INTRODUCTION:

Among the many acute abdominal conditions that confront the general surgeon, disorders involving the vascular system are in the minority. Yet these conditions are often highly lethal if undiagnosed or inappropriately treated. Since operations involving vascular exposure, control and repair are uncommon in the practice of most abdominal surgeons a straightforward plan to identify and manage these conditions is required for optimal success. This chapter will concern itself with the general diagnosis of acute vascular abdominal conditions, principles of vascular control and repair and a discussion of the management of the three most common types of vascular emergency: mesenteric ischemia, rupture abdominal aneurysm, and abdominal vascular trauma. Whenever possible emphasis will be placed on general principles that can be applied to a variety of conditions. Acute pathology of the gastrointestinal tract which results in hemorrhage (e.g. bleeding ulcer, esophageal varices, bleeding diverticula) will not be considered within this Chapter.

General Diagnostic Considerations:

Acute vascular conditions can be divided into those associated with hemorrhage and those accompanied by vascular thrombosis. The presentation within each of these two broad categories is generally distinct. Conditions associated with hemorrhage present with evidence of blood loss including shock. Hemodynamic alterations, e.g. hypotension and tachycardia, predominate over physical findings. Signs of an “acute abdomen”, specifically peritoneal irritation, are often absent. While abdominal pain is usually present, it is often focal, and may be associated with a palpable abdominal mass. Signs of shock in the absence of generalized peritonitis or visceral perforation should prompt the consideration of a vascular emergency. In contrast, vascular thrombosis leads to intestinal ischemia and perforation. The clinical presentation of vascular thrombosis is often identical to that of other acute non vascular abdominal conditions which cause an acute abdomen. Presence of stigmata of cardiovascular disease, e.g. peripheral vascular occlusions, history of cardiac disease, atrial fibrillation, vascular bruits, advanced age, should all increase the clinical suspicion of a vascular event as the underlying cause of symptoms. Nevertheless, thrombotic vascular complications often remain undiagnosed until the time of laparotomy.

While physical examination may help to identify patients with intraabdominal or retroperitoneal bleeding (signs of hemorrhagic shock, absence of peritonitis), routine laboratory evaluations are less helpful. Acute hemorrhage may not result in changes in hemoglobin in its early stages. Laboratory studies are generally useful in excluding other acute inflammatory states such as pancreatitis, and acute processes of the biliary tree or intestine. Plain films of the abdomen may reveal vascular calcifications or suggest hemorrhage (loss of psoas shadow) but are often non diagnostic. Computed Tomography (CT) scanning, when available, is the most useful preoperative diagnostic study.
Scanning should be performed with oral and intravenous contrast unless absolutely contraindicated. CT angiography can identify vascular calcifications, aneurysms and pseudoaneurysms, localize and quantify blood loss, and often identify thrombosis of major arterial and venous structures. Refinements in CT angiography, such as 3 dimensional reconstructions, have markedly reduced the need for diagnostic angiography and streamlined the evaluation of all patients with acute abdominal problems. In addition to visualizing vascular structures, non vascular findings on CT scan may raise the suspicion of an acute vascular emergency. Thickening of the bowel wall and pneumatosis intestinalis may be present without an identifiable lesion in the mesenteric arterial or venous system. Evidence of visceral embolization, particularly in the spleen or liver should suggest a proximal embolic source, most often from endocarditis. Evidence of a shrunken kidney is a sign of visceral atherosclerosis and, while a non specific finding, should increase suspicion of disease in other visceral beds.

CT scanning cannot identify all acute vascular conditions, particularly when intravenous contrast is not administered, and scans may not be performed before laparotomy in a number of cases. Under these circumstances, the diagnosis of an acute vascular emergency is made at the time of laparotomy. Most often this diagnosis is obvious on clinical grounds, identification of a mesenteric or retroperitoneal hematoma, presence of free blood in the abdomen, or the presence of infarcted bowel without evidence of internal hernia.

