheath with a latex tip containing sterile gel. After the operative field

has been flooded with warm saline, B-scan images are first obtained in longitudinal projection. Care is taken to image the entire upper abdominal aorta and renal artery origins along the entire length of the repair. All defects seen in longitudinal projection are imaged in transverse projection to confirm their anatomic presence and estimate associated luminal narrowing. Doppler samples are then obtained just proximal and distal to the imaged lesions in longitudinal projection, determining their potential contribution to flow disturbance. The author's (KJH) criteria for major B-scan defects (>60% stenosis) of a peak systolic velocity greater than 2.0 m/s associated with a distal turbulent waveform have been validated in a canine model of graded renal artery stenosis.²⁷ They have also proved valid in a retrospective study when preoperative radiographic studies were compared with intraoperative duplex images before surgical repair.¹⁹ In more than 1000 open renal artery repairs, intraoperative duplex sonography has been 86% sensitive and 100% specific for technical defects associated with postoperative stenosis and occlusion of direct aortorenal reconstruction.^{3,4,13}

Designation of B-scan defects according to Doppler velocity criteria provides accurate information to guide decisions about intraoperative revision. However, there are special circumstances that deserve comment. Unlike the case in surface duplex sonography, in which the Doppler sample volume is large relative to the renal artery diameter, a small Doppler sample volume can be accurately positioned within midcenter stream flow. Despite a small, centered Doppler sample, renal artery repairs demonstrate at least moderate spectral broadening. Transaortic endarterectomy gives the audible Doppler signal an oscillating characteristic, which is normal and not associated with anatomic defects. In addition, an infrequent intraoperative study will demonstrate peak systolic velocities that exceed criteria for critical stenosis when no anatomic defect exists. In these cases, the peak systolic velocities will be elevated uniformly throughout the repair, there will be no focal velocity change, and there will be no distal turbulent waveform. This scenario is most commonly encountered immediately after renal artery reconstruction for nonatherosclerotic renovascular disease. Moreover, renovascular repair to a solitary kidney will frequently demonstrate increased velocities throughout. Finally, an increase in peak systolic velocity is observed in transition from the main renal artery to the segmental renal vessels after branch renal artery repair; however, no distal turbulent waveform will be observed.

Finally, there are B-scan abnormalities observed in conjunction with renal endarterectomy that deserve comment. Infrequently, an irregular B-scan abnormality will evolve during performance of a completion scan. This B-scan finding may be associated with either increased or decreased (blunted) peak velocities but reflects formation of intra-arterial thrombus. Unlike acute venous thrombus, which is usually echolucent, acute arterial platelet aggregates are characterized by irregular echogenic material. Regardless of the associated velocity estimates, the endarterectomy site should be reopened and revised immediately. Last, a B-scan defect

otherwise minor by velocity criteria may be revised according to its location and appearance. A mobile flap longer than 2 mm at the distal endpoint of an endarterectomy site is usually revised on the basis of its mere presence and potential for dissection or thrombosis. Intraoperative revision based on ultrasound findings may require thrombectomy, extension of endarterectomy, or patch angioplasty depending on the anatomy.

RESULTS OF OPEN OPERATIVE MANAGEMENT

Anatomic Results

Our center's cumulative open operative experience from January 1987 through January 2012 has included more than 1700 renal artery repairs in more than 1000 patients. Our consecutive operative experience for atherosclerotic renovascular disease from January 1987 through June 1997 is described in Table 128.1.²⁵ During this 10.5-year period, 720 renovascular reconstructions and 57 primary nephrectomies were performed in 534 patients. Postoperative stenosis or thrombosis occurred in 3.3% of renal artery repairs, resulting in recurrent hypertension and declining renal function in 3.7% of patients during a mean follow-up of 36 months.³ However, because complete anatomic failure of repair (i.e., thrombosis) may result in blood pressure benefit equivalent to nephrectomy, anatomic failure is potentially more common than the rate of recurrent hypertension or reoperation.²⁷ To examine the authors' rate of anatomic failure, the results of 277 postoperative duplex studies in 128 consecutive patients were reviewed.²⁵ During a mean follow-up of 22 months, 6 of 177 (3.4%) operative renal artery repairs developed stenosis.³

TABLE 128.1 Summary of Operative Management (N = 534 patients)

Total renal reconstructions	720
Aortorenal bypasses	445
Vein	288
PTFE	127
Dacron	19
Hypogastric arteries	11
Ex vivo	33
Reimplantations	52
Thromboendarterectomies	223
Total nephrectomies	57
Total kidneys operated	777

PTFE, polytetrafluoroethylene.

From Hansen KJ, Deitch JS, Oskin TC, et al. Renal artery repair: consequence of operative failures. *Ann Surg*. 1998;227:678–690.

Operative Morbidity and Mortality

Perioperative morbidity accounting for prolonged hospital stay has been observed in 15% to 20% of patients.^{3,4} Less than half of 1% of patients with ischemic nephropathy have undergone operation resulting in permanent dialysis dependence within 1 month of repair.⁴ Perioperative mortality within 30 days of surgery has a significant and independent association with advanced age and depressed left ventricular systolic function (i.e., ejection fraction <25%).³ Overall, during the past 10 years, mortality after isolated renal artery repair has averaged 0.8%, compared with 1.6% for bilateral renal artery repair and 3.3% for bilateral repair combined with aortic repair for correction of atherosclerosis. This compares with 6.9% when visceral, renal, and aortic repairs are combined as one procedure.⁶²

Blood Pressure Response

Considering open renal reconstruction for atherosclerotic renal artery disease and criteria for response including both blood pressure measurements and medication requirements at least 2 months after operation, 85% of surgical survivors had been cured or improved and 15% had no blood pressure response.³ When patients with atherosclerosis were stratified according to blood pressure response and dialysis-free survival, only blood pressure cured was significantly associated with improved estimated survival.³

Renal Function Response

Considering renal function response in consecutive patients treated for atherosclerotic renovascular disease, renal function improved significantly when all patients are considered. In patients with atherosclerotic renovascular disease and ischemic nephropathy, defined by a preoperative serum creatinine concentration above 1.8 mg/dL, 58% demonstrated improved renal function at 3 weeks after repair.^{3,4} When 45 patients considered permanently dependent on dialysis were examined, 70% were permanently removed from dialysis dependence after open operation.^{4,13} Although other authors have considered that recovery of renal function is limited by elevated serum creatinine concentration, when patients are selected on the basis of severe hypertension and rapidly deteriorating renal function, the proportion of patients improved increases with increasing severity of preoperative renal dysfunction. This association between increased preoperative serum creatinine concentration and improved postoperative renal function is independent and significant ($P < 0.0001$).^{3,4}

Hypertension and Renal Function Benefit: Clinical Outcome

Both preoperative parameters and postoperative blood pressure and renal function response are associated with progression to death or dialysis among patients with atherosclerotic

renovascular disease. Preoperative factors that are associated with death or dialysis include diabetes mellitus, severe aortic occlusive disease, and poor renal function (creatinine level of 1.3 mg/dL or greater).³ Postoperatively, significant associations were noted for blood pressure cured compared with blood pressure improved or worsened (Fig. 128.11). Moreover, improved postoperative renal function is associated with increased dialysis-free survival compared with postoperative unchanged renal function. The relationship between each category of renal function response and dialysis-free survival demonstrated significant interaction with preoperative renal function. For patients with renal function unchanged, an increased risk of death or dialysis was observed for those with poor preoperative renal function.⁴ For patients who worsened, an increased risk of death or dialysis was significant for those with preoperative

renal function at median values of estimated glomerular filtration rate or greater. These significant interactions are shown for predicted dialysis-free survival according to preoperative renal function in Figure 128.12.

Consequence of Failed Open Renal Artery Repair

Our secondary management of surgical repairs has included 10 repeat reconstructions and 10 nephrectomies for nonreconstructable renovascular disease.²⁵ Regardless of the type of management (repeat reconstruction versus nephrectomy), and regardless of the blood pressure or renal function benefit after reintervention, the product-limit estimates for dialysis-free survival for patients experiencing failure of open operative renal artery repair were decreased compared with patients with primary patency. Patients requiring secondary intervention also demonstrated a significant and independent risk of eventual dialysis dependence (relative risk: 12.6; confidence interval: 4.5, 34.9; $P < 0.001$) and decreased dialysis-free survival (relative risk: 2.4; confidence interval: 1.1, 5.4; $P = 0.035$).²⁵ Whether failed balloon angioplasty with endoluminal stenting demonstrates significant associations with eventual dialysis dependence or death is not known.³⁰

This experience with failed open renal artery repair reinforces two management strategies. First, irretrievable loss of excretory renal function observed after failed renal artery repair supports the view that renal revascularization should be performed for clear clinical indications, not as a prophylactic procedure in the absence of either severe hypertension or renal insufficiency.^{3,6,62} Second, the direct aortorenal reconstruction used in the majority of these patients is durable.²⁵ The short graft length and high blood flow characterizing aortorenal repair has favored prolonged patency.

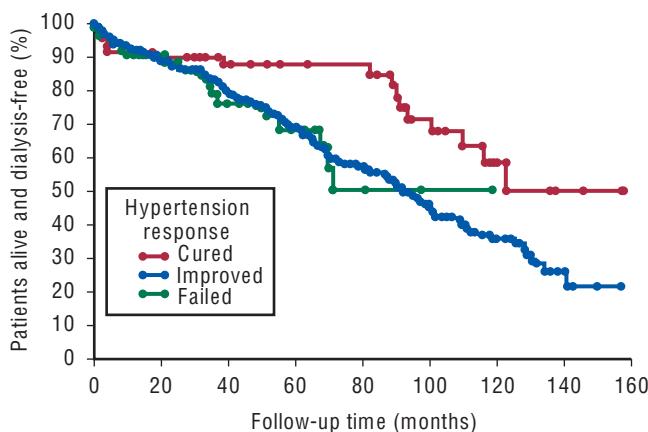


Figure 128.11 Product-limit estimates of time to death or dialysis according to blood pressure response to operation. (From Cherr GS, Hansen KJ, Craven TE, et al. Surgical management of atherosclerotic renovascular disease. *J Vasc Surg*. 2002;35:236–245.)

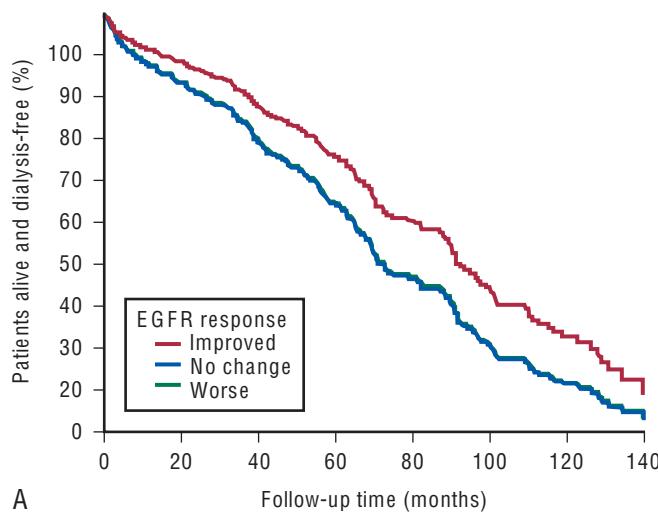
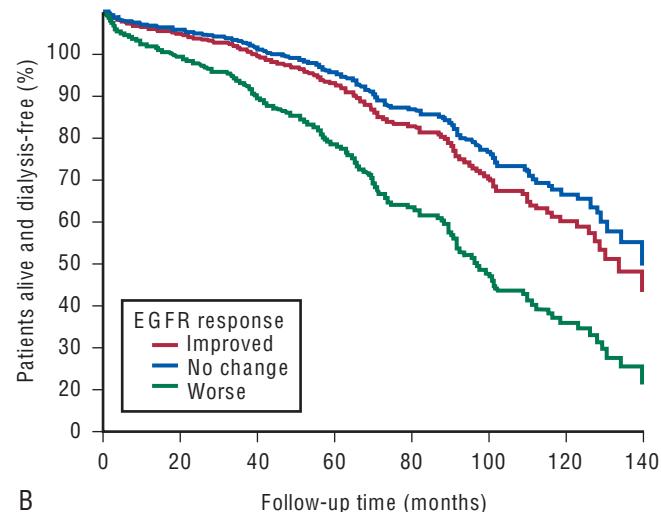
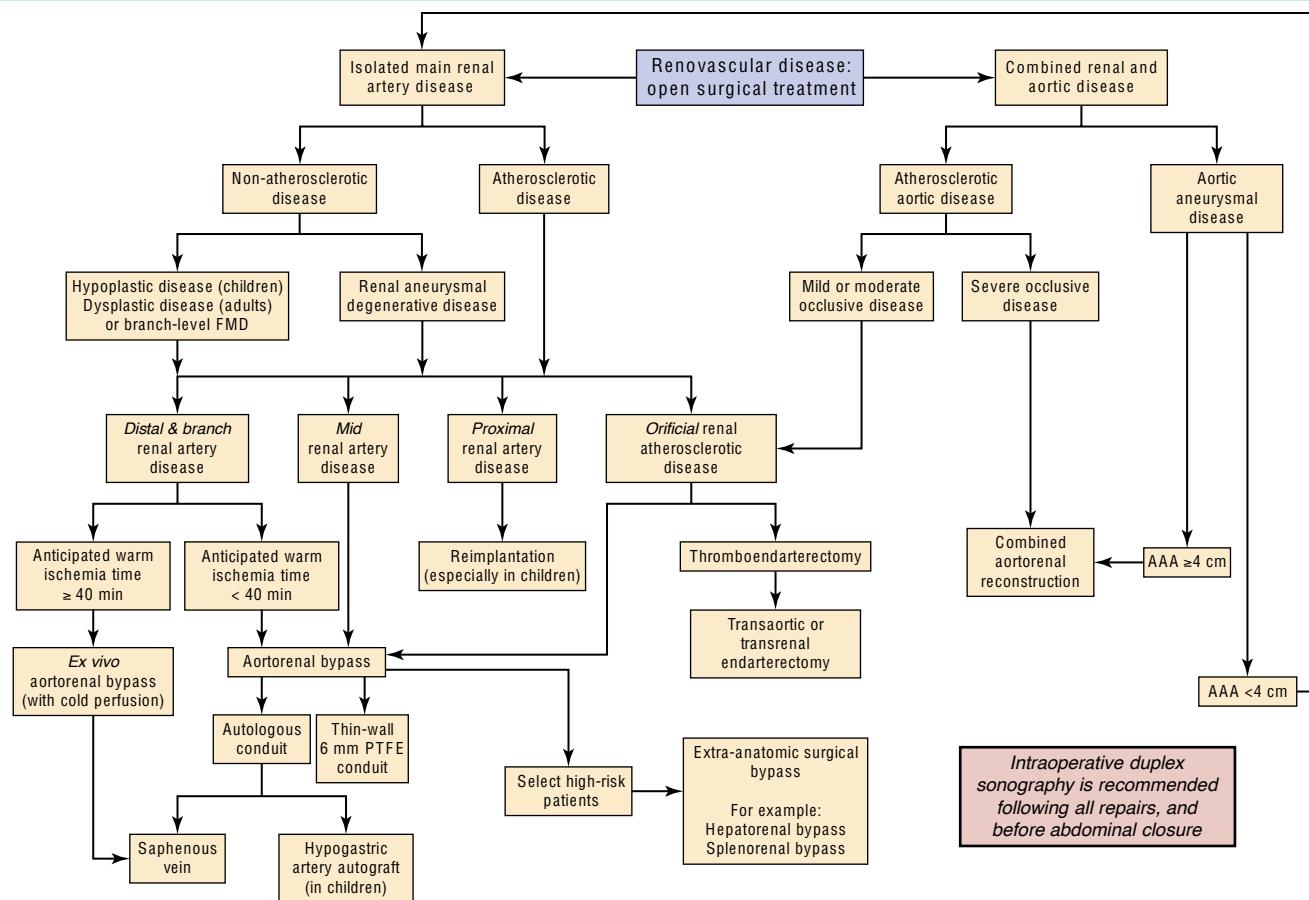


Figure 128.12 Predicted dialysis-free survival according to postoperative renal function response for patients with a preoperative estimated glomerular filtration rate (EGFR) of 25 mL/min per m^2 (25th percentile, A) or 39 mL/min per m^2 (median value, B). The interaction between preoperative EGFR and renal function response for dialysis-free survival was significant and independent. (From Cherr GS, Hansen KJ, Craven TE, et al. Surgical management of atherosclerotic renovascular disease. *J Vasc Surg*. 2002;35:236–245.)



CHAPTER ALGORITHM



SELECTED KEY REFERENCES

Cherr GS, Hansen KJ, Craven TE, et al. Surgical management of atherosclerotic renovascular disease. *J Vasc Surg*. 2002;35:236–245.

Management of atherosclerotic renovascular disease has focused solely on hypertension. Along with the article by Hansen and colleagues (later in this list), this work establishes an incremental increase in excretory renal function as the dominant determinant of dialysis-free survival on follow-up. Among patients with severe hypertension and renal insufficiency, changing renal function at 3 weeks predicts dialysis-free survival – a relationship that is stable at 10 years of follow-up.

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The renin–angiotensin system has been demonstrated in virtually every tissue studied. This report summarizes the classic experiments of Goldblatt and provides an overview of the importance of tissue renin and angiotensin in both health and disease.

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See the annotation for Cherr and colleagues.

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Stanley JC, Criado E, Upchurch Jr GR, et al. Pediatric renovascular hypertension: 132 primary and 30 secondary operations in 97 children. *J Vasc Surg*. 2006;44:1219–1229.

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A complete reference list can be found online at www.expertconsult.com.

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Renovascular Disease: Endovascular Treatment

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INTRODUCTION

The endovascular treatment of renovascular disease remains controversial. Despite several adequately powered randomized trials, there is no current evidence to support the broad use and efficacy of renal artery stenting. However, renal revascularization remains a commonly used treatment option for selected patients who have clearly failed adequate medical therapy.^{1–4} Endovascular techniques offer the benefits of decreased morbidity, mortality, patient recovery time, and hospital resource utilization compared with conventional open surgical revascularization.^{5–10} This chapter provides an overview of the technical aspects involved in the performance of endovascular renal revascularization as well as current data concerning the technical results, clinical outcomes, and associated complications.

INDICATIONS AND CONTRAINDICATIONS

Indications

The indications for renal artery revascularization are controversial in light of the absence of level I data demonstrating

the efficacy of such therapy in improving adverse event-free survival in patients with renovascular disease. Early data in the late 1990s, from the Dutch Renal Artery Stenosis Intervention Cooperative Study Group trial (DRASTRIC) compared renal artery angioplasty to medical therapy in a small cohort of patients.¹¹ No significant improvement in hypertension was shown with angioplasty, but the study was flawed with a significant patient cross-over. In the late 2000s, two clinical trials conducted in Europe, the Angioplasty and Stenting for Renal Artery Lesions (ASTRAL) trial¹² and the stent placement and blood pressure and lipid-lowering for the prevention of progression of renal dysfunction caused by Atherosclerotic Ostial Stenosis of the Renal Artery (STAR) trial,¹³ followed in 2014 by the North American Cardiovascular Outcomes in Renal Atherosclerotic Lesions (CORAL) trial¹⁴ demonstrated no evidence to support the widespread application of renal stents to patients with renovascular hypertension or ischemic nephropathy. However, both of the European trials have been criticized for methodologic shortcomings that limit the generalizability of their results and conclusions. Those limitations aside, the data presented in ASTRAL strongly suggest that the application of renal revascularization therapy to patients without strong clinical

indications (i.e., prophylactic or “drive by” revascularization) lacks demonstrable benefit for the patient.

The results from the CORAL trial, which was more scientifically rigorous and robust, also demonstrate no clinical benefit for the widespread application of revascularization to patients with renal artery stenosis and significant hypertension with or without associated renal dysfunction. Furthermore, no interactions were observed for bilateral renal stenosis, degree of stenosis, or other clinical factors in terms of clinical benefit. Subsequent secondary analyses of the CORAL data and other reports have directly examined the prognostic value of trans-lesion pressure gradients, baseline blood pressure, urine albumin to creatinine ratio, stenosis severity, and fractional flow reserve; no evidence of prognostic value for these parameters was observed.^{15–17} Despite the existing evidence, practice guidelines and consensus statements formed in conjunction with several societies help delineate recommendations for intervention in patients not representative of the CORAL study population. In general, those documents recommend renal revascularization based upon the presence of hemodynamically significant renal stenosis in the setting of severe, difficult-to-control hypertension with or without associated renal dysfunction or in instances of hypertension-associated cardiac disturbance syndrome.¹⁸

Hypertensive indications and manifestations of renovascular disease include a spectrum of clinical presentations, such as chronic, severe hypertension that is refractory to pharmacologic management; renovascular hypertension associated with cardiovascular disturbance; and more acute hypertensive emergencies, which refer to acute blood pressure elevations associated with target organ damage, including flash pulmonary edema, hypertensive encephalopathy, myocardial infarction, and acute renal failure. Given the findings of ASTRAL and CORAL,¹² which demonstrated no reduction in adverse cardiovascular or renal events despite significant blood pressure decreases after RA-PTAS, as well as other large case series demonstrating a lack of survival benefit in association with blood pressure improvement alone,^{5,7,8,19,20} many authorities on renovascular disease are advocating the application of renal revascularization only to cases of truly refractory hypertension and those cases of hypertension complicated by defined end-organ effects.^{15,21,22}

Renal dysfunction in patients with hemodynamically significant atherosclerotic renovascular disease is commonly referred to as ischemic nephropathy,^{3,23} although definitive establishment of a causal relationship between anatomic renovascular disease and renal dysfunction can be challenging in an individual patient despite extensive diagnostic testing. Conditions that would support a causative relationship between anatomically defined renal artery stenosis and disease manifestations are primarily based on reported predictors of response to intervention. Predictors include a significant observed decline in renal function over a short period of time and the presence of anatomic disease affecting perfusion to the entirety of the functioning renal mass (i.e., bilateral renovascular disease or renovascular disease affecting a solitary kidney).^{7,24–28} However, a causative relationship

can be proved only by a physiologic response after revascularization. Available data from numerous authors indicate that documented improvement in renal function after renal revascularization is the most important predictor of clinical benefit, suggesting that renovascular disease associated with renal dysfunction may represent the most appropriate contemporary indication for RA-PTAS.^{5,7,29–31}

Contraindications

Contraindications to the treatment of renovascular disease by endovascular means can be broadly classified into anatomic reasons, cases in which open revascularization is optimal, and prophylactic treatment. Anatomic contraindications involve situations in which the disease exists in renal arteries not easily treated with currently available endovascular devices or, more commonly, those arteries unable to be treated with any reasonable expectation of durable result. Renovascular disease extending into the terminal portion of a main renal artery or involving the branches and disease involving a very short main renal artery represent instances in which treatment with currently available technology may be problematic (Fig. 129.1). Renovascular disease existing in the branch arteries beyond the main bifurcation, lesions in multiple small renal arteries (Fig. 129.2), and lesions in children (most commonly hypoplastic lesions) are poor candidates for endovascular treatment because of durability concerns. When encountered, these situations are likely best treated with open surgical revascularization unless the patient represents a prohibitive surgical risk. Surgical revascularization should also be considered in patients with indications for renal revascularization who require open aortic surgery for other reasons, such as aneurysm repair.



Figure 129.1 Arteriogram demonstrating early bifurcation of the *right* main renal artery.

With regard to prophylactic revascularization, the high rate of technical success and low incidence of periprocedural complications associated with endovascular renal artery revascularization have led to aggressive application of this technique. There is currently no evidence supporting prophylactic renal revascularization (i.e., endovascular treatment of renal artery stenosis in patients with normal renal function and hypertension that is well controlled with conventional medical therapy). Furthermore, the results of ASTRAL,¹² the conduct of which approximated prophylactic treatment in a large percentage of its participants, confirmed the lack of efficacy for such an approach in terms of blood pressure control, protection of renal function, reduction of cardiac events, or improved survival.

The natural history of atherosclerotic renal artery stenosis is associated with anatomic progression in only 10% to 30% of patients and progression to occlusion in 0% to 7%.^{32–36} When anatomic disease progression does occur, it is not consistently correlated with increases in blood pressure or serum creatinine concentration.³⁴ Considering the benign clinical course of asymptomatic renal artery stenosis in the majority of patients together with the risk of procedure-associated deterioration in renal function with intervention and the recent results from the CORAL trial, we support a nonoperative, noninterventional management strategy for asymptomatic patients with anatomic renovascular disease, normal or well-controlled blood pressure, and normal renal function.

RESULTS

Definition of Success

Criteria for procedural technical success include reduction in stenosis as assessed by completion angiography and, when endoluminal stenting has been performed, complete coverage of the stenotic lesion by the deployed stent. Postintervention

residual stenosis of less than 30% has been suggested as a threshold for technical success versus failure.³⁷ When contrast angiography demonstrates anatomically successful treatment of the stenotic lesion, these findings should be further confirmed with transduced pressure measurement immediately proximal and distal to the treated lesion. We consider a persistent systolic pressure gradient of 10 mm Hg or more to be suggestive of inadequate treatment and will repeat angioplasty in this setting in the absence of a significant residual stenosis on the completion angiogram.

Whereas technical success implies delivery of the intended treatment with satisfactory anatomic and hemodynamic results, determination of whether patient benefit has been derived requires consideration of the indications for intervention and the clinical responses observed. Approaches to the assessment of hypertension response have evaluated systolic, diastolic, and mean blood pressure and number of antihypertensive medications (both as continuous outcome measures and in a categorical fashion) using a combination of blood pressure and medication criteria.³⁷ The categorical definitions generally describe hypertension responses as cured, improved, or failed. Serum creatinine, cystatin C, estimated glomerular filtration rate (eGFR), and renal length and volume all represent continuous outcome measures indicative of renal function response to intervention. Categorization of renal response based on these measures has generally been determined as improved, unchanged, or worsened on the basis of postintervention change within a predefined range. We have generally used an increase or decrease of 20% or more in eGFR to define improvement or worsening in renal function, respectively, with all other patients categorized as unchanged. Alternative analytic approaches have evaluated the impact of renal revascularization on the rate of decline in renal function, defining worsened or unchanged rates of eGFR decline as failure and attributing procedure-related benefit to patients experiencing improvement, stabilization, or slowed rate of decline in eGFR.

Unfortunately, none of the previously discussed parameters assesses the impact of intervention on patient morbidity or survival. Survival free from dialysis, renal death, or cardiovascular morbidity represents an outcome that provides the most direct means of assessing the true benefit of intervention and likely represents the outcome of greatest importance in measuring the true value of renal revascularization to the patient.

Early Outcomes

A large body of literature exists reporting results of RA-PTAS in adult patients.^{24,26,27,30,31,38–68} Technical success rates ranged from 88% to 100% and periprocedural 30-day mortality was 0% to 5%. Procedure-related complications associated with RA-PTAS occurred in 0% to 43% of patients. In general, these early results mirror those of ASTRAL and STAR.^{12,13} In CORAL, technical success rates exceeded 95%, with no early patient deaths. Procedural complications were rare (less than 5% of cases) with the most common being dissection of the treated artery.

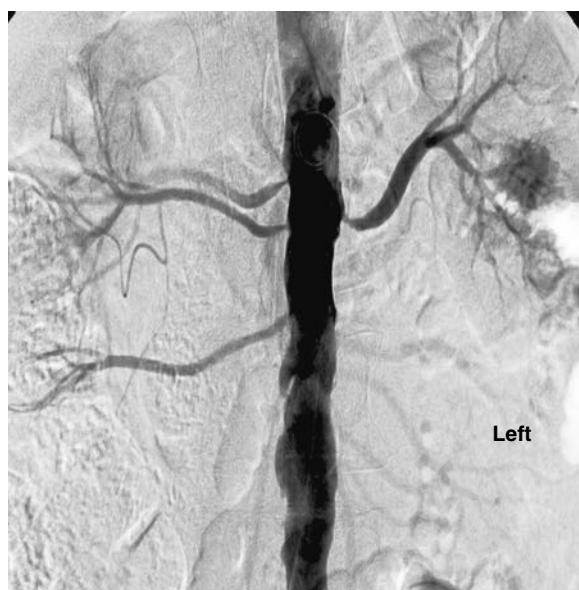


Figure 129.2 Abdominal aortogram demonstrates hemodynamically significant stenosis involving multiple renal arteries.

Late Outcomes

Late outcomes associated with endovascular renal artery intervention may be considered in terms of hypertension control, renal function change, anatomic durability, and survival free of dialysis or cardiovascular morbidity. From an anatomic perspective, primary patency of renal stenting has been described to be as high as 80% at 5 years.³⁹

Hypertension Response

Long-term hypertension response to RA-PTAS performed for atherosclerotic renovascular disease is most often improvement or failure, whereas cure (defined as normal blood pressure off all antihypertensive medications) is very uncommon. In the case series data,^{24,26,27,30,31,38–68} hypertension improvement was seen in a majority of patients, but failure to improve blood pressure control was also a common outcome. In ASTRAL, no significant improvement in systolic blood pressure was observed when stenting was directly compared with medical therapy. A modest decrease in diastolic blood pressure was observed, and a statistically (although probably not clinically) significant decrease in the number of antihypertensive medications administered was also observed.¹² In CORAL, a small but significant decrease in systolic blood pressure was observed with no resultant decrease in medications.¹⁴

Reported predictors of hypertension response have included severity and duration of preoperative hypertension,^{24,26,27} age of the patient,⁶⁹ percentage angiographic stenosis,²⁴ bilateral disease,²⁶ female gender,²⁷ and preoperative brain natriuretic peptide level,⁷⁰ although the brain natriuretic peptide finding is now in question.⁷¹ Durable hypertension responses have been reported by several authors,^{44,51,52,63} but others have observed a loss of initial hypertension response at 6-month post-RA-PTAS follow-up associated with a return to the preoperative number of blood pressure medications.^{67,72} Given the data from ASTRAL¹² and CORAL¹⁴ and the lack of association between blood pressure improvements and improved adverse event-free survival reported by multiple authors, there is increasingly limited enthusiasm for RA-PTAS for management of hypertension in the setting of normal renal function. For hypertensive indications, we would recommend limiting intervention for patients with truly uncontrollable and severe hypertension (which is increasingly uncommon given the potency of contemporary antihypertensive medications) and severe hypertension associated with target organ damage.

Renal Function Response

Although several case series have reported improvement as the most frequent categorical renal function response to RA-PTAS,^{27,57,64} others have observed postoperative deterioration with a frequency equal to or greater than that of improvement,^{43,48,50,51,56,63,65,66} and renal function was unchanged in the majority of patients after intervention. Reported rates of post-intervention improvement versus deterioration in renal function vary widely in these series.

The heterogeneous results may be partly attributable to differing patient selection criteria, particularly regarding the percentage of patients with baseline renal dysfunction (because patients with normal baseline renal function have no prospect for improvement through intervention). Weighted averages across series of unprotected RA-PTAS demonstrate renal function improvement in approximately 25% of patients, with a roughly equal proportion experiencing post-interventional decline.

Rather than assessing renal function response as mean or categorical change in outcome measures, several studies have instead analyzed renal responses to RA-PTAS using breakpoint analysis based on the slope of either eGFR or reciprocal of serum creatinine concentration, with categorization of effect as improvement, stabilization, or failure.³⁷ Whereas these studies have demonstrated that RA-PTAS can reduce the rate of renal function decline,^{38,58,64,72} interpretation of clinical benefit based on breakpoint analysis is controversial. Reciprocal creatinine analysis was employed in ASTRAL,¹² and RA-PTAS demonstrated no benefit in slowing the rate of renal function decline relative to the use of best medical management alone. One notable finding from ASTRAL that deserves to be stressed was the observation of a much lower rate of renal function decline in the medical therapy arm than expected. This most likely represents the effects of improving medical management on the natural history of atherosclerotic renovascular disease.

Case series data suggest that improvement in renal function after renal revascularization is associated with improved survival. Hansen et al.⁷ observed improved dialysis-free survival after open surgical revascularization in patients with early postoperative improvement in renal function, but no similar survival effect was noted in patients with unchanged renal function. Similar associations with improvement in renal function have also been demonstrated after RA-PTAS.^{29,30,38,73} Although patients with increases in post-operative eGFR (or decline in serum creatinine concentration) appear to experience a survival benefit, clinical benefit resulting from stabilization of renal function or reduction in rate of decline is not clear. We therefore believe that in interpreting either categorized change in eGFR or breakpoint analysis data, patient benefit can be assumed only for patients with improvement, whereas presuming benefit in patients with unchanged or stable renal function may be conceptually flawed. Reported predictors of renal function response to RA-PTAS have included bilateral disease,³¹ elevated baseline serum creatinine concentration,^{27,31} higher levels of chronic kidney disease,^{7,29} rapid preoperative decline in renal function,^{25,38} impaired left ventricular function,²⁷ absence of metabolic syndrome,⁷⁴ smaller pre-revascularization renal parenchymal volume,^{75,76} and improved renal volume after revascularization.^{76,77}

Renal Artery Restenosis After Intervention

Restenosis after RA-PTAS has been defined on the basis of angiography,^{43,48,67} duplex ultrasound,⁶³ or both.^{56,65,78}

Reported rates of restenosis after RA-PTAS range from 5% to 66% and vary with duration of follow-up and criteria for repeated imaging.^{40,42–44,48–51,56–58,65,67,68,78} The frequency of restenosis is variably reported in the literature given the variations in techniques and follow-up. Restenosis is more common in women and in patients exhibiting metabolic syndrome⁷⁹ and less common in patients treated perioperatively with statins.⁶ Other factors that may occasionally contribute to restenosis include misplacement of the stent, most frequently inadequate proximal coverage of an ostial lesion, stent fracture, and severe calcification resulting in stent compression.

Anatomically identified recurrent stenosis must be considered in combination with clinical manifestations in formulating a management strategy. Conversely, acute worsening of hypertension or decline in renal function should prompt suspicion of disease recurrence. Lederman et al.⁴³ identified recurrent disease twice as frequently when anatomic imaging was performed in the setting of clinical suspicion as opposed to routine post-intervention screening (42% vs. 21%, respectively). The relatively frequent occurrence of recurrent disease should be considered when primary intervention is being contemplated, particularly in asymptomatic patients, and raises additional concerns about the long-term benefits achieved in patients with unchanged renal function after RA-PTAS.

Survival

Long-term survival data for patients undergoing RA-PTAS are presently limited to retrospective studies, ASTRAL, and CORAL.¹² Dorros et al.⁵² observed a 74% 3-year survival rate after RA-PTAS; although renal failure was not a frequent cause of death, they observed decreased survival among patients with both baseline renal dysfunction and bilateral disease. Similar overall survival was reported by Kashyap et al.,³⁸ who also observed a 63% rate of dialysis-free survival; lack of post-intervention improvement in renal function was associated with subsequent dialysis in that series. Similar relationships between baseline renal dysfunction, renal function response, and survival have been described by Kennedy et al.³⁰ Survival benefit associated with angiotensin-converting enzyme treatment⁸⁰ and statin use after RA-PTAS has also been described,^{30,81} whereas preoperative congestive heart failure, coronary artery disease, and chronic obstructive pulmonary disease are additional factors that adversely affect post-intervention survival.^{30,43,81}

Similar data have come from ASTRAL and STAR.^{12,13} In ASTRAL, 5-year survival was 58% for the entire study sample, with no improvement demonstrated for those undergoing RA-PTAS. Improvement in diastolic blood pressure and reduced medication use observed for the stent arm participants were not demonstrated to have any effect on survival. As previously stated, no differences were observed in renal function, rate of renal function decline, or systolic blood pressure. In the results from STAR, 2-year survival was slightly in excess of 80%, with

no differences observed according to post-revascularization blood pressure control or renal function.

DESCRIPTION OF TECHNIQUE

Relevant Anatomy

The renal arteries originate laterally from the abdominal aorta and commonly assume an oblique orientation in their proximal course relative to the aortic axis (Fig. 129.3). Because aortography performed in a straight anteroposterior position can produce contrast overlap between the aorta and proximal renal arteries, resulting in inadequate visualization of ostial disease, oblique imaging is frequently necessary for renal arteriography and intervention. Multiple renal arteries are present in 18% of patients and may not be accurately characterized with renal artery duplex ultrasound.^{34,81,82} Accessory arteries may therefore be an unanticipated finding in patients imaged with duplex ultrasound alone before intervention and must be assessed for occlusive disease during procedural planning. Distal renal artery diameter in patients undergoing endovascular intervention is usually 5–6 mm.^{41,43} Post-stenotic renal artery dilatation is common⁴³ and may result in size mismatch between the luminal diameter and stent or protection device; small-diameter arteries (<4 mm) increase the technical difficulty of ostial engagement and endovascular treatment. Endovascular treatment of atherosclerotic disease is most often performed for lesions of the ostial and proximal main renal artery. Branch vessel atherosclerosis is less frequently encountered and is seldom ideal for endovascular management.

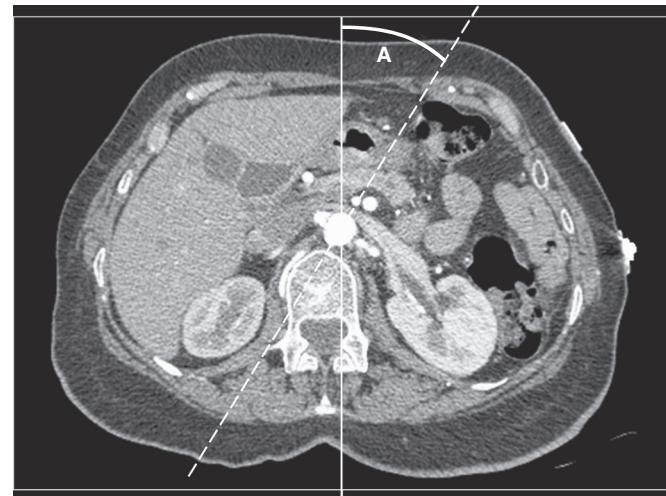


Figure 129.3 Oblique Orientation of Proximal Renal Arteries. Computed tomographic angiography demonstrates the axis perpendicular to the proximal renal arteries (dashed line) relative to the axis perpendicular to straight anteroposterior imaging (solid line). The resulting angle (*A*) indicates the oblique correction needed to avoid artifact resulting from contrast overlap between the aorta and the proximal renal artery, which could result in underestimation of the degree of renal artery stenosis.

Operative Planning and Options

Contrast Considerations

Angiographic visualization of arterial anatomy is a fundamental element of both diagnosis and endovascular treatment of atherosclerotic renovascular disease. In addition to associated renal dysfunction and hypertension, common conditions among patients with renovascular disease that may predispose to contrast-induced renal function impairment include diabetes mellitus, congestive heart failure, and anemia.^{43,83–88} Chronic volume depletion may represent an additional risk factor in patients taking diuretic medications. Procedure-related factors affecting risk of contrast-induced nephropathy include the volume, osmolality, and viscosity of the contrast agent administered.^{86,89,90} Accordingly, procedural planning should incorporate assessment of the likelihood of contrast-induced nephropathy and implementation of protective measures for high-risk patients. Prophylactic strategies for the prevention of contrast-induced nephropathy have included a variety of measures directed toward maximizing renal perfusion, reducing oxidative stress, and inhibiting vasoconstriction. Hydration is a generally accepted prophylactic measure, but evidence supporting the routine use of additional measures such as *N*-acetylcysteine, sodium bicarbonate, and ascorbic acid is inconsistent.^{87,91} Currently, we prepare all patients for aortorenal arteriography with preoperative hydration using isotonic saline (reduced in the setting of significant congestive heart failure) and routine use of half-strength iso-osmolar, nonionic contrast material at the lowest volume possible. In the setting of severe renal dysfunction (eGFR <30 mL/min per 1.73 m²), carbon dioxide can be used as the primary contrast agent or as an adjunct during initial aortography and selective cannulation to reduce the administered volume of iodinated contrast material (see Ch. 46, Systemic Complications: Renal).^{92,93}

Medical Management

In the setting of acute symptomatic presentations of renovascular disease (i.e., hypertensive emergency and acute renal failure), clinical stabilization is usually possible through medical management, making it possible to perform intervention in a lower-risk elective setting. Acute renal failure in the setting of renovascular disease can be precipitated by the initiation of angiotensin-converting enzyme inhibitors or angiotensin receptor antagonists in patients with bilateral disease or hypovolemia.^{94,95} Deferring renal intervention until functional recovery is complete in these patients avoids additional renal insult from the administration of nephrotoxic contrast material.