**VASCULAR EXPOSURE AND CONTROL:**

Expeditious vascular exposure and control is essential for optimal management of vascular emergencies. The principles of operative vascular control are well established: proximal and distal control in a relatively normal area of the vessel. Proximal control should always be established before the lesion is addressed. When attempts to establish distal control would result in excessive dissection or cause damage to adjacent tissues and organs, the vessel is opened after proximal control is established and distal control established intraluminally by placing balloon catheters to control back bleeding. Increasingly, intraluminal techniques are being used for establishing proximal arterial control from remote access sites. *Antegrade* intravascular balloon control can be established without concern for balloon migration from arterial pulsation. A good example of this is placement of an arterial occlusion balloon in the suprarenal abdominal aorta through the arm vessels.(ref) When the balloon catheter is placed from a site distal to the artery (retrograde control) the balloon must be buttressed to avoid migration as a result the repetitive force of arterial pressure. This can be done by supporting the catheter and balloon by a rigid sheath on which the balloon can rest .(Figure). Balloon catheters can by used to tamponade proximal collateral bleeding if the main arterial inflow has otherwise been controlled. The most common example of this is the combination of supraceliac clamping coupled with placement of a foley catheter to control of collateral visceral back bleeding during repair of ruptured aortic aneurysm.
In cases of active hemorrhage or when dissection is difficult, initial venous control is usually obtained by external pressure. Extensive venous dissection is usually avoided to reduce iatrogenic venous damage. Circumferential venous dissection must be meticulous because of the many venous tributaries and the fragility of the vein wall. Intraluminal balloons can be combined with external compression for both proximal and distal control in cases of venous injury, since this is a low pressure system and catheter dislodgement is not a problem.

Endovascular techniques have been applied across all aspects of vascular surgery, and management of abdominal vascular emergencies is no exception. However the application of most of these techniques requires dedicated angiographic facilities and significant endovascular experience. In routine practice the most expeditious way to achieve control remains open exposure. Endovascular techniques remain most useful when they replace extensive or dangerous open dissection. While endovascular options will be discussed within the context of each disease process, these approaches will not be described in detail within this chapter. References to endovascular textbooks in which the technical details of these approaches can be found are provided in the bibliography (refs). What follows is a description of the open surgical approach to control of the major abdominal vessels.

**Exposure of the Aorta:**

**Supraceliac Exposure:**
Expeditious supraceliac control of the abdominal aorta is the most important and versatile technique in the management of abdominal vascular emergencies. While suprarenal, intrarenal and occasionally supra mesenteric control of the aorta are all possible, there is no evidence that these prove superior to supraceliac aortic control as long as visceral ischemia is limited to 45 minutes or less. (REF). Supraceliac aortic control can be achieved rapidly with very little risk of damage to adjacent organs such as the intestines, pancreas or vena cava or the visceral vessels. Finally, the supraceliac aorta is most likely to be free of either aneurismal or atherosclerotic vascular disease. For this reason exposure and control of the aorta at that level is easier and safer than control between the visceral vessels. Supraceliac control of the aorta through a left retroperitoneal approach has been well described (ref) but is not germane in this situation, since it precludes evaluation of the abdominal viscera. Therefore only the transabdominal exposure of the supraceliac aorta is described.

The supraceliac aorta is approached through the gastro hepatic ligament, which is divided between clamps. The left lobe of the liver is mobilized by dividing its diaphragmatic attachments if necessary. Division of the gastrohepatic ligament brings one directly down on to the esophagus and aorta as they course through the diaphragmatic hiatus. The aorta lies to the right of the esophagus and should be easily palpable. In the event that the two organs are not easily distinguishable, a nasogastric or orogastric tube may be placed in the esophagus to aid in distinguishing between the two organs, but this is rarely required in our experience. Once the aorta has been identified, the key to obtaining control is complete division of the fibers of the left crus of the diaphragm as they cross the anterior
aspect of the aorta. This can be done by placing either the index finger or a large angled clamp between the aorta and the crural fibers as they cross over its anterior aspect. The fibers are divided, slightly to the left of the midline (“2 o’clock” position) to avoid bleeding, either with scissors or electrocautery. The phrenic arteries are identified and either clipped or, preferentially, spared. One cannot overemphasize the importance of completely dividing these fibers and clearing the anterior, medial and lateral aspects of the aorta prior to applying the vascular clamp. If this is not done, any aortic clamp will slip anteriorly, resulting in loss of aortic control with disastrous results. Once the crura are divided the aorta is encircled between the thumb and index finger of the operating surgeon’s right hand. The aorta is then lifted gently off the spine to be sure that it has been completely mobilized. A clamp can then be reliably placed across the aorta. More extensive dissection of the aorta is not required and we avoid passing angled clamps and loops under the aorta to minimize damage to intercostals vessels. Use of the index finger and a straight aortic clamp are all that is required.