For elective revascularization, nonsteroidal anti-inflammatory drugs, diuretics, metformin, and anticoagulants are withheld during the periprocedural period. Routine antihypertensive medications should be continued perioperatively. Preoperative antiplatelet and statin therapy is initiated in all patients unless contraindicated. Dual antiplatelet medication is recommended for patients who undergo renal artery stenting. Of note, in the CORAL trial, optimal medical management included an aspirin, statin, calcium channel blocker

(amlodipine), angiotensin II receptor blocker (candesartan), and a vasodilator (hydralazine) if required.

Primary Angioplasty Versus Angioplasty and Stenting

Whereas primary angioplasty is currently considered appropriate endovascular management for renal artery fibromuscular dysplasia, primary endoluminal stenting for the treatment of ostial atherosclerotic renovascular disease is associated with superior technical success and a lower incidence of recurrent stenosis versus angioplasty alone.^{9,40} Primary angioplasty is still commonly used for the management of recurrent disease. Nonostial atherosclerotic lesions may also respond well to angioplasty alone, but secondary stent placement should be considered if primary angioplasty is unsuccessful.

Bilateral Atherosclerotic Renovascular Disease

In patients with bilateral, hemodynamically significant atherosclerotic renovascular disease and significant renal dysfunction, one can initially proceed with unilateral treatment. If the clinical response is inadequate and there is no evidence of recurrent stenosis, contralateral RA-PTAS can be performed in a staged fashion. Compared with simultaneous bilateral RA-PTAS, a staged management approach can reduce the volume of contrast material administered during a single procedure setting and decrease the likelihood of acute renal injury. Downsides to this approach include the additional costs of two separate procedures, the incremental risk of vascular access complications, and patient inconvenience. However, this strategy may be useful in patients who are at a high risk for contrast-related complications.

Access

Ultrasound-guided common femoral artery access is safe, versatile, and our preferred access method for RA-PTAS. When selective renal artery cannulation is planned, selection of the femoral location contralateral to the targeted renal artery for access facilitates ostial cannulation through a tendency for the catheter to preferentially track along the contralateral aortic wall (Fig. 129.4). The left brachial artery is an alternative access site that may be preferred in patients with aortoiliac occlusive disease, in patients with renal arteries with significant inferior angulation, or when selective cannulation through a femoral approach has been unsuccessful. Compared with the femoral artery, disadvantages of brachial access include higher incidence of access-related complications and limitations of the catheter or sheath diameter.^{47,96} Recently, successful use of radial artery access for RA-PTAS has been described, although the technical challenges can be formidable.⁹⁷

Angioplasty and Stenting

After initial aortography, the patient is systemically heparinized and the target renal artery is accessed. Access can be gained by use of a selective catheter in the form of a guide

catheter or a diagnostic catheter (Fig. 129.5). If a diagnostic catheter is used for engagement, the selective catheter and sheath are exchanged over a 0.014- to 0.035-inch guide wire for a guide sheath, which is advanced to the origin of the

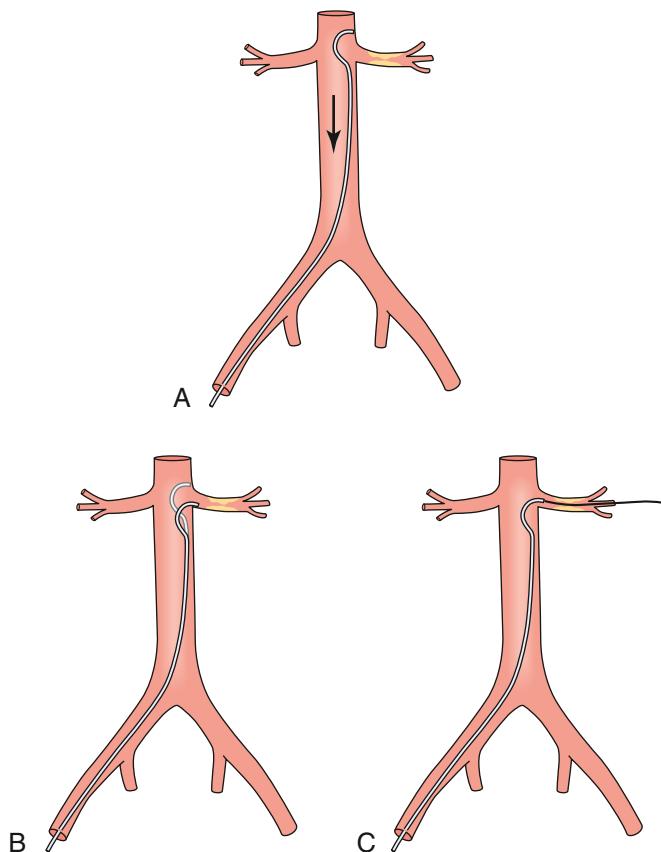


Figure 129.4 (A–C) Contralateral femoral access often facilitates selective renal cannulation. (From Schneider PA. More about how to get where you are going: selective catheterization. In: Campbell B, ed. *Endovascular Skills: Guidewires, Catheters, Arteriography, Balloon Angioplasty, Stents*. St. Louis: Quality Medical Publishing; 1998:71.)

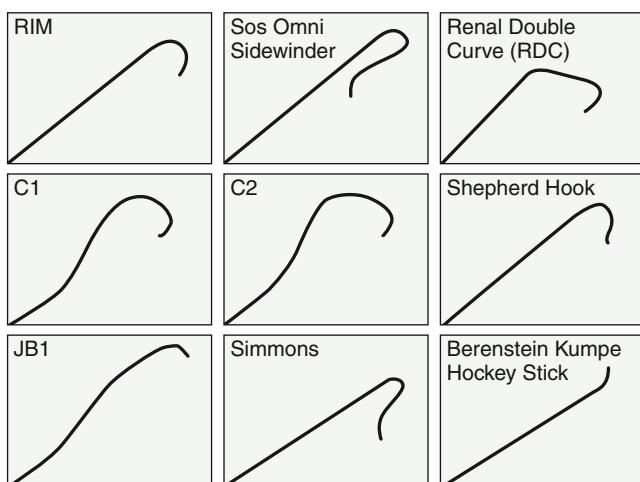


Figure 129.5 Various selective catheters commonly used to cannulate a renal artery.

renal artery. Guide sheath and guide catheter access platforms both provide mechanical support during guide wire exchange and facilitate delivery of therapeutic devices to the targeted lesions. The choice of platform type is a matter of preference. Multiple device configurations are commercially available, and a luminal diameter of 6 F will accommodate the majority of angioplasty and stenting systems. The tip of the guide sheath or catheter is positioned at or within the orifice of the targeted renal artery, and then a guide wire is passed across the stenotic lesion. We believe that smaller wire systems minimize renal artery trauma, limit the risk of atheroembolism, and are associated with technical advantages in crossing critical lesions. We prefer a 0.014-inch guide wire with a floppy, radiopaque tip for lesion passage. Once guide wire access across the stenotic lesion has been obtained, care must be taken to avoid advancement of the non-tapered guide sheath or catheter, which could potentially result in injury to the luminal surface of the renal artery.

If renal artery distal embolic protection is being used, a wire system incorporating a distal renal artery occlusion balloon or filter is used for crossing the lesion and is subsequently deployed distally. If an occlusion balloon device is used, complete renal artery occlusion is confirmed by hand injection of contrast material (Fig. 129.6). Angioplasty with stenting is then performed, followed by aspiration of the static column of blood distal to the treated lesion, irrigation with heparinized saline, and repeated aspiration. The distal occlusion balloon is then deflated, and completion angiography is performed. Filter devices permit ongoing distal renal artery flow in their deployed configurations and use a porous membrane to trap embolic material. After angioplasty and stenting, the filter is collapsed to trap any captured embolic material before device withdrawal.

Both coaxial and monorail systems are available for RA-PTAS. For critical lesions, initial predilatation angioplasty may be necessary to permit subsequent endoluminal stenting. Angioplasty balloon diameter should be selected on the

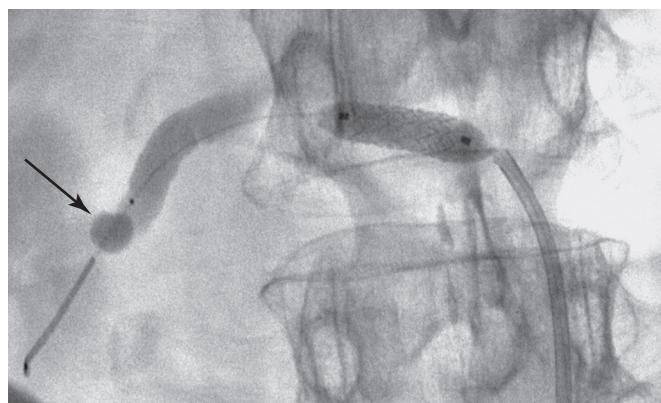


Figure 129.6 Renal angioplasty and stenting with distal renal artery balloon occlusion for embolic protection. A static column of contrast material is visible proximal to the occlusion balloon (arrow) during stent deployment.

basis of quantitative angiography measurements; we select angioplasty balloons sized to the adjacent normal artery for primary RA-PTAS and select smaller balloons if predilatation is required (we commonly use a 2- to 4-mm × 20-mm low-profile balloon for predilatation). For predilatation, the angioplasty balloon should be inflated to nominal pressure for 15 to 30 seconds before deflation; noting the location of the balloon's "waist" before full inflation is helpful for subsequent endoluminal stent positioning. Patients may experience pain or discomfort during angioplasty that resolves with balloon deflation; symptoms persisting after deflation suggest renal artery injury; in the worst cases, they coincide with renal artery rupture. Guide wire access is maintained during balloon deflation and removal.

RA-PTAS is most commonly performed with balloon-expandable stents that have greater radial strength and can be deployed with precision; self-expanding stents offer greater flexibility but are used less frequently. Stents are placed with 1 to 2 mm of extension into the aorta for treatment of ostial or proximal disease; more distal stent placement frequently fails to support the true renal artery orifice; it is associated with increased risks of both technical failure and recurrent stenosis^{44,45,49,98} and also increases the technical difficulty of subsequent surgical revascularization.⁹⁹ The shortest stent that will adequately cover the lesion should be used, and positioning can be guided by frequent hand injection of small volumes of contrast material into the guide catheter or sheath. A balloon-expandable stent is deployed by fully inflating the angioplasty balloon. Usage of a covered or bare-metal stent are both reasonable options. However, no data currently supports efficacy of covered stents over bare-metal for renal arteries specifically. Currently available stents are supplied premounted on low-profile delivery systems that often permit primary RA-PTAS without pre-dilatation. If a residual waist is visible on fluoroscopy or completion angiography after stent deployment, the narrowed area can be post-dilated with an appropriately sized angioplasty balloon (Fig. 129.7). However, dense calcifications sometimes preclude full stent expansion, and overly vigorous attempts to fully dilate the vessel can lead to the risk of vessel rupture. In all cases, the vessel should be dilated only to the diameter of the nondilated segments, not to the diameter of areas of poststenotic dilatation.

After RA-PTAS, technical results may be assessed by completion angiography, intravascular ultrasound, or measurement of the pressure gradient. When RA-PTAS has been performed with protection of the distal renal artery by balloon occlusion, we find it useful to measure pressure gradients through the aspiration catheter, which has already been positioned distal to the treated lesion before balloon deflation. Use of the aspiration catheter to check pressure gradients during its withdrawal therefore avoids the need for passage of an additional catheter past the newly stented lesion, which could potentially lead to stent deformation or migration. Pressure gradients of the renal artery to the aorta

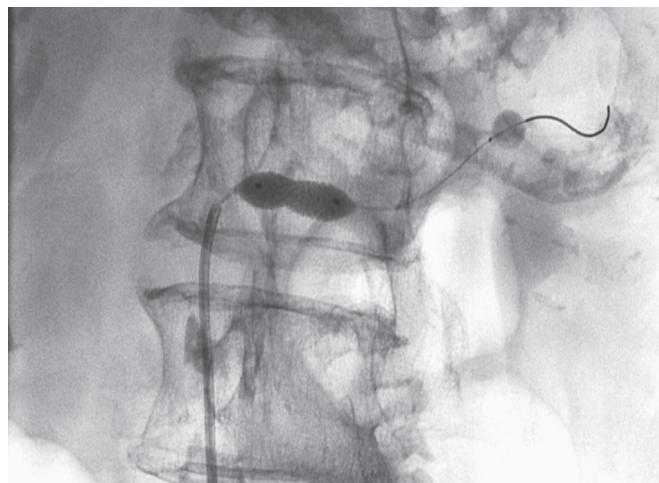


Figure 129.7 "Waisting" of an endoluminal stent being treated with post deployment dilatation. The distal renal artery occlusion balloon is also visible.

can also be measured by pressure-sensing guide wires,^{99,100} which also afford the advantage of allowing both pre- and post-revascularization pressure measurement without catheter exchanges. Once a satisfactory result has been obtained, the guide wire and sheath are removed and hemostasis is obtained. An adequate result includes a residual stenosis of less than 30% on angiography and a pressure gradient of less than 10 mm Hg between the distal renal artery and the aortic lumen. Intravascular ultrasound can also be used as an adjunct measure to assess lesion coverage, stent extension into the aorta, and patency.

Embolectic Protection

Atheroembolization during RA-PTAS may occur, with the liberation of debris sufficient enough to occlude the arterioles of the kidney.^{54,101} It has been postulated that atheroembolism may play a role in post-procedural deterioration of renal function. Embolic protection devices can be used during RA-PTAS, although none are currently approved by the U.S. Food and Drug Administration for such a purpose. Several case series have been published demonstrating infrequent deterioration of acute renal function and higher rates of renal function improvement after RA-PTAS when embolic protection devices were used.^{53,54,59–61,102} One prospective randomized trial of the use of an embolic protection device has been published, the Randomized Comparison of Safety and Efficacy of Renal Stenting (RESIST) trial.¹⁰³ RESIST demonstrated an improvement in the rate of adverse renal events by embolic protection with the Angioguard filter device (Cordis Johnson & Johnson, Bridgewater, NJ), but only when the filter device use was combined with the potent glycoprotein IIb/IIIa inhibitor abciximab. No benefit was seen in use of the filter alone. The beneficial effects were most pronounced in those patients with greater degrees of renal dysfunction.¹⁰⁴

Post-Procedure Management

After intervention, patients are monitored for several hours for access site problems and hemodynamic instability. Dual antiplatelet therapy is started at least seven days preoperatively and continued postoperatively. Clopidogrel is continued after intervention for a minimum of 30 days; aspirin and statin therapy is continued indefinitely.

Clinical follow-up with surveillance renal duplex ultrasonography is performed within the first month after intervention and subsequently at 6-month intervals for 2 years and annually thereafter. Serum creatinine is measured at each of these surveillance intervals as well. We consider a renal artery peak systolic velocity of more than 180 cm/s suggestive of disease recurrence¹⁰⁵ and consider all anatomically identified recurrent lesions in combination with their clinical manifestations. For patients experiencing anatomic recurrence in the setting of maintained blood pressure or renal function response to intervention, we increase the frequency of clinical follow-up and imaging surveillance but do not intervene unless clinical manifestations occur. When repeated intervention for recurrent disease is required, an endovascular approach is most often used.⁷⁹ In-stent stenosis due to intimal hyperplasia should be suspected with early disease recurrence; this pathologic process is difficult to manage with routine balloon angioplasty alone; technical results may be improved by initial dilatation with a cutting balloon¹⁰⁶ or by the use of a balloon-expandable covered stent.¹⁰⁷

COMPLICATIONS

Reported major complications include hemorrhage, access site pseudoaneurysm, bowel or extremity ischemia, myocardial infarction, renal artery thrombosis, and acute renal failure.^{108,109} Hemodynamic instability after endovascular renal intervention must be presumed to be hemorrhagic in etiology until proven otherwise, and computed tomography scanning is a useful tool for the identification and localization of bleeding. Hemorrhage is most commonly related to bleeding at the site of arterial access, but potentially fatal bleeding can also occur at the site of renal artery rupture secondary to angioplasty balloon, or guide

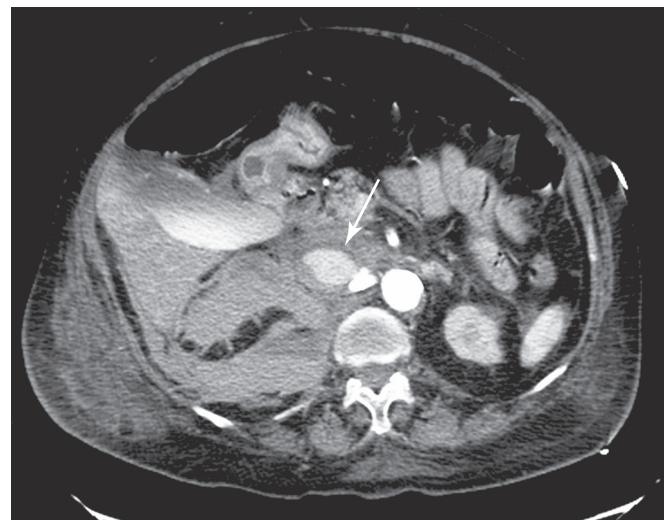
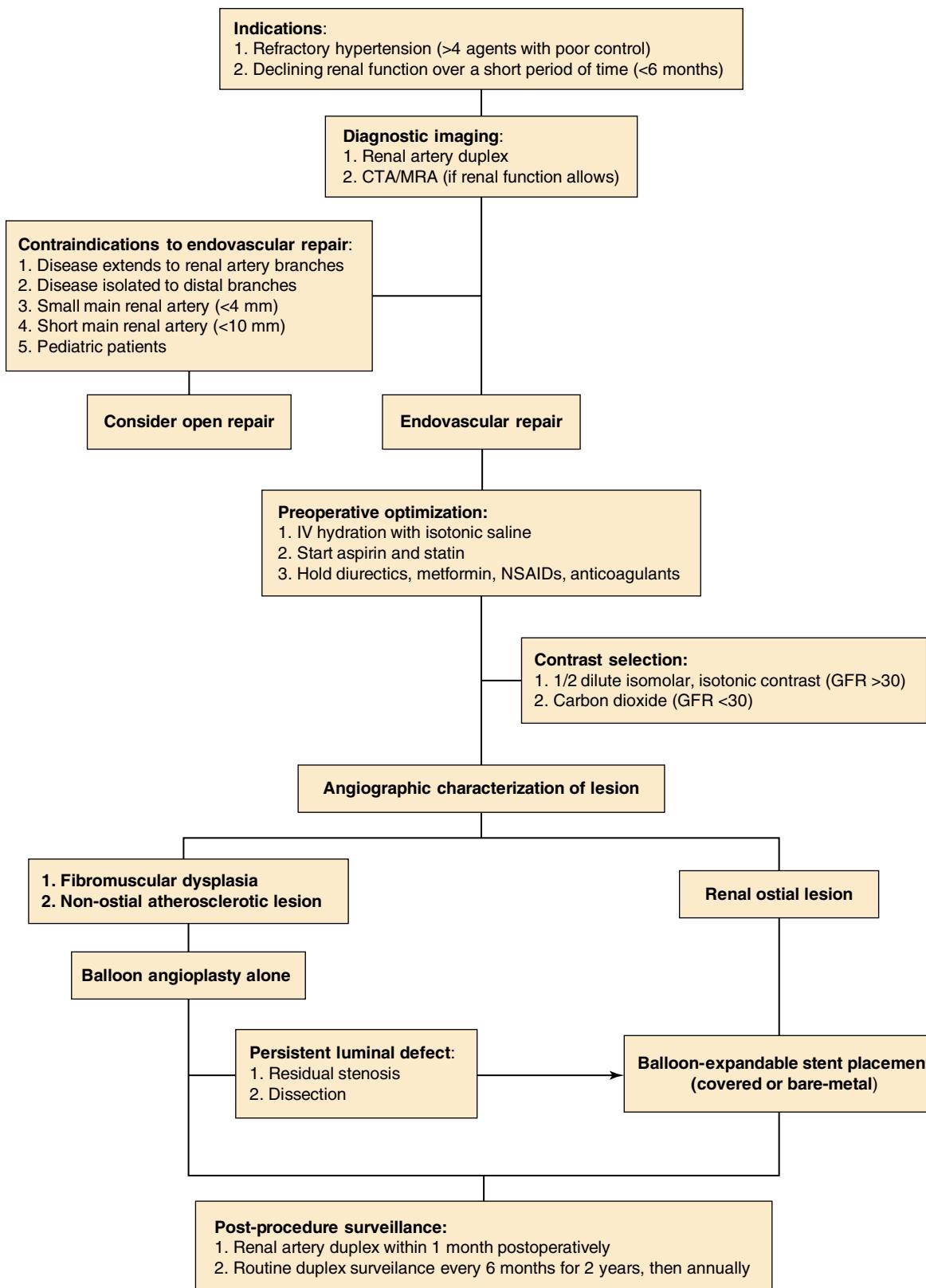


Figure 129.8 Right renal artery rupture resulting from angioplasty and stenting. A retroperitoneal pseudoaneurysm (arrow) is visible adjacent to the deployed right renal artery stent on a contrast-enhanced computed tomography scan. The patient required surgical exploration to achieve hemostasis.

wire perforation¹¹⁰ of the parenchyma of the kidney, resulting in a perinephric hematoma. Retroperitoneal or perinephric hematoma accumulation that is undetectable on physical examination may be identified by non-contrast-enhanced computed tomography (Fig. 129.8); if no apparent bleeding is identified, other causes (such as myocardial infarction, heart failure, or reaction to the contrast agent) should be investigated. Bleeding due to renal artery perforation may be managed most commonly with endovascular techniques; surgical exploration is rarely required. A perinephric hematoma due to guide wire perforation with hemodynamic instability is a medical emergency and should be treated immediately and definitively with either endovascular embolization, coiling, or open surgical treatment. Acute renal artery stent thrombosis after intervention is a rare complication. Successful endovascular treatment of procedure-related renal artery thrombosis by thrombolysis has also been described.¹⁰⁹ Late renal artery stent thrombosis occurs infrequently and thrombolysis with re-stenting as an endovascular option has been shown to be effective.¹¹¹

CHAPTER ALGORITHM



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Bax L, Woittiez AJJ, Kouwenberg HJ, et al. Stent placement in patients with atherosclerotic renal artery stenosis and impaired renal function: a randomized trial. *Ann Intern Med.* 2009;150:840–841.

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This report details the results of the CORAL trial, which represents the most robust and rigorous investigation of renal revascularization therapies.

Edwards MS, Corriere MA. Contemporary management of atherosclerotic renovascular disease. *J Vasc Surg.* 2009;50:1197–1210.

This review provides an excellent overall summary of existing thought on current management of atherosclerotic renovascular disease.

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A complete reference list can be found online at www.expertconsult.com.

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Renovascular Disease: Acute Occlusive and Ischemic Events

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INTRODUCTION

Acute renovascular ischemia is defined as a sudden interruption of arterial and/or venous renal blood flow; it may involve either partial or complete ischemia of one or both kidneys. In the setting of acute renal ischemia, salvage of a functioning kidney requires a high index of suspicion, rapid diagnosis, and prompt implementation of a definitive treatment plan. Acute renal ischemia can progress rapidly to irretrievable loss of renal function. Appropriate clinical management is therefore quite time-sensitive, and the duration and severity of renal ischemia are key considerations for decision-making.

Causes of acute renal ischemia include renal artery thrombosis, embolism, trauma, aortic or renal artery dissection, iatrogenic injury, and renal vein thrombosis (Box 130.1).

Evidence supporting specific management strategies for acute renal ischemia is limited and primarily comes from case reports and small cohort studies.

PATHOPHYSIOLOGY

Ischemic renal injury is characterized by glomerular collapse and tubular necrosis, resulting in reduced glomerular filtration and loss of tubular function.¹ Post-ischemic reperfusion further exacerbates endothelial dysfunction through excess cytokine secretion and the expression of adhesion molecules that promote leukocyte influx. Reperfusion injury also leads to production of nitric oxide and reactive oxygen species, creating additional parenchymal damage and impaired autoregulation of renal blood flow.²

BOX 130.1**Causes of Acute Renal Ischemia**

1. Renal artery thrombosis
 - Atherosclerotic disease
 - Aneurysmal disease
 - Dissection (aorta or renal artery)
 - Fibromuscular dysplasia
 - Arteritis (e.g., Takayasu)
 - Stent thrombosis
 - Hypercoagulable state
2. Renal artery embolism
 - Chronic atrial fibrillation
 - Valvular heart disease
 - Aortic plaque
 - Paradoxical embolism in setting of patent foramen ovale
 - Cardiac tumor
3. Trauma
4. Iatrogenic
 - Direct injury or embolization
 - Ostial occlusion by aortic endograft
5. Renal vein thrombosis
 - Hypercoagulable state
 - Mechanical obstruction
 - Malignancy

Baseline kidney function and the duration of warm renal ischemia affect the potential for recovery of function and are therefore important considerations when revascularization is being considered. Age and diabetes have been identified as risk factors associated with increased susceptibility to injury from acute renal ischemia in experimental models.^{3,4}

Duration of Ischemia

In a normal kidney, 1 hour of warm renal ischemia is associated with loss of 70% to 80% of function, but complete recovery is possible within weeks. Increasing the ischemia duration to 2 hours limits long-term recovery to 30% to 50% of baseline function.⁵ *In vivo* models of acute, total renal artery occlusion have demonstrated irreversible ischemia after 3 to 4 hours of warm ischemia, but this varies by species.^{6,7} Figure 130.1 shows serum creatinine levels in a porcine model after warm renal ischemia lasting 15, 60, 90, and 120 minutes.⁸ Prolonged ischemia time (>45 minutes) has been identified as a predictor of poor early graft function following human living donor kidney transplantation,⁹ and more than 90 minutes of acute warm ischemia has been suggested as a cut point for the retrieval of renal function.¹⁰ Technically successful revascularization performed after longer durations of acute ischemia may not achieve functional recovery, especially when overt renal failure or anuria is already present.^{10,11}

Gradual Versus Acute Renal Occlusion

In contrast to acute renal artery occlusion, gradual main renal artery occlusion (e.g., main renal artery stenosis that progresses in severity over months or years) may induce development of collaterals and lessen the severity of the ischemic insult. Renal artery collaterals originate from the inferior adrenal, gonadal, ureteral, internal iliac, lumbar, intercostal, and capsular arteries (Fig. 130.2).^{12–15} Less commonly, collaterals may also originate

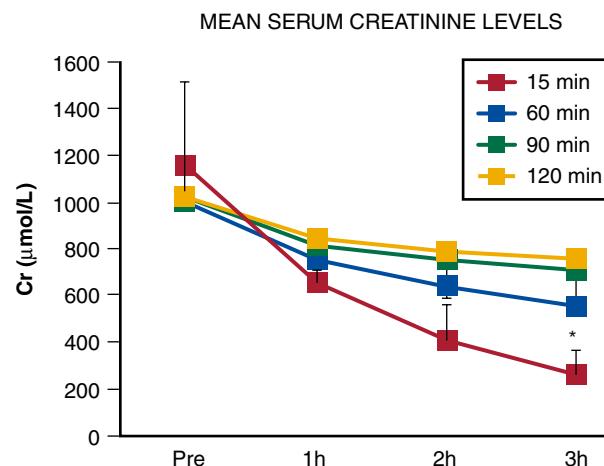


Figure 130.1 Duration of Warm Ischemia and Mean Serum Creatinine. Mean serum creatinine levels following 15, 60, 90, and 120 minutes of warm ischemia and 2 hours (h) of static cold storage. (From Hosgood SA, Shah K, Patel M, and Nicholson ML. The effect of prolonged warm ischaemic injury on renal function in an experimental ex vivo normothermic perfusion system. *J Transl Med*. 2015;13:207.)

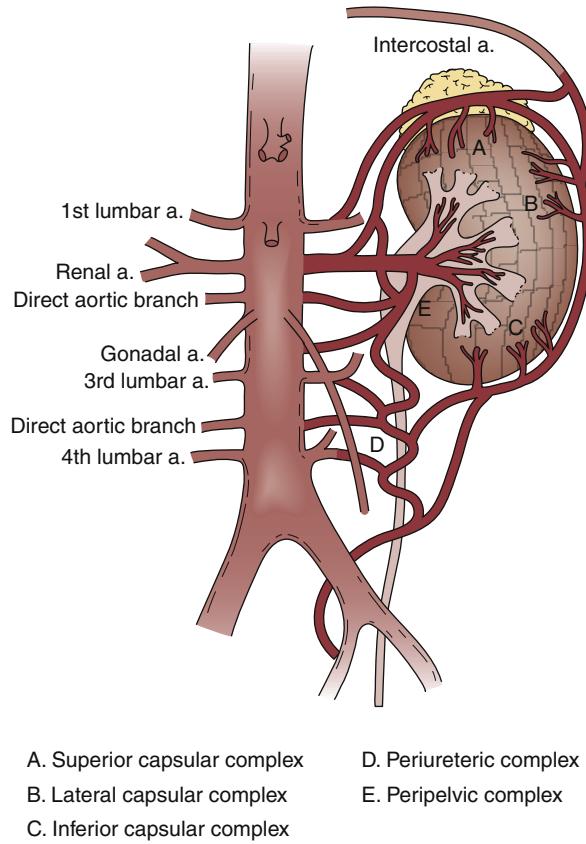


Figure 130.2 Collateral Circulation of the Kidney. (From Flye MW, Anderson RW, Fish JC, Silver D. Successful surgical treatment of anuria caused by renal artery occlusion. *Ann Surg*. 1982;195:346–353.)

from the inferior mesenteric artery.¹⁶ Pre-renal communication may exist at the hilum, while intrarenal communication occurs through capsular perforating arterioles. As much as 80% of renal arterial collateral communication may be independent of the main renal artery,⁷ potentially allowing maintained functional viability despite main renal artery occlusion (as in acute

thrombosis of a chronic critical stenosis).^{14,17} Accessory renal arteries (alternatively referred to as aberrant, duplicated, or extra renal arteries) have a prevalence of 24% to 42% based on imaging and cadaver studies and may also preserve direct flow from the aorta.^{18–21}

With gradual reduction of renal perfusion, maintained tubular function (reflected by low fractional excretion of sodium and normal *N*-acetyl-glucosaminidase excretion) has been observed despite reductions in both glomerular filtration rate and filtration fraction.¹ In a canine model of gradual renal artery occlusion induced incrementally over 7 weeks, collateral circulation developed and was sufficient to maintain both renal viability and life.²² Clinical reports of functional recovery following periods of main renal artery occlusion exceeding 24 hours underscore the importance of collateral perfusion and demonstrate that the potential for kidney salvage is not solely a function of warm ischemia time.^{23–31} Main renal artery occlusion accompanied by the visualization of good renal vein flow on duplex ultrasound suggests the presence of collateral arterial flow.³¹

Renal Vein Thrombosis

Renal vein thrombosis can also produce acute renal ischemia, and may occur primarily or in association with inferior vena cava thrombosis. As in acute arterial ischemia, the degree of renal functional impairment and severity of clinical presentation depend on the acuity of venous occlusion and collateral flow.³² When renal vein thrombosis occurs rapidly in the absence of developed collaterals, congestion, and edema of the affected kidney can produce pain from capsular distension as well as hemorrhagic infarction.^{33,34} Experimental models have demonstrated that renal injury and blood flow reduction are both greater from venous than arterial occlusion.³⁵

CLINICAL PRESENTATION

Symptoms and signs of acute renal ischemia include abdominal or back pain, dyspnea, nausea, vomiting, hematuria, anuria, and acute hypertension.^{24,26,29,30,36–39} Since many of these symptoms are nonspecific, the differential diagnosis for acute renal ischemia is broad and includes a variety of nonrenal pathologies. Renal colic with nephrolithiasis is a common initial presumptive diagnosis with a similar presentation, and several reports have recommended that absence of renal calculi on an unenhanced computed tomography (CT) scan performed for suspected renal colic should prompt a contrast-infused study to rule out acute ischemia.^{40–43} Other diagnoses with symptoms similar to renal ischemia include pyelonephritis, renal carcinoma, mesenteric ischemia, cholecystitis or biliary colic, gastritis, splenic infarction, myocardial infarction, and pulmonary embolism.^{24,30,37,40,44}

Clinical presentation of acute renal ischemia is often delayed for hours or days after symptom onset, adversely affecting the possibility of functional salvage through revascularization. In a series of patients with embolic renal artery occlusion secondary to atrial fibrillation described by Hazanov et al., the

majority of patients presented over 24 hours after symptom onset⁴⁰; Huang et al. reported a median time of 31 hours between symptom onset and emergency department presentation for acute renal infarction.³⁶

Renal vein occlusion may also present with unilateral congestion and tenderness, although the enlarged kidney is seldom palpable in adults.³³ In a retrospective cohort study of neonates with renal vein thrombosis, only 13% of patients had the “classic triad” of flank mass, gross hematuria, and thrombocytopenia.⁴⁵

DIAGNOSTIC EVALUATION

Laboratory Tests

No single laboratory test is diagnostic of acute renal ischemia, so tests must be selected and interpreted in combination with the clinical presentation and physical exam findings. Laboratory abnormalities reported with acute renal ischemia include leukocytosis, elevated lactate dehydrogenase (LDH), gross or microscopic hematuria, proteinuria, elevated D-dimer, and eosinophilia.^{26,28,30,36,40,41,46,47} Eosinophilia may be indicative of atheroembolism.^{47,48} In a series of 20 patients with acute renal ischemia, 19 patients had an elevated serum LDH, while 16 had the triad of flank/abdominal pain or tenderness, elevated serum LDH, and proteinuria.³⁶ Only half of patients presenting with acute renal ischemia have an elevated serum creatinine,⁴¹ so a normal serum creatinine does not exclude this diagnosis. In the setting of a contralateral kidney with normal function, asymptomatic unilateral renal ischemia may be identified as an incidental imaging finding of unknown acuity.

Imaging Tests

Imaging plays a critical role in the diagnosis of acute renal ischemia because the history, physical exam, and laboratory findings are often nonspecific. The ideal diagnostic imaging test for renal ischemia would be rapid, widely available, noninvasive, inexpensive, and with no need for either radiation exposure or contrast administration. Although no imaging technique shares all of these characteristics, consideration of these attributes can be helpful in choosing the most appropriate study.

Computed Tomography

Computed tomography angiography (CTA) is widely available, rapid, and useful for ruling out other soft tissue abnormalities when acute renal ischemia is suspected. CTA is less time-consuming than MRA and has 80% sensitivity for acute renal ischemia (which is superior to ultrasound).⁴⁰ CTA also evaluates aortic and iliac anatomy, which is useful for planning revascularization if indicated. CTA should include both noncontrast and venous phase images, and is also the study of choice for renal vein thrombosis, with a sensitivity and specificity that both approach 100%.³⁴ Features of a renal infarct on CTA imaging include areas of hypoattenuation with associated mass effect (Fig. 130.3) or a “cortical rim sign,” described as a rim of functioning nephrons supplied by capsular collaterals surrounding an otherwise nonfunctioning kidney.^{40,41,49,50}

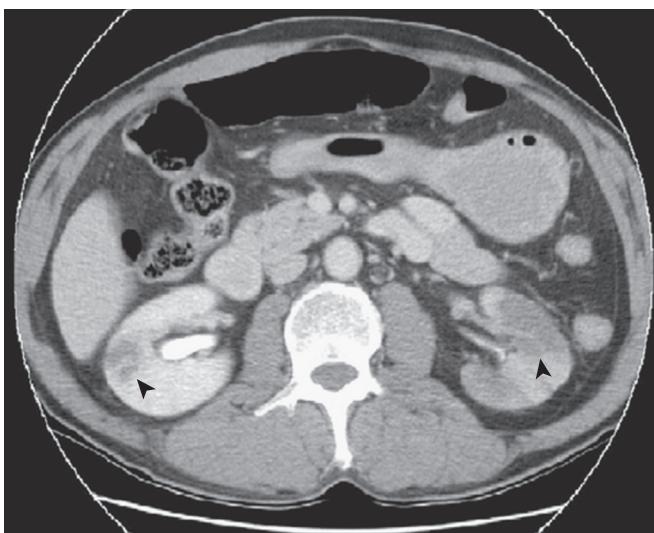


Figure 130.3 Computed Tomography Angiography Demonstrating Bilateral Renal Infarcts. Patient had sudden-onset bilateral flank pain with a history of atrial fibrillation. Bilateral wedge-shaped, hypodense lesions (arrowheads) are consistent with embolic renal infarcts. (From Huang C-C, Lo H-C, Huang H-H, et al. ED presentations of acute renal infarction. *Am J Emerg Med*. 2007;25(2):164–169.)

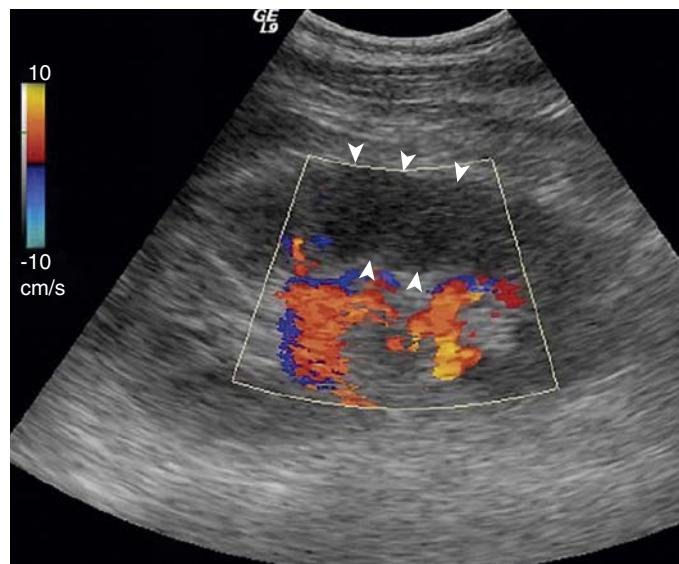


Figure 130.4 Renal Infarction Demonstrated by Duplex Ultrasound. Renal infarction caused by arterial embolism appears as a wedge-shaped hypoechoic area with absence of blood flow signal (arrowheads). (From Cai S, Ouyang YS, Li JC, et al. Evaluation of acute renal artery thrombosis or embolism with color Doppler sonography. *Clin Imaging*. 2008;32(5):367–371.)

Magnetic Resonance Angiography

Renal MRA is capable of the anatomic evaluation plus physiologic assessment through flow-dependent imaging.⁵¹ Unenhanced MRA evaluation for renal artery disease has similar reliability to CTA; its capability for evaluating renal segmental arteries and parenchymal disease make it potentially useful when contrast imaging is contraindicated.⁵² These advantages have resulted in increasing utilization of MRA for the evaluation of chronic renal artery disease, but MRA is used less frequently as first-line imaging for acute renal ischemia. MRA has practical drawbacks when rapid imaging is required because it is more time-consuming and may require extensive image post-processing by a technologist. Compared with CTA and/or ultrasound, access to rapid MRI is more limited. MRI/MRA may have a benefit when assessing for renal cell carcinoma concomitant with renal vein thrombosis.