Exposure of the Visceral Aorta:
This area of the aorta will rarely need to be exposed for acute vascular emergencies. Transperitoneal control of the visceral aorta requires a left medial visceral rotation (ReF). The left colon is mobilized along Toldt’s line, the retroperitoneal and phrenic attachments of the spleen are divided and the spleen, colon and tail of the pancreas are reflected medially, leaving the left kidney down. This results in exposure of the anterior aspect of the aorta, the origins of the renal, celiac and superior mesenteric arteries. Exposure of the visceral vessels more distally is described below.

Infrarenal aortic exposure:
This technique is familiar to most surgeons and involves incision of the ligament of Treitz and mobilization of the 4th portion of the duodenum superiorly and to the right. When encountered, the inferior mesenteric vein may be divided between clamps. This sometimes improves exposure and is preferable to leaving an intact vein under tension with the risk of avulsion. The left renal vein serves as a reference to identify the superior extent of dissection. This vein almost never requires division. Should additional mobilization be required, the gonadal, and lumbar veins can be divided for superior mobility and the adrenal vein is divided if the vein is to be retracted inferiorly. If these collaterals are divided and the renal vein is subsequently sacrificed, it must be repaired, either primarily or with an interposition graft. If the left renal vein is not encountered during this dissection, one must consider the possibility of an aberrant renal vein coursing posterior to the aorta, which occurs in 1% (ref) of patients. In that case the vein is at risk for damage during aortic cross clamping. Lymphatic and areolar tissue anterior to the aorta is cauterized or divided and ligated between clamps. It is better to ligate large lymphatics to prevent chyle leak postoperatively. As with the suprarenal aorta, the vessel is encircled using the thumb and index finger and lumbar vessels usually do not require division. We are more inclined to place a tape around the aorta in the infrarenal location, since visualization is optimal, but this is not required. As described above, the aorta is circumferentially mobilized digitally, raised off the spine, and an aortic cross clamp is placed under direct vision.
Exposure of the Iliac Arteries:

The common and external iliac arteries are controlled after entering the retroperitoneum. For proximal iliac control the small bowel mesentery is reflected to the right and the aortic bifurcation is exposed. For more distal control, particularly of the external iliac arteries, the right or left colon is mobilized along Toldt’s line and reflected toward the midline. It is important to be mindful of the ureter as it crosses over the iliac bifurcation. Control of the iliac arteries at the aortic bifurcation can be dangerous because of the confluence of the iliac veins behind the right iliac arteries. This is one of the most common sites of iatrogenic vascular injury during aortoiliac surgery. Whenever possible, the venous structures are gently separated from the arteries by use of blunt dissection (sponge on stick, kuttner dissector or digital dissection). Whenever possible we avoid use of clamps to dissect around the iliac vessels. Once the vessels are separated from the adjacent venous structures they can be encircled with vessel loops and clamped. Relatively blind clamping of the iliac arteries without dissection away from surrounding veins is discouraged as venous injury may result with disastrous consequences.

The hypogastric arteries and distal external iliac arteries can be difficult to expose, particularly in a deep pelvis. The hypogastric artery in particular may present challenges with the risk of injury to deep pelvic veins. This artery can usually be controlled by retrograde balloon tamponade and oversewn. The very distal external iliac artery can be controlled with an intravascular balloon and if necessary oversewn. Vascular continuity can be restored by a bypass to the common femoral artery.

Exposure of the Celiac Artery and its branches:

Exposure of the proximal celiac artery can be obtained through the gastrohepatic ligament, as described for the suprarenal aorta, or by left medial visceral rotation. We prefer the former approach whenever possible. The celiac artery is identified as it originates from the aorta at the diaphragmatic hiatus. Division of diaphragmatic fibers facilitates proximal exposure. More distal control is achieved by careful dissection along the anterior aspect of the vessel with caudal traction on the stomach and superior border of the pancreas. The tissue surrounding the vessel is carefully divided and ligated. By opening the gastrohepatic ligament along the lesser curvature of the stomach one can trace and isolate the Common Hepatic Artery superior to the pancreas. The Proper Hepatic Artery courses in the portal triad anterior and medial to the portal vein. The standard techniques for exposure of the portal hepatis will serve to identify and isolate this structure. The Splenic Artery is exposed by entering the lesser sac and reflecting the pancreas inferiorly and anteriorly. The multiple branches of this vessel which supply the pancreas must be ligated for adequate exposure. The distal splenic artery is best exposed by mobilizing the spleen as for splenectomy.
Exposure of the Superior mesenteric Artery (SMA):