Ultrasound

Ultrasound requires neither contrast nor radiation exposure but is less sensitive than CTA for renal ischemia. Renal infarcts typically appear as wedge-shaped, hypoechoic lesions with absent blood flow on duplex ultrasound (Fig. 130.4). Ultrasound imaging is technician-dependent and may be challenging in patients who are obese or have excessive bowel gas. In a series of 44 patients with renal embolism, only 3 of 27 (11.1%) ultrasounds were positive. In a retrospective review of 10 patients with renal artery thrombosis or embolism, Cai et al. concluded that color Doppler ultrasonography was useful for the detection of large infarcts resulting from main renal artery occlusion but that other imaging modalities were needed to confirm smaller infarcts.⁵³ They also reported decreased renal artery peak systolic velocity as a finding that may be associated with intrarenal embolism. In addition to renal vein thrombus,

other ultrasound findings indicative of ischemia due to venous thrombosis may include unilateral kidney edema or congestion.

Nuclear Renal Scan

Nuclear renal isotope scanning has been suggested as the most sensitive imaging technique for the diagnosis of acute renal ischemia, but this modality is seldom practical because it is time-consuming and less widely available. The reported sensitivity of nuclear imaging for acute renal ischemia is 97%,⁴⁰ and infarcts are characterized by a marked reduction of renal blood flow with preserved kidney size.⁵⁴

Angiography

Although catheter angiography potentially allows simultaneous endovascular treatment of renal ischemia during the same procedure, angiography for diagnostic purposes should be limited to situations where other imaging studies are equivocal or the index of suspicion is especially high (e.g., suspected iatrogenic injury).

RENAL ARTERY EMBOLISM

Epidemiology

Renal artery embolism is the most common cause of acute renal ischemia.^{40,55} Risk factors for renal artery embolism include atrial fibrillation, ischemic heart disease, cardiomyopathy, previous arterial thromboembolism, valvular heart disease, cardiac tumor, atherosclerotic aortic plaque, and paradoxical embolism in the setting of a patent foramen ovale (Fig. 130.5). Renal artery embolism is uncommon, with an incidence among hospitalized patients of 0.007% (or 6.1 per million per year).⁵⁶

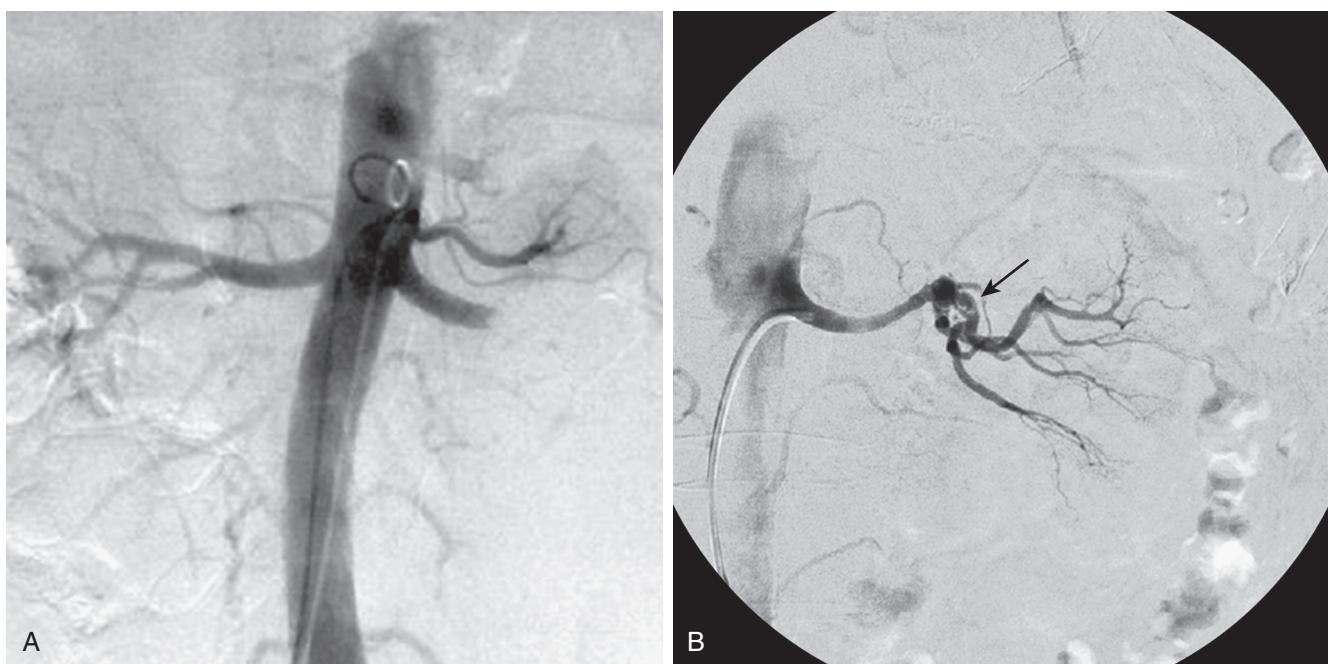


Figure 130.5 Renal Artery Embolism. (A) Contrast angiogram demonstrating left main renal artery occlusion resulting from embolism in a patient with atrial fibrillation. (B) Contrast angiogram demonstrating branch renal artery thrombosis resulting from cardiac tumor embolus. (A, From Syed MI, Shaikh A, Ullah A, et al. Acute renal artery thrombosis treated with t-PA power-pulse spray rheolytic thrombectomy. *Cardiovasc Revasc Med*. 2010;11(4):264.e1–7. B, From Boggetto-Graham L, Chavanon O, Hacini R, et al. An uncommon cause of renovascular hypertension. *Can J Cardiol*. 2012;28(3):397.e1–3.)

Renal artery branches are more commonly occluded by emboli than the main renal artery, making it challenging to distinguish between embolism and thrombosis in the setting of branch occlusion with a partial infarct unless an embolic source is identified.

Cholesterol crystal embolization (CCE) is a distinct type of renal embolism characterized by multiple small emboli from cholesterol debris. Chronic CCE has an incidence of 6.2 per million people per year; risk factors include male gender, hypertension, and atherosclerosis.⁵⁷ CCE may also occur during arterial catheterization, especially with repeated instrumentation of a diseased thoracic or abdominal aorta. CCE is associated with a 30% incidence of progression to dialysis within 2 years and increased mortality (see Ch. 106, Atheromatous Embolization and Its Management).⁵⁸

Anticoagulation

Heparin anticoagulation should be initiated immediately once embolic renal artery occlusion is suspected to prevent thrombus propagation and additional embolic events. Besides anticoagulation, patients with renal embolism also require thorough evaluation for the embolic source to determine whether additional treatment (such as repair of proximal aneurysmal disease, thrombectomy, repair of patent foramen ovale, or heart valve replacement) is warranted for definitive management and reduction of risk for repeat embolism. Among patients presenting with renal artery embolism, over 20% have a history of previous thromboembolism.^{40,56,59}

Although successful management with anticoagulation alone has been described,^{12,40} revascularization may be considered in

patients presenting with acute ischemia and potentially salvageable renal function, especially in the setting of bilateral embolism. Conversely, anticoagulation alone can be used for definitive management in patients with unilateral embolism and limited potential for renal salvage (e.g., patients with pre-existing chronic kidney disease or other renal pathology). In the report of Hazanov et al., over 80% of patients presenting with renal embolism were managed using anticoagulation alone, without procedural intervention.⁴⁰ One-month mortality was 11% in this series; 61% of patients had normal renal function at long-term follow-up, and only 8% required dialysis.

Endovascular Treatment

Catheter-directed thrombolysis can be a valuable treatment strategy for acute renal ischemia resulting from arterial embolism, where the occlusion is typically located at the renal artery bifurcation or distal segmental branches (Fig. 130.6).^{11,37,60,61} Although thrombus resolution can be achieved with catheter-directed thrombolysis alone,⁶² adding mechanical thrombectomy may permit definitive management in a single procedure without need for extended infusion.^{28,38} Aspiration thrombectomy, angioplasty, and stenting may also be utilized as adjuncts to thrombolysis in the setting of embolic renal artery occlusion.³⁰ Despite treatment with low-dose thrombolysis in 14 patients, Blum et al. did not observe recovery of renal function in any patients presenting with complete main renal artery embolic occlusion, whereas partial functional recovery was observed following either incomplete or segmental branch occlusion.¹⁰

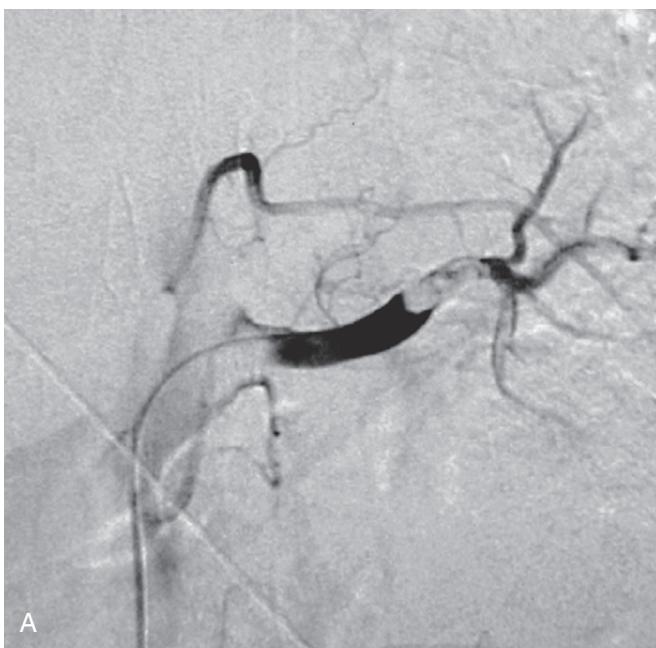


Figure 130.6 Management of Renal Artery Embolism with Catheter-Directed Thrombolysis. (A) Distal left renal artery embolus demonstrated by angiography. (B) Patent left renal artery demonstrated following catheter-directed thrombolysis. (From Robinson S, Nichols D, Macleod A, et al. Acute renal artery embolism: a case report and brief literature review. *Ann Vasc Surg.* 2008;22(1):145–147.)

Open Surgery

Surgical embolectomy is utilized infrequently for acute renal embolism because it is relatively invasive and time-consuming compared with endovascular treatment. Surgical embolectomy can be performed through a midline or transverse incision (see Ch. 56, Abdominal Vascular Exposures).^{63,64} Following renal artery exposure, the artery and vein are mobilized and encircled with vessel loops. Embolectomy can then be performed through either a transverse or longitudinal incision in the renal artery, with removal of thrombus using forceps and a Fogarty catheter. Following removal of the embolism and associated thrombus, the arteriotomy can be closed either primarily or with a patch angioplasty.



Figure 130.7 Thrombotic Bilateral Renal Artery Occlusion. The patient presented with acute renal failure and anuria. Nuclear medicine study demonstrated renal perfusion by collateral vessels and indicated viable kidneys.

Intraoperative completion duplex ultrasound should then be considered to evaluate patency and rule out residual stenosis or other technical complications. There is a paucity of mortality data following operative treatment of acute renal artery embolism. The largest study to date was published in 1977 by Lacombe, who reported a 25% mortality associated with surgical management of renal artery embolism. These authors noted a similar mortality but a lower rate of renal function recovery among patients managed medically.⁶⁵ Lack of more recent series suggests that thromboembolectomy is infrequently used for acute renal ischemia in contemporary practice.

RENAL ARTERY THROMBOSIS

Epidemiology

In situ (i.e., non-embolic) renal artery thrombosis may result from a variety of both renal and extrarenal disease processes, including atherosclerotic renal artery stenosis, aortic aneurysmal or atherosclerotic disease, aortic or renal artery dissection, renal artery aneurysm, fibromuscular dysplasia (FMD), arteritis (e.g., Takayasu), stent thrombosis, hypercoagulable state, or trauma. Iatrogenic thrombosis may also result from renal artery coverage or injury during endovascular procedures. Other less common causes of renal artery thrombosis include antiphospholipid antibody syndrome,^{66,67} factor V Leiden mutation,⁶⁸ heparin-induced thrombocytopenia,⁶⁹ and Behçet disease.⁷⁰

Thrombotic occlusion of pre-existing atherosclerotic disease is most common among individuals over age 55 (Fig. 130.7). Although atherosclerotic renal artery stenosis has an incidence of 7% among elderly individuals,⁷¹ progression to thrombosis is uncommon in the absence of other contributing factors, such as dehydration or hypotension.^{54,72} In a study by Caps et al., the 5-year incidence of renal artery thrombosis in the setting of known stenosis was less than 5%. Risk factors

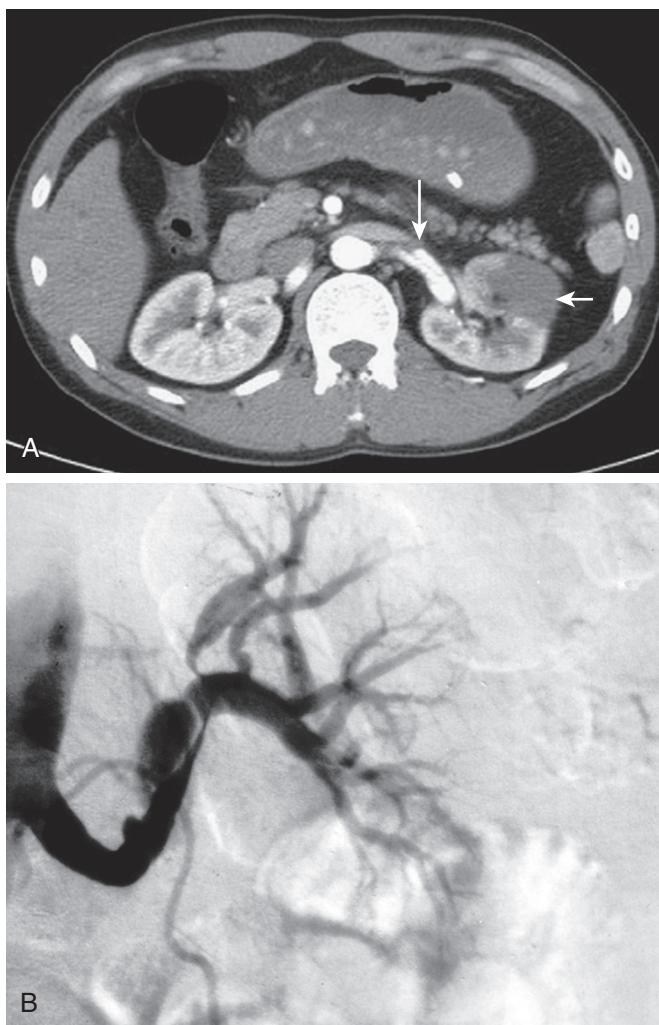


Figure 130.8 Spontaneous Renal Artery Dissection. (A) Axial computed tomography angiographic image shows left renal artery dissection (long arrow) with ipsilateral upper pole renal infarction (short arrow). (B) Contrast angiogram demonstrating spontaneous renal artery dissection extending into the hilar branches of the left kidney. (A, From Tsai TH, Su JT, Chao CC et al. Spontaneous renal artery dissection complicating with renal infarction. *Urology*. 2010;76:1371–1372. B, From van Rooden CJ, van Baalen JM, van Bockel JH. Spontaneous dissection of renal artery: long-term results of extracorporeal reconstruction and autotransplantation. *J Vasc Surg*. 2003;38(1):116–122.)

for thrombosis included systolic blood pressure greater than 160 mm Hg, diabetes mellitus, and the presence of high-grade ($\geq 60\%$) stenosis.⁷³

Spontaneous renal artery dissection occurs more commonly among men (4:1 versus women).^{74,75} Spontaneous renal artery dissection was the second most common cause of acute renal ischemia (embolism was most common) in an institutional series reported by Yoon et al.⁷⁶ An intimal flap is the most characteristic imaging finding (Fig. 130.8) but may not be apparent if the false lumen is thrombosed; other findings may include renal artery enlargement or a long, tapered stenosis. Dissection may be unilateral or bilateral at the time of presentation.⁷⁷ Other risk factors include fibromuscular dysplasia,^{74,78–80} Ehlers–Danlos syndrome,^{75,81} cocaine use,⁸² and strenuous exercise.^{83–85} Iatrogenic renal artery dissection has been reported following a variety of procedures including

diagnostic angiography,⁸⁶ peripheral or aortic endovascular intervention,^{87,88} and extracorporeal lithotripsy.⁸⁹

Medical Treatment

Medical treatment – including systemic anticoagulation, control of hypertension, and assessment and management of intravascular volume status – is an important component of the management of acute renal ischemia regardless of whether revascularization is undertaken. Systemic anticoagulation should be initiated in the setting of acute presentation to prevent propagation of thrombus and preserve collateral flow.

Endovascular Treatment

Endovascular options for the management of acute renal ischemia include angioplasty and stenting, thrombolysis, mechanical catheter thrombectomy, and aspiration thrombectomy, and these treatments are frequently used in combination.

Angioplasty and stenting are useful for the treatment of thrombosis of a critical primary renal artery or in-stent lesion.^{26,31,90} Use of catheter-directed thrombolysis in combination with angioplasty and stenting can restore patency of thrombosed distal branch vessels distal to a main renal artery occlusion.⁹¹ Aspiration thrombectomy combined with angioplasty and stenting plus intraoperative bolus thrombolytic administration may also restore renal perfusion.³⁰ Catheter-directed thrombolysis for treatment of an acutely thrombosed renal artery aneurysm has also been reported, permitting subsequent staged aneurysm repair.⁶⁰ Systemic thrombolysis prior to angioplasty and stenting has also been described.⁹² In a series of 10 patients with renal artery thromboembolism from a variety of causes treated by Salam et al. with catheter-directed thrombolysis, adjunctive angioplasty and/or stenting was necessary in half of the procedures and the rate of anatomically successful revascularization (defined by completion angiography) was 70%.¹¹ Only one of four patients in this series who presented with acute renal failure, however, recovered renal function after intervention.

Renal artery dissection may resolve with medical management.⁹³ Angioplasty and stenting have also been used to treat spontaneous renal artery dissection. Jiang et al. reported technical success in 14/15 attempted cases.⁹⁴ Serum creatinine and estimated glomerular filtration rates improved from presentation over a median follow-up of 41.5 months in this series; 29% of patients did not require any postoperative antihypertensive medications, and 57% who still required medication had improved blood pressure. Similar findings have been reported by others.⁹⁵ These results suggest that endovascular treatment of renal artery dissection is reasonable for patients who are symptomatic and cannot be managed medically. Intravascular ultrasound may be useful for confirming the diagnosis and identifying the true versus false lumen during treatment (Fig. 130.9).⁸⁸

Open Surgery

Surgical revascularization options include renal artery thrombectomy, bypass, and/or endarterectomy (Fig. 130.10).^{63,64}

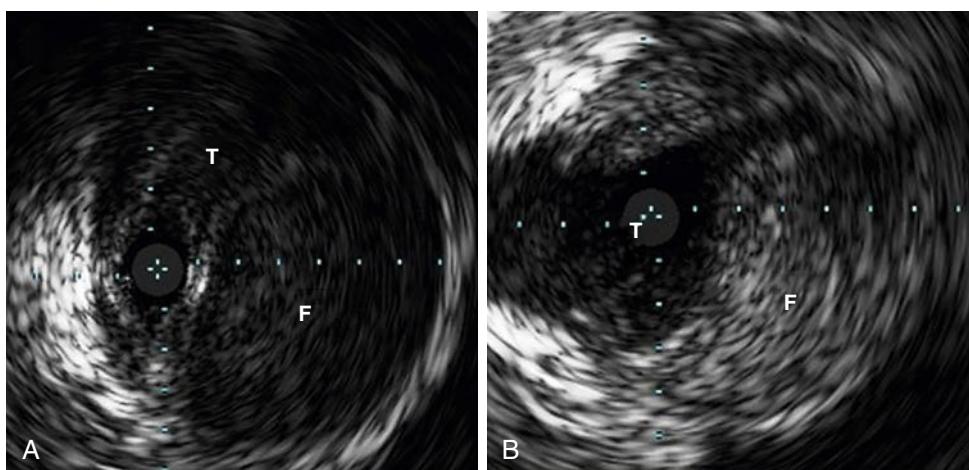


Figure 130.9 Intravascular ultrasound revealing renal artery dissection flap with compromised true lumen. (A) Proximal portion of left renal artery. (B) Distal portion of left renal artery. F, false lumen; T, true lumen. (From Watanabe Y, Aramoto H, Asano R, Furuichi S et al. Iatrogenic renal artery dissection uncommon complication during aortic endovascular repair. *JACC Cardiovasc Interv.* 2010;3(9):986–987.)



Figure 130.10 Bilateral Aortorenal Vein Graft Bypasses Performed for Thrombotic Occlusion. (Preoperative angiogram is shown in Figure 130.7.) Renal biopsy showed partial infarction, but renal function recovered immediately following revascularization and gradually returned to normal over several months.

Because surgical revascularization is more invasive and time-consuming (therefore necessitating a longer duration of ischemia), these procedures are typically reserved for situations where the chance of renal salvage is deemed substantial (limited ischemia, normal pre-existing kidney function, acute thrombosis of pre-existing chronic stenosis with existing collaterals, etc.) and where endovascular treatment has failed or may be ill-advised owing to technical or anatomic considerations.

Surgical exposure for renal artery revascularization is similar to that described earlier for embolectomy. Revascularization techniques – including endarterectomy, aortorenal bypass, and extra-anatomic bypass (see Ch. 128, Renovascular Disease: Open Surgical Treatment). Renal artery bypass for ischemia resulting from delayed thrombosis after renal artery angioplasty and/or stenting has been reported with successful salvage of renal function.^{96,97} As with endovascular treatment, the

maintenance of collateral perfusion is critical to functional kidney salvage. In a series that included 16 patients treated with surgical revascularization, Ouriel et al. noted that renal salvage with thrombectomy and aortorenal bypass was dependent on a distally reconstituted renal artery regardless of the duration of ischemia, but recovery of renal function among patients with embolism or traumatic injury in this series was uncommon.⁹⁸

Perioperative mortality associated with surgical revascularization for acute renal artery thrombosis is 15% to 25%, with salvage of renal function in approximately 65% of patients.^{99,100} Surgical management may also be preferred over endovascular therapy when thrombus burden is extensive or thrombolysis is contraindicated. Chevalier et al. described complete recovery following surgical thrombectomy in a patient with acute aortic and renal artery thrombosis due to heparin-induced thrombocytopenia.⁶⁹

There is inadequate evidence to support a particular management strategy for spontaneous renal artery dissection. Reported outcomes associated with surgical revascularization are predominantly derived from series of elective procedures performed for hypertension associated with dissection-related stenosis rather than acute thrombosis.^{101,102} Resolution of symptoms with preservation of renal function has been reported in patients presenting with dissection and segmental renal infarction managed nonoperatively with antihypertensive agents and/or anticoagulation.^{103,104}

RENOVASCULAR TRAUMA

Epidemiology

Renovascular injury has a prevalence of less than 1% in the setting of blunt abdominal trauma (see Ch. 182, Abdominal Vascular Trauma).^{105,106} In addition to renovascular injury, approximately 75% of patients have concomitant abdominal solid organ injury involving the kidney, liver, or spleen, and over 17% have multiple abdominal vascular injuries (most commonly renal vein or inferior vena cava).¹⁰⁶ The mechanism of traumatic renal artery injury frequently involves rapid deceleration or compression between the abdominal wall and spine,

resulting in subintimal dissection and thrombosis. Renovascular injury may also be associated with injury to the urinary collection system (Fig. 130.11).^{107–109}

Treatment

A selective approach to revascularization is warranted for renovascular trauma. Revascularization for traumatic injury should be considered in the setting of bilateral injury or injury to a solitary functioning kidney.

In contrast to nontraumatic renal ischemia, where the time of symptom onset may be ill-defined and patients often present more than 24 hours later, the duration of ischemia is often known with some degree of accuracy in the setting of traumatic injury, thus facilitating relatively informed decisions regarding revascularization. In addition to prolonged warm ischemia,

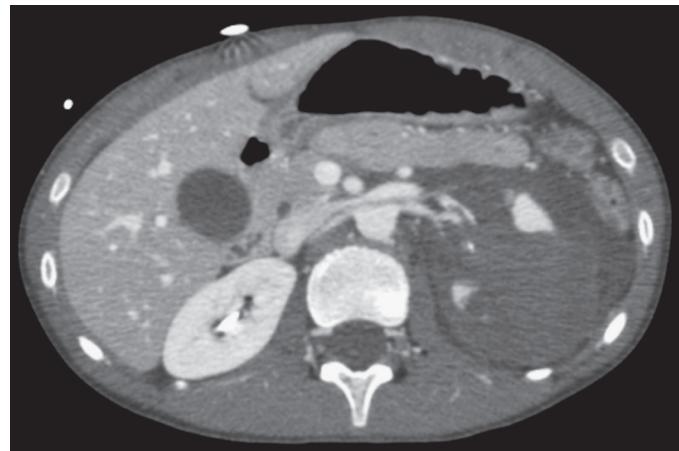


Figure 130.11 Traumatic Injury to the Left Kidney. Contrast CT demonstrates large left renal perfusion defect caused by acceleration-deceleration trauma of the renal pedicle, resulting in vascular injury and kidney rupture as well as injury of the urinary collecting system.

other factors favoring management without revascularization include hilar injury, extensive kidney injury, and a normal contralateral kidney in a patient with multiple other injuries and/or hemodynamic instability.^{110,111} Warm ischemia time lasting over 2–3 hours becomes progressively unsalvageable. Thorough evaluation for other injuries is also important to determine whether intraoperative anticoagulation can be safely administered during revascularization.

Selection of a revascularization approach requires consideration of renal and other solid organ injuries, hemodynamic stability, and anatomic characteristics of the vascular injury. Management options for blunt injury other than revascularization include nephrectomy, embolization, and observation with or without anticoagulation. Even complex injuries may heal without revascularization (Fig. 130.12).

Results

A meta-analysis of blunt renal artery injuries treated with percutaneous revascularization identified primary stenting as the most frequently utilized technique (>90%).¹¹² Complete renal artery occlusion was associated with technical failure in this analysis, and embolization was selectively used as a bail-out technique in <5% of cases.¹¹² In a review of 517 renal artery injuries from the National Trauma Data Bank by Sangthong et al., 73% of patients were managed nonoperatively, 18% had immediate nephrectomy, 9% underwent surgical revascularization, and 1.5% were treated with stenting.¹⁰⁶ Surgical revascularization techniques in this series included direct primary repair and autotransplantation. Overall mortality was 21%, the majority of which occurred within 24 hours of admission; these deaths were attributed to other injuries. Mortality among patients without other major abdominal injuries was 10.3%. Despite similar injury severity scores, patients who underwent revascularization or nephrectomy had longer hospital and

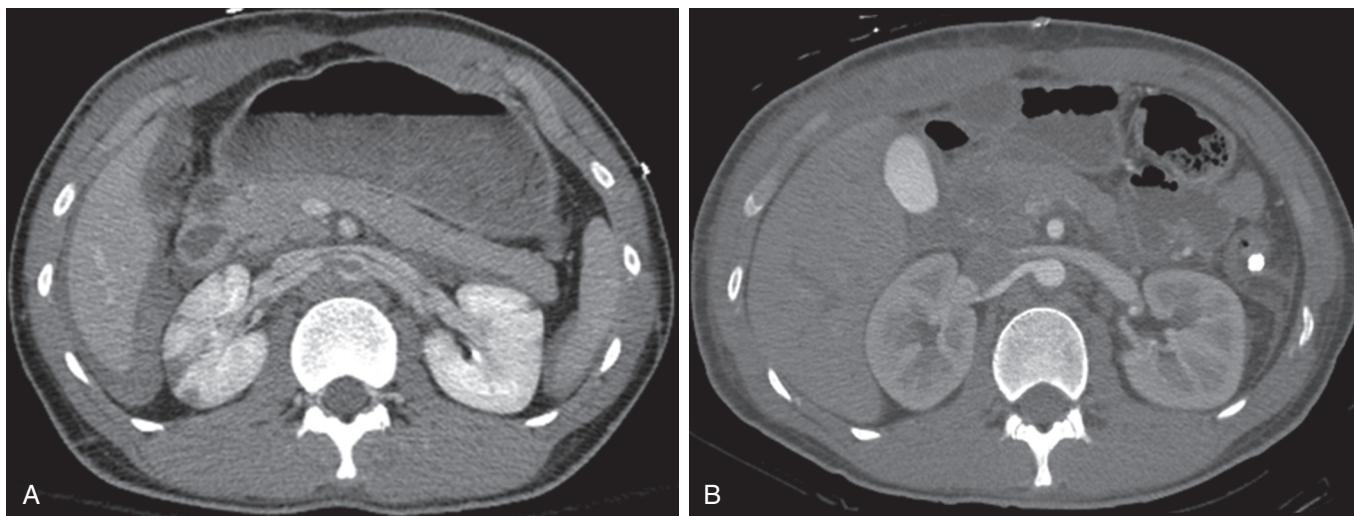


Figure 130.12 Renal Artery Dissection and Infarcts Associated with Blunt Traumatic Injury. Renal artery dissection associated with aortic dissection and multiple right renal infarcts visible on contrast computed tomography at the time of initial presentation (A). Follow-up scan 1 month later, after initial management with observation and anticoagulation, demonstrates healed dissection without residual perfusion defects (B).

intensive care stays, leading the authors to conclude that revascularization should be reserved for bilateral or solitary kidney injury to avoid long-term dialysis. A similar conclusion was reached by Haas and Spirnak, who reported a kidney salvage rate of approximately 25% in the setting of unilateral renal artery trauma.¹¹³

A registry study of severe kidney injuries by Knudson et al. identified blunt mechanism and severity of injury (categorized based on American Association for the Surgery of Trauma grade IV/V injuries) as predictors of poor outcomes. Interestingly, neither physician specialty nor time to definitive surgery were associated with the composite outcome (renal failure requiring dialysis, serum creatinine >2 mg/dL, <25% function of the injured kidney on renal scan, postinjury hypertension, or nephrectomy).¹¹⁴ Ten percent of patients in this study developed renal failure or hypertension postoperatively, and arterial repairs had inferior outcomes when compared with repair of isolated venous injuries.

Lopera et al. reviewed eight consecutive patients presenting with renal artery occlusion or dissection following blunt abdominal trauma who underwent endovascular treatment.¹¹⁵ Intervention resulted in contrast extravasation requiring embolization of the main renal artery in two patients in this study. Of the remaining six who underwent technically successful recanalization and stenting, half required nephrectomy or embolization during a follow-up period ranging from 2 to 24 months.

RENAL VEIN THROMBOSIS

Epidemiology

Renal vein thrombosis has an incidence of less than one case per million annually among adults, and is the most common non-catheter-related thrombosis in neonates.^{116,117} Risk factors include malignancy (especially renal cell carcinoma, lymphoma, and other retroperitoneal tumors), nephrotic syndrome, inherited thrombophilia, local surgery or inflammation, oral contraceptive use, pregnancy, and infection.^{32,118} Malignancy and nephrotic syndrome accounted for renal vein thrombosis in 66% and 20% of patients, respectively, in a single-center review of 218 adult patients by Wysokinski et al.¹¹⁹ Mean age at presentation in this cohort was 55 years, and 35% were women. Iatrogenic renal vein thrombosis may also occur in the presence of a vena cava filter or central venous catheter. Among children, more than 80% of renal vein thrombosis is diagnosed during the first month of life; risk factors include central vein catheters, dehydration, hypercoagulable state, and prolonged hypotension.^{45,117}

Anticoagulation

Anticoagulation is the treatment of choice in the majority of patients with renal vein thrombosis. As with treatment of renal artery embolism, goals for the treatment of renal vein thrombosis include both preservation of renal function and prevention of additional thromboembolism.³³

Therapy should be initiated with unfractionated or low-molecular-weight heparin at the time of diagnosis. The duration of long-term anticoagulation depends on the presumed etiology and identified risk factors. Patients with a temporary or modifiable risk factor (such as infection, hormone use, pregnancy, or immobilization due to surgery) can be treated for 3 to 6 months with a vitamin K antagonist or other oral anticoagulant, while those with idiopathic thrombosis or ongoing long-term risk factors (such as inherited thrombophilia or malignancy) should be managed, in the absence of contraindications, with lifelong anticoagulation.³²

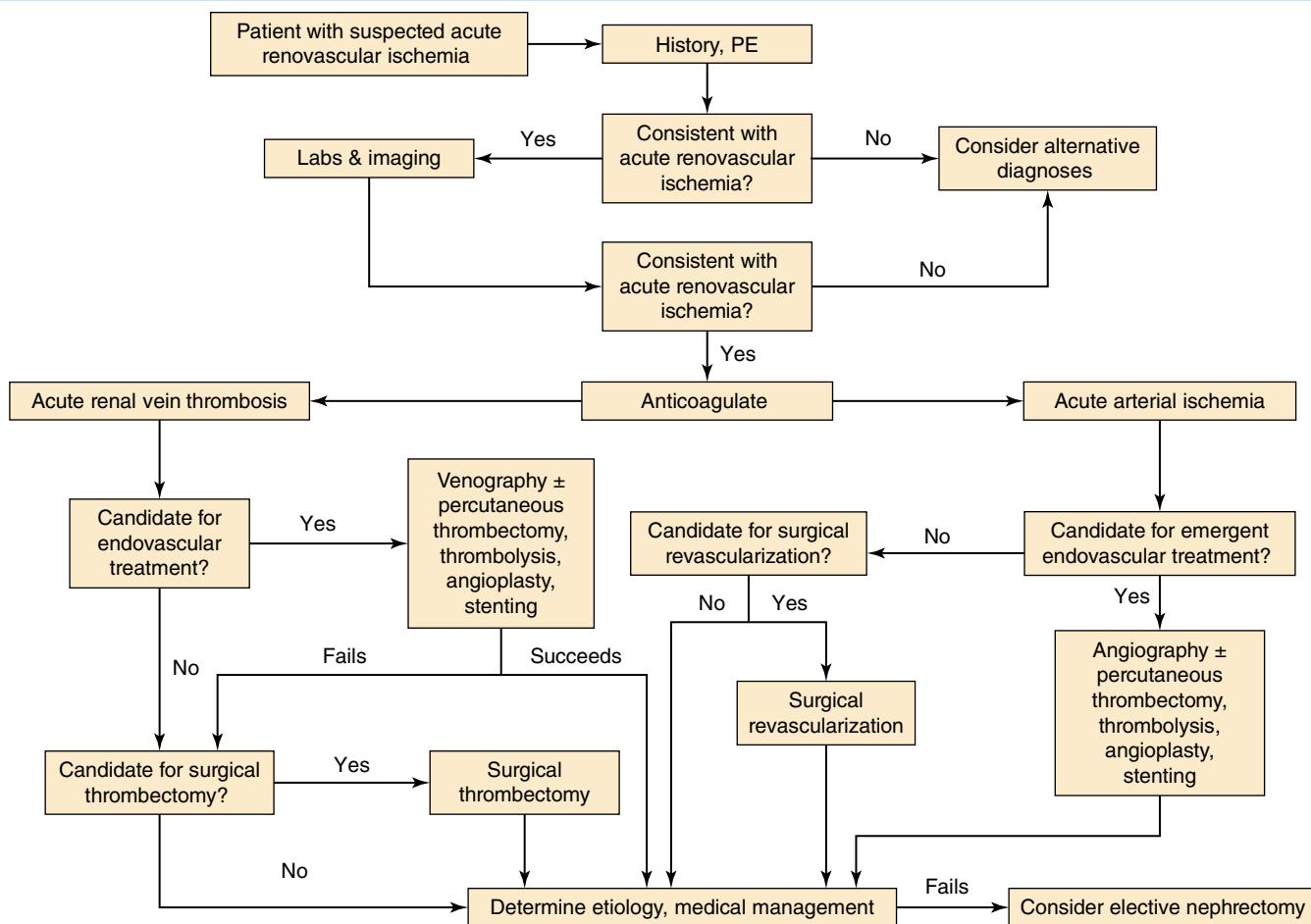
The overall incidence of recurrent renal vein thrombosis is 1% per patient year of follow-up. Survival following renal vein thrombosis is inferior to that of individuals with lower extremity deep vein thrombosis.¹¹⁹ Laville et al. reported a 6-month mortality rate of approximately 40% following diagnosis of acute renal vein thrombosis.¹²⁰ Malignancy and infection negatively affect survival, whereas warfarin therapy is associated with a survival advantage.¹¹⁹

Anticoagulation is also definitive treatment for renal vein thrombosis in neonates. Zigman et al. observed long-term renal functional impairment in one third of patients treated with heparin anticoagulation versus 100% of those who were not anticoagulated.⁴⁵ No patients in this series required dialysis or surgical intervention for renal salvage.

Thrombectomy/Thrombolysis

Use of thrombectomy or thrombolysis in the setting of acute renal vein thrombosis should be reserved for select situations, including failure or complication of oral anticoagulation (such as thrombus propagation or pulmonary embolism), bilateral thrombosis or thrombosis of a solitary kidney, associated caval thrombosis, acute renal failure, or persistent severe symptoms (most commonly flank pain).^{34,121,122} Thrombectomy should be considered in the setting of a contraindication to anticoagulation, failure of thrombolysis, or when nephrectomy for an associated renal malignancy is anticipated. Nephrectomy has a limited role but may be the treatment of choice in the setting of postinfarction hemorrhage.

CHAPTER ALGORITHM



SELECTED KEY REFERENCES

Asghar M, Ahmed K, Shah SS, et al. Renal vein thrombosis. *Eur J Vasc Endovasc Surg*. 2007;34(2):217–223.

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Hazanov N, Somin M, Attali M, et al. Acute renal embolism. Forty-four cases of renal infarction in patients with atrial fibrillation. *Medicine (Baltimore)*. 2004;83:292–299.

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A complete reference list can be found online at www.expertconsult.com.

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Renovascular Disease: Aneurysms and Arteriovenous Fistulae

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RENAL ARTERY ANEURYSMS

Epidemiology

Few vascular surgeons have extensive experience with the clinical management of renal artery aneurysms.^{1,2} Autopsy studies have revealed an incidence of 0.01% to 0.09%, which is probably an underestimation because renal artery aneurysms may be small, intrarenal, or not specifically sought.^{1,3} In two catheter-based arteriographic studies, renal artery aneurysms were documented in 0.73% (7/965) to 0.97% (83/8525) of arteriograms.^{4,5} We documented a lower incidence of asymptomatic renal artery aneurysm of only 0.12% (1/845) in a series of abdominal aortograms performed at Pennsylvania Hospital (Philadelphia, PA).⁶ In a series of computed tomographic arteriography (CTA) scans, the incidence was 0.7% (6/862).⁷

Renal artery aneurysms are bilateral in about 10% of cases.^{1,5} If fibrodysplastic cases are omitted, there is an equal incidence in males and females.^{1,2,8}

Pathogenesis

Types of renal artery aneurysms include true (saccular and fusiform), false, dissecting, and intrarenal aneurysms.

True Aneurysms

More than 90% of true renal artery aneurysms are extraparenchymal.^{2,9–11} The peak incidence is in patients between the ages of 40 and 60 years. Stanley² and Stanley et al.¹¹ have suggested that true aneurysms are probably due to either atherosclerosis or a congenital defect. Although arteriosclerotic changes have

been identified in most aneurysms in patients with multiple lesions, these aneurysms are more likely due to a congenital medial degenerative process with weakness of the elastic lamina.^{8,11} Lesions typically occur at the primary or secondary renal artery bifurcations and are rarely confined only to the main trunk of the renal artery, often making treatment challenging.

Approximately 75% of true renal artery aneurysms are saccular. Saccular aneurysms occur almost invariably at the main renal artery bifurcation.¹² Fusiform aneurysms are usually associated with atherosclerosis or are a result of a poststenotic dilatation distal to a hemodynamically significant renal artery stenosis, the latter of which results from atherosclerosis or fibromuscular disease.^{8,9,12,13} Fusiform aneurysms usually affect the main renal artery trunk, which may be associated with more straightforward endovascular or open surgical intervention.⁹

Arterial fibrodysplasia is often a direct contributor to the development of renal artery aneurysms.^{2,11} Medial fibroplasia is typically associated with multiple stenoses and poststenotic dilatation of the distal two-thirds of the renal artery. Renal artery aneurysms in association with fibromuscular dysplasia are generally only a few millimeters in diameter. The typical angiographic appearance of a renal artery involved with medial fibroplasia is a “string of beads.” In a series of 23 aneurysms that were examined histologically, 16 (70%) were found to be of dysplastic origin.¹⁴ Larger aneurysms can also occur, however, and in one study, renal artery microaneurysms were found in 9.2% of adults with fibromuscular dysplasia.¹¹ A rare cause of renal artery aneurysms is Ehlers–Danlos syndrome. This disorder is associated with extreme arterial fragility and spontaneous rupture.¹⁵

False Aneurysms (Pseudoaneurysms)

False aneurysms of the renal artery arise from blunt or penetrating trauma and occasionally from iatrogenic causes such as renal artery catheterization or after nephrectomy. They represent contained ruptures of the renal artery, with only inflammatory and fibrous tissue encasing the leak.

Dissections

Spontaneous dissections confined to the renal artery that do not arise from the adjacent aorta are rare. However, primary dissections causing pseudoaneurysms affect the renal arteries more than any other peripheral artery.^{2,12,16–19} Poutasse¹² and Stanley et al.¹¹ reported that 14 of 57 cases of renal artery aneurysms were due to spontaneous dissection. An intimal defect of the renal artery due to atherosclerosis is probably the underlying cause of spontaneous renal artery dissection causing aneurysms, along with dysplastic renovascular disease and trauma.⁹ The incidence of dissection in patients with fibrodysplastic renal arteries ranges from 0.5% to 9.0%.^{2,16} Dissection often extends into the branches of the renal artery and may pose particularly challenging reconstruction problems.

Traumatic renal artery dissection can occur secondary to blunt abdominal trauma or catheter-induced injury. Blunt trauma accounts for the higher prevalence of dissection in men

and is more likely to result in right-sided injuries, possibly because of ptosis-related physical stresses affecting the renal pedicle.² Blunt trauma can cause renal artery dissection by either severe stretching of the artery, with fracture of the intima, or compression of the artery against the vertebra. Renal artery dissection caused by guide wires or catheters can occur, but is rare, having been observed in only 4 of 2200 selective renal artery arteriograms.¹⁶

Intrarenal Aneurysms

Less than 10% of renal artery aneurysms are intraparenchymal.^{10,11} Intrarenal aneurysms are usually multiple and may be congenital, are associated with collagen vascular disease, or are posttraumatic. They may be associated with arteriovenous fistulae (AVFs), possibly as a result of spontaneous closure of a fistula. Intrarenal aneurysms can occur with polyarteritis nodosa and are usually in the renal cortex.^{20,21}

Clinical Manifestations and Diagnosis

Most renal artery aneurysms are asymptomatic and are found on imaging studies, such as arteriography, ultrasonography, magnetic resonance angiography (MRA), or CT performed to investigate other intraabdominal pathology.^{2,22,23} Clinical manifestations of renal artery aneurysms include rupture, hypertension, pain, and hematuria.

The most dreaded complication of renal artery aneurysm is rupture. Clinical symptoms include acute abdominal pain and distention and hypotension. Intuitively, a stable renal artery aneurysm should not cause pain or symptoms, similar to a stable abdominal aortic aneurysm, so the clinician should hesitate to attribute chronic abdominal pain to a stable, noninflammatory renal artery aneurysm.

Renal artery aneurysms may be associated with severe hypertension. Macroaneurysms were found in 2.5% of arteriograms performed for the evaluation of hypertension.¹¹ Renal artery aneurysms may cause renovascular hypertension by distal embolization with segmental hypoperfusion, renin-mediated vasoconstriction, and fluid retention. Compression of an adjacent renal artery branch or luminal stenosis due to extensive thrombus may also lead to renin-mediated hypertension. Frequently, significant renal artery stenosis causes a post-stenotic fusiform aneurysm, and the renal artery stenosis is responsible for the hypertension. Saccular and intrarenal aneurysms are much less likely to be associated with hypertension. We urge that caution be exercised in attributing the cause of hypertension to a renal artery aneurysm, especially if intervention is being considered. Just as renal artery stenosis without an aneurysm is currently less likely being treated with intervention and more likely treated with medical management, the clinician should hesitate to recommend endovascular or surgical intervention to treat hypertension potentially due to a renal artery aneurysm.

Patients with renal artery aneurysms caused by dissection may present with severe flank pain, hematuria, or acute hypertension, although most dissections are asymptomatic. CT angiography or MRA is essential to detect dissection.

Intrarenal aneurysms may rupture into calices.³ In addition to pain, microscopic or gross hematuria may occur. Similarly, renal artery aneurysms may rarely cause obstruction of the collecting system.

Indications for Intervention

Indications to repair a renal artery aneurysm are related to the risk of rupture, hypertension, acute dissection, and other clinical symptoms. Because of the lack of controlled data, controversy persists regarding indications for repair of asymptomatic renal artery aneurysms. The optimal method of repair is also controversial.

Rupture and Prevention of Rupture

Rupture of a renal artery aneurysm is an indication for emergency intervention, as it is for virtually any arterial aneurysm. Probably less than 3% of renal artery aneurysms rupture.^{2,11} This complication is associated with a mortality rate of approximately 10% in men and nonpregnant women.^{2,11,24,25} In a hemodynamically stable patient, an emergent CT scan may reveal the pathology and allow the surgeon to plan the operative repair.

Prevention of rupture is the most common indication for intervention in cases of asymptomatic renal artery aneurysms. Traditionally, repair has been recommended for renal artery aneurysms greater than 2 cm in diameter.^{9,13} We believe this recommendation is overly aggressive, and more conservative guidelines for intervention based on more recent studies with longer follow-up have been suggested.

Older series suggested higher rates of rupture. Harrow and Sloane²⁶ reported one of the highest rates of rupture of renal artery aneurysms, noting 14 ruptures in 100 cases. In another series of 126 renal artery aneurysms, six ruptured.²⁷ However, most other series of asymptomatic renal artery aneurysms in men and nonpregnant women report a much lower incidence of rupture. Only 1 of 62 patients with aneurysms 4 cm in diameter or smaller ruptured after follow-up from 1 to 17 years.²⁸ None of 19 small aneurysms in another series ruptured.²⁴ A group of 21 patients was observed for an average of 3 years without rupture.²³ In another series of 18 patients, no renal artery aneurysms ruptured that were less than 2.6 cm who were followed for 1 to 16 years.¹ There were no ruptures in a series of 32 patients (who eventually underwent surgery) with renal artery aneurysms that ranged from 0.7 to 9 cm.¹⁵ Of 83 renal artery aneurysms found on arteriography and followed without surgery,²⁹ none ruptured or became symptomatic after a mean of 4.3 years.⁵ In a pooled analysis, there were no ruptures in more than 200 renal artery aneurysms observed for up to 17 years.⁸ Because of the very low risk of rupture and the relatively high rate of significant postoperative complications, especially with open surgery, we agree with Coleman and Stanley that a more conservative approach be used for these cases and that a 2-cm guideline for intervention is too aggressive and unwarranted.³⁰ We also agree with the recommendations of the Vascular Low-Frequency Disease Consortium that identified 865 renal artery aneurysms in 760 patients at 16 institutions.³¹ The

authors recommended that repair should be considered for asymptomatic renal artery aneurysms >3 cm diameter in men and in women of non-childbearing age.

Besides size, other factors may play a role in the consideration of elective surgery for asymptomatic renal artery aneurysms. Calcification of the aneurysm has been thought to protect against rupture. Poutasse¹³ suggested that a heavily calcified renal artery aneurysm may be less likely to rupture than a noncalcified or minimally calcified one. In a review of cases through 1959, 14 of 100 noncalcified aneurysms ruptured.²⁶ In a more recent series, 15 of 18 ruptured renal artery aneurysms were noncalcified.³² However, in a series of 62 solitary aneurysms less than 4 cm in diameter, one-third were not calcified, and only one aneurysm in the entire series ruptured after 1 to 17 years of follow-up.²⁵ Because of these conflicting data, some authorities believe that the presence or absence of calcification is not relevant when predicting the risk of rupture.² The Vascular Low-Frequency Disease Consortium found that calcification does not protect against enlargement or rupture of renal artery aneurysms.³¹

Most authorities agree that pregnancy is associated with a significantly increased risk of rupture for renal artery aneurysms.^{2,11,22,33} Pregnancy may increase the risk of rupture because of a hyperdynamic state with increased blood volume and cardiac output, hormonal influences, and increased intraabdominal pressure due to the gravid uterus.^{1,11} Cohen and Shamash³³ reported 18 cases of rupture during pregnancy. In another series of 18 patients who underwent surgery for renal artery aneurysms, the only two ruptures were in women at childbirth; both of these aneurysms measured only 1 cm in diameter.¹ In a review of 43 ruptured renal artery aneurysms, 81% occurred in women; 21 of the 35 women in this series were younger than 40 years old, and 18 were pregnant. Of the 18 aneurysms of known size, three ruptured when they were less than 2 cm.³⁴ Rupture of renal artery aneurysms in pregnancy has been associated with a maternal mortality rate of 55% and a fetal death rate of 85%.^{33,35} Vascular surgeons should maintain an aggressive surgical or endovascular approach for pregnant women with renal artery aneurysms of any size, namely more than 1.5 times the diameter of the normal adjacent artery.

It seems prudent to recommend repair of renal artery aneurysms in good-risk men and women older than childbearing age when the diameter is at least greater than 3 cm in good-risk patients when there is reasonable certainty that nephrectomy will not be required.^{26,29} In a recent review by the Vascular Low-Frequency Consortium of 865 renal artery aneurysms from 16 institutions, the authors concluded that these aneurysms rarely rupture when asymptomatic (even if >2 cm), major complications occurred in 10% of patients postoperatively, and recommended repair for renal aneurysms greater than 3.0 cm in asymptomatic patients of non-childbearing age.³¹ An even more conservative approach reserving repair for aneurysms greater than 4 cm has been suggested; in our opinion, this is not unreasonable.³⁶ As previously mentioned, any renal artery aneurysm in women of childbearing age should be repaired – namely, more than 1.5 times the diameter of the normal adjacent artery.

Hypertension

Although the prevalence of hypertension in patients with renal artery aneurysms is approximately 80%, there is no conclusive evidence that the aneurysms themselves are the direct cause of hypertension, unless there is an associated stenosis or compression of an adjacent artery.^{1,11,22} In a series of 39 patients with renal artery aneurysms, 26 had diastolic hypertension, but only nine (23%) patients had hypertension that was of renovascular origin.¹ The indication for intervention for renovascular hypertension due to renal artery stenosis continues to be failure of medical management. The same criterion should be applied when a renal artery aneurysm is present (if large size alone is not otherwise an indication for repair). If the primary indication to repair a renal artery aneurysm is poorly controlled hypertension, a nephrology consult should be routinely obtained to be sure there are firm indications for intervention for this reason. Both the stenotic artery and the aneurysm should be repaired.

Dissection

Emergent intervention is required for dissections that cause renal artery aneurysms and threaten the viability of the kidney. Nephrectomy is frequently required because of the extensive damage to the renal branch vessels and the limited time available to salvage a previously healthy kidney that cannot tolerate prolonged periods of ischemia. If hypertension is the only manifestation of a chronic dissection and the hypertension is well controlled by blood pressure medications, or if the patient is asymptomatic and a renal artery dissection is found incidentally (without an associated aneurysm), endovascular or surgical repair is not justified.¹¹

Other Clinical Manifestations

If a patient with an intact renal artery aneurysm as documented by CT or magnetic resonance imaging (MRI) is symptomatic, repair may be indicated. Acute onset of symptoms is more worrisome than a patient referred for months of abdominal pain or fullness, unless the renal artery aneurysm is very large. Symptoms may be a harbinger of impending rupture. Embolization of mural thrombus from the aneurysm to the renal parenchyma may also account for these symptoms.¹⁰ Nonetheless, it is difficult to attribute chronic abdominal pain to an intact 2-cm diameter renal artery aneurysm. Other causes need to be thoroughly investigated.

Treatment: Medical, Open Surgical, and Endovascular

Repair of a Ruptured Renal Artery Aneurysm

If emergent surgery is required for a ruptured renal artery aneurysm, a sizable juxtarenal hematoma may not allow safe exposure of the proximal renal artery or even of the suprarenal aorta for clamping to obtain proximal control. Therefore, in many cases, a midline approach and supraceliac aortic control may be temporarily required. If the bleeding is quickly controlled, the patient is hemodynamically stable, and the proximal and distal renal arteries lend themselves to a relatively quick and

straightforward bypass, consideration can be given to reconstruction. In most cases, however, nephrectomy is required because of the instability of the patient or the limited warm ischemic time of the kidney (less than 30–60 minutes in healthy patients). The prolonged ischemia of the kidney and the technical and time-consuming nature of surgical repair with a bypass generally result in a nonsalvageable kidney.^{2,11,12,28} If the aneurysm extends into the renal parenchyma or if a “bench” repair of the kidney is required, the patient is generally best treated by nephrectomy, as long as the contralateral kidney is intact with normal function.

A stable patient with a ruptured renal artery aneurysm may be treated with endovascular techniques. The use of a stent graft may be the preferred treatment for lesions not involving the distal renal branches, as reported by Bloemsma et al.,³⁷ and Routh et al.³⁸ Others have reported thrombosis of a leaking saccular aneurysm using Gianturco coils, thrombin, and buccylate.^{39,40}

Elective Repair of Renal Artery Aneurysm

Open surgical

Open repair of a renal artery aneurysm is usually more challenging than revascularization for renal artery stenosis. Most renal artery aneurysms extend past the bifurcation of the main renal artery and frequently extend into the renal parenchyma. For *in situ* repairs of a renal artery aneurysm, the left kidney can be exposed through a left retroperitoneal or midline transperitoneal approach. The right kidney can be exposed through a midline transperitoneal approach with a Kocher maneuver to reflect the right colon and duodenum medially or with a right subcostal incision (see Ch. 56, Abdominal Vascular Exposures).

Several methods have been used to repair renal artery aneurysms. The most straightforward technique for saccular aneurysms involves aneurysmorrhaphy with primary repair or patching. In three combined series of patients who underwent surgical repair of renal artery aneurysms, approximately one-third (6/18, 3/10, and 6/23) of the aneurysms were able to be repaired in this manner.^{1,10,22} A common renal arterial reconstruction is an end-to-side anastomosis of a small renal artery branch to the main renal artery or a side-to-side anastomosis of two small renal arteries to create a common inflow channel with a single, larger diameter lumen, which can then be anastomosed to the more proximal renal artery (or to the distal end of a bypass).³⁰ Because the small branches of the main renal artery are often involved with the aneurysm, a branched autologous graft is preferred to reconstruct these lesions. The internal iliac artery is an excellent choice in these reconstructions because of its multiple small side branches, and its durability in young patients.^{29,41} Alternatively, the saphenous vein may be used as conduit in adults.^{9,22} The proximal anastomosis of the graft is usually the infrarenal aorta. Useful alternative reconstructions include a splenorenal bypass for a left-sided renal artery aneurysm and hepatorenal bypass for a right-sided aneurysm.

If multiple branch vessels are involved and renal ischemic time is expected to exceed 30 to 60 minutes, and especially if the cause of the renal artery aneurysm is dissection resulting

in a friable vessel, extracorporeal or bench surgery may be required.^{42,43} *Ex vivo* surgery requires nephrectomy, followed by hypothermic perfusion of the kidney with a heparinized renal preservation solution. The kidney can then be auto-transplanted to its original bed, as Dean et al.⁴⁴ and Crutchley et al.⁴⁵ prefer, or to the iliac fossa. For renal autotransplantation into the iliac fossa, a flank incision with a retroperitoneal approach is used for exposure of the kidney, ureter, and iliac artery. Gonadal and adrenal veins are divided to obtain an adequate length of renal vein. If the reconstruction can be safely performed by placing the kidney on the anterior abdominal wall, the ureter does not need to be divided. The procedure is occasionally best performed at a separate table after dividing the ureter and removing the kidney from the operative field. Perfusion is carried out through the main renal artery to preserve the kidney, while selected branches are individually repaired and other branches are perfused. The kidney may be perfused with a heparinized crystalloid solution, such as Collins solution or lactated Ringler's solution with heparin 1000 U/L with 12.5 g of mannitol, while the kidney is wrapped with gauze and placed in a chilled solution at 4°C.^{9,38,46} The use of continuous pulsatile perfusion is controversial.³⁸

When performed for proper indications by well-trained vascular surgeons, elective repair of renal artery aneurysms has been associated with very low mortality.^{2,10,46,47} English et al.⁴⁸ reported a 1.7% perioperative mortality rate in 62 patients with 72 renal artery aneurysms with a 4-year patency of 96% and cured or improved hypertension in three quarters of patients. Pfeiffer et al.,⁴⁹ from Germany, reported similar excellent long-term surgical results in a series of 94 patients. A series of 26 renal artery aneurysms that required reconstruction of first- or second-order branches were repaired primarily with *in situ* techniques. Autogenous vein bypasses resulted in a mortality of 0%, with long-term patency of 95% at an average follow-up of 99 months (range, 1–300 months).⁵⁰ A recent small series of 4 patients with renal artery aneurysms involving distal renal artery branches treated *in situ* using a "Y-shaped" pantaloon greater saphenous vein graft created on-bench showed 100% patency at 64 months.⁵¹ *Ex vivo* repairs have been shown to be safe and effective by Dean et al.,⁴⁴ Crutchley et al.,⁴⁵ and others.⁴² In a review of *ex vivo* repairs, postoperative mortality rates ranged from 0% to 9.6%.⁵² A recent series of 26 renal artery aneurysms involving 20 kidneys in 19 patients were treated between 2009 and 2018 by *in situ* open repair in 16 (80%) cases with no mortality.¹⁴ The primary and secondary patency rates were 90% and 95% after a mean of 54 months. The use of bifurcated internal iliac artery autografts was also highly successful in a series of 11 patients, most with fibrodysplastic aneurysms who were treated by *in situ* or bench repair.⁴¹

Recent technologic and surgical developments have allowed less invasive surgical repair of complex renal artery aneurysms, especially those involving the hilum. Both laparoscopic and robot-assisted techniques have been described.^{53–55}

Endovascular

An alternative approach to the treatment of renal artery aneurysms includes the use of endovascular techniques.^{29,39,56,57}

Degenerative renal artery aneurysms have been treated with transcatheter embolization with detachable platinum coils that occlude the aneurysms but maintain renal flow.^{39,57,58} In a small series of endovascular repairs, Klein et al.³⁹ treated 12 renal artery aneurysms using selective endovascular embolization with nondetachable microcoils or Guglielmi detachable coils. In another series, 13 patients with renal artery aneurysms were successfully treated using a variety of endovascular techniques with combinations of coil embolization, liquid embolization, stenting, and stent grafts, with no complications after an average follow-up of 43 months (range, 13–103 months).⁵⁹ A series of 18 renal artery aneurysms were also treated with endovascular intervention.⁶⁰ Sixteen of the 18 aneurysms were embolized utilizing adjunctive techniques to protect embolizing into the main renal artery. There was a 100% technical success rate, with only one aneurysm showing residual minor flow into the sac and with no long-term complications. In a recent series from China of 17 patients treated for 26 renal artery aneurysms between 2009 and 2016, 12 aneurysms were treated with simple coil embolization and the remaining cases underwent other endovascular techniques without major complications.⁶¹ Alternatively, several authors have recently reported using ethylene vinyl alcohol copolymer to ablate renal artery aneurysms.^{40,62,63}

Endovascular stent graft exclusion as a definitive treatment for renal artery aneurysms has been widely reported. Many aneurysms extend to branch vessels, making this approach potentially risky or ill-advised, but it may be ideal for aneurysms of the main renal artery not involving branches. Advances in stent-graft technology, with smaller diameter devices and lower profile delivery systems, have made definitive treatment of renal artery aneurysms feasible. A review of the English literature analyzing the use of stent grafts to treat renal artery aneurysms up to 2010 yielded 22 cases with a technical success rate of 91% (20 cases), without any deaths or significant morbidity reported.⁶⁴ Although most published series have been small, results have been excellent, and this approach will undoubtedly become more common as technology improves.^{65–69}

A nationwide inpatient sample of 6234 isolated renal artery aneurysms from 1998 to 2011 revealed an increase in endovascular repairs without a decrease in open repairs.⁷⁰ Although the in-hospital mortality was low for both endovascular intervention (1.8%) and open repair (0.9%), complication rates were greater than 10% for both. The authors concluded that indications for repair of renal artery aneurysms should be re-evaluated and possibly be less aggressive.

Fibromuscular Dysplasia

Post-stenotic dilatation causing hypertension resulting from fibromuscular disease can be treated by balloon angioplasty of the stenotic lesion. When the lesion extends into the branches of the main renal artery, surgery can yield excellent results. Dean et al.⁴⁴ reported 24 patients with fibromuscular disease, many of whom had branch aneurysms; all but one did well.

Intrarenal Aneurysms

Intrarenal aneurysms represent particularly challenging lesions. Frequently, a partial nephrectomy is required.⁵² Intrarenal aneurysms in association with polyarteritis nodosa have also been successfully treated with renal artery embolization, with preservation of the kidney.⁷¹

See the Chapter Algorithms for management of renal artery aneurysms.

RENAL ARTERIOVENOUS MALFORMATIONS AND FISTULAE

Arteriovenous malformations (AVMs) and AVFs are uncommon lesions that can be associated with hematuria, hypertension, renal dysfunction, high-output congestive heart failure, and even rupture. More than 200 cases have been reported since the first description in 1928.⁷² Fistulae may be congenital or acquired. Multiple diagnostic modalities are now available, although conventional selective arteriography remains critical in therapeutic interventions. Many asymptomatic lesions do not require treatment. In the past, symptomatic lesions were treated surgically, but endovascular treatment has now supplanted surgery in most cases.

Epidemiology and Pathogenesis

Congenital Arteriovenous Malformations

True congenital AVMs of the kidney are rare, with an incidence of only 0.04%.^{73,74}

In a large series, only one congenital AVM was noted in 30,000 autopsies.⁷⁵ These lesions represent approximately one-fourth of all renal AVFs.^{76,77} The right kidney is involved more often than the left, and although multiple lesions can occur, a single focus is more common.⁴² The angiographic appearance of the lesions is similar to that of AVMs elsewhere, with large coils of dilated vessels. An early “blush” is noted and correlates with the degree of arteriovenous shunting.

These lesions have been described as cirsoid or varix-like, and are generally focal and located in the renal medulla. AVMs are not neoplastic, but enlargement presumably can occur because of vessel dilatation and hypertrophy associated with high-flow volume from arteriovenous shunting. Symptomatic AVMs have been reported in pregnancy,^{72,78} and it is thought that the hyperdynamic state of the gravida leads to increased AVM flow and symptoms. Histologically, involved vessels have irregular fibrosis or intimal hyperplasia, as well as medial hypertrophy.

Acquired Arteriovenous Fistulae

Acquired AVFs may occur spontaneously. Spontaneous AVFs have been documented in association with fibromuscular dysplasia⁷⁹ and are thought to develop when a dysplastic or aneurysmal renal artery erodes into a neighboring vein.⁸⁰ This may also occur with renal malignancy, and significant arteriovenous shunting is a hallmark of renal cell carcinoma.⁸¹ With arteriography, it can be difficult to differentiate a renal malignancy from a congenital or acquired AVF, although CT and MRI

generally reveal a mass distinct from the renal parenchyma in malignancy.

Traumatic AVFs are the most common lesions, accounting for more than 70% of all renal AVFs.⁷² These lesions may occur after nephrectomy, and are related to the erosion of the arterial stump into the vein with mass ligature,^{75,82,83} after renal artery angioplasty,⁷² after blunt⁸⁴ or penetrating⁸² trauma, after nephrostomy,⁸⁴ and most commonly, after percutaneous renal biopsy (Fig. 131.1). With the routine use of needle biopsy for the diagnosis of rejection in renal allografts, the incidence of acquired AVFs has grown, although only 1% to 2% of patients who undergo needle biopsy develop symptomatic AVFs.^{85,86} However, the true incidence of AVF is 15% to 18% when arteriography is routinely used.^{87,88} Similarly, Ozbek et al.⁸⁹ found AVFs in 8 of 64 patients (12.5%) monitored by color duplex ultrasonography, whereas only 5% developed AVFs in the study of Rollino et al.⁹⁰ In the prospective study of Merkus et al.,⁸⁷ who used routine color duplex surveillance, 10% of patients who underwent biopsy developed AVFs.

Cho et al.⁹¹ proposed a classification for AV malformations for nontraumatic lesions. Type I is defined as no more than a few arteries into a single draining vein. Type II describes multiple arterioles shunted into a single draining vein. In type III there are multiple connections between arterioles and venules, forming a complex vascular network. Type III can be further subdivided into those without venous dilatation (IIIa), and cirsoid (IIIb). This classification scheme is depicted in Table 131.1.

Clinical Presentation

The majority of both congenital and acquired AVFs do not produce clinical symptoms, and many lesions are noted incidentally in studies done for other reasons. The most common symptom of congenital AVM is hematuria, which occurs in 72% of cases.⁷⁵ Hematuria occurs when subepithelial varices erode transitional epithelium into the collecting system. A dramatic presentation with massive hematuria can occur,^{78,92,93} although minor or microscopic hematuria is more common. Hypertension occurs in congenital AVMs and is also the primary abnormality in most acquired AVFs that are described as symptomatic. The hypertension is renin-mediated, based on diminished glomerular filtration pressure distal to the fistula because of arterial “steal.”^{77,94,95}

Although AVFs are generally painless, intermittent periumbar discomfort has been reported in some patients.^{81,83} This discomfort is generally associated with hematuria and may represent as renal colic. In addition, dyspnea and other symptoms of congestive heart failure may be the primary complaint in some patients; this is more common with acquired lesions, and only with those having a large communication between the artery and vein. This “high-output” type of heart failure manifests as tachycardia, left ventricular hypertrophy, and cardiomegaly, and a palpable thrill in the flank. A continuous abdominal bruit is a hallmark of acquired AVFs and is frequently noted with congenital AVMs as well. Retroperitoneal or intraabdominal hemorrhage rarely occurs with AVMs and

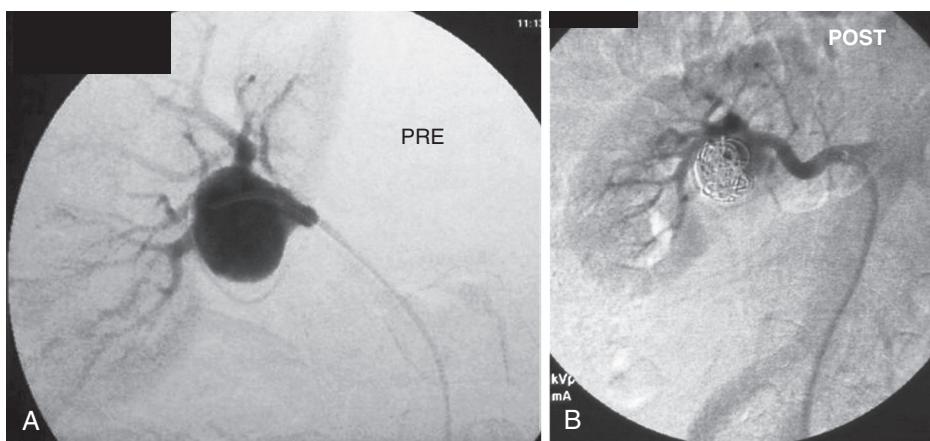


Figure 131.1 (A) Arteriogram showing a post-traumatic arteriovenous fistula. This patient experienced a stab wound to the flank and presented with hematuria. (B) After Gianturco coil embolization of multiple arterial branches, venous communication is no longer present. Hematuria resolved, and the patient recovered uneventfully.

TABLE 131.1

Cho Classification Scheme for Renal Arteriovenous Fistulae

Cho Classification	Arterial	Venous
Type I	one or few arteries	single draining vein
Type II	multiple arterioles	single draining vein
Type IIIa	multiple arterioles	multiple venules (no dilatation)
Type IIIb	multiple arterioles	multiple venules (+ dilatation – cirloid)

From Cho SK, Do YS, Shin SW, et al. Arteriovenous malformations of the body and extremities: analysis of therapeutic outcomes and approaches according to a modified angiographic classification. *J Endovasc Ther.* 2006;13(4):527–538.

AVFs.^{92,96} However, patients with rupture present with severe abdominal and flank pain and shock, a clinical picture indistinguishable from ruptured abdominal aortic aneurysms.

Diagnosis

Excretory urography is performed in many patients presenting with hematuria or flank pain. A filling defect may be noted in the kidney, and dilated vessels can compress the collecting system, although these findings are not specific.

CT can usually define AVFs and AVMs within the kidney, but it is not always possible to differentiate these lesions from other hypervascular abnormalities (e.g., renal cell carcinoma). Similarly, radionuclide imaging can demonstrate early augmented perfusion, but differentiation from malignancy is not possible.⁹⁷ In contrast, CT angiography has significantly improved the noninvasive imaging of AVFs and AVMs.⁹⁸ Likewise, contrast-enhanced MRA allows three-dimensional reconstruction that can provide visualization not possible with conventional angiography.⁹⁹

Color duplex imaging is also of growing importance in the diagnosis of AVMs and AVFs. Because it is inexpensive and noninvasive, it is the ideal study for screening purposes. Color duplex imaging has been used liberally to assess for AVFs after

percutaneous renal biopsy.^{87,100,101} Marked turbulence is noted on color examination, and Doppler spectral analysis reveals an elevation of peak systolic flow velocity and a larger increase in end-diastolic flow velocity compared with the normal renal artery, with a resultant low resistive index.^{89,100}

In the past, contrast arteriography was the definitive diagnostic modality for renal AVMs and AVFs. Rapid opacification of the inferior vena cava is noted. Depending on the size of the fistula, the nephrogram may be diminished distal to the AVF. With congenital AVMs, multiple segmental and interlobar arteries communicate with varix-like veins, whereas a single arterial communication is generally present with acquired AVFs.⁷⁵

Treatment: Medical, Open Surgical, and Endovascular

The majority of both congenital and acquired AVFs do not cause symptoms and do not require treatment. However, patients may become symptomatic, and even if asymptomatic, should be closely observed for the development of hypertension, hematuria, or high-output cardiac failure. Most AVFs occurring after percutaneous renal biopsy close spontaneously.^{87,89,94,101,102} Periodic duplex surveillance, along with clinical follow-up for the development of hypertension or renal insufficiency, is indicated. If a postbiopsy AVF persists at 1 year, it is not likely to close spontaneously,⁸⁵ although intervention should still be delayed until the development of symptoms.^{87,95} Spontaneous regression of AVMs unrelated to trauma has also been reported.^{103,104} Although hypertension related to an AVF may be readily controlled with angiotensin-converting enzyme inhibitors,⁷⁹ the long-term effect on renal function is not known. In most published reports, patients with hypertension have undergone surgical or endovascular therapy; thus the natural history of medically treated patients with hypertension secondary to AVFs remains undefined.

Open Surgical

For patients with symptomatic AVFs, surgery remains an option, although being performed less frequently today.^{105–107}

Except for very peripheral lesions, a transperitoneal approach is preferred to establish proximal arterial and venous control at the renal pedicle. Because of the frequent presence of thin-walled dilated veins and channels, surgery can be challenging. With surgery, ligation of the feeding vessel or vessels alone is often not possible, and partial or total nephrectomy is often required. The resultant loss of functional renal mass, as well as the morbidity of the operation itself, makes endovascular treatment an attractive approach.

Endovascular

There are now more than 3 decades of experience with percutaneous arterial embolization therapy for congenital and acquired AVFs.¹⁰⁸ Because renal arteries are “end arteries,” they are especially amenable to therapeutic occlusion using gelatins, glues, alcohols, silicon, steel and platinum coils, and detachable balloons.^{25,76,96,101,109,110} The development of co-axial catheter systems has allowed highly selective embolization, which can preserve renal function; loss of functional renal parenchyma is reportedly between 0% and 30% with modern techniques. In general, smaller AVFs are treated with glues or macroparticles, whereas coils and balloons are used for larger vessel fistulae (see Fig. 131.1).^{111,112} Because Gelfoam (Pfizer, New York) and autologous clot resorb, recanalization with recurrence of symptoms can occur in up to 50% of cases.^{113,114} For this reason, and because of the perception that microcoils are associated with less indiscriminate embolization than glues and alcohols, recent trends favor the use of microcoils even for smaller AVFs and AVMs^{25,96,101}; however, the success rate has been reported to be higher if liquid agents are used in addition to coils.¹¹⁵

For very large AVF, covered stent exclusion has been described,¹¹⁶ and others have utilized bare metal stents to provide

a barrier to coil extrusion.¹¹⁷ Radiofrequency ablation has recently been described as well.¹¹⁸

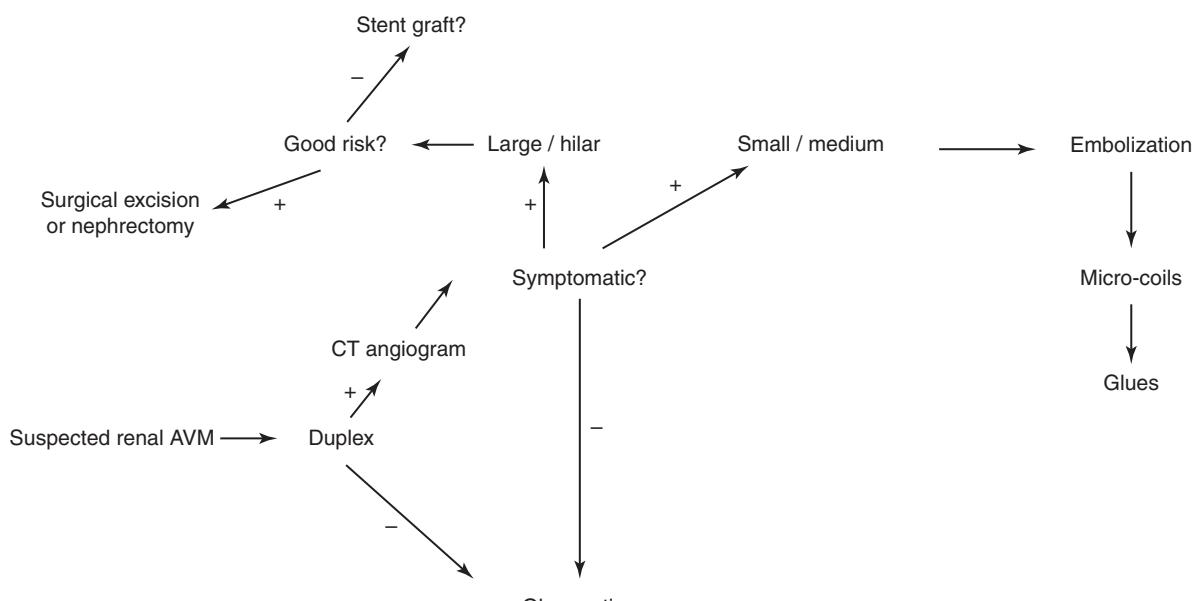
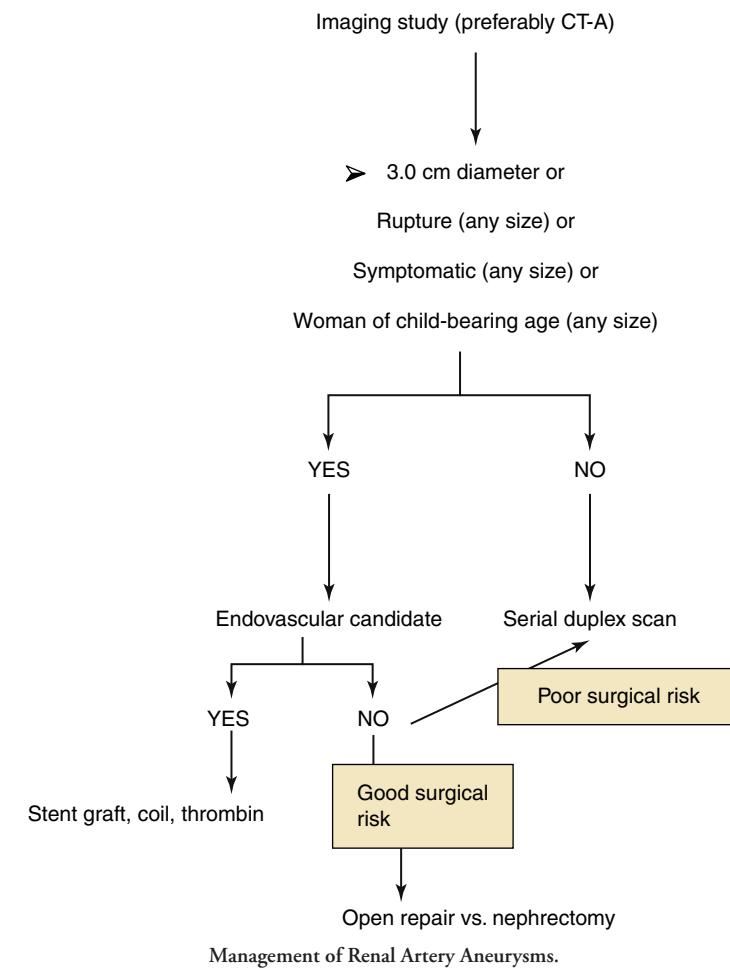
Very large AVFs may present a technical challenge because of the risk of central embolization. Some authors recommend surgery in this setting.^{105,106,119,120} Others have reported success in this setting using the Amplatz spider device (Cook Medical, Bloomington, IN) to provide a scaffolding that can then engage other embolic materials.^{76,82} Staging the procedure – beginning with large coils, followed weeks later by smaller coils and other materials to close off persistent flow channels – may also be an effective strategy.¹²¹ Because very large arteriovenous communications tend to be at the renal pedicle rather than intraparenchymal, surgical treatment is feasible and probably preferable for good-risk patients. In the case of surgical therapy, *ex vivo* techniques may be of value.

Complications of embolization are unusual but not insignificant. In addition to arterial access site morbidity and contrast agent toxicity, pulmonary or peripheral arterial embolization can occur. Large AVFs require large devices such as coils or detachable balloons, but even these can embolize centrally. Gelfoam, alcohol, and various glues may be more appropriate for very small communications, but the delivery is less precise, and renal parenchymal infarction seems to be greater with these materials.⁹⁴ More recently, adjunctive balloon occlusion of inflow and outflow vessels has been reported with the use of n-butyl-2-cyanoacrylate embolization.¹²²

It is common for patients to have transient fever, leukocytosis, and even hypertension after embolization.⁸⁸ With modern techniques, endovascular treatment is successful in more than 80% of patients, although repeat treatments are sometimes necessary^{123,124} and is clearly the treatment of choice for symptomatic congenital and most acquired AVFs.