Transabdominal control of the superior mesenteric artery at its origin requires medial visceral rotation of the left colon, spleen and tail of the pancreas (ref). Exposure of the more distal mesenteric vessel can be done through the base of the small bowel mesentery or by approaching the vessel on its posteromedial aspect after reflecting the small bowel mesentery to the right (as in standard aortic exposure). In the former approach the transverse colon is elevated and the middle colic vessel is traced down to the SMA in the small bowel mesentery. The anterior aspect of the vessel is cleared, taking care not to injure the adjacent vein. In the latter approach the vessel is palpated in the root of the small bowel mesentery and dissection proceeds on the lateral aspect of the vessel. In either case, dissection requires meticulous division and ligature of small venous, arterial and lymphatic branches, and the preservation of as many major arterial and venous branches as possible.

Exposure of the Renal Arteries:

Transperitoneal control of the renal arteries can be achieved in a variety of ways depending on the area of the artery to be controlled. The Left Renal Artery is exposed in the same manner as the infrarenal aorta. The artery is usually superior and posterior to the left renal vein. The renal vein may require mobilization, including division of its lumbar, gonadal or adrenal tributaries. Occasionally the retroperitoneal attachments at the inferior border of the pancreas must be incised so the pancreas can be retracted in a cephalad fashion. The renal artery can be traced distally from its origin at the aorta. If the distal renal artery, near the hilum of the kidney, requires exposure, this is most easily done by mobilizing the left colon toward the midline. This may require mobilization of the splenic flexure and occasionally the tail of the pancreas, although this is not always the case. The Proximal Right Renal Artery can be exposed for a short segment between the aorta and inferior vena cava. The first part of the exposure is similar to that for the infrarenal aorta. Since the right renal artery runs behind the inferior vena cava, significant proximal exposure of this vessel requires mobilization of the vena cava and retracting it to the right. This requires careful division of one and often two sets of lumbar veins. Even with this maneuver, only the most proximal portion of the renal artery is exposed. As a result the right renal artery is most often exposed by an extended Kocher maneuver; which reflects the duodenum, ascending colon and hepatic flexure toward the midline. The artery again lies posterior and inferior to the renal vein, which often requires mobilization.

Exposure of the Venous structures:

The visceral veins are exposed by the same approaches as their corresponding arteries. Exposure of the Vena Cava and Iliac Veins requires some discussion. In general these vessels are not involved in acute abdominal vascular emergencies outside the trauma setting. However the vena cava is the vascular structure most commonly involved in penetrating abdominal trauma.(REF) The inferior vena cava and confluence of the iliac
veins is generally exposed by a right medial visceral rotation. This involves mobilization of the right colon along with the duodenum and pancreas when more proximal venous exposure is required. When exposing venous structures one must be exceedingly cautious of the fragility of the vessel, and in particular disrupting small, posterior, lumbar vessels. As a consequence, and because the venous system is a “low pressure” system, compression plays a greater role in control of the vena cava and iliac veins than it does in exposure and control of the corresponding arterial segments. Circumferential mobilization of the veins is avoided if possible, as is the application of clamps. Use of blunt instruments such as sponge sticks, can usually provide adequate hemostasis. Fine clamps such as Allis clamps, can be used to coapt cut ends of vessels and facilitate either suture or control by applying partial occlusion clamps. Whenever possible only the anterior segments of the vein are exposed, to avoid dissection around the lumbar vessels. Exposure of isolated posterior injuries involves significant mobilization and rotation of the vena cava and often requires ligation of multiple tributaries. Ligation is liberally applied in cases of extensive venous injury.

**PRINCIPLES OF ARTERIAL REPAIR:**

Several factors dictate the approach to emergency arterial repair. These include the presence or absence of contamination, size of the arterial defect and the adequacy of collateral circulation. The following are principles which should guide the choice of procedure.

1) When possible, primary repair is indicated. While most circumstances do not lend themselves to this approach, lateral repair or primary end to end anastomosis, or even arterial reimplantation, is associated with good long term results and avoids use of a conduit.

2) When adequate collateral circulation exists, ligation without repair is indicated. This is the case with most splenic artery aneurysms and selected aneurysms of the hepatic and superior mesenteric arteries.

3) In the absence of contamination, prosthetic conduits provide the best choice for bypass of major intraabdominal arteries. The high flow in the aorta and major visceral arteries along with their relatively large diameters is associated with good long term patency of prosthetic bypass. Prosthetic conduits have the advantage of adequate diameter and ready availability, which makes them preferable to saphenous vein in the absence of any contraindication. Occasionally when reconstruction of a small to medium diameter (<6 mm) vessel is required, saphenous vein may be the preferred conduit.

4) In the presence of contamination, autogenous material should be used when vascular reconstruction is required. The risk of prosthetic graft infection with rupture precludes its routine use. For small to medium sized vessels (<6 mm), or when a patch closure is feasible, saphenous vein is usually adequate. For larger vessels, deep veins (femoral, popliteal, or jugular) should be considered. Short segment arterial repairs (e.g. visceral
and renal vessels) can be performed with hypogastric artery. Aortoiliac repair in the face of contamination should be performed with either deep leg veins, or more often arterial ligation and extra-anatomic bypass to restore perfusion.

MANAGEMENT OF VASCULAR EMERGENCIES

Acute Mesenteric Insufficiency:

Presentation:

Patients with acute mesenteric insufficiency generally present initially with abdominal pain out of proportion to their physical findings. However if undiagnosed, acute ischemia will progress to intestinal infarction with the attendant signs of peritonitis. Laboratory investigations include complete blood count, electrolytes, lactic acid, liver panel, amylase and lipase. In general findings are nonspecific early in the course of the disease and consist of a leukocytosis and perhaps some evidence of hemoconcentration. Liver panel, amylase and lipase are most useful to exclude other acute abdominal conditions. Elevated lactic acid is usually a late sign and associated with a poor prognosis. Plain radiographs are non specific. An ileus may be present and occasionally edema of the bowel wall (“thumb printing”) may be present. Computed tomography (CT), with intravenous contrast, has emerged as the most useful imaging modality. CT scans can identify abrupt arterial cutoffs, particularly when 3-D reconstructions are available. In addition, late phase CT angiography is the most reliable means to identify mesenteric vein thrombosis. Occasionally, angiography may be required, particularly when non occlusive mesenteric ischemia is suspected. In these cases, angiography may be both diagnostic and therapeutic.

Treatment of mesenteric insufficiency relies on prompt diagnosis and initiation of therapy before extensive bowel infarction occurs. This is dependent on a high index of suspicion. Mesenteric ischemia results from a variety of conditions, including arterial embolism, arterial thrombosis, low flow states and venous occlusion.. Prompt effective fluid resuscitation is important in all cases of mesenteric ischemia, along with the initiation of broad spectrum antibiotics. Patients with signs of an acute abdomen should be taken to the operating room as soon as they have been adequately resuscitated. Beyond this however, the specific management of each type of mesenteric ischemia differs somewhat according to the etiology. Therefore they will be discussed separately.

*Acute mesenteric embolization* presents with the sudden onset of severe abdominal pain in the setting of a relatively normal abdominal examination. Most emboli are of cardiac origin and the patient may have an irregular pulse, cardiac murmur, or a history of prior myocardial infarction. Many patients may have a history of atrial fibrillation and/or prior
embolic events. Because of the flow characteristics of the visceral vessels, most emboli preferentially go to the Superior Mesenteric Artery. While some emboli lodge at the origin of this vessel, most end up distal to the first jejunal branches. An abrupt cutoff of flow in the SMA distal to the first jejunal branches on catheter angiography or CT angiogram is diagnostic of this condition. Treatment is generally laparotomy and embolectomy. Characteristically the most proximal jejunum is viable in the case of SMA embolus, since the occlusion occurs distal to the first jejunal branches. This is a helpful but not foolproof way to differentiate mesenteric embolization from mesenteric thrombosis.