See Chapter Algorithms for management of renal arteriovenous fistulae.

CHAPTER ALGORITHMS



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Renovascular and Aortic Developmental Disorders

DAWN M. COLEMAN, JONATHAN L. ELIASON, and JAMES C. STANLEY

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INTRODUCTION

Developmental renal artery and abdominal aortic disease is complex and the underlying pathogenesis in the majority of cases is poorly understood. Nevertheless, certain contributing factors are well recognized and the treatment of most lesions has become better defined in the past few decades.^{1–6} The majority of children will benefit from appropriately planned and performed interventions, either endovascular or open surgical procedures, when undertaken in properly selected individuals.

DEVELOPMENTAL ABDOMINAL AORTIC COARCTATION

Classification

Abdominal aortic coarctation was first described by Quain in 1847.⁷ It was considered an oddity until it was more frequently recognized as aortic imaging evolved in the latter years of the 20th century. In recent decades it has become known as the middle aortic syndrome (MAS) or midaortic syndrome, and its anatomic character became important in clinical practice. A contemporary classification of developmental abdominal aortic

coarctation is based on the most cephalad extent of the affected aorta. This distinguishes suprarenal (69%) from intrarenal (23%) and infrarenal (8%) narrowing (Figs. 132.1–132.3).⁵ These coarctations occur as limited focal or segmental narrowing and are to be differentiated from diffuse aortic hypoplasia, which is often considered separately.^{5,8}

Embryologic Factors

Overfusion of the two dorsal aortas during the fourth week of gestation is thought to be the cause of most developmental abdominal aortic coarctations. Prior to this event, both dorsal aortas normally extend along the entire length of the fetus, with each segment of the paired aortas containing dorsal intersegmental, lateral segmental, and ventral segmental branches. As the two dorsal aortas fuse, these branches persist, involute, or join together to form the named aortic branches found at birth.

In support of the overfusion theory is the loss of paired lumbar arteries within the narrowed aortic segment, with only a single lumbar artery trunk exiting the dorsal aorta.^{5,9} A developmental etiology for aortic hypoplasia is also supported by the frequent presence of multiple renal arteries to one or both kidneys

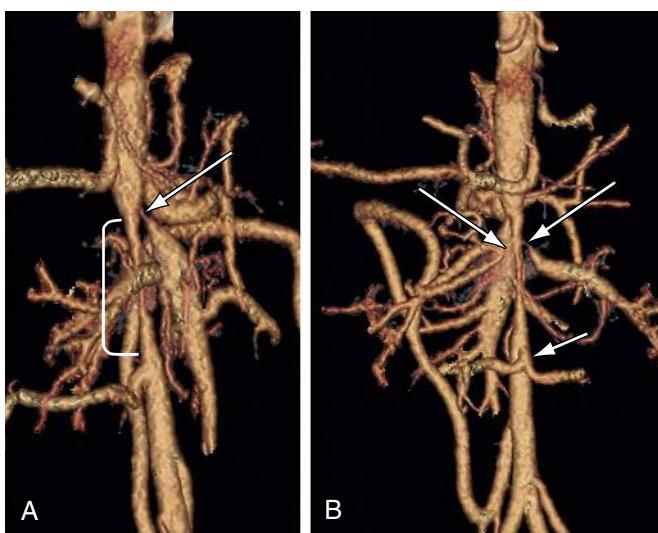


Figure 132.1 (A) Suprarenal abdominal aortic coarctation (bracket) with superior mesenteric artery stenosis (arrow). (B) Bilateral renal artery stenoses (arrows). Note common trunk of lower lumbar artery (arrow) on posterior projection. (From Stanley JC, Criado E, Eliason JL, et al. Abdominal aortic coarctation: surgical treatment of 53 patients with a thoracoabdominal bypass, patch aortoplasty, or interposition aortoaortic graft. *J Vasc Surg*. 2008;48:1073–1082.)

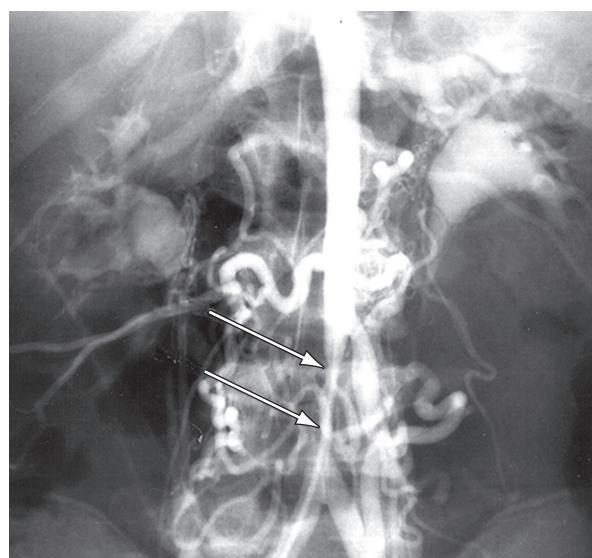


Figure 132.3 Infrarenal abdominal aortic coarctation manifest by tubular stenosis extending from a dilated inferior mesenteric artery to the aortic bifurcation (arrows). (From Stanley JC, Eliason JL. Pediatric arterial disease. In: Coran AG, Caldamone A, Adzick NS, et al., eds. *Pediatric Surgery*, 7th ed. Philadelphia: Elsevier Saunders; 2012:1631–1645.)

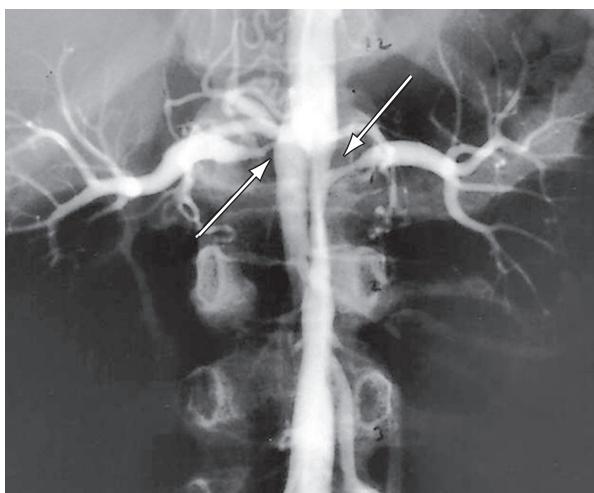


Figure 132.2 Intrarenal abdominal aortic coarctation with bilateral artery stenosis (arrows). (From Stanley JC, Criado E, Eliason JL, et al. Abdominal aortic coarctation: surgical treatment of 53 patients with a thoracoabdominal bypass, patch aortoplasty, or interposition aortoaortic graft. *J Vasc Surg*. 2008;48:1073–1082.)

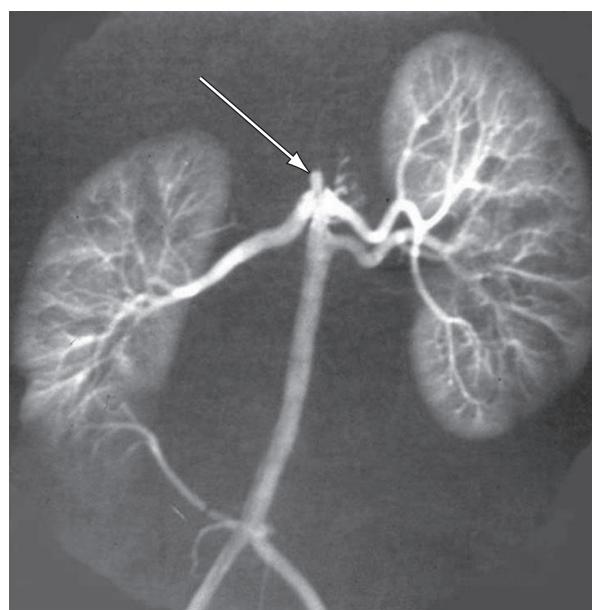


Figure 132.4 Multiple bilateral renal arteries in a patient with a near-occlusive coarctation of the suprarenal aorta (arrow).

compared to the general population.^{5,10,11} It is known that aortic development occurs concurrent with the involution of multiple metanephric arteries to leave a single renal artery to each kidney in the majority of fetuses. It is plausible that developmental aortic coarctations cause flow disturbances near what would usually be the principal renal artery; this diminishes its hemodynamic advantage and allows adjacent metanephric channels to persist as multiple polar or accessory renal arteries (Fig. 132.4).

Genetic, Inflammatory, and Infectious Factors

Certain genetic syndromes have been associated with abdominal aortic coarctations. The most frequent is neurofibromatosis-1 (NF-1).^{5,11,12–16} Others include Williams syndrome,^{17–19}

Alagille syndrome,^{20,21} and tuberous sclerosis.^{22,23} It is theorized that each of these diseases inhibit cell growth or cause cellular death of vascular tissues during fetal development.

The NF-1 phenotype is highly variable, and a wide spectrum of vascular involvement exists, including aneurysms, arteriovenous malformations, cardiac valvular anomalies, and direct neural tumor invasion or compression of vascular structures. However, hypertension due to renal artery stenotic disease or abdominal aortic narrowing is the most common vascular manifestation of NF-1.^{24–27} It is difficult to find a unifying cause for this phenotype of NF-1, although the influence of modifying genes in addition to a primary NF-1 mutation, as

well as alterations in the tissue-specific elastin gene transcription, may play a role.^{28,29} In our experience, more than 25% of patients with abdominal aortic coarctation or hypoplasia carry a confirmed diagnosis of NF-1.⁵ Among patients with NF-1-related vascular involvement, 13% exhibited abdominal aortic coarctations.²⁷

Takayasu aortoarteritis is a well-recognized inflammatory cause of abdominal aortic coarctation that in its quiescent phase must be differentiated from developmental narrowings, although it is more likely to involve the aortic arch and proximal descending thoracic aorta.³⁰ Such an aortoarteritis may be the most common cause of abdominal aortic narrowings encountered in the subcontinents. Maternal rubella during the first trimester is an infectious disorder that has been associated with abdominal aortic hypoplasia.^{31–33} The cellular destruction properties of the virus responsible for rubella may eliminate certain vascular cells needed for normal aortic and arterial growth. Nondevelopmental abdominal aortic coarctation has also been associated with umbilical artery catheterization during the neonatal period, although aortic thromboses and mycotic aneurysms have been reported more frequently than aortic narrowings.^{34,35}

DEVELOPMENTAL RENAL AND SPLANCHNIC ARTERIAL STENOSES

Abdominal aortic coarctations are associated with stenoses of the splanchnic and renal arteries, in 87% and 62% of cases, respectively.⁵ Most of these stenoses in North American Caucasian children are developmental,^{1,6,11} compared with the inflammatory aortoarteritis and Takayasu-related stenoses encountered in Asia, Africa, and South America.^{36–39} The underpinnings of the developmental arterial stenoses appear to be the same as those causing abdominal aortic coarctations,^{1,2} and that many of these patients have NF-1.^{6,27,40–42}

The vast majority of developmental renal artery narrowings are ostial in location, representing true arterial hypoplasia. These arteries are very small, with one report documenting external and luminal diameters of the stenotic ostial segments in children with renovascular hypertension averaging 2.2 mm and 0.4 mm, respectively.⁴³ These are very abnormal arteries, exhibiting intimal fibroplasia, internal elastic lamina disruptions, diminutive and discontinuous media, and excessive adventitial elastin (Fig. 132.5).

Similar ostial stenoses of the celiac artery (CA) and superior mesenteric artery (SMA) also occur in areas of abdominal aortic coarctations (Fig. 132.6). These stenotic narrowings usually are recognized during studies for suspected aortic or renal artery pathology.⁴⁴ Their etiology appears related to embryonic phenomena affecting the reorganization of the ventral segmental vessels associated with the cephalic roots of the vitelline arteries that normally form the CA and SMA.

Certain renal, CA, and SMA stenoses that are not critical at a young age may exhibit limited growth. It is believed that these vessels may not grow as the child grows. In such

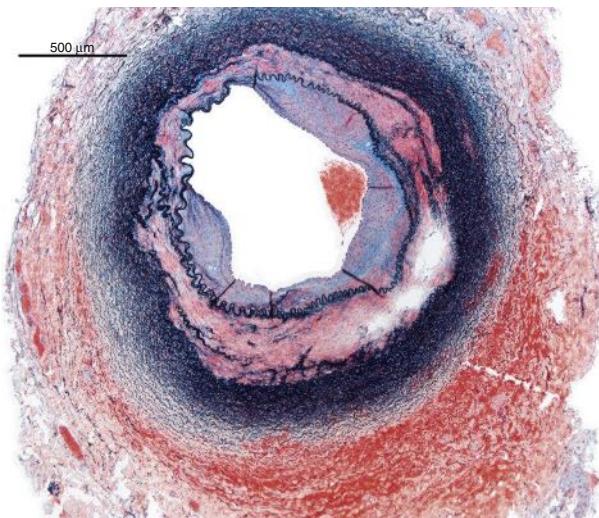


Figure 132.5 Typical developmental renal artery ostial stenosis exhibiting abnormalities in all three vessel layers: intimal fibrodysplasia, duplication of the internal elastic lamina, disorganized fibrodysplastic media, and disproportionate accumulations of elastin in the adventitia (Movat stain). (From Coleman DM, Heider A, Gordon D, et al. Histologic and morphologic character of pediatric renal artery occlusive disease. *J Vasc Surg*. 2021;73:161–171.)



Figure 132.6 Severe celiac artery and superior mesenteric artery ostial stenoses evident on a lateral aortogram (arrows). (From Stanley JC, Eliason JL. Pediatric arterial disease. In: Coran AG, Caldamone A, Adzick NS, et al., eds. *Pediatric Surgery*, 7th ed. Philadelphia: Elsevier Saunders; 2012:1631–1645.)

circumstances, the artery's origin becomes proportionately narrower as the adjacent renal or splanchnic artery becomes bigger, until the narrowing represents a critical stenosis. This entire process suggests a major growth arrest of the aortic origins of the arteries supplying blood to the kidney and intestine.

CLINICAL MANIFESTATIONS

Renal artery occlusive disease is the third most common cause of pediatric hypertension, ranking behind parenchymal renal disease and coarctation of the thoracic aorta.^{45–47} The exact incidence of renovascular hypertension as a cause of childhood hypertension is unknown, but is likely to account for 8% to 10% of those exhibiting marked elevations of their blood pressure.⁴⁸ In this regard, renovascular disease accounts for a much larger proportion of hypertension in children than it does in the adult population.

Hypertension due to suprarenal or intrarenal aortic narrowings may be compounded by occlusive lesions of the renal artery. The resulting secondary hypertension in these cases is often refractory to simple pharmacologic control. Presentations of such patients who are receiving three or more antihypertensive agents are commonplace. Lower extremity fatigue with exercise due to abdominal aortic narrowing has been reported infrequently as the presenting complaint, and true claudication is rare.^{1,5}

Despite the high rate of CA and SMA occlusive involvement with developmental narrowing of the abdominal aorta, symptomatic intestinal ischemia affects only 6% of these cases.¹ However, when these children do have functionally relevant splanchnic arterial narrowings, they present with postprandial intestinal angina manifest by periumbilical discomfort that lasts for the duration of small bowel transit of ingested food. These children develop an aversion for food (sitophobia) and exhibit weight loss from a failure to eat.

Most children with renovascular hypertension are asymptomatic. If they do become symptomatic, initial manifestations are varied including: headache, seizure, epistaxis, and visual disturbances. An isolated facial nerve palsy (Bell's palsy) is an uncommon presentation, usually occurring with severe blood pressure elevations.^{48,49}

Chronic childhood renovascular hypertension may arrest kidney growth and cause chronic renal insufficiency.⁵⁰ Hemorrhagic stroke, hypertensive encephalopathy with impaired cognitive development, and failure to thrive may occur in unrecognized and untreated children with hypertension.^{4,51–53} Poorly controlled hypertension also may result in severe diastolic dysfunction and left ventricular hypertrophy (LVH). The resulting hypertensive cardiomyopathy can progress to heart failure. If the entire renal mass is involved, flash pulmonary edema associated with renal insufficiency may occur, although it is an uncommon complication.

Definition of Hypertension in Children

The National Health and Nutrition Examination Survey (NHANES) data are the basis for definitions of hypertension in children and adolescents. Blood pressure tables for the 50th, 90th, 95th, and 99th percentiles by sex, age, and height are included in *The Fourth Report on the Diagnosis, Evaluation, and Treatment of High Blood Pressure in Children and Adolescents* published by the US Department of Health and Human Services (http://www.nhlbi.nih.gov/health/prof/heart/hbp/hbp_ped.pdf).⁵⁴ Normal pressure is defined as systolic blood pressure (SBP) or diastolic

blood pressure (DBP) that is less than the 90th percentile for sex, age, and height. Hypertension is defined as an average SBP or DBP that is greater than or equal to the 95th percentile for sex, age, and height on at least three separate occasions. Children older than 3 years of age should have their blood pressure measured, preferably by auscultation, whenever they are seen in the medical setting. It is important that an appropriately sized cuff be used for the size of the child's upper arm. Children younger than 3 years of age should also have their blood pressure measured if they require hospitalization, have hereditary illnesses like neurofibromatosis or tuberous sclerosis, or have diseases affecting their urologic tract or heart.

DIAGNOSTIC EVALUATION

Renal sonography is the first-line study to detect renin-mediated hypertension caused by aortic or main renal artery disease, with up to 90% sensitivity and 68% specificity.⁵⁵ But imaging requires significant detail to accurately define renal artery stenotic disease.^{5,7,55–58}

In this regard, axial imaging with both computed tomography angiography (CTA) and magnetic resonance angiography (MRA) are being used increasingly as first-line imaging modalities to diagnose pediatric renovascular hypertension.⁵⁶ Although there are concerns about using CTA with radiation in children due to its cumulative effects, contemporary low-dose CTA protocols reduce radiation exposure in this sensitive age group, without compromising diagnostic imaging. Time-resolved three-dimensional (3D) cine phase-contrast MRI may permit the evaluation of renal blood flow pre- and postintervention.^{59,60} Cerebral vascular abnormalities are described in a small percentage of patients with MAS (3%–13%), including intracranial arterial occlusive disease and cerebral aneurysms.^{4,61} For this reason, screening children with contrast-enhanced axial imaging (CTA or MRA) should be considered.

Catheter-based digital angiography should be pursued if a strong suspicion exists that renovascular disease is causing a child's hypertension. Conventional angiography remains the most valuable diagnostic study for children with renovascular hypertension and it is an essential element in the planning of an endovascular or open surgical intervention.^{1,5,6,57,58,62,63}

MEDICAL MANAGEMENT

All children with secondary hypertension should be treated. The goal of therapy is reduction of the pressure to the 95th percentile for sex, age, and height, unless LVH or other target organ damage is identified, in which case the goal should be less than the 90th percentile. Treatment of hypertension in a very young child may be difficult and requires frequent and careful monitoring.

Antihypertensive Drug Treatment

The nuances of pharmacologic treatment of high blood pressure in children and renovascular hypertension in particular are complicated.^{51,64,65} Certain cautions in treating pediatric renovascular hypertension with drugs deserve note.

First, angiotensin-converting enzyme inhibitors or angiotensin receptor blockers are potent drugs. They may control high blood pressures with monotherapy, whereas multiple agents from other classes of drugs fail. However, drugs like these that block any part of the renin–angiotensin system may be hazardous when used in children with bilateral renal artery disease or unilateral disease affecting a solitary kidney, causing elevated creatinine levels. It is imperative to monitor serum creatinine levels early after initiating treatment with these agents in order to recognize any deterioration in renal function. If such occurs, discontinuing use of these agents usually results in a return to baseline renal function.

Second, rebound hypertension and tachycardia seen with the sudden withdrawal of clonidine is particularly troublesome in children. Patches used in a very young child may be accidentally detached, resulting in dramatic fluctuations in blood pressure and heart rate. One should confirm the adequacy of the patch placement before undertaking a complex search for the cause of abrupt changes in blood pressure control. Finally, diuretics should be used only as combination therapy, as their isolated use may increase renin release and exacerbate hypertension.⁶⁶

SURGICAL TREATMENT

Isolated abdominal aortic coarctations are preferentially treated by patch aortoplasty when technically feasible, whereas thoracoabdominal bypasses are undertaken in patients in whom the diseased aorta is too narrow to facilitate placement of a patch, or in cases of complex disease affecting the renal and splanchnic arteries.^{1,5,67} Expanded polytetrafluoroethylene (ePTFE) grafts are favored over woven or knitted grafts for both patch aortoplasty and thoracoabdominal bypass grafting, due to the greater stability of ePTFE regarding post-implantation dilatation.

We have identified younger age at operation as an independent predictor of reoperation; more specifically, age at operation significantly affected hazards of a reoperation in that as age

increased by 1 year, the rate of reoperation decreased by about 10%. Acknowledging this observation, tempered durability along with the added challenges of surgical reconstruction in the very young introduces (i.e., vessel size, conduit challenges, vasospasm, and projected somatic growth), medical management is favored for the very young (i.e., 3–4 years) with the understanding that definitive surgery would likely be more successful in an older and larger child.

Patch Aortoplasty

Patch aortoplasty is performed when the narrowed segment of the aorta has a large enough diameter to allow completion of the anastomosis without an overlap of sutures from the opposite side of the patch. There are important rheologic benefits related to renal artery blood flow that favor the performance of a patch aortoplasty over a thoracoabdominal bypass when treating children with abdominal aortic narrowings.⁶⁸ Aortic patches in children are sized large enough to match the growth potential of the remaining aorta, but not so large as to create low-velocity flow with low shear stress and potential thrombus formation (Fig. 132.7). Aortoplasty is often accompanied by complex renal and splanchnic arterial reconstructive procedures (Fig. 132.8).

Thoracoabdominal Bypass

Thoracoabdominal bypass grafts should originate above the narrowed area, either from the distal thoracic aorta above the diaphragm using a thoracoabdominal approach or from the supraceliac aorta near the diaphragmatic hiatus by an abdominal approach (Fig. 132.9). The grafts are positioned behind the left kidney, terminating in the distal aorta. The intent is to use oversized grafts compared with the aorta, recognizing anticipated growth. Otherwise, the graft may be too small to maintain normal distal pressures and flow as the child grows into adolescence

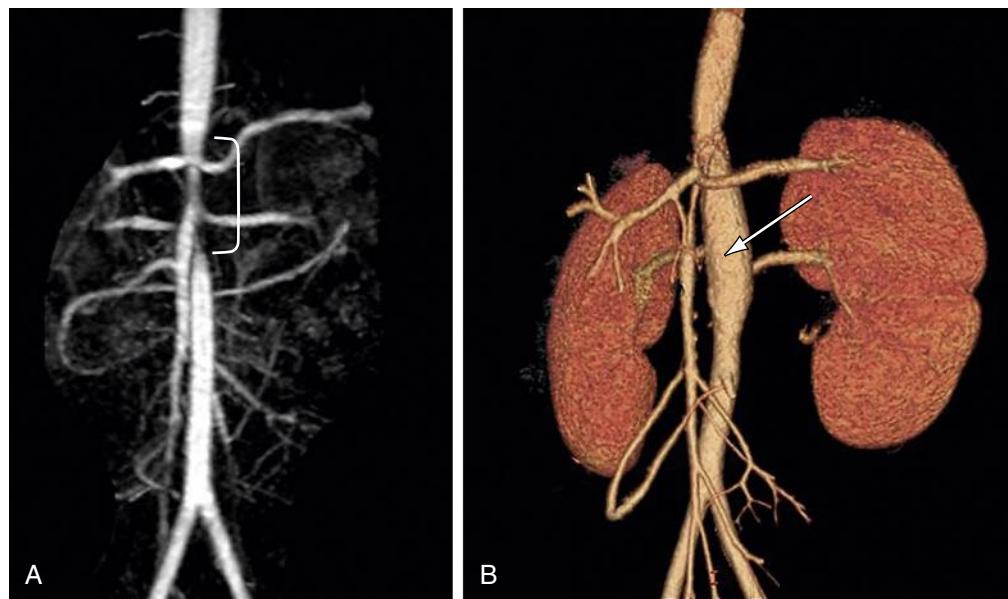


Figure 132.7 (A) Suprarenal coarctation (bracket) with bilateral renal artery ostial stenoses. Preoperative magnetic resonance angiography. (B) Subsequent patch aortoplasty (arrow), with aortic diameter exceeding that of uninvolved proximal and distal aorta. Reimplantation of the renal arteries accompanied the aortic reconstruction. (From Stanley JC, Criado E, Eliason JL, et al. Abdominal aortic coarctation: surgical treatment of 53 patients with a thoracoabdominal bypass, patch aortoplasty, or interposition aortoaortic graft. *J Vasc Surg*. 2008;48:1073–1082.)

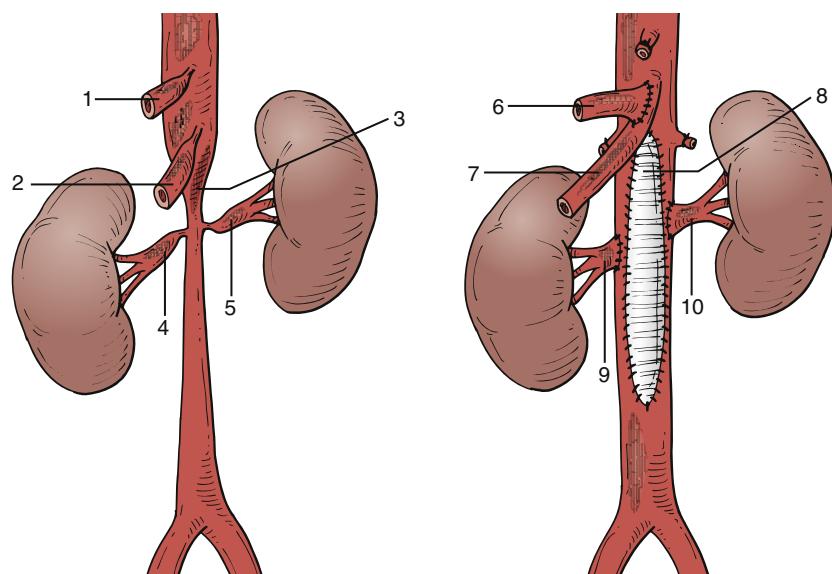


Figure 132.8 Complex aortic, splanchnic, and renal arterial reconstruction: 1, celiac artery (CA) stenosis; 2, superior mesenteric artery (SMA) stenosis; 3, interrenal midabdominal aortic coarctation; 4 and 5, right and left renal artery stenosis; 6, CA implanted onto stenotic SMA origin; 7, widely patent SMA; 8, polytetrafluoroethylene patch aortoplasty; 9 and 10, bilateral implantation of renal arteries onto aorta. (From Upchurch GR, Jr, Henke PK, Eagleton MJ, et al. Pediatric splanchnic arterial occlusive disease: clinical relevance and operative treatment. *J Vasc Surg*. 2002;35:860–867.)

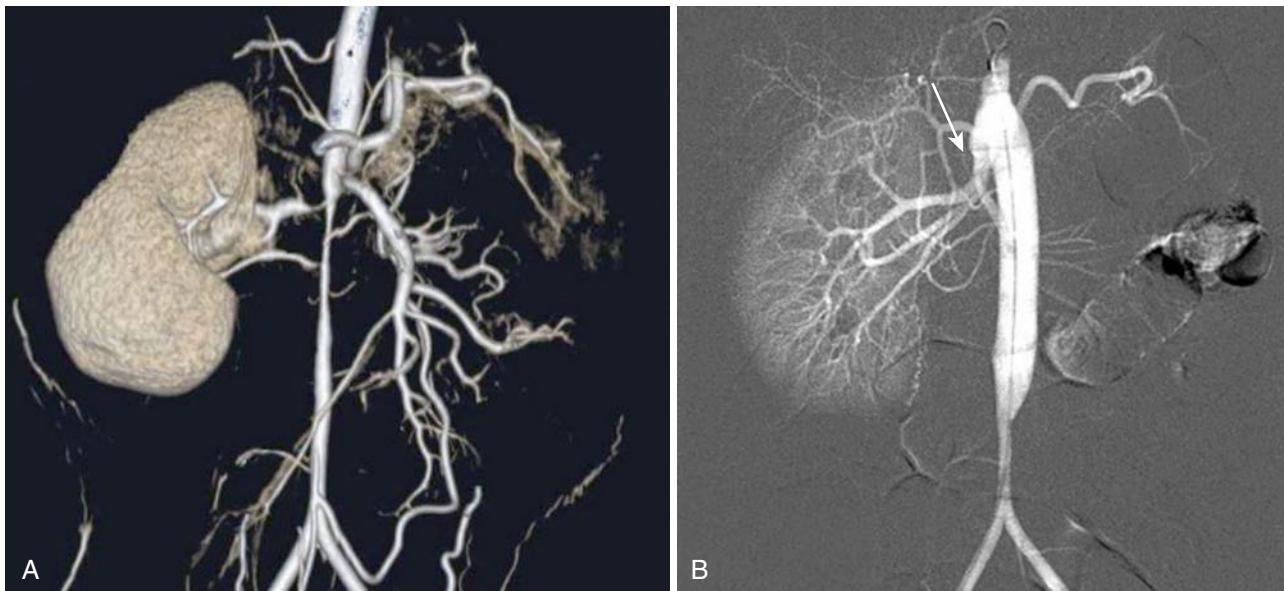


Figure 132.9 Thoracoabdominal aortic bypass. (A) Preoperative evidence of suprarenal abdominal aortic coarctation, solitary kidney with ostial stenoses of two renal arteries. (B) Postoperative aortoaortic bypass with reimplantation of both renal arteries into proximal aorta (arrow). (From Coleman DB, Eliason JL, Beaulieu R, et al. Surgical management of pediatric renin-mediated hypertension secondary to renal artery occlusive disease and abdominal aortic coarctation. *J Vasc Surg*. 2020;72(6):2035–2046.e1.)

and adulthood. General guidelines include: 8- to 12-mm grafts in young childhood; 12- to 16-mm grafts in early adolescence; and 14- to 20-mm grafts in late adolescence. It is not essential to leave significant graft redundancy in older children, in that axial growth from the diaphragm to the pelvis is insignificant after reaching 9 or 10 years of age. These bypass procedures are frequently undertaken with concurrent renal and splanchnic artery reconstructions (Figs. 132.10 and 132.11). Similar grafts are often used in treating pediatric abdominal aortic aneurysms in concert with aortic branch reconstructions.⁶⁹

Open surgical treatment of abdominal aortic coarctations benefits more than 90% of patients when treated with a patch aortoplasty or a thoracoabdominal bypass.^{1,5} Postoperative

morbidity is low, with infrequent bleeding and postsurgical pancreatitis being the most noteworthy. Surgical mortality should approach zero. The patency of patch aortoplasty, thoracoabdominal aortic bypass, or endovascular treatments (see below) may be followed long-term by noninvasive blood pressure measurements of the upper and lower extremities, with and without exercise. If the ankle–brachial index measurements are noted to decline, then detailed imaging should be considered. MRA is favored to avoid excessive radiation exposure accompanying CT studies. In very early childhood, the use of large conduits may not be possible.^{1,5} Therefore, aortic reoperations may be required for anastomotic narrowings, or if a patient outgrows the primary reconstructive procedure. The incidence

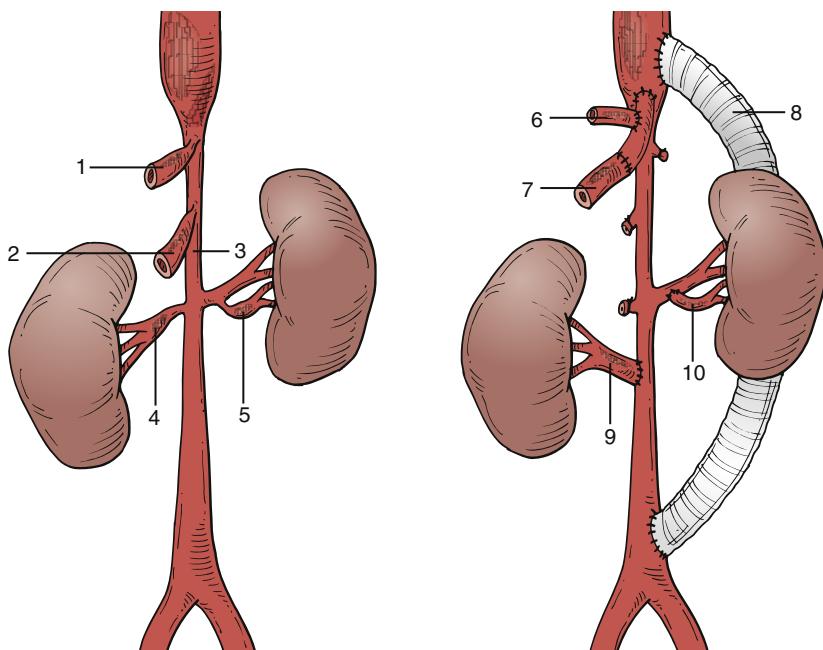


Figure 132.10 Complex aortic, splanchnic, and renal arterial reconstruction: 1, celiac artery (CA) stenosis; 2, superior mesenteric artery (SMA) stenosis; 3, suprarenal midabdominal aortic coarctation; 4, right renal artery ostial stenosis; 5, left segmental renal artery stenosis; 6, CA implanted onto aorto-SMA bypass (with autogenous internal iliac artery graft); 7, reconstructed SMA; 8, thoracoabdominal aortic bypass; 9, right renal artery reimplantation onto aorta; 10, left segmental renal artery implantation onto adjacent main renal artery. (From Upchurch GR, Jr, Henke PK, Eagleton MJ, et al. Pediatric splanchnic arterial occlusive disease: clinical relevance and operative treatment. *J Vasc Surg*. 2002;35:860–867.)

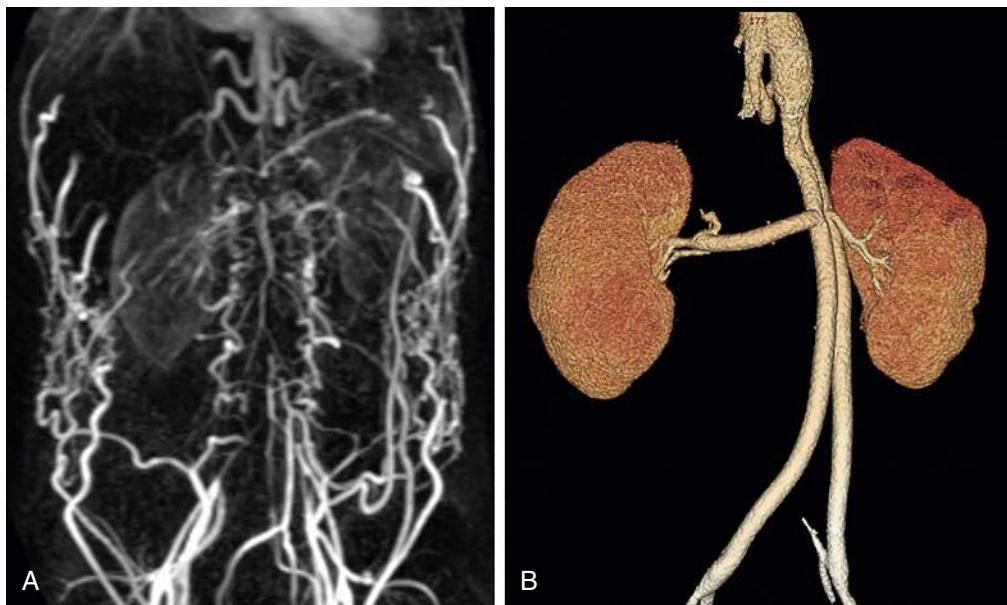


Figure 132.11 (A) Absence of the abdominal aorta and agenesis of the proximal main renal arteries, with reconstitution of the distal renal arteries and iliac arteries by way of extensive collateral vessels. (B) Complex thoracic aortobi-iliac bypass with bilateral renal artery reconstructions.

of such secondary aortic operations at the authors' institution following surgical treatment of abdominal aortic coarctation or segmental hypoplasia approached 10%, and were performed 5 to 12 years after the initial reconstruction.⁵

Retroaortic Tissue Expander

A unique means of treating abdominal aortic coarctation involves placement of a retroaortic tissue expander to induce longitudinal growth of the normal distal aorta.⁷⁰ The expander is sequentially inflated through a subcutaneous port over the course of many months, following which the coarctation may be excised and the elongated aorta anastomosed directly to the normal aorta above the narrowed segment. The value of this novel therapy awaits greater experience and longer follow-up.

Renal Artery Reconstruction

The surgical options currently used to reconstruct renal arteries that are stenotic or occluded are designed to minimize renal ischemia in the operating room and restore sustainable and normal renal blood flow (Figs. 132.12 and 132.13).^{1,6,11} A certain degree of caution surrounds the technical challenges in reconstructing the very small and highly vasoactive renal arteries in infants. Although uncontrolled severe hypertension or renal failure in rare instances may require an operation in the very young, revascularizations are more likely to be successful after the age of 3 years.¹ Deferring renal artery reconstructive procedures in infants when possible is reasonable and appropriate.

Optimal surgical therapy depends on the character of the renal artery disease being treated, the length of the stenotic

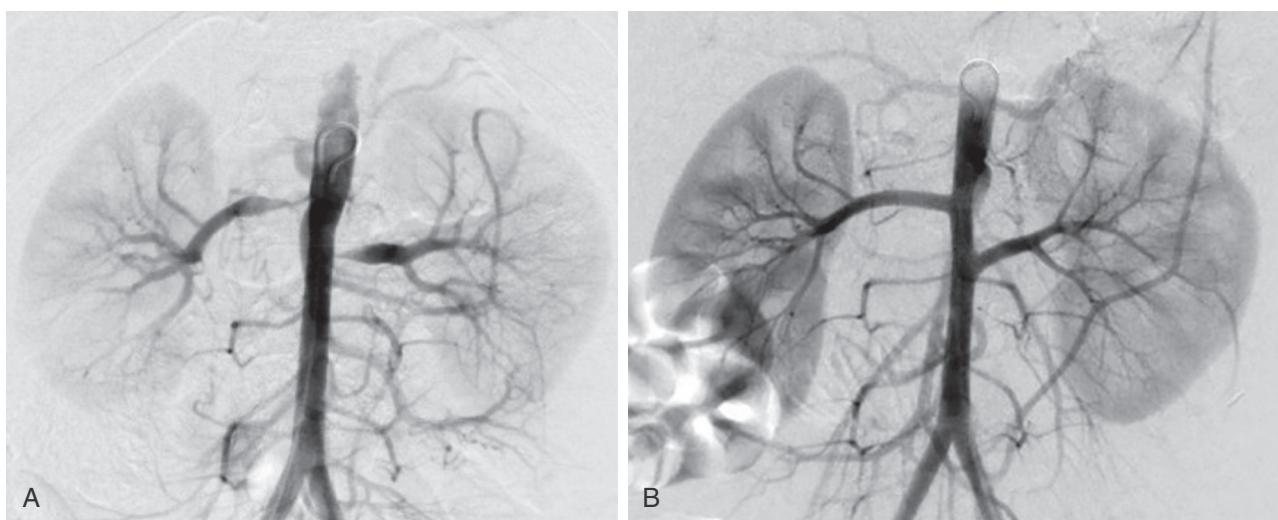


Figure 132.12 Renal artery–aortic implantation. (A) Preoperative bilateral proximal ostial stenoses. (B) Postoperative bilateral renal artery implantations. (From Coleman DM, Eliason JL, Beaulieu R, et al. Surgical management of pediatric renin-mediated hypertension secondary to renal artery occlusive disease and abdominal aortic coarctation. *J Vasc Surg*. 2020;72(6):2035–2046.e1.)