The superior mesenteric artery is exposed as described earlier in this chapter. The artery is usually soft and the site of the embolus is readily apparent. While a transverse arteriotomy with primary repair can be done, we prefer a longitudinal arteriotomy, and patch closure in most circumstances. The longitudinal arteriotomy can be extended if necessary and will allow through examination of the vessel and meticulous closure. In addition, it facilitates bypass should this be required. Once the artery is opened 3F and 4F embolectomy catheters are passed both proximally and distally to reestablish flow. If necessary, papaverine, 1mg/kg or 100 mcg of nitroglycerine can be instilled in the distal vessels to reduce vasospasm. When there is concern about residual distal thrombus, 250 mgm of Urokinase or 4-5 mgm Tissue Plasminogen Activator (TPA) in 50cc saline can be instilled in the distal vascular bed (REF). If there is clinical evidence of atherosclerosis in the artery, a longitudinal arteriotomy and patch closure is mandatory. If bowel resection is required then proximal saphenous vein should be used for arterial reconstruction.

In unusual circumstances catheter directed thrombolysis can be used as an alternative to open embolectomy. The patient should have no signs of peritonitis and angiography should demonstrate distal emboli (not easily retrieved by an embolectomy catheter) or a partially occluding proximal embolus which permits distal flow to continue during thrombolysis. In these rare circumstances an infusion of TPA (dose needed) directly into the inferior mesenteric artery, can be attempted. Mechanical thrombolysis should not be attempted because of the danger of distal embolization. During lysis the patient must be observed carefully for signs of deterioration and any concern over bowel viability will promote laparotomy. Lysis should not continue for more than 12-18 hours in most cases and in no case beyond 24 hours.

The clinical signs of acute mesenteric thrombosis are indistinguishable form those of acute embolic occlusion, however there are often differences in the history and some physical findings. History of arterial occlusive disease (stroke, claudication, myocardial infarction) is common and atrial fibrillation or prior embolic episodes are unusual. Careful questioning may elicit a history of chronic post prandial pain and weight loss, characteristics of chronic mesenteric ischemia. Physical examination often reveals stigmata of atherosclerosis, e.g. absent pulses and vascular bruits. Angiographic findings usually reveal diffuse atherosclerosis of the aorta and visceral vessels, with multivessel involvement. When vascular occlusion occurs it is usually at the origin of the mesenteric vessel.

The operative approach to acute mesenteric ischemia from thrombosis differs from that of embolic occlusion. Mesenteric flow cannot be restored by a simple embolectomy and
alternatives are required. The most common procedure required is bypass of the superior mesenteric artery usually from the infrarenal aorta or from one of the iliac arteries. While suprarenal bypass is preferred in elective surgery for chronic ischemia, an infrarenal origin of the bypass is more expeditious in the acutely ischemic patient and avoids the acute hemodynamic consequences of suprarenal clamping in a patient already acutely ill and often hemodynamically compromised. Since bowel resection is usually required, autogenous saphenous vein is the preferred conduit and should be harvested from the proximal thigh. When the bypass is performed there should be sufficient redundancy to allow a “lazy C” loop, traveling from right to left in the abdomen, to avoid sharp kinking. (NEED A FIGURE HERE) The bypass is usually performed on the lateral side of the SMA slightly posterior, so that it can lie without compromise when the viscera are returned to the abdomen. While it is tempting to use very short bypasses, these may be prone to kinking and perioperative thrombosis. In the acute setting revascularization is usually restricted to the SMA alone.

When there is no suggestion of intestinal necrosis and angiography reveals high grade stenosis rather than vascular occlusion, an endovascular approach may be attempted. Endovascular recanalization should not be attempted when vessels are completely occluded because of the possibility of causing distal embolization. While the target lesion remains the SMA, it is reasonable to perform angioplasty of multiple visceral arteries if the patient remains stable. The visceral vessels may be engaged either transfemorally, or more often via a transbrachial approach. The latter facilitates access to the origin of the vessel and passage of angioplasty balloons and stents as required. If there is any indication of intravascular thrombus, lytic infusion should be performed prior to any attempt at angioplasty, to avoid the possibility of distal embolization. Once the possibility of thrombus is excluded then angioplasty with the placement of a balloon expandable nitinol stent is performed. Use of a short (15-20 mm) 5-6 mm diameter balloon expandable stent allows precise deployment. The stent should completely traverse the area of narrowing and extend a few mm’s out into the aorta. This is important since the lesion in this case usually has its origin in the aorta. Selecting an endovascular approach does not mean that laparotomy is avoided, since bowel ischemia may be present. Any signs of peritonitis require prompt laparotomy and inspection of the bowel for viability.