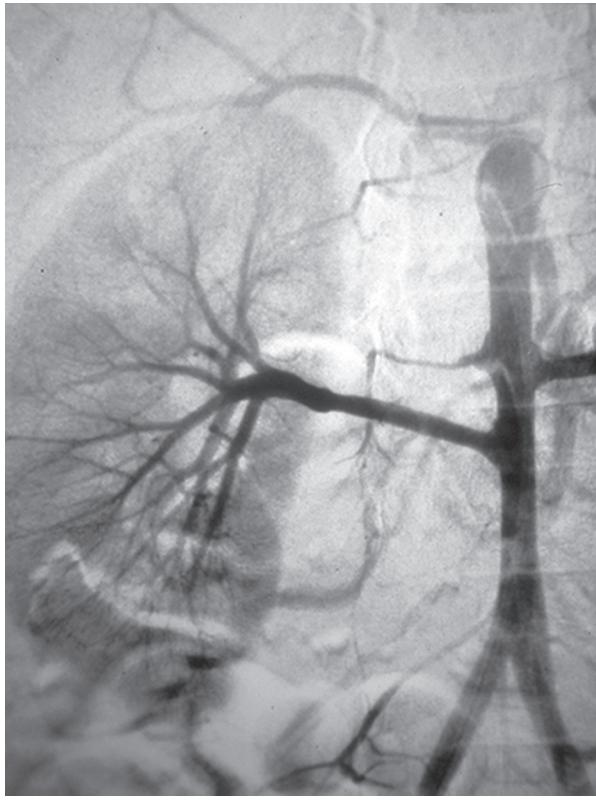


Figure 132.13 Aortorenal bypass with an internal iliac artery graft. (From Stanley JC, Zelenock GB, Messina LM, Wakefield TW. Pediatric renovascular hypertension: a thirty-year experience of operative treatment. *J Vasc Surg*. 1995;21:212–227.)

disease, and whether segmental renal branches are also narrowed. The most common method of surgical reconstruction has been renal artery implantation of the normal artery beyond an ostial stenosis, into the aorta or an adjacent normal renal artery.^{1,6} When reimplantation cannot be performed, such as occurs with a distal main renal artery or branch stenosis, a

bypass is appropriate. Use of the internal iliac artery for an aortorenal graft has proven to be a suitable conduit.^{1,6} The use of an autologous vein segment for a bypass graft, as utilized in the past, is no longer recommended because of their aneurysmal degeneration when used in young patients.⁷¹

Less common renal artery reconstructive procedures include resection of stenotic segments followed by a primary reanastomosis, a focal arterioplasty, or open operative dilations. The frequent presence of splanchnic arterial narrowings in patients with renal artery stenoses and the propensity for the CA to become narrowed as it passes through the aortic hiatus, are reasons why splenorenal or hepatorenal reconstructions are not favored in the pediatric population.

Nephrectomy may be undertaken in certain children having unreconstructable renovascular disease. Indications for nephrectomy include multiple intrarenal stenoses not amenable to open *in situ* or endovascular treatment, or a diminutive non-functioning kidney when the contralateral kidney is assumed to be sufficient in preventing the patient from going into renal failure. Partial nephrectomy may offer a nephron-sparing cure for segmental renovascular hypertension, as diagnosed by selective renal vein renin sampling.⁷²

Large clinical experiences with pediatric renovascular hypertension are uncommon.^{1,6,11,73–80} A recently published University of Michigan series included 169 children (76 girls and 93 boys), ages 3 months to 18 years, who underwent operation from 1991 to 2017.¹ All but one patient had refractory hypertension not responsive to medical therapy. Among these children, abdominal aortic coarctations occurred in 45%, renal artery stenoses were ostial in 66%, and the celiac or superior mesenteric arteries were stenotic in 49%. Nearly a third had experienced failed open surgical or endovascular attempts to reconstruct their aorta or renal artery elsewhere before being treated at Michigan. In this series, open surgical procedures included 146 renal artery reimplantations into the aorta or adjacent renal artery, 55 aortorenal bypasses, 10 alcohol ablations

of a kidney segment, 25 partial or total nephrectomies, 30 splanchnic arterial reconstructions, 32 patch aortoplasties, and 32 thoracoabdominal aortic bypasses. Reoperations were required in 21% of cases. There was no postoperative renal failure requiring dialysis or operative mortality during a mean follow-up of 4.1 years. These primary and secondary interventions resulted in hypertension being cured in 44% of cases, improved in 45%, and unchanged in 10%. These outcomes were less salutatory than reported in earlier Michigan experiences.^{6,11} This likely reflects the recent increases in aortic disease requiring operation and greater numbers of children presenting with prior PTA failures. Prior to the aforementioned 2020 Michigan publication, a 2006 report described 97 children with less complex disease and fewer failed interventions before undergoing treatment, with superior outcomes.⁶ Specifically, in that series hypertension was cured in 70%, improved in 27%, and was unchanged in 3%.

Another large open surgical experience was reported from the Hospital Beaujon in France, including 78 children ranging in age from 1.4 to 18 years.⁷⁴ There were 91 primary revascularization procedures and 15 nephrectomies. These numbers were quite similar to the 2006 University of Michigan series. A third series from the Cleveland Clinic involved 56 children ranging in age from 8 months to 21 years.⁷⁵ They reported 46 primary operations and 10 primary nephrectomies. A fourth series, from both Vanderbilt University and the Children's Hospital of Philadelphia, included 50 children ranging in age from 5 months to 16 years.⁷⁶ The authors reported on 28 primary reconstructive procedures and an additional 12 primary nephrectomies. The latter two series, unlike the experiences at the University of Michigan or Hospital Beaujon, did not contain many patients treated for aortic or splanchnic arterial disease.

When blood pressure control outcomes were analyzed in the three major reports on pediatric renovascular hypertension from the University of Michigan in 2006, Vanderbilt University–Children's Hospital of Philadelphia, and the Cleveland Clinic, a great deal of similarity was found in the results. Respective cure rates were 70%, 70%, and 66%; reported improvement rates were 27%, 26%, and 23%; and failure rates were 3%, 4%, and 11%, respectively. Each of these reports reflected the open surgical management of renal artery stenoses in children.

Renal artery bench repairs and autotransplantation of the kidney have been suggested in case reports and more contemporary retrospective single institution series as safe and efficacious for complex and segmental renal artery disease, without associated mortality, negligible morbidity, and reasonable long-term follow-up.^{80–83} Hypertension cure rates in the latter setting may approach 62% with concurrent preservation of renal function.

Splanchnic Artery Reconstruction

Splanchnic artery repair depends on whether obstructions of the CA and SMA are symptomatic. A relative indication to prophylactically reconstruct these vessels exists when



Figure 132.14 Superior mesenteric artery implantation into infrarenal aorta (arrow), as treatment of intestinal ischemia due to an ostial stenosis of this artery, in a child with near occlusion of the supraceliac abdominal aorta who underwent a concomitant thoracoabdominal bypass to the distal aorta (not visualized on this lateral aortogram).

performance of an aortoplasty or renal revascularization would make a subsequent CA or SMA revascularization exceedingly difficult (Fig. 132.14).^{44,84} Aortic reconstructions remote from the mesenteric vessels make prophylactic mesenteric revascularization less likely to be warranted.

The operative treatment of pediatric splanchnic arterial occlusive disease is complex. Internal iliac artery grafts are used to treat lengthy CA or SMA stenoses in young patients. In other children, aortic implantation of the CA or SMA after spatulation of the transected vessel beyond its stenotic origin is favored over an aortosplanchnic bypass. Symptomatic patients treated by both methods are quickly able to gain weight and are rapidly freed from abdominal discomfort. Percutaneous transluminal balloon angioplasty with or without stenting in children for these splanchnic ostial lesions is not appropriate and is ill-advised.

Splanchnic arterial occlusive disease encountered in childhood has an uncertain natural history. Long-term follow-up of these children with periodic imaging studies among those operated upon seems appropriate, and regular clinical assessment of those not subjected to surgical therapy is recommended. With regard to the latter, it is important to remember that most CA and SMA stenoses are asymptomatic because of the inferior mesenteric artery serving effectively as a source of collateral circulation. Unoperated patients should be aware of their splanchnic arterial anatomy and be able to pass this information to a surgeon performing a later abdominal operation. Inadvertently interrupting the collateral circulation might cause catastrophic intestinal ischemia in these individuals.

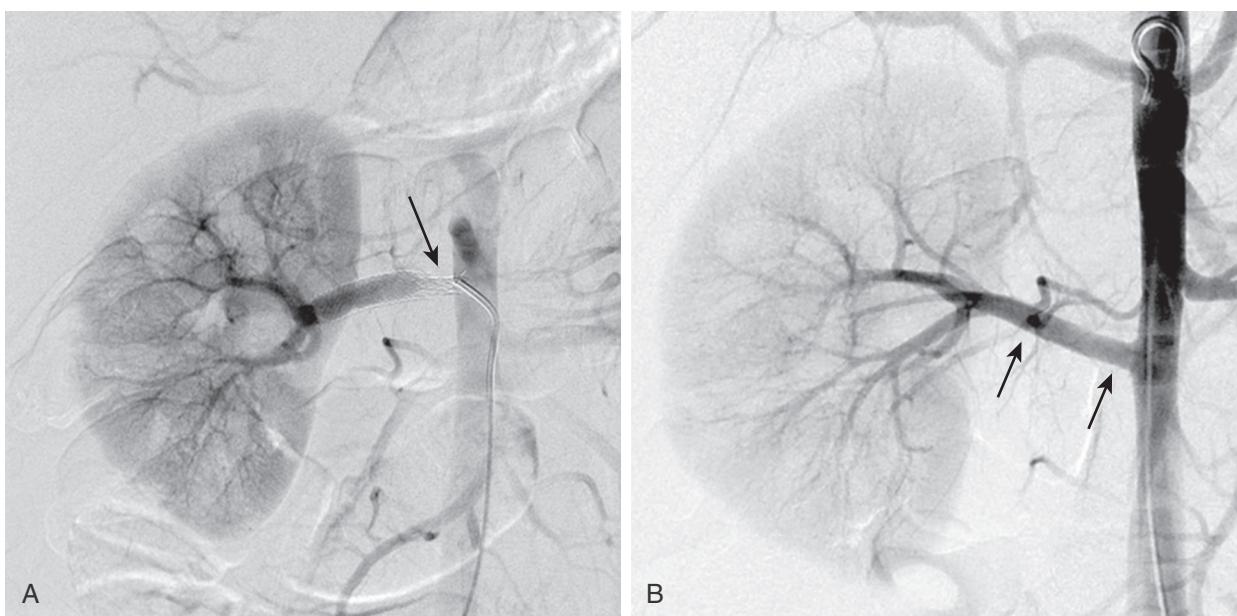


Figure 132.15 (A) Severe proximal in-stent restenosis following PTA with placement of two stents, one of which failed to expand (arrow). (B) Treated by an aortorenal bypass with segment of the internal iliac artery. (From Eliason JL, Coleman DM, Criado E, et al. Remedial operations for failed endovascular therapy of 32 artery stenosis in 24 children. *Pediatr Nephrol*. 2016;31:809–817.)

ENDOVASCULAR TREATMENT

Endovascular treatment of abdominal aortic narrowings may be successful in the procedural setting at decreasing a pressure gradient across a stenosis, but at the cost of increased perioperative morbidity in comparison to open surgery. These interventions have complication rates that approach 30%, including aortorenal injury (“tears”), aneurysm formation at the site of angioplasty, and stent embolization.^{4,63} In addition, endovascular treatments are associated with an increased incidence of restenosis and reintervention with a reported freedom from reintervention of only 55% at 1 year and 33% at 5 years.⁶³

Endoluminal interventions may have utility in the treatment of select focal abdominal aortic coarctations distant from the renal and splanchnic branches, and this therapy has been most commonly employed for the treatment of stenotic disease due to an aortitis.^{85–89} Developmental aortic narrowings are prone to significant recoil from the hypoplastic and fibrotic segments, and stenting may be required to overcome this.^{90,91} Unfortunately, the long-term benefits of endovascular interventions for developmental aortic narrowings remain unproven in children, and subsequent aortic operations after endovascular failures are considerably more difficult.

Endoluminal interventions have provided major benefits in treating renovascular disease, particularly in young adults with fibromuscular dysplasia, and are being utilized with increasing frequency in the treatment of pediatric renovascular disease with several contemporary reports suggesting very good results.^{92–96} However, it warrants mention that in one of the larger experiences with endoluminal angioplasty, including 114 procedures, the cure, improved, failure rates were 23%, 40%, and 37%, respectively.² Similar failure rates, ranging from 24% to 44%, have been reported by others.^{93,96,97} These results may be anticipated, given the underlying developmental arterial derangement associated with

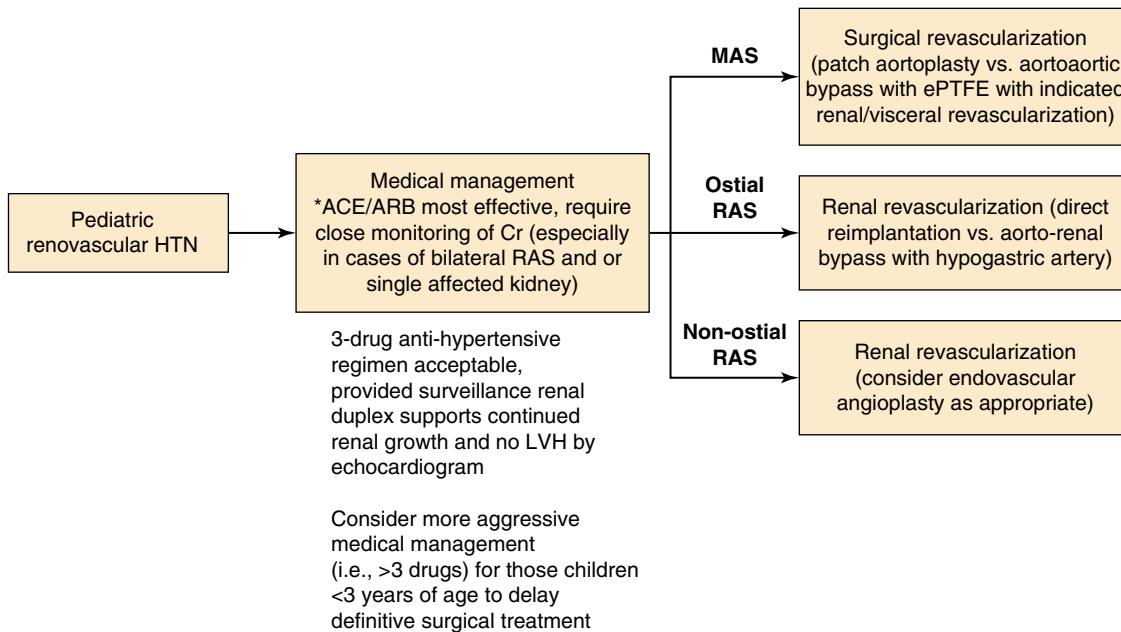
unyielding intimal fibrosis and excessive elastic tissue which may result in early postdilation arterial recoil. Importantly, the excessive degree of radial force required to dilate a fibrotic lesion and the accompanying injury to a diminutive renal artery risks thrombotic complications. Although one may consider endovascular intervention as a bridge to definitive open surgery once the patient grows, there may be irreversible complications from endovascular treatment that cannot be underestimated. Therefore, the role of endovascular interventions in the treatment of pediatric renal artery stenoses remains controversial.^{1,98–100}

Remedial operations following failed endovascular therapy are more complex (e.g., distal bypass in lieu of direct aortic reimplantation) and are not as successful as primary operations.¹⁰⁰ Outcomes following remedial operations for endovascular failures included hypertension cure rates of only 25% and improvement rates of 54% (in comparison to 70% and 27%, respectively, with open procedures). Patient selection for endovascular intervention should consider the small vessel size and fibrotic nature of these lesions. Stents are to be avoided, as they increase the risk of neointimal hyperplasia and recurrent stenosis, and they do not take into account later necessary growth of the artery, both in diameter and length (Fig. 132.15). Finally, endovascular therapies should only be performed at high volume centers by an experienced team.

CONCLUSION

The management of pediatric aortic, renal, and splanchnic artery disease remains a complex process that is best managed with the collaboration of multiple specialties. Vascular surgery, nephrology, diagnostic and interventional radiology, and cardiology all can contribute valuable insights into the diagnosis, follow-up, and management of these children. A disease specific, multidisciplinary, patient- and family-centered focus will afford the most salutary outcomes.

CHAPTER ALGORITHM



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Acute Mesenteric Ischemia: Epidemiology, Pathophysiology, Clinical Evaluation, and Management

SARA L. ZETTERVALL and MARC L. SCHERMERHORN

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Acute arterial mesenteric ischemia may be challenging to diagnose and treat, resulting in high rates of mortality despite advances in operative technique and critical care. The first superior mesenteric artery (SMA) embolectomy for acute mesenteric ischemia was reported by Klass in 1951.¹ Over the next two decades several reports demonstrated increasing success with SMA thromboembolectomy and thromboendarterectomy for the treatment of acute embolic occlusion; however mortality rates reached 70% to 90%.^{2,3} Early and liberal use of angiography was championed by Boley and Clark in the early 1970s.⁴⁻⁶ With an aggressive surgical approach, including the use of vasodilators, they demonstrated a reduction in the mortality rate to approximately 50%. Recent reviews have shown significant

declines in the in-hospital mortality rate among patients treated for acute mesenteric ischemia; however they remain high at 17%–21% among patients requiring revascularization.^{7,8} While open embolectomy remains a mainstay of treatment, endovascular techniques including percutaneous and retrograde open mesenteric stenting have increased dramatically and now account for nearly 50% of interventions for acute mesenteric ischemia.

INCIDENCE AND RISK FACTORS

Acute mesenteric ischemia accounts for less than 1 of every 1000 hospital admissions.⁹ This disease process affects

women three times as frequently as men and is typically seen in elderly patients with multiple comorbidities.⁷ Clinical risk factors often provide clues to the specific pathophysiology. Patients at risk for mesenteric embolus include those with a history of atrial fibrillation/flutter, recent myocardial infarction, congestive heart failure, or peripheral arterial emboli. Alternatively, a history consistent with chronic mesenteric ischemia – such as postprandial abdominal pain, weight loss, and food intolerance or previous intervention for chronic mesenteric ischemia – should raise the suspicion of an acute or chronic thrombosis of a pre-existing mesenteric artery stenosis (see Ch. 134, Chronic Mesenteric Ischemia: Epidemiology, Pathophysiology, Clinical Evaluation, and Management). In contrast, patients with nonocclusive mesenteric ischemia (NOMI) are likely to be critically ill and in shock from alternative sources, having suffered significant hemodynamic insults including hypotension or dehydration in the preceding hours to days. Postoperative cardiac surgery and hemodialysis patients are classically at highest risk for NOMI. Dissection of the visceral arteries from aortic dissection or spontaneous visceral dissection can also cause acute mesenteric ischemia; however these topics will be addressed in full detail in other chapters (see Ch. 135, Mesenteric Arterial Dissection).

PATHOPHYSIOLOGIC CLASSIFICATION

Arterial Embolism

Historically, arterial embolism was considered the common pathophysiology of acute mesenteric ischemia, accounting for 40% to 50% of cases.^{10,11} Nearly one-third of patients with a mesenteric embolus have had an antecedent embolic event, most commonly arising from a cardiac source including atrial tachyarrhythmia, low ejection fraction (congestive heart failure, cardiomyopathy), recent myocardial infarction, and ventricular aneurysm, while as high as 68% of patients have synchronous embolisms in other locations.^{10,12,13} Although less common than a cardiac source, proximal arterial sources, such as in instances of a cardiac valvular disease, endocarditis, aortic aneurysm or mural thrombi, and recent catheter-based angiography may also be sources of embolization.

Due to the oblique angle of the SMA origin from the visceral aortic segment, thromboembolism most commonly lodges in the SMA, specifically in the proximal SMA, just beyond the first few jejunal branches as the SMA tapers. However embolization to the celiac artery may also cause mesenteric symptoms, particularly in patients with chronic SMA disease. A minority (15%) may lodge at the SMA origin, but 50% lodge distal to the middle colic artery,^{9,14} creating a classic pattern of ischemia that spares the first portion of the small intestine and the ascending colon (Fig. 133.1). Atheroembolic emboli, in contrast, tend to be smaller and therefore lodge in the more distal mesenteric circulation; as a result, they affect bowel perfusion less often and in more localized areas.

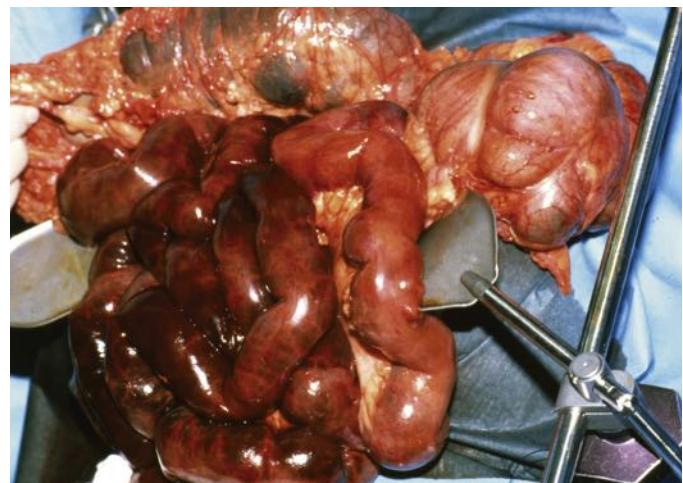


Figure 133.1 Intraoperative photograph of a patient with a superior mesenteric artery embolus. Note the relative sparing of the proximal jejunum and proximal transverse colon. (Courtesy R. M. Zwolak.)

Arterial Thrombosis

In patients with a thrombotic etiology, acute-on-chronic presentation is common and occurs when an arterial thrombosis is superimposed on pre-existing severe atherosclerotic disease. While historical data noted arterial embolism to be the most common cause of acute mesenteric ischemia, recent studies have suggested that *in situ* thrombosis of chronic lesions may now account for greater than 50% of cases of acute mesenteric ischemia.⁸ Autopsy studies have shown that up to 29% of people have significant stenosis in at least one mesenteric artery, including 60% of patients older than 80 years of age.¹⁵ In those with peripheral vascular disease, the incidence may be as high as 27%.¹⁶ In acute-on-chronic ischemia, the presentation of bowel infarction may be more insidious in onset due to a pre-existing collateral network that maintains viability until an event such as a small embolism or hypotension results in occlusion of a critically stenotic vessel or collateral.

Patients with previous intervention for mesenteric ischemia are also at heightened risk for thrombotic complications. These include patients who have in-stent restenosis or hyperplasia of a previous bypass graft. While immediate endovascular failures – including distal embolization, vessel perforation, dissection, stent migration, and thrombosis – result in higher mortality, morbidity, and longer hospital length of stay, late occlusion may also occur in patients treated for chronic mesenteric ischemia who have developed restenosis or intimal hyperplasia over time.¹⁷ For these reasons, short- and long-term follow-up is critical for maintaining the patency of both open- and endovascular-treated vessels.

Nonocclusive Mesenteric Ischemia

Mesenteric vasospasm, usually in the distribution of the SMA, is the *sine qua non* of Nonocclusive Mesenteric Ischemia (NOMI). This form of AMI accounts for approximately 20% of presentations, but carries the highest mortality rates. NOMI is morbid due to its difficulty to diagnose and treat and because

of its frequent association with multisystem organ failure.^{18,19} Initial descriptions were in postmortem observations of small intestinal gangrene in patients who had shown no evidence of arterial or venous occlusive disease.²⁰ NOMI is frequently described in patients with severe cardiac failure, and while concomitant visceral atherosclerosis may be present, the underlying etiology is related to vasospasm. These observations formed the basis for the hypothesis that cardiac failure, peripheral hypoxemia, paradoxical splanchnic vasospasm, and reperfusion injury may all contribute to the development of NOMI. Perhaps resulting from excessive sympathetic activity during cardiogenic shock or hypovolemia, the vasospasm represents a homeostatic mechanism that attempts to maintain cardiac and cerebral perfusion at the expense of visceral and peripheral organs. Vasopressin and angiotensin are the likely neurohormonal mediators of this process.²¹ In the current era, vasoactive medications such as epinephrine, norepinephrine, and vasopressin have also been associated with the development of NOMI.²² Once mesenteric vasospasm is initiated, it may persist even after correcting the initiating event. Although intestinal auto-regulation may initially offset reductions in blood flow, the auto-regulatory capacity is exceeded after several hours.

COMMON PATHWAY OF BOWEL ISCHEMIA

The degree of reduction in blood flow that the bowel can tolerate without permanent cellular damage is significant. Only one-fifth of the mesenteric capillaries are open at any given time, and normal oxygen consumption can be maintained with only 20% of maximal blood flow.²³ Proposed mechanisms that result in the preservation of splanchnic tissue perfusion include direct arteriolar smooth muscle relaxation and a metabolic response to adenosine and other metabolites of mucosal ischemia.²⁴ In addition, the intestinal mucosa is able to extract increasing amounts of oxygen during hypoperfusion to preserve mucosal integrity during periods of metabolic insult. Prolonged ischemia, regardless of the pathophysiologic cause, leads to disruption of the intestinal mucosal barrier, primarily through the actions of reactive oxygen metabolites and polymorphonuclear neutrophils.¹⁰ The mucosal surfaces are affected first due to the high mucosal metabolic demand as compared to the serosa. Clinically, this may present with malabsorption and heme-positive diarrhea before the onset of other symptoms.

EVALUATION OF ACUTE MESENTERIC ISCHEMIA

Clinical Presentation

Patients presenting with acute mesenteric ischemia often have vague abdominal symptoms. These may include diffuse abdominal pain, nausea and vomiting, melena, and generalized malaise. Tenderness to palpation typically does not occur until transmural ischemia has developed, causing peritoneal irritation. Once patients have bowel necrosis they may present with

tachycardia, hypotension, fever, and other signs of systemic sepsis. For patients who present with embolism, the onset is often more abrupt with rapid clinical decline due to the lack of an established collateral circulation. In contrast, patients with a thrombotic etiology may present with an insidious development and progression of symptomatology as a result of a developed collateral network. The subacute presentation may start days or weeks before the final acute insult that prompts the patient to seek medical attention. Patients may have abdominal pain, distention, diarrhea, acidosis, sepsis, or gastrointestinal bleeding. NOMI patients have the most unpredictable presentation and typically present with a protracted clinical course. These patients are often critically ill and being treated for concurrent organ failure or were found to be ill at home for days prior to presentation, making history difficult to obtain. In these patients, systemic sepsis, laboratory abnormalities, and abdominal pain and distention may be the initial clues to the underlying diagnosis of acute mesenteric ischemia; however, imaging is necessary to evaluate for occlusive flow and definitive cause.

The signs and symptoms of acute mesenteric ischemia can easily be mistaken for other, more common intra-abdominal pathologies such as pancreatitis, cholecystitis, appendicitis, diverticulitis, or bowel obstruction, and early diagnosis may be missed in up to 60% of patients.^{25,26} Delays in diagnosis and treatment remain the greatest challenge to reducing morbidity and mortality for all forms of mesenteric ischemia. It has been shown that survival decreases from approximately 50% to 30% when the diagnosis of SMA embolism is made more than 24 hours after the onset of symptoms.¹⁴ A high index of suspicion in the setting of a compatible history and physical examination serves as a cornerstone of prompt treatment. Once it is suspected, the clinician should quickly move to appropriate imaging to confirm the diagnosis and proceed to the operating room.

Laboratory Evaluation

While laboratory studies provide valuable information on disease severity and resuscitative efforts, acute mesenteric ischemia should not be diagnosed or dismissed as a result of these findings. Leukocytosis, acidosis, and elevated lactate may occur in patients with mesenteric ischemia due to ongoing insult to the intestine. However, such abnormalities are not essential to the diagnosis, and normal values do not rule out acute mesenteric ischemia. Patients are often hypovolemic on presentation, reflected by laboratory abnormalities such as hemoconcentration, leukocytosis, and an anion gap. High amylase, aspartate aminotransferase, and lactate dehydrogenase can also be observed, but are neither sensitive nor specific for the diagnosis of mesenteric ischemia. Other laboratory markers have also been studied in an effort to better assist in this challenging diagnosis. These included d-dimer, which when elevated may help differentiate between mesenteric ischemia and bowel obstruction, as well as urinary and plasma intestinal fatty acid-binding protein (iFABP) levels which may provide early suggestion of intestinal ischemia.^{26,27} However, neither value is routinely used in clinical practice as a diagnostic tool.

Diagnostic Imaging

Historically, early and aggressive use of diagnostic arteriography was advocated as the gold standard for the evaluation of mesenteric ischemia. However, 24-hour access to high-resolution computed tomographic angiography (CTA) has altered this practice. While alternative modalities including MR angiography, ultrasound, and angiography all provide valuable data in the assessment of the visceral vessels, the diagnosis of mesenteric embolization or thrombotic occlusion as well as evaluation of the intestines and alternative sources of abdominal pain are best evaluated with a CTA.

Computed Tomography

The use of CT angiography (CTA) for the evaluation of both acute and chronic mesenteric ischemia is well described, with

a sensitivity and specificity of 93% and 96%, respectively.²⁸ The widespread availability of high quality CT scanners has advanced the diagnostic algorithm for AMI, providing a significant amount of information about the central arterial and venous circulations as well as intestinal disease. Accurate timing of contrast injection and fine slices (0.5–1.5 mm) through the upper abdomen provide excellent visualization of the celiac artery and SMA as well as their early branches (Figs. 133.2 and 133.3). CT also may exclude or identify alternate causes of abdominal pain including perforation and obstruction and can provide some information as to bowel perfusion and edema, further guiding intervention (Table 133.1). While a traditional CTA is sufficient to allow assessment of the visceral vessels, a biphasic CTA, including an arterial phase and delayed phase, can improve sensitivity by allowing time-based visualization of the portal venous system and bowel wall perfusion. Findings

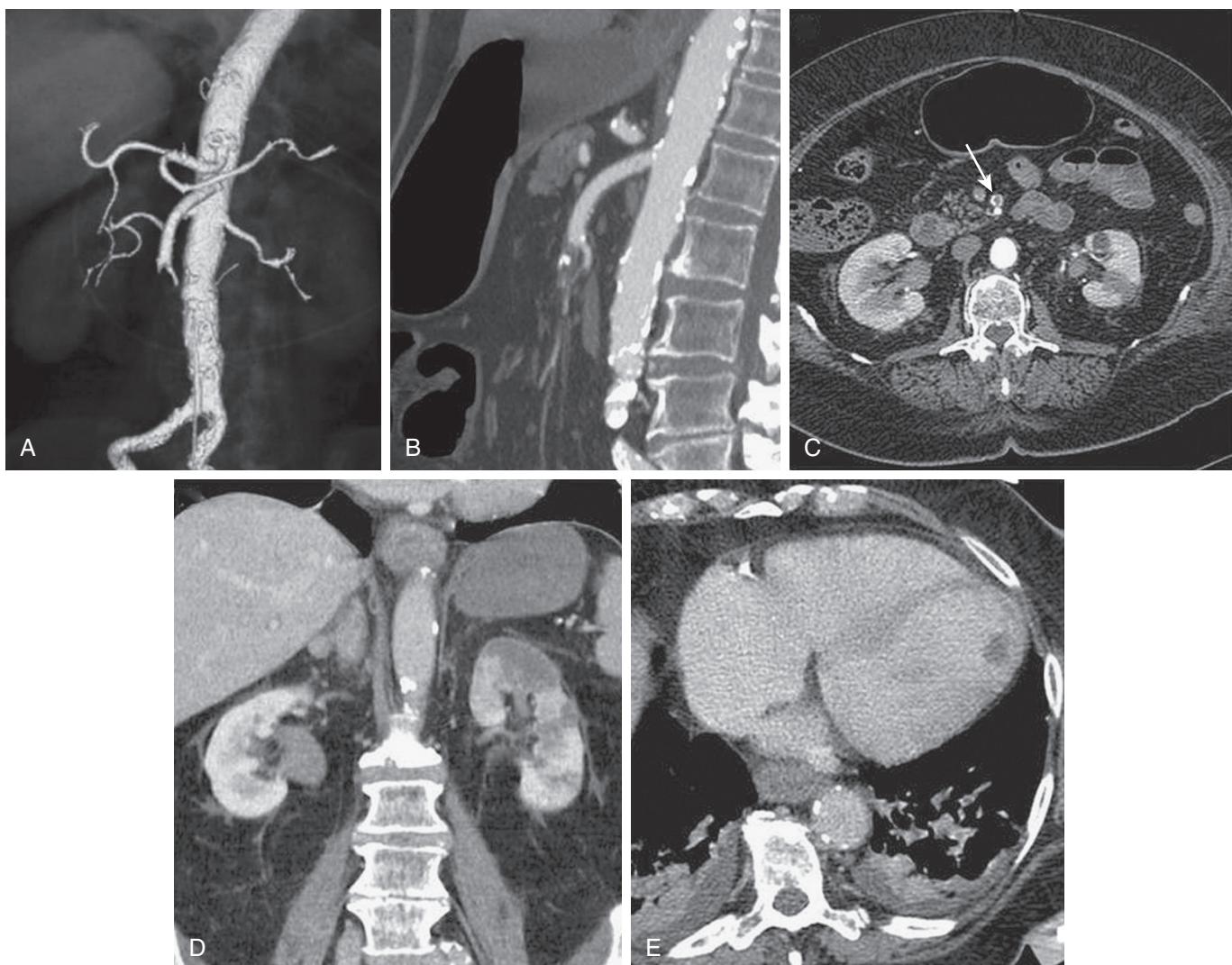


Figure 133.2 (A) Three-dimensional volume rendering of arterial-phase multidetector computed tomographic angiography (MDCTA) shows an abrupt mid-superior mesenteric artery (SMA) occlusion consistent with embolus. (B) Sagittal multiplanar reformat shows the same occlusion. (C) SMA occlusion seen on an axial CTA slice (arrow). (D) Coronal multiplanar reformat reveals left kidney infarction. (E) Transverse portal venous-phase MDCTA depicts thrombus in the left ventricle as the likely embolic source. (From Aschoff AJ, Stuber G, Becker BW, et al. Evaluation of acute mesenteric ischemia: accuracy of biphasic mesenteric multi-detector CT angiography. *Abdom Imaging*. 2009;34:345–357.)



Figure 133.3 Computed tomographic angiography sagittal multiplanar reformat shows an acute thrombotic occlusion of the proximal superior mesenteric artery. Bowel resection was performed 3 days before this image was obtained. The concomitant celiac stenosis suggests an acute-on-chronic presentation of acute mesenteric ischemia. (From Aschoff AJ, Stuber G, Becker BW, et al. Evaluation of acute mesenteric ischemia: accuracy of biphasic mesenteric multi-detector CT angiography. *Abdom Imaging*. 2009;34:345–357.)

such as changes in bowel wall thickness, pneumatosis, portal venous gas, mesenteric venous thrombosis, and mucosal or bowel wall enhancement patterns all suggest intestinal compromise and can be well assessed using CTA (Fig. 133.4A–C).

Magnetic Resonance Angiography

Magnetic resonance angiography (MRA) is an additional imaging modality that can incorporate both functional and anatomic evaluations of the mesenteric vasculature. This valuable tool can accurately evaluate for vascular stenosis and other nonatherosclerotic vascular pathologies such as fibromuscular dysplasia. It also has the benefit of avoiding the risk of ionizing radiation and nephrotoxicity associated with iodinated contrast agents. However, MRA is a lengthy study that requires significant post-processing, and is often not readily available for rapid emergency evaluation of these critically ill patients. In addition, the secondary signs of acute mesenteric ischemia, such as indurated fat or bowel wall thickening, which are routinely delineated by CT, are more difficult to assess with MRA.

Alternative Imaging Modalities

Although traditional catheter-based arteriography (Fig. 133.5) has been replaced by CTA as the definitive diagnostic study for acute mesenteric ischemia, its utility in the treatment of patients with both chronic and acute mesenteric ischemia has increased dramatically in recent years through the use of endovascular and hybrid approaches to the management of these patients. Additionally, while rarely performed due to the efficacy and availability of high quality CT imaging, angiography may have a limited role in identifying NOMI in patients where

TABLE 133.1 Mesenteric CT Angiography Findings of Acute Mesenteric Ischemia

Finding	Patients with AMI (n = 26)	Control Group (n = 36)	Sensitivity (%)	Specificity (%)
Pneumatosis intestinalis	11	0	42	100
SMA or combined celiac and IMA occlusion ^a	5	0	19	100
Arterial embolism	3	0	12	100
SMA or portal venous gas	3	0	12	100
Focal lack of bowel wall enhancement	11	1	42	97
Free intraperitoneal air	5	2	19	94
Superior mesenteric or portal venous thrombosis	4	2	15	94
Solid organ infarction	4	2	15	94
Bowel obstruction	3	2	12	94
Bowel dilation	17	6	65	83
Mucosal enhancement	12	7	46	81
Bowel wall thickening	22	10	85	72
Mesenteric stranding	23	14	88	61
Ascites	19	24	73	33

^aPatients with both celiac and IMA occlusion also had evidence of distal disease in the SMA distribution. AMI, acute mesenteric ischemia; IMA, inferior mesenteric artery; SMA, superior mesenteric artery.

From Kirkpatrick ID, Kroeker MA, Greenberg HM. Biphasic CT with mesenteric CT angiography in the evaluation of acute mesenteric ischemia: initial experience. *Radiology*. 2003;229:91–98.

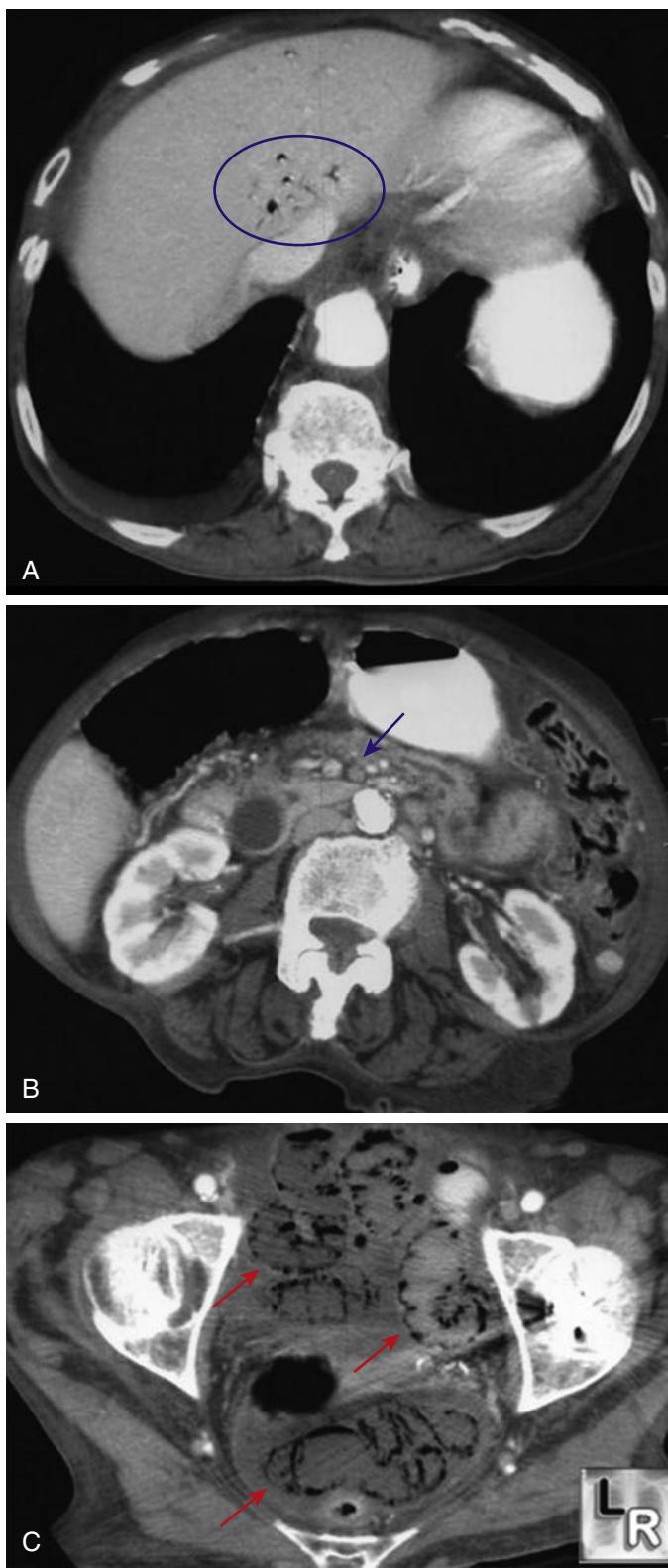


Figure 133.4 (A) Computed tomographic angiography demonstrating hepatic venous air (circle). (B) The superior mesenteric artery is occluded (arrow). (C) There is extensive colonic pneumatosis and ascites (arrows). (From: <http://www.learningradiology.com/notes/ginotes/mesentericischemiapage.htm>.)

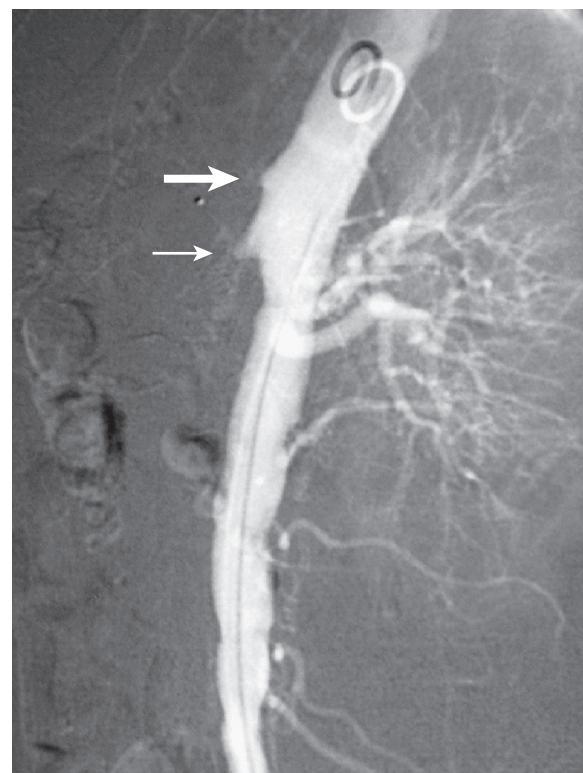


Figure 133.5 Lateral aortogram of a patient with acute mesenteric ischemia due to superior mesenteric artery thrombosis (small arrow). Note the chronic occlusion of the celiac artery (large arrow).

the diagnosis may be of question. Classic features of NOMI can be subtle. However, four arteriographic criteria have been described for the diagnosis of mesenteric vasospasm: (1) narrowing of the origins of multiple branches of the SMA; (2) alternate dilation and narrowing of the intestinal branches – the “string of sausages” sign (Fig. 133.6); (3) spasm of the mesenteric arcades; and (4) impaired filling of the intramural vessels (Fig. 133.7).²⁹

Despite being frequently ordered as the initial study for abdominal pain, abdominal radiographs are neither sensitive nor specific for the diagnosis of acute mesenteric ischemia and are frequently normal. Findings such as ileus or bowel wall edema (“thumb printing”) and pneumatosis may be seen in abdominal radiographs; however, their role in diagnosing mesenteric ischemia is limited.

Duplex ultrasonography is a frequently used and valuable tool for the assessment of visceral vessels. It accurately identifies high-grade stenosis of the celiac artery and SMA and may also identify alternative sources of abdominal pain such as biliary pathology. However, in the acute setting its role is limited by its inability to assess for emboli beyond the proximal segment and intestinal malperfusion. Additionally, optimal evaluation requires a fasting state with a highly trained technologist specializing in vascular lab assessment, which may not be available at all hospitals or during off-hours. Nonetheless, arterial duplex remains the noninvasive diagnostic study of choice in the initial workup of chronic mesenteric ischemia.

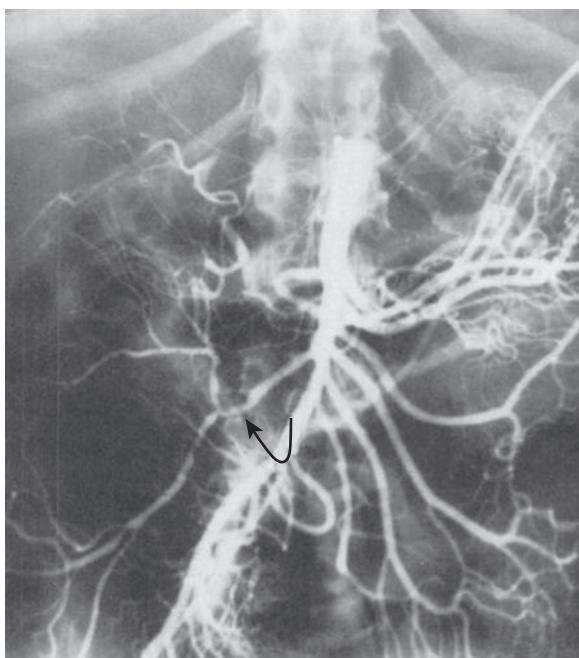


Figure 133.6 Selective superior mesenteric artery arteriogram in a patient with nonocclusive mesenteric ischemia. Note the “string of sausages” appearance of some of the ileocolic branches (arrow). (From Clark RA, Colley DP, Jacobson ED, et al. Superior mesenteric angiography and blood flow measurement following intra-arterial injection of prostaglandin E1. *Radiology*. 1980;134:327–333.)

TREATMENT

The treatment of acute mesenteric ischemia involves early revascularization for patients with embolic and thrombotic disease, source control through resection of necrotic bowel, frequent second look evaluation of bowel, and critical care management including aggressive resuscitation. The latter is of the utmost importance in patients with NOMI for whom revascularization is not indicated.

Initial Resuscitation and Critical Care

Patients with acute mesenteric ischemia often present with sepsis and early sequela of organ dysfunction. While critical care is a crucial component to management, revascularization and source control through resection of ischemic bowel should not be delayed in an effort to obtain normal physiologic and laboratory parameters. Resuscitation of a patient with acute mesenteric ischemia should begin immediately upon diagnosis with crystalloid solution and correction of electrolyte imbalances. Simultaneously, broad-spectrum antibiotics should be administered early to assist with management of sepsis from translocated bacteria and ischemic bowel. Finally, therapeutic intravenous heparin should also be administered to prevent further propagation of thrombosis. In patients with hypotension, vasopressors may be necessary but should

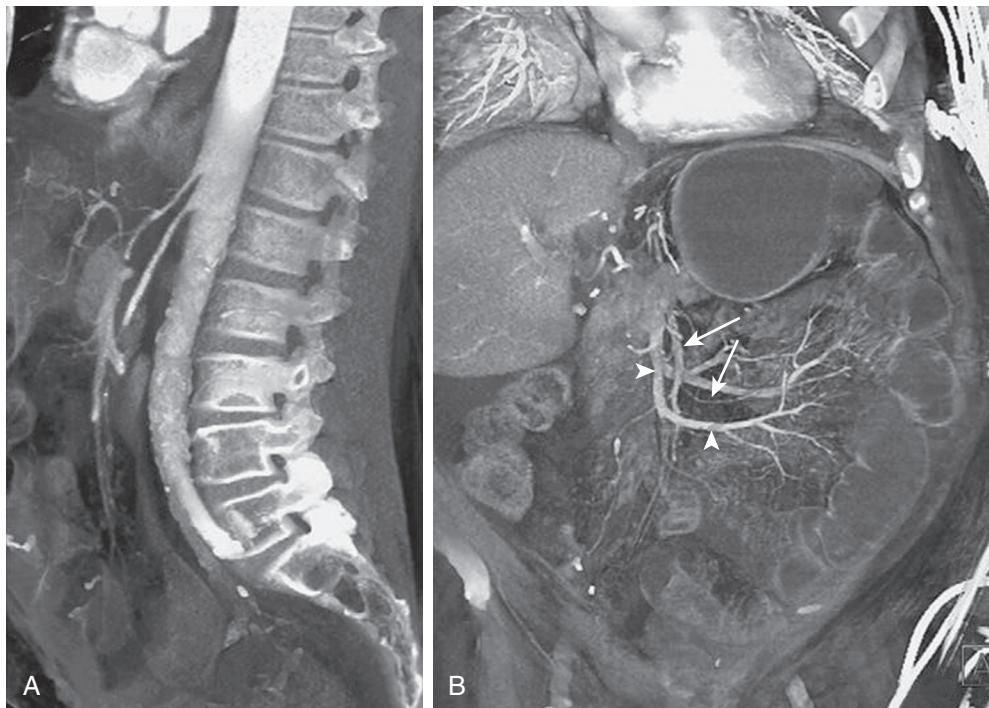


Figure 133.7 Computed Tomographic Angiography Appearance of Nonocclusive Mesenteric Ischemia. (A) Sagittal volume-rendered image shows diffuse narrowing of the main celiac and superior mesenteric arteries. (B) Coronal oblique volume-rendered image of the same patient shows pruning of the superior mesenteric artery branches (arrows). Note the dilated fluid-filled small intestine and the superior mesenteric vein (arrowheads). (From Horton KM, Fishman EK. Multidetector CT angiography in the diagnosis of mesenteric ischemia. *Radiol Clin North Am*. 2007;45:275–288.)

be administered in conjunction with aggressive fluid resuscitation, as splanchnic vasoconstriction may worsen ischemia in marginally viable bowel and exacerbate visceral vasospasm. If vasopressors are absolutely necessary to improve cardiac function, those with less impact on splanchnic flow including low-dose dopamine, dobutamine, and milrinone should be selected.³⁰ Pure alpha-adrenergic agents should be avoided if possible, even after successful revascularization.³⁰

While surgical management of embolic and thrombotic etiologies of mesenteric ischemia is critical to patient survival, the prognosis for patients with NOMI is poor despite the absence of organic obstruction in the principal arteries. Moreover, given a lack of vascular occlusion, the primary treatment for NOMI is the correction of the underlying cause including optimization of cardiac output, decreasing pressor requirements, hold offending medications such as digoxin and cardiac glycosides, as well as supportive care. While the use of catheter-directed vasodilators, including prostaglandin analogs and papaverine, have been reported to improve mortality in small studies and case reports, conflicting evidence and the risk for further hypotension in already clinically labile patients has prohibited this treatment from becoming standard practice.^{31,32} Moreover, animal models have suggested that infusion of vasodilators may actually be detrimental to intestinal viability. Therefore, in patients with NOMI operative exploration is typically reserved for symptoms and laboratory values that suggest the presence of gangrenous bowel that requires resection.

Open Surgical Treatment

The mainstay of treatment for patients with embolic or thrombotic mesenteric ischemia is rapid revascularization and assessment of the bowel with resection of any frankly necrotic bowel. This differs from NOMI, which is managed primarily with aggressive medical care as discussed above. Since 2000, the frequency of surgical treatment of acute mesenteric ischemia has increased nearly twofold from 2.6 to 4.0 cases per million population, driven primarily by a threefold increase in endovascular intervention, while open revascularization procedures have remained stable.⁷ Importantly, this increase in surgical revascularization has been associated with a congruent decline in population mortality from acute mesenteric ischemia over the same time period.

Embolectomy

Embolectomy of the visceral vessels is an important approach to revascularization whose frequency has not declined despite advances in endovascular treatment. Anterior exposure for SMA embolectomy is achieved by elevating the omentum and transverse colon and retracting the small intestine to the patient's right. A horizontal incision is made in the peritoneum at the base of the transverse mesocolon (see Fig. 133.8, dotted line). Careful dissection in the mesentery initially uncovers venous tributaries of the superior mesenteric vein, autonomic nerve fibers, and small lymphatic vessels that are divided to gain exposure of the SMA, which lies to the left of the superior mesenteric vein. Exposure of the more proximal segments

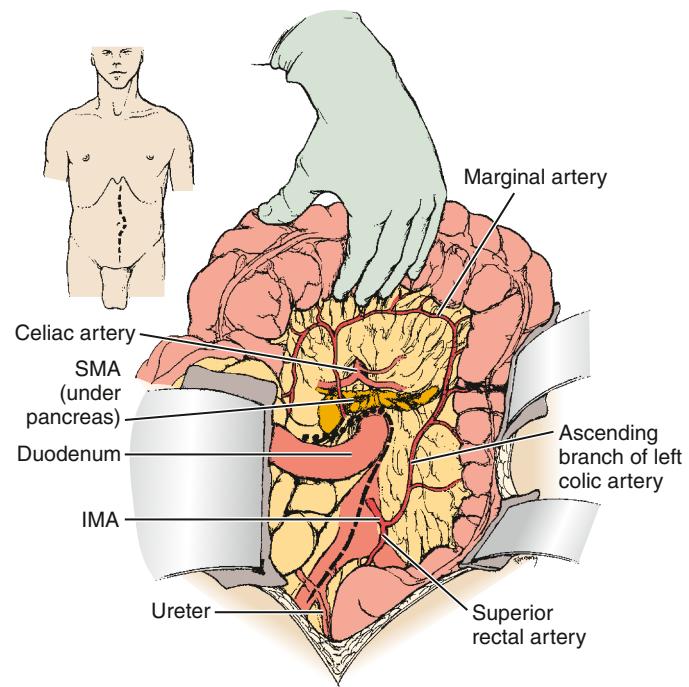


Figure 133.8 Operative Exposure of the Infra-Pancreatic Superior Mesenteric Artery (SMA). IMA, inferior mesenteric artery. (From Kazmers A. Operative management of acute mesenteric ischemia. *Ann Vasc Surg*. 1998;12:187–197.)

is possible by judicious mobilization of the inferior pancreatic border, the nearby splenic vein, and its tributaries from the pancreas.

After systemic heparinization, the artery is opened transversely (Fig. 133.9) in a segment of sufficient diameter to allow direct repair with interrupted sutures. For diminutive or atherosclerotic vessels, a short longitudinal arteriotomy with patch closure with vein or bovine pericardium may be considered. The proximal SMA is vented to allow any clot to be expelled without the use of an embolectomy catheter if possible. When necessary, catheter embolectomy is typically performed with a 3- to 5-F balloon catheter proximally, and a smaller Fogarty balloon catheter distally. With extraction of the embolus, torrential, pulsatile inflow should be expected. If the balloon embolectomy catheter cannot be passed distally due to vessel diameter or atherosclerotic disease, surgeons may place a hand on either side of the mesentery and "milk" thrombotic material out of the vessels. When all thrombus has been removed, the arteriotomy is closed primarily with interrupted sutures or with a patch if necessary and flow is reestablished. If distal thrombus is not completely removed, 0.5–1 mg of tissue plasminogen activator (TPA) can be infused through the arteriotomy to the distal vessels.

Bypass

The superior mesenteric artery bypass is most commonly performed for chronic mesenteric ischemia; however, its role remains important for patients with acute mesenteric ischemia due to thrombotic disease. In these patients proximal or long segments of atherosclerotic disease may prevent adequate or safe revascularization from embolectomy or stenting alone.

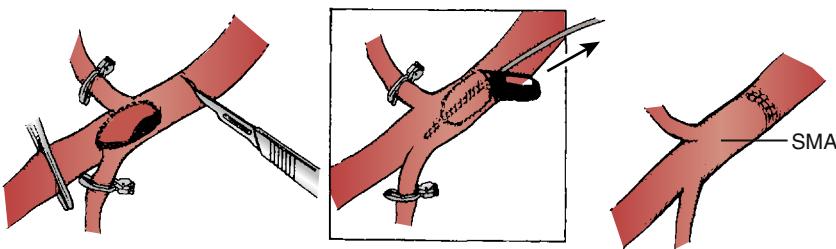


Figure 133.9 Traditional Transverse Arteriotomy for Superior Mesenteric Artery (SMA) Embolectomy. (From Kazmers A. Operative management of acute mesenteric ischemia. *Ann Vasc Surg*. 1998;12:187–197.)

Unlike embolectomy, when bypass is planned, the lateral portion of the SMA is exposed, rather than the more limited exposure provided by a strictly anterior approach. The peritoneum is opened lateral to the duodenum, anterior to the aorta, and onto the left or right common iliac artery (see Fig. 133.8, *dashed line*). Several combinations of graft orientation, inflow vessel, and conduit may be considered with the decision influenced largely by the suitability of potential inflow vessels for a proximal anastomosis and by the presence or absence of necrotic bowel, respectively.

The choice of inflow vessel is influenced by the degree of atherosclerosis and occlusive disease present in the inflow vessels, the overall lie of the graft, as well as the patient's overall physiologic state. In critically ill patients the hemodynamic shifts and physiologic insult associated with an aortic cross-clamp typically preclude an aortic inflow source. Rather in the emergent setting a common iliac inflow source provides a preferable option. In this situation, most surgeons prefer a retrograde graft orientation with its origin from the right common iliac artery in a "lazy C" configuration (Fig. 133.10). This avoids aortic clamping and usually provides a good lie to prevent kinking. However, when iliac disease precludes this orientation a left iliac artery or distal infrarenal aorta may be considered. With any of these configurations the graft lie is improved by increasing the graft length and performing an end-to-end graft-to-SMA anastomosis, as pictured in Figure 133.11. Alternatively, a very short retrograde bypass using a larger-diameter graft (8–10 mm) can be configured from the immediately infrarenal aorta (Fig. 133.12). When distal inflow sources are unable to be clamped, heavily diseased, or aneurysmal, an antegrade bypass can be considered. Advantages of the antegrade bypass include the fact that the supraceliac aorta is often relatively free of disease and that the straighter graft orientation is less prone to kinking. Dissection of the supraceliac aorta is technically more demanding, requires more time, and adds the hemodynamic and physiologic insult of a proximal aortic cross-clamp if a partially occluding clamp is not possible. It should be acknowledged that most side-biting clamps placed to allow sufficient access for creation of an anastomosis will still functionally occlude the aorta.

The choice of graft conduit is challenging and multifaceted. In patients without evidence of necrotic bowel, synthetic bypass grafts of 6- to 8-mm Dacron or externally supported polytetrafluoroethylene are commonly selected due to the ease of size match, handling, kink resistance, and the avoidance of the extended operative time needed for vein

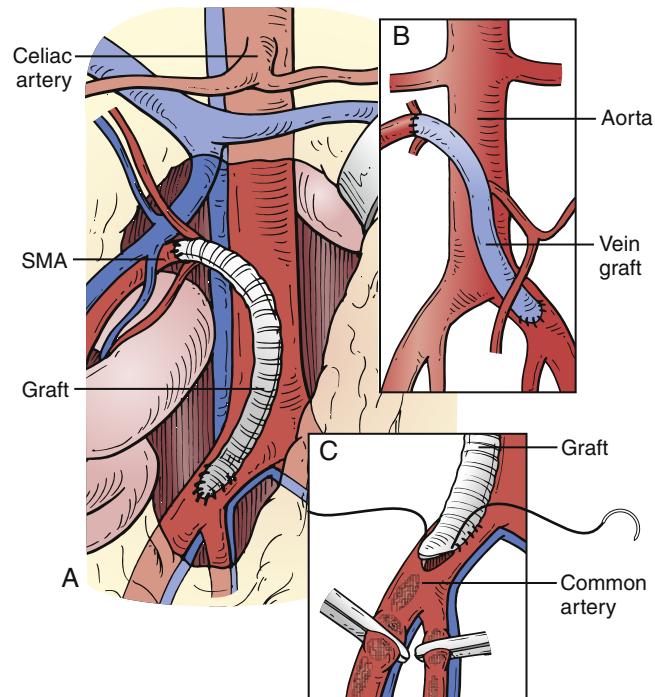


Figure 133.10 Optimal Orientation for Retrograde Superior Mesenteric Artery (SMA) Bypass. (A) Prosthetic. (B) Vein. (C) Proximal iliac graft anastomosis. (From Kazmers A. Operative management of acute mesenteric ischemia. *Ann Vasc Surg*. 1998;12:187–197.)

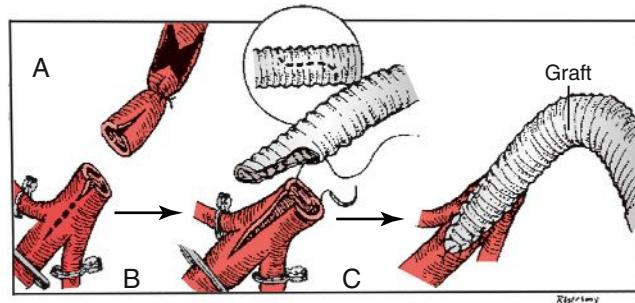


Figure 133.11 (A–C) Creation of a beveled, end-to-end anastomosis.

harvest. However, in the setting of abdominal contamination due to a bowel resection, perforation, or necrosis, autologous vein is preferred if suitable in size and quality, with greater saphenous and femoral vein most commonly utilized. Importantly, caution must be used in fashioning vein grafts because their smaller diameter and thin-walled nature make them

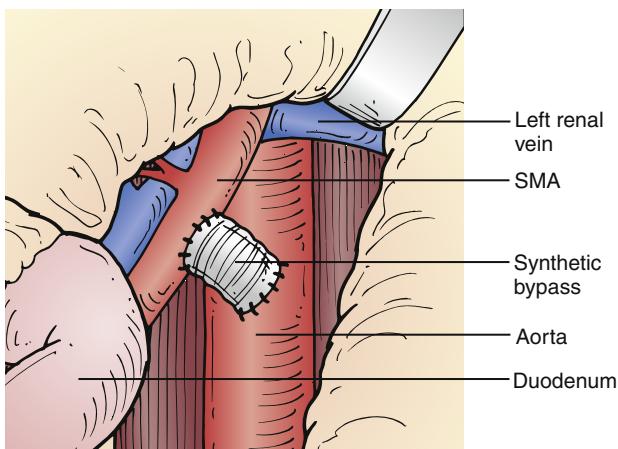


Figure 133.12 Short retrograde aorta–superior mesenteric artery (SMA) bypass. (From Valentine RJ, Wind GG. *Anatomic Exposures in Vascular Surgery*, 2nd ed. Philadelphia: Lippincott Williams and Wilkins; 2003:281.)

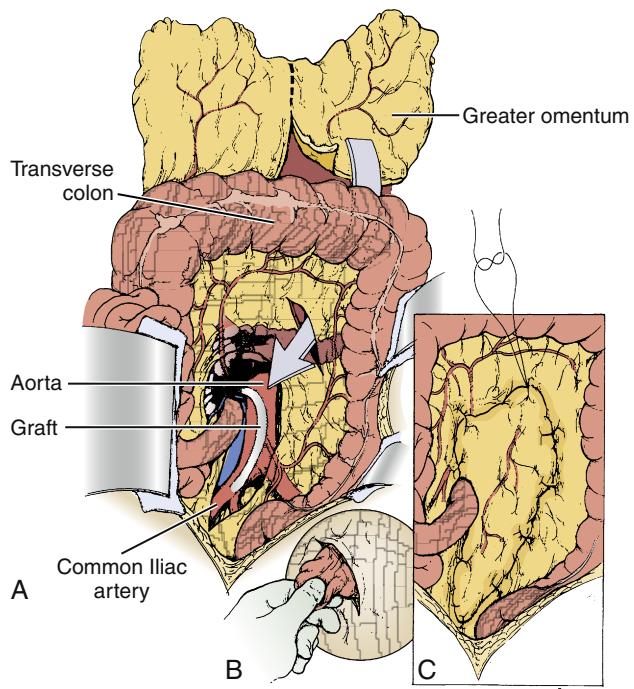


Figure 133.13 (A–C) Technique for omental flap coverage of a retrograde superior mesenteric artery bypass. (From Kazmers A. Operative management of acute mesenteric ischemia. *Ann Vasc Surg*. 1998;12:187–197.)

more prone to kinking and external compression than synthetic grafts. While few studies have evaluated the long-term effects of mesenteric graft infection, it can be presumed that such occurrence risks catastrophic consequences and therefore prosthetic material should be avoided in contaminated abdomens if possible. Omental coverage should be used in all cases when available, either to cover the prosthetic graft, or to buttress the vein graft to decrease the risk of kinking. Kazmers and colleagues illustrate a useful technique to provide coverage of a retrograde bypass by bringing an omental flap through the transverse mesocolon (Fig. 133.13).³³

Endovascular Management

Endovascular treatment for mesenteric ischemia has been well described for subacute or chronic presentations, especially in patients at high operative risk or as a bridge to elective surgical bypass after the acute illness has resolved.^{34,35} The endovascular approach was not historically applied to patients with acute mesenteric ischemia who require emergent revascularization. However, endovascular treatment of acute mesenteric ischemia has expanded dramatically since 2000 and now surpasses open surgery as the most prevalent revascularization option for acute mesenteric ischemia.⁷ While likely multifactorial in nature, this increase was associated with a concurrent decline in mortality from acute mesenteric ischemia during the same time period. Endovascular treatment options include thrombolysis, pharmacomechanical embolectomy, angioplasty, and stenting. Although successfully described in the literature, the use of chemical thrombolysis alone for true acute mesenteric ischemia is risky due to the time required to achieve adequate result and risk to further bowel necrosis during that period. Moreover, the ongoing use of tissue plasminogen activator (TPA) is contraindicated due to bleeding risk in those with open abdomen, recent surgery, and need for bowel resection or further surgery, precluding nearly all patients with acute mesenteric ischemia (see Ch. 43, Thrombolytic Agents). Even when a laparoscopic or open surgery can be avoided, TPA can exacerbate bleeding from friable or ischemic intestinal mucosa. Moreover, studies with successful use of thrombolysis caution that this was used as an adjunctive therapy to eliminate residual thrombus burden after an initial procedure for acute mesenteric ischemia.³⁶

Percutaneous stenting is another highly effective and safe modality for mesenteric ischemia. It provides rapid restoration of flow in a minimally invasive fashion and is particularly helpful to improve inline flow to the visceral vessels in patients with underlying proximal stenosis. This technique can be performed in antegrade or retrograde fashion (described below) (Fig. 133.14). Recently, utilization of covered stents has been advocated for the treatment of chronic mesenteric ischemia due to higher long-term patency and lower rates of reintervention as compared to bare metal stents, likely due to more aggressive dilation in the setting of a covered stent.³⁷ However, in the setting of necrotic bowel, the potential benefits of a covered stent should be weighed again the risk of stent graft infection. We would rarely recommend use of angioplasty alone; however it may be suitable for patients with occlusion or stenosis of a previously placed stent.

Percutaneous or basket embolectomy may be used in the acute setting using filters designed for carotid stenting beyond the embolism to retract and capture the embolism. A similar approach using pharmacomechanical thrombectomy provides the benefit of rapid treatment of the disease process using a short burst of lysis followed by mechanical or suction thrombectomy. Both approaches provide the benefit of a less invasive endovascular approach without the risk of prolonged catheter-directed thrombolysis and can be advantageous. However, such approaches may also lead to distal embolization, which can be challenging to treat. While multiple small studies

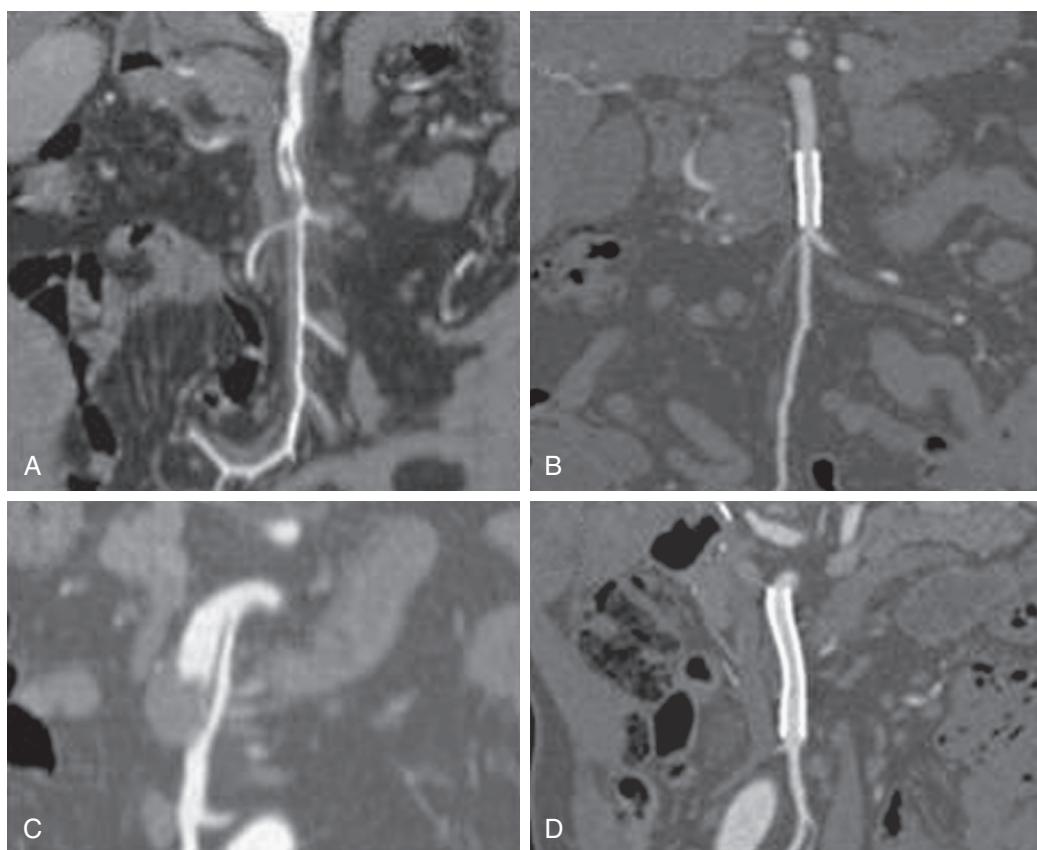


Figure 133.14 Reconstructed Computed Tomography Angiography (CTA) Findings Indicated for Primary Endovascular Stenting. (A) Severe compression of the true lumen and (B) a patent superior mesenteric artery stent and complete obliteration of the false lumen at 60-month follow-up CTA in patient no. 12. (C) Large dissecting aneurysm (20.0 mm in diameter) and (D) near complete disappearance of the aneurysm at 36-month follow-up CT in patient no. 13. (From Min SI, Yoon KC, Min SK, et al. Current strategy for the treatment of symptomatic spontaneous isolated dissection of superior mesenteric artery. *J Vasc Surg*. 2011;54(2):461–466.)

suggest the efficacy and safety of these techniques, large studies have not been performed to assess the outcomes of this approach.^{38,39}

Endovascular techniques are less common historically, but recent reports indicate improved morbidity and mortality with this strategy. Nonetheless, careful patient selection is necessary, with the highest reductions in mortality identified in those patients with thrombotic rather than embolic disease.³⁶ Finally, an important consideration for all patients treated with endovascular surgery is the importance of bowel assessment, which should not be neglected despite the minimally invasive nature of endovascular intervention. All patients with possible bowel ischemia should be assessed with laparoscopy or laparotomy.

Hybrid Procedure: Retrograde Open Mesenteric Stenting

Milner et al. and Wyers et al. have reported a hybrid open-interventional approach for the treatment of acute atherosclerotic SMA thrombosis that involves an efficient, less invasive mesenteric revascularization.^{40,41} Retrograde open mesenteric stenting (ROMS) is performed through a laparotomy incision with subsequent proximal and distal control of the mesenteric

vessel. The vessel, most commonly the SMA, is then palpated for calcification and accessed utilizing retrograde approach using a micropuncture needle and wire, which is then upsized to a stiffer wire and a 6- or 7-F sheath is placed. A hand injection is performed to identify the area of disease as well as the aortic origin and a balloon expandable stent is placed, and a completion angiogram performed. Closure can be performed primarily, or with vein or bovine pericardial patch angioplasty with or without concurrent thromboendarterectomy of the access site (Figs. 133.15 and 133.16). In a multicenter study from the Vascular Low Frequency Disease Consortium (VLFDC), this technique has been shown to be a highly successful (98%) safe alternative to surgical bypass or percutaneous stenting with primary and secondary patency of 76% and 90%, respectively, at 2 years.⁴² This approach may be of particular value in patients who have flush occlusions, or other anatomical features prohibit percutaneous stent or bypass such as tortuosity or calcification. In addition, the ability to place a stent intraluminally during exploratory laparotomy identifying necrotic bowel can have less risk of infection compared to prosthetic bypass.

Importantly, much like percutaneous stenting, recurrent stenosis after ROMS occurs frequently and therefore close duplex surveillance should be performed following intervention,

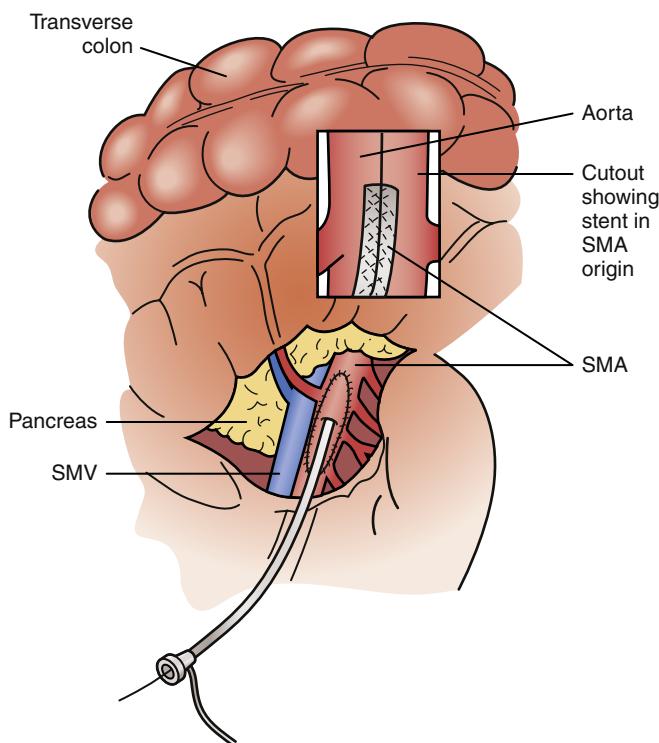


Figure 133.15 Retrograde open mesenteric stenting with a long 6-F sheath placed through the patched superior mesenteric artery (SMA). The inset illustrates a stent deployed in the proximal SMA. SMV, superior mesenteric vein.

at 6 months, 12 months, and annually thereafter. Most patients with recurrent stenosis can be retreated with a percutaneous approach as outpatients. Many of these patients remain poor operative candidates and have limited life expectancies because of comorbidities.⁴³

Assessment of Bowel Viability and Second Look Surgery

An important consideration for all patients with acute mesenteric ischemia is the timing of bowel assessment and revascularization. For the vast majority of patients, revascularization should be performed first to allow time for the bowel to regain perfusion and better assess viability. The exception to this practice is those patients with frank perforation or evidence of free air on imaging studies. In these patients, resection of the shortest possible segment necessary to achieve source control should be performed immediately to decrease septic burden. Revascularization should then be undertaken, and the bowel should be reassessed after reperfusion is established. The alternative consideration is those patients in whom complete bowel necrosis is suspected. This is a non-survivable insult, and in these patients revascularization is not warranted, rather the abdomen should be explored then closed if complete bowel death is identified and the patient allowed to pass without additional interventions.

Following revascularization, bowel assessment may be done either with laparoscopy or exploratory laparotomy. In those patients who have already undergone an open revascularization,

the bowel should be revascularized first, and then assessed for viability and possible resection through the open abdomen. However, for those treated with an endovascular approach, a skilled surgeon may easily assess the bowel using a laparoscopic approach with a satisfactory completion angiogram precluding the need for tactile evaluation of the vasculature. Alternatively, a traditional laparotomy may be utilized for assessment of bowel viability, with the approach dictated by surgeon preference. Regardless of approach, frankly dead or necrotic bowel should be resected. However, great efforts should be undertaken to preserve as much bowel as possible, and bowel with questionable viability should not be aggressively resected up front.

Second look surgery with a temporary abdominal closure device or laparoscopic re-exploration in 24–36 hours, or sooner with any clinical deterioration, has an important role in the management to avoid catastrophic consequences of short gut from excessive small bowel resection. Temporary abdominal closures allow for easy assessment and return to the operating room in a rapid fashion, and should be used for patients with questionable bowel viability or in whom further ICU resuscitation is necessary for systemic sepsis or hemodynamic instability. In these patients leaving the bowel in discontinuity with a planned return to the operating room for a second look and delayed reconstruction is recommended to preserve maximal bowel length.

OTHER CONSIDERATIONS

Intraoperative Vasodilators

Splanchnic vasospasm can occur following mesenteric intervention and may contribute to continued bowel ischemia after a successful revascularization. Low flow after ischemia can also result from relative hypovolemia and septic shock, and therefore aggressive fluid resuscitation and minimization of vasoconstricting pressors is advisable. To treat local vasospasm in the mesenteric arcades, papaverine can also be administered selectively in the SMA during open or endovascular intervention. Alternatively, intravenous glucagon administration increases cardiac output and flow to all layers of the small and large intestine as well as the liver while inhibiting gastrointestinal motility and secretory function. This has been shown to improve survival in an animal model, but has not been tested extensively in humans.^{44,45} Moreover, vasodilators may actually be counter-productive by worsening hypotension and therefore should be used with caution and coupled with additional volume resuscitation to avoid vasodilation-mediated hypotension.

Long-Term Surveillance

Long-term surveillance after mesenteric intervention is recommended by the Society of Vascular Surgeons clinical practice guidelines due to the risk of death and mesenteric ischemia from failed intervention. Clinical follow-up alone is not sufficient to identify failing mesenteric reconstructions,

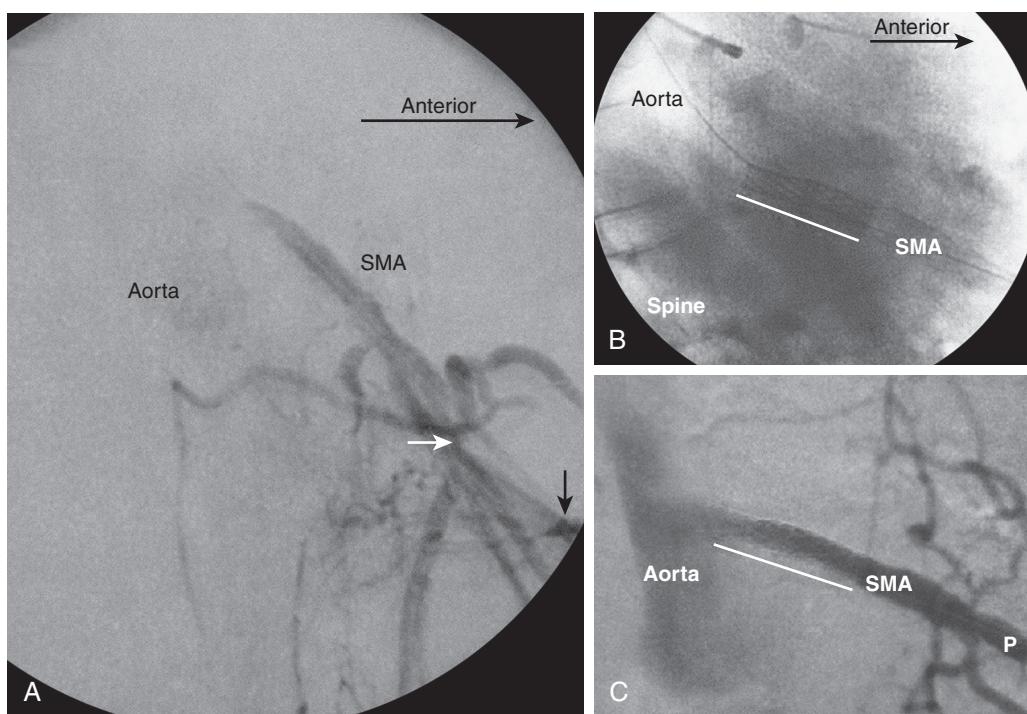
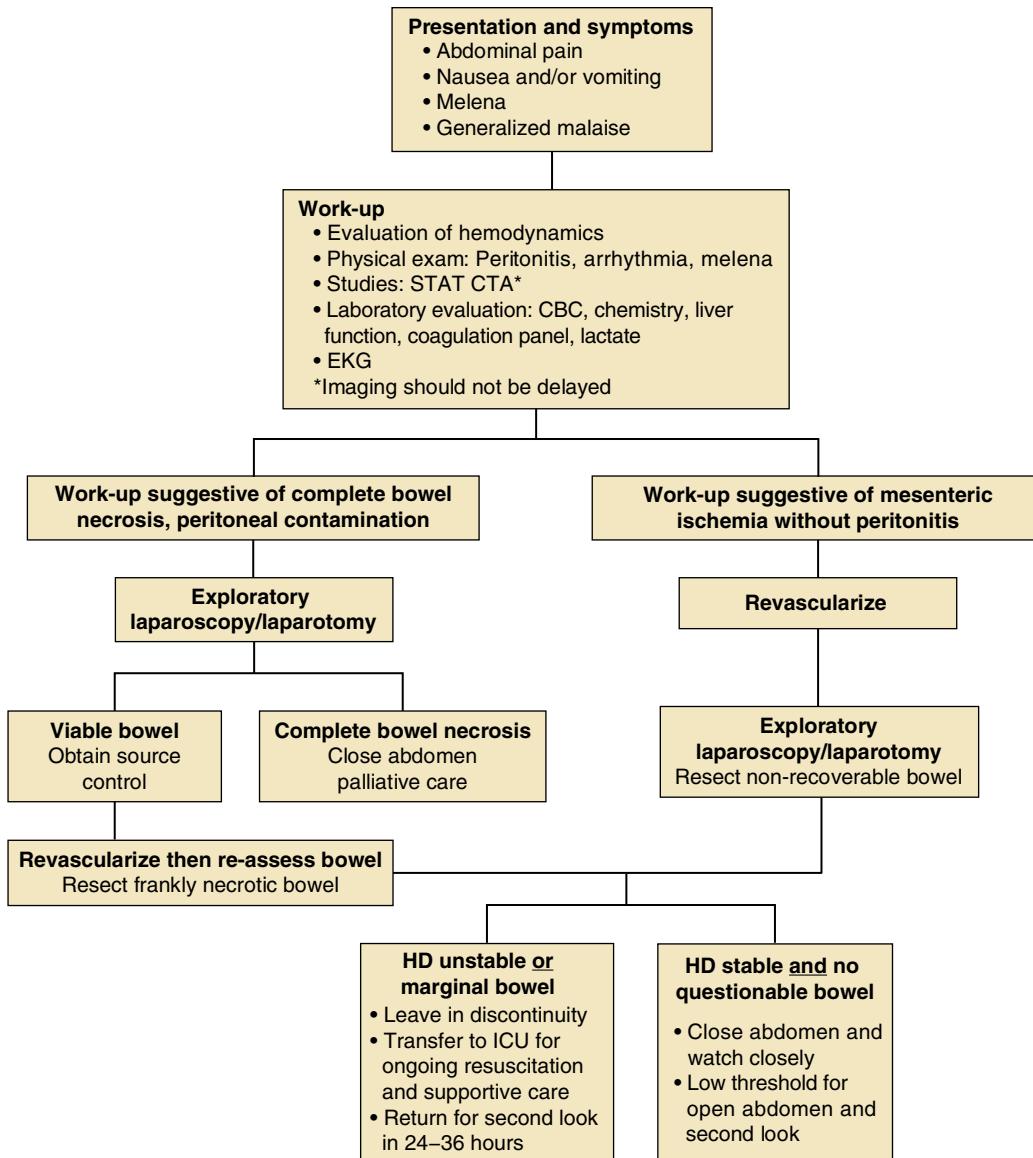


Figure 133.16 Retrograde Open Mesenteric Stenting. (A) Intraoperative arteriogram during retrograde superior mesenteric artery (SMA) injection. Note the proximity of the sheath's point of entry (black arrow) and the sheath's tip (white arrow) to the proximal SMA occlusion. There is no reflux of contrast material into the aorta. (B) Intraoperative lateral fluoroscopic image of two stents (underscored by a white line) deployed in the SMA origin with the 0.018-in wire still in place. Note the lumbar vertebral bodies to the left. (C) Completion retrograde arteriogram with free reflux of contrast material into the aorta and no residual angiographic stenosis. P, approximate location of SMA patch angioplasty. (From Wyers MC, Powell RJ, Nolan BW, Cronenwett JL. Retrograde mesenteric stenting during laparotomy for acute occlusive mesenteric ischemia. *J Vasc Surg*. 2007;45:269–275.)

therefore imaging is necessary.⁴⁶ Visceral duplex ultrasound is the optimal imaging modality for surveillance after both open and endovascular intervention. However, reliable ultrasound remains technologist-dependent and may not be universally available. There are no standardized criteria to diagnosis restenosis after reconstruction and studies have shown traditional criteria used to identify stenosis of the SMA and celiac artery may overestimate the degree of stenosis in a bypass or stent. It is possible that elevated velocities may also represent residual stenosis or calcification.⁴⁷ Therefore, given the potential danger of restenosis, current guidelines recommend duplex ultrasound to establish a new

baseline velocity following intervention as well as 6 months, 12 months, and annually thereafter. Patients with recurrent symptoms of acute or chronic mesenteric ischemia should undergo contrast-imaging studies to better assess the mesenteric vasculature. Intravascular ultrasound or pressure measures may be considered to better assess the degree of stenosis when elevated velocities are identified following intervention. Finally, despite limited evidence, contrast studies are also recommended for those patients with a substantial velocity increase from post-treatment baseline as well as a peak systolic velocity greater than 370 cm/s in the celiac artery and 420 cm/s in the superior mesenteric artery.⁴⁶

CHAPTER ALGORITHM



Algorithm for Diagnosis and Management of Acute Mesenteric Ischemia

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Chronic Mesenteric Ischemia: Epidemiology, Pathophysiology, Clinical Evaluation, and Management

MARK F. CONRAD

Based on a previous edition chapter by Gustavo Oderich and Mauricio Ribeiro

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INTRODUCTION

Clinically significant chronic mesenteric ischemia (CMI) is a relatively uncommon disease that was characterized by G.H Goodman in 1918 as “abdominal angina.”¹ In contemporary practice, the term *intestinal angina* is used to describe the classic symptom of chronic abdominal pain that occurs after meals that eventually leads to food fear and weight loss, which is the

hallmark of CMI. Current estimates indicate that CMI accounts for less than 1 per 100,000 hospital admissions in the United States and less than 2% of all admissions for gastrointestinal conditions.² Shaw and Maynard described the first successful mesenteric endarterectomy in 1958 and since then, techniques of revascularization have greatly evolved.³ With the evolution of less invasive endovascular techniques, primary mesenteric stenting has become the most frequently utilized

initial treatment for CMI, with open surgery reserved for patients who fail endovascular therapy or have complex lesions not amenable to endovascular intervention.⁴ This chapter provides a comprehensive review of the clinical evaluation, treatment options, and results of mesenteric revascularization for patients with CMI.

PATOPHYSIOLOGY

Approximately 20% of the cardiac output goes through the mesenteric arteries under fasting conditions and this can increase to 35% after a meal.⁵ Blood flow to the gastrointestinal tract increases drastically shortly after food is ingested and can reach levels approaching 100% to 150% of normal (2000 mL/min) that is sustained over the next 3 to 6 hours.⁶ Postprandial intestinal hyperemia is a locally mediated vascular response to the presence of digested food products in the lumen. The degree of response is dependent upon the makeup of the meal as fatty acids and products of protein digestion lead to vasodilation and produce profound and sustained intestinal hyperemia.⁷

Patients with CMI become symptomatic when the combined primary and collateral circulation is inadequate to provide the postprandial hyperemic response that is required to supply oxygen for the metabolic processes of secretion and absorption, and for increased peristaltic activity. Poole et al. showed that when splanchnic blood flow is compromised by 50%, the addition of a fatty meal to the small intestine leads to a significant decrease in pH and when a second meal is added to the stomach, the pH decreases further suggesting that the food in the stomach leads to steal from the rest of the intestines. This sequence of events likely explains the temporal nature of the pain experienced with CMI.⁸ A similar phenomenon occurs in the patient with coronary demand ischemia. The symptoms of angina pectoris occur because of an inadequate blood supply to the heart that starves the cardiac muscle of oxygen; similarly, intestinal angina results from the relative imbalance between tissue supply and demand for oxygen and other metabolites. Because the mesenteric circulation has a rich collateral network, most patients only develop symptoms when at least two of the three mesenteric arteries are severely narrowed or occluded (Fig. 134.1). However, this is not an absolute requirement and some patients present with single vessel disease, particularly if the SMA is involved.^{9,10} Ultimately, the degree to which a patient becomes symptomatic from CMI correlates with the extent of disease, timing of progression of stenosis and adequacy of collateral pathways in preventing arterial steal.

ETIOLOGY

The most common cause of CMI is arterial obstruction from atherosclerotic disease and this accounts for about 90% of cases of symptomatic CMI. Atherosclerotic lesions usually develop as an extension of plaque from the aorta and thus most are located at the origin of the mesenteric arteries and can extend 2 to 3 cm into the branches. Non-atherosclerotic lesions can also cause CMI and these patients tend to be younger than those with atherosclerotic disease. Non-atherosclerotic pathologies that can

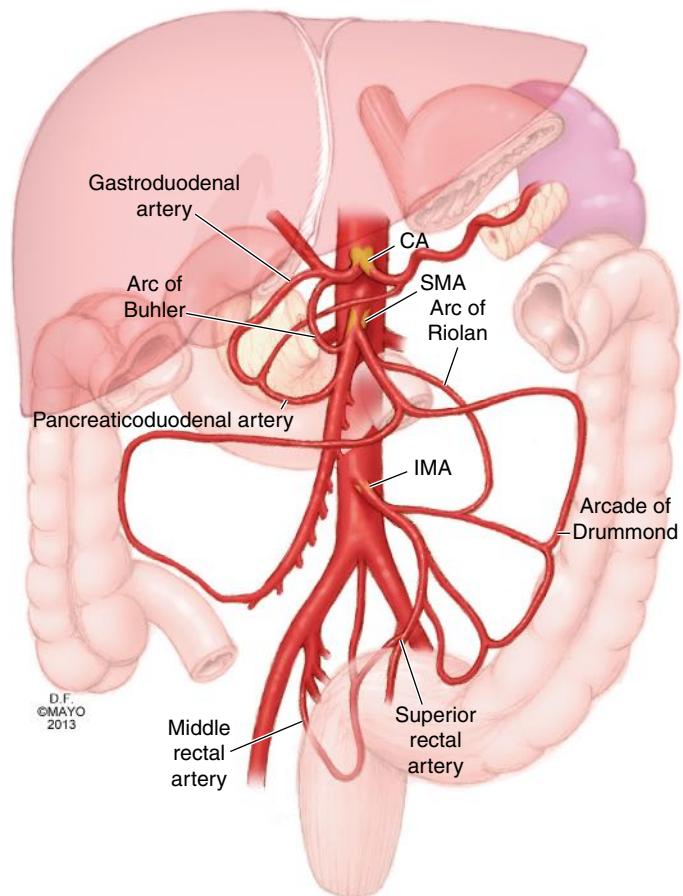


Figure 134.1 Mesenteric artery circulation and common collateral pathways in patients with severe occlusive mesenteric artery disease. Note severe disease at the celiac axis (CA), superior mesenteric artery (SMA), and inferior mesenteric artery (IMA). Common collateral pathways include the arc of Riolan between the left colic artery (IMA) and middle colic artery (SMA). The CA and SMA have collaterals via pancreaticoduodenal arcade (arc of Buhler) and the gastroduodenal arteries. © D.F. Mayo 2013

affect the mesenteric arteries include vasculitis (including giant cell arteritis, Takayasu disease, and polyarteritis nodosa), systemic lupus, Buerger disease, spontaneous dissections, fibromuscular dysplasia, neurofibromatosis, radiation arteritis, aortic coarctation, mesenteric venous stenosis or occlusion, and drug-induced arteriopathy from cocaine or ergot use.

NATURAL HISTORY

Asymptomatic stenosis of one mesenteric artery is often an incidental finding that has a benign clinical course. However, 15% to 50% of patients who present with mesenteric ischemia with bowel gangrene have developed an acute thrombosis of a preexisting chronic lesion with no antecedent warning signs.^{11,12} As part of a prospective cardiovascular health study, Wilson and associates screened the mesenteric arteries of 553 elderly patients with duplex ultrasound and found that 18% of patients had some degree of SMA or celiac disease but none reported symptoms consistent with CMI.¹³ Alternatively, Thomas and associates reviewed 980 aortograms of patients without mesenteric symptoms and found that 82 (8%) had >50% stenosis of at least one mesenteric artery and 15 of the 82 had involvement of all three visceral vessels.¹⁴ The patients were followed for a

range of 1–6 years and 86% of patients with three-vessel mesenteric disease developed acute mesenteric ischemia, became symptomatic or died, leading them to conclude that early revascularization should be considered in these patients.

CLINICAL PRESENTATION

Patients who present with clinically significant CMI secondary to atherosclerosis are usually in their 60s but the age can range from 40 to 90 years old and the female to male ratio is 3:1.^{15,16} The main symptoms associated with CMI include abdominal pain, weight loss, and “food fear.” The classic abdominal pain is postprandial and begins within 10–15 minutes of ingestion of a meal. The pain can persist for as long as 5 to 6 hours. It is usually located in the mid-abdominal location, and its character can range from sharp and debilitating to cramping or a dull ache. Patients often describe intolerance to certain types of food, and consequently they alter their eating habits to avoid foods that precipitate symptoms. In some cases, patients will not endorse postprandial pain because they have adapted their eating habits to prevent the pain from occurring, but careful questioning will reveal minimal caloric intake. Patients may also present with complaints of postprandial nausea, vomiting or diarrhea. Unintentional weight loss is the result of the altered eating patterns, progressing to malnutrition and cachexia, which is then an indication for intervention. Indeed, patients who endorse postprandial pain but have experienced no weight loss may not benefit from revascularization. A recent review of experience with CMI at the Massachusetts General Hospital showed that over 90% of patients presented with abdominal pain and 65% had significant weight loss prior to intervention.¹⁶ In addition, the average time from onset of symptoms to revascularization was 18 months. The pattern of symptoms varies as symptoms can occur intermittently, occur consistently after every meal or progress in severity. CMI may present in a form of subacute mesenteric ischemia that is characterized by progression of pain from intermittent to unremitting or continuous over days to weeks. Sub-acute mesenteric ischemia may be an ominous presentation that warrants immediate revascularization. Physical examination is often nonspecific and does not commonly reveal any pathognomonic findings, but can point to the diagnosis. Many individuals are thin at the onset of their symptoms and progress to cachexia. These patients have physical signs of malnutrition including muscle wasting, and a flat or scaphoid abdomen. Pain may be present but not localized or aggravated by abdominal palpation; the finding of pain out of proportion to physical findings is more consistent with acute mesenteric ischemia (AMI) than CMI (see Ch. 133, Acute Mesenteric Ischemia: Epidemiology, Pathophysiology, Clinical Evaluation, and Management). An abdominal bruit may be noted in up to 50% of patients, and differs from the bruit caused by compression of the celiac axis (CA) by the median arcuate ligament, which is elicited by deep expiration and elevation of the diaphragm. A complete vascular examination may document diminished peripheral pulses and bruit. Laboratory tests are nonspecific or unremarkable in the absence of acute symptoms but may demonstrate malnutrition.¹⁷

The clinical presentation can be ambiguous in some patients. Vague abdominal pain, nausea, vomiting, or a change in bowel habits such as diarrhea or constipation, without the classic postprandial component to the pain, may be subtle complaints that coupled with weight loss could support the diagnosis of CMI. If upper or lower endoscopy is performed, diffuse small ulcerations in the stomach or proximal duodenum or patchy areas of ischemia in the colon are not uncommon and some patients will present with abnormal liver function tests. Other common risk factors include hypertension, hyperlipidemia, and a history of smoking.^{15,16,18} Indeed, atherosclerosis can affect multiple vascular beds and patients often have disease in the coronary (50%–70%), cerebrovascular (20%–45%), and peripheral arteries (20%–35%). Concomitant renal artery disease with difficult to control hypertension or ischemic nephropathy is present in about 20% of patients.^{16,19}

DIAGNOSTIC IMAGING

It is not infrequent for patients with CMI to undergo an extensive evaluation to rule out other causes of chronic abdominal pain and weight loss. The differential diagnosis is extensive, including inflammatory, infectious, and malignant disease. The investigation often includes upper and lower gastrointestinal endoscopy and cross-sectional studies of the abdomen. Often the finding of mesenteric artery stenosis in an imaging study is the first clue to the diagnosis.

Mesenteric Duplex Ultrasound

For patients with suspected CMI, mesenteric duplex ultrasound is the recommended screening study of choice (see Ch. 22, Vascular Laboratory: Arterial Duplex Scanning). A negative duplex ultrasound study essentially excludes the diagnosis of mesenteric artery disease, however this study can be limited by operator experience, patient obesity and overlying bowel gas. Moneta and associates published validated mesenteric diagnostic criteria where a peak systolic velocity greater than 275 cm/s was consistent with >70% stenosis of the superior mesenteric artery (SMA) and greater than 200 cm/s was predictive of a severe stenosis of the celiac artery (CA).²⁰ For the SMA, the threshold velocity of 275 cm/s has a sensitivity of 92% and a specificity of 96% with a positive predictive value of 96% and a negative predictive value of 99%. In the CA, the threshold velocity of 200 cm/s was not as accurate. The Bowersox criteria uses end diastolic flow to predict >50% arterial stenosis and found that a threshold of 45 cm/s in the SMA had a 90% sensitivity and 91% specificity for stenosis.^{21,22} Reversal of flow in the hepatic and splenic arteries may also predict CA stenosis.

Multidetector Computed Tomography

Cross-sectional imaging of the abdomen provides an anatomic analysis to plan revascularization.^{23,24} The choice of CTA or MRA can be driven by individual expertise or institutional preference. Multidetector computed tomography (MD-CT) technology is readily available in most centers, has the highest spatial resolution and finest image detail, and is considered by

most surgeons to be the best study to evaluate anatomic characteristics (calcification, thrombus, diameters, and lengths) that are important to plan mesenteric interventions (Fig. 134.2). In patients with classic symptoms, CTA may supplement or even replace duplex ultrasound, and is often the only imaging study that is obtained prior to intervention. It is also useful to objectively assess patency of grafts and stents.

Magnetic Resonance Angiography

Gadolinium-enhanced MRA has advanced in recent years to provide improved imaging resolution with shorter acquisition times.^{25,26} This imaging modality is used to a lesser extent, but provides functional information by integrating flow dynamics, and blood oxygen saturation techniques, with the anatomic detail, which can be useful in patients with symptoms that are inconstant with a clinical diagnosis of CMI.^{27,28} In patients with previous stents or heavily calcified arteries, MRA may not provide adequate detail due to signal loss.

Contrast Arteriography

Diagnostic catheter-based arteriography has long been considered the “gold-standard” diagnostic study for evaluating mesenteric artery disease. However, its role as the confirmatory test that is used for planning revascularization has essentially been replaced by the aforementioned axial imaging modalities. In most cases, angiography is only used as part of a planned endovascular intervention. Exceptions include patients with suboptimal imaging studies and those with extensive calcification, small vessels, or multiple prior stents that may cause metallic artifact.

Other Ancillary Studies

Endoscopy can demonstrate inflammatory and/or ischemic changes, most noticeable in the stomach, duodenum, or right colon. Erosive ischemic gastritis, gastroduodenitis, or ischemic colitis noted on endoscopy have also been described in association with CMI.²⁹ Gastric tonometry has also been shown to be a valuable diagnostic test to assess intestinal perfusion.^{29–31} Tonometry can be performed as part of a 24-hour monitoring study during fasting and postprandial state, or as an exercise test using a small nasogastric tonometry catheter with serial pCO₂ measurements in the stomach, duodenum, or upper jejunum. Finally, visible light spectroscopy is a new technique that enables noninvasive measurements of mucosal mixed venous oxygen saturation during endoscopy using white light from a fiber-optic probe. This has been used to evaluate the degree of bowel mucosal ischemia at a given location based upon percentage of saturation where a lower number is associated with CMI.^{32,33}

TREATMENT STRATEGIES

The goal of treatment in patients with chronic mesenteric ischemia should be to resolve abdominal symptoms, promote weight gain and prevent deterioration to AMI. The timing and type of intervention is dependent upon the severity of patient symptoms and the patient’s overall surgical risk. There may be a role for medical therapy with bowel rest and parenteral nutrition in select patients with symptomatic CMI as a short-term bridge to revascularization. However, there is no role for a prolonged treatment with chronic parenteral nutrition and noninterventional therapy in these patients. Indeed, excessive delays in proceeding with definitive revascularization or use of parenteral nutrition alone have

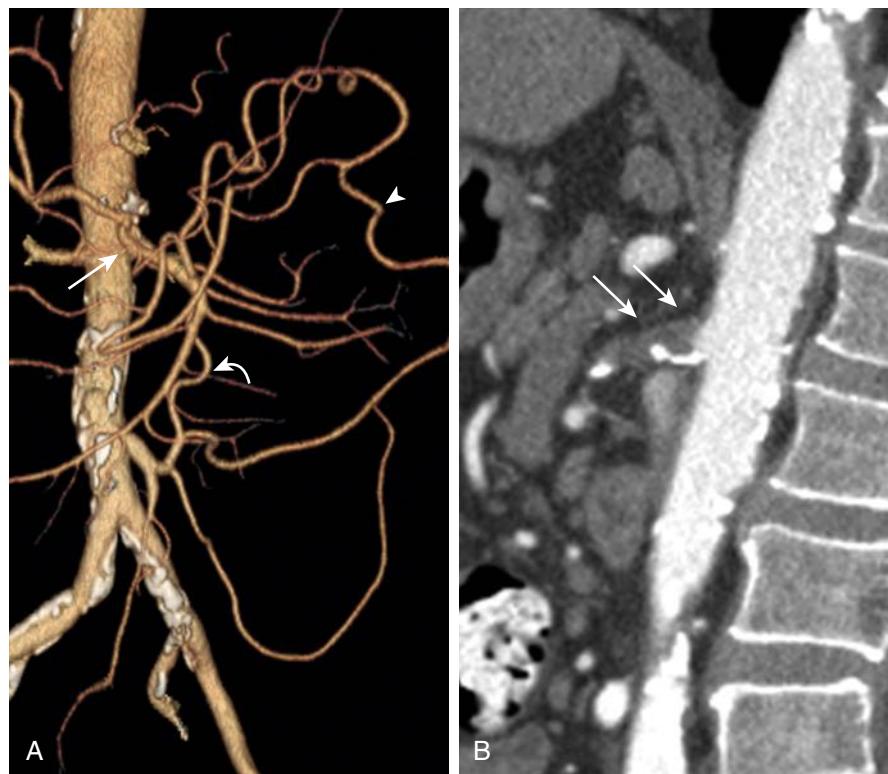


Figure 134.2 Computed tomography angiography with three-dimensional reconstruction in a patient with severe three-vessel mesenteric occlusive disease. Note occlusion of the celiac axis (CA) and superior mesenteric artery (SMA), with collateral flow via a large inferior mesenteric artery (IMA) and meandering artery. (A) Collateralization is shown from the IMA to SMA via the arc of Riolan (curved white arrow) and the arcade of Drummond (arrowhead), and from the SMA to CA via large gastroduodenal artery (straight white arrow). (B) Axial views of the SMA demonstrate occlusion with a small stump (double arrow).

been associated with clinical deterioration, bowel infarction, and risk of sepsis from catheter-related complications.^{34,35} In addition, a recent study that looked at the quality of life of patients who were treated with long-term parenteral nutrition found that it was significantly worse in patients with CMI when compared to those with Crohn's disease.³⁶

Indications for Revascularization

The Society for Vascular Surgery (SVS) practice guidelines recommend revascularization in all patients with CMI who have symptoms of weight loss, food fear, diarrhea, or postprandial pain.³⁷ Treatment goals are to relieve symptoms, restore normal weight, and prevent the development of AMI with bowel infarction. The indication of prophylactic revascularization in patients with asymptomatic disease remains controversial. Based on the report by Thomas and colleagues, there may be a role for prophylactic revascularization in patients with severe three-vessel disease, particularly for those with difficult access to medical care who live in remote or underserved areas.¹⁴ The current

recommendation for the management of these patients is close surveillance for the development of symptoms and counseling with shared decision-making between the patient and provider regarding options for revascularization.³⁷ With ongoing surveillance, surgeons should have a low threshold to proceed with revascularization if any gastrointestinal symptoms (e.g., bloating, diarrhea, atypical pain) arise. Finally, revascularization has been advised in asymptomatic patients with severe three-vessel disease undergoing aortic reconstruction for other indications.³⁷

Open Versus Endovascular Revascularization

The approach to mesenteric revascularization has evolved over the last 20 years. The number of mesenteric revascularizations has increased 10-fold in the United States in the last decade, largely because of improved diagnostic imaging and decreased morbidity of endovascular therapy.⁴ Primary stenting is currently the first choice of treatment in greater than 80% patients with CMI who have suitable lesions, independent of their clinical risk (Fig. 134.3). A careful review of the pre-procedure

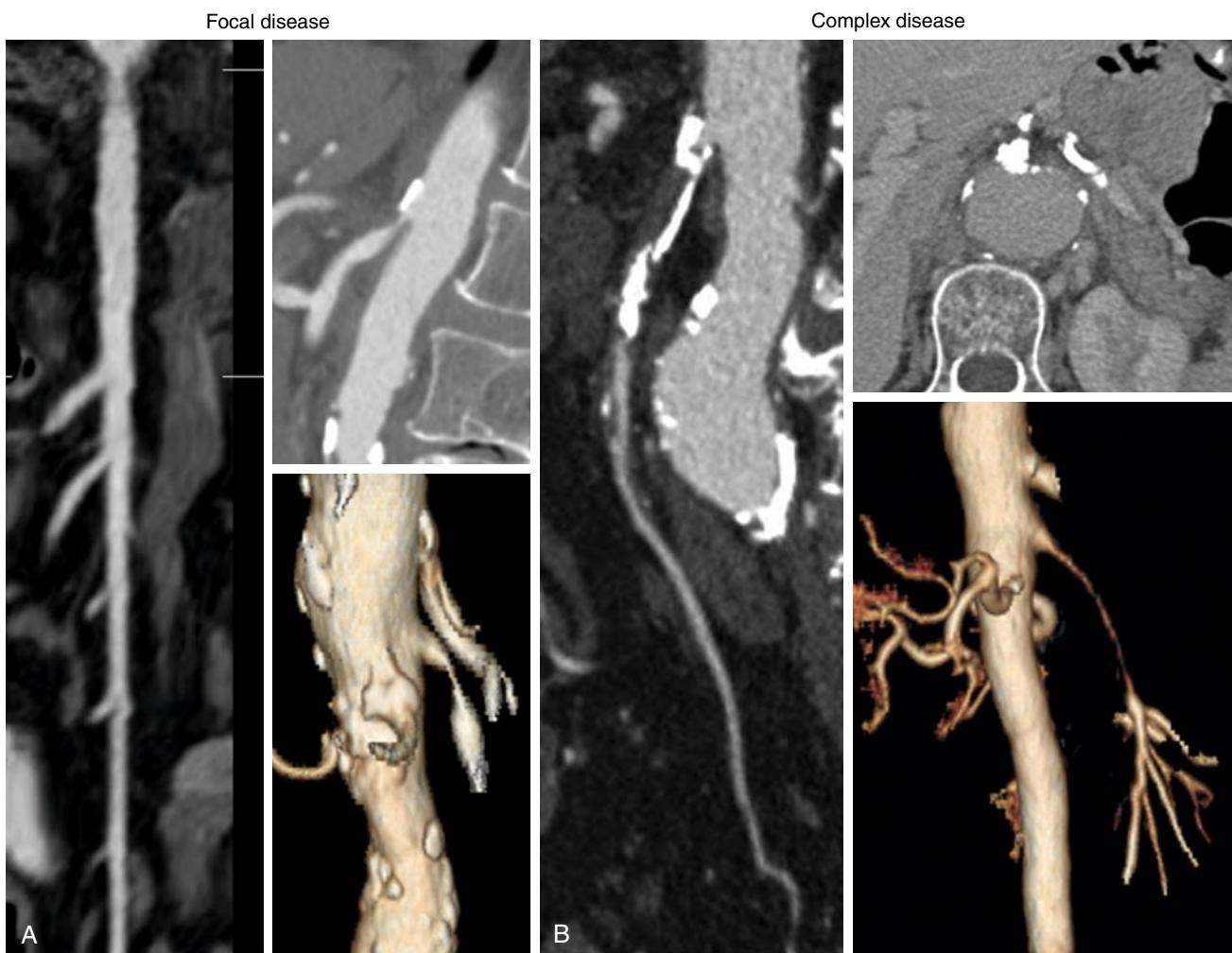


Figure 134.3 Computed tomography angiography is the most useful imaging study to plan revascularization. Anatomic characteristics of the superior mesenteric artery can be used to identify patients with focal disease where angioplasty and stenting is favored (A), or patients with complex disease where endovascular therapy is technically more challenging (B). Lesions with unfavorable anatomy for stenting include heavily calcified occlusions, long segment occlusions, and long segment stenosis involving multiple branches.

CTA with attention to anatomic factors will drive the selection of an open or endovascular approach. The SMA is the primary target for revascularization, and as such the anatomy of the SMA is the most important determinant of choice of therapy. The ideal lesion for angioplasty and stenting is a short, focal stenosis or occlusion with minimal to moderate calcification or thrombus. The CA and IMA should be considered secondary targets and when CA lesions are to be addressed, an endovascular approach with angioplasty and stenting is associated with a higher rate of restenosis than in the SMA.³⁸ Indeed, this should not be performed if there is active compression by the median arcuate ligament, unless the patient first undergoes surgical release.

The technical difficulty of endovascular procedures is increased by the presence of severe eccentric calcification, a flush occlusion with the aorta, longer occlusions, small outflow vessels, and tandem lesions affecting branches. Although these anatomic features do not contraindicate the use of stents, the initial technical outcome is often not optimal, and there is increased risk of arterial complications (e.g., distal embolization, dissection) and restenosis.^{39,40} The SVS clinical practice guidelines recommend that patients in lower operative risk groups undergo open revascularization if the anatomy is unfavorable for angioplasty and stenting.^{37,41} Mesenteric bypass has also been indicated in patients who have failed a percutaneous intervention, in those with recurrent in-stent stenosis after multiple failed reinterventions, and for nonatherosclerotic lesions.

PREOPERATIVE EVALUATION

Evaluation of the patient prior to mesenteric revascularization should focus on a critical review of surgical risk, nutritional status, and anatomic factors that will affect choice of

reconstruction. Mesenteric revascularization carries definitive risk, regardless of which technique is used. These patients should undergo a comprehensive medical evaluation to identify and optimize cardiovascular risk factors and their nutritional status. The evaluation should be tailored but often includes noninvasive cardiac stress test, pulmonary function tests, carotid ultrasound, and noninvasive lower extremity arterial studies. Optimal medical therapy in these patients ideally should include cessation of cigarette smoking, acetylsalicylic acid, and beta-blocker and cholesterol-lowering medication, preferentially a statin.

Although a detailed preoperative work-up is warranted, revascularization should not be excessively delayed. Patients who present with acute deterioration of symptoms should be admitted, given nothing by mouth, started on intravenous heparin, and treated urgently within 24 to 48 hours. Patients with iodinated contrast allergy should be premedicated with steroid and antihistamine preparations. Those with chronic kidney disease with a serum creatinine level greater than 1.5 to 2.0 mg/dL (133–177 mmol/L) should be pre-treated with intravenous hydration with sodium bicarbonate and normal saline, starting the day prior to intervention.

ENDOVASCULAR REVASCULARIZATION

Diagnostic Arteriography

Diagnostic angiography (Fig. 134.4) used to be the gold standard for diagnosis of CMI but now it is most often reserved for planned interventions. Angiography can be performed through either a femoral or brachial approach. The brachial approach is most useful for occlusions of the SMA because the downward



Figure 134.4 Abdominal aortogram with right anterior-oblique view demonstrates large patent inferior mesenteric artery (IMA). Selective IMA angiography confirms collateralization to the superior mesenteric artery via arc of Riolan (arrow) (A), and collateralization to the celiac axis via gastroduodenal artery (arrow) (B).

angle facilitates the ability to cross the occlusion with the wire. Brachial artery access may require open exposure for small arteries, and to reduce the risk of post-procedure hematoma. Access in either artery is established using ultrasound guidance and 0.035-inch guide-wire system or a micropuncture access system. A 5-F sheath is positioned in the external iliac artery, and a 5-F diagnostic flush catheter is advanced to T12 level over a 0.035-inch guide wire. Modest intravenous heparinization (40 units/kg) is recommended prior to selective catheterization of the mesenteric arteries. Low-osmolar contrast agent (e.g., Visipaque) minimizes abdominal discomfort during selective injections. Choice of catheter shape is dependent upon access site, angle of origin, and individual preference. The MPA catheter is ideal for selective catheterization via the brachial approach, whereas a secondary curve catheter (e.g., SOS or Simmons) or a catheter with more acute curve (e.g., Cobra 2) can be used for interventions done via a femoral approach. If the lesion is slightly distal from the ostium, a Kumpe can be used to select the SMA from the femoral approach without the need to reform the catheter after selection. A complete study includes abdominal aortogram with anterior–posterior and lateral views to define the location, severity, and extent of visceral artery involvement, and to identify concomitant lesions in the aorta, renal, or iliac arteries. The optimal projection to display the proximal CA and SMA is a lateral view, and for the origin of the inferior mesenteric artery (IMA), it is a 15-degree

right lateral–oblique view. Selective angiography is necessary to confirm the severity of disease and to identify tandem lesions and collateral patterns. In patients with questionable lesions, pressure gradients can be measured using pressure wire, “pull-back,” or simultaneous pressure measurement technique.⁴²

Angioplasty and Stenting

The primary goal of percutaneous treatment is to restore antegrade flow into at least one of the three mesenteric arteries, preferentially the SMA. Routine stenting is recommended because of elastic recoil and restenosis that occurs with angioplasty of ostial lesions. In patients with CA compression by the median arcuate ligament, there is risk of stent fracture and compression. There is no proven benefit that routine two-vessel stenting provides more durable relief, and a second intervention adds cost and potential risk of complications.^{38,43}

The intervention can be done via a brachial or femoral approach. Access via the brachial artery is preferred for patients with a very acute origin of the SMA from the aorta and in those with occlusions. The authors favor the brachial artery approach whenever possible (Fig. 134.5). This offers excellent support with smaller profile system and precise stent deployment in patients with an acute SMA angle. To minimize the risk of puncture-related complications with brachial access, a

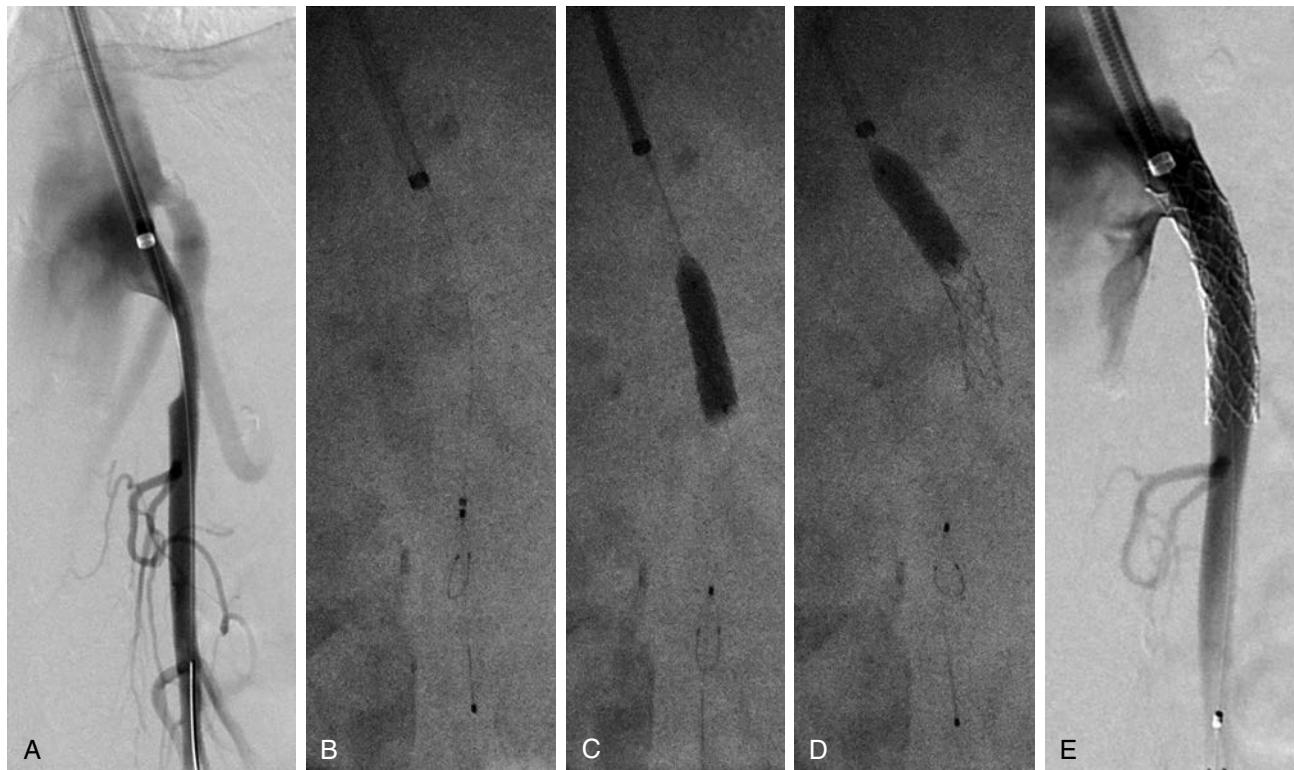


Figure 134.5 Angioplasty and stenting of a focal stenosis of the superior mesenteric artery (SMA) stenosis using brachial approach. After selective angiography (A), the lesion is crossed and a 0.014-inch Spider Rx filter wire (Covidien, Plymouth, MN) is deployed in the main trunk of the SMA (B), avoiding jejunal branches. The entire lesion is treated by a balloon-expandable stent (C), which is extended 1–2 mm into the aorta and flared proximally (D). Completion angiography demonstrates the patency of the stent without embolization or dissection (E).