Retrograde endovascular recanalization of a proximal SMA lesion has been reported at the time of celiotomy. (REF) This technique involves a longitudinal arteriotomy was made in the Superior Mesenteric Artery and a wire was passed retrograde into the aorta under fluoroscopic guidance. Balloon angioplasty of the proximal lesion is performed as an alternative to bypass and the arteriotomy was closed with a patch. While reports are anecdotal, this procedure is of interest since it avoids the possibility of distal embolization and may be performed more expeditiously than a vein bypass.

Non Occlusive Mesenteric ischemia may occur as the result of low flow, without evidence of acute arterial thrombosis or embolization. In one form of this condition the colon, in whole or in part, is involved. The arterial supply of the colon is less robust than that of the small bowel and, particularly in elderly patients, the inferior mesenteric artery may be diseased or occluded. System illness with reduced visceral blood flow, or abrupt interruption of the inferior mesenteric artery, such as with aortic resection, may
precipitate infarction of marginally perfused areas of the colon. This is most common in the sigmoid colon and the splenic flexure. The rectum is often spared in this process, because of its dual supply through the hemorrhoidal vessels. The small bowel is also usually spared. In these situations, resection of the infarcted colon, with exteriorization and diversion as necessary, is all that is required. The SMA and celiac arteries are usually normal and no attempt at revascularization of the inferior mesenteric artery is indicated. Mesenteric ischemia without an underlying visceral lesion may also involve the SMA and Celiac distribution. This has been called “non occlusive mesenteric ischemia” (NOMI) and is associated with severe systemic illness, hypotension, and spasm of the mesenteric vessels without evidence of an obstructive lesions. (REF) Patients with “NOMI” are often already in an intensive care unit and have had a cardiac event requiring vasocative drug infusions. Some patients may have been on digitalis preparations, which themselves are known to reduce visceral blood flow. Angiography, when performed, shows “pruning” of the mesenteric vessels, without discrete obstruction. (FIGURE) Management of these patients is directed at overall cardiovascular support, treatment of the underlying acute condition(s) and broad spectrum antibiotics. There are reports of intra-arterial administration of papaverine to relieve vascular spasm (REF), although this is not always effective and may be complicated by preexisting systemic hypotension. In general, “NOMI” usually portends a bad outcome, which is related as much to the underlying illness as to mesenteric compromise. Laparotomy should be reserved for patients in whom intestinal infarction is suspected and often will not influence the outcome in this disease.

Mesenteric venous thrombosis may results in acute intestinal ischemia, although this accounts for only about 5 percent of all cases. Patients are a distinct subgroup, being younger (30-50 years.) and predominantly female (REF). Associated hypercoagulable state can be identified in more than three quarters of patients and a history of prior venous thrombosis is not uncommon. Common inherited states include deficiencies of Protein C and S, anti-thrombin III, Activated Protein C resistance, and Factor V Leyden mutation. Acquired pro thrombotic states include profound dehydration, polcythemia, cancer, pelvic or abdominal inflammation, and hormone use. Mesenteric venous occlusion is most readily diagnosed by venous phase CT angiography, which can demonstrate thrombus in the superior mesenteric vein and portal system. Operative findings suggestive of this condition are edematous beefy red bowel with thrombus in veins of the mesentery. The primary mode of therapy is anticoagulation, operative intervention is rarely indicated. Most patients can be managed supportively, although significant volume resuscitation may be required. There are anecdotal reports of mesenteric and portal vein thrombectomy and thrombolysis,(REFS) but these are anecdotal and do not reflect the standard of care for most patients.

Determining Intestinal Viability: the role of “second look” surgery:

A major challenge in managing patients with intestinal ischemia is assessing the need for and extent of intestinal resection. Preoperatively, colonoscopy can be used to assess the viability of the large intestine in questionable situations. Friable red mucosa suggests viability and a grey mucosa which readily sloughs indicates the need for resection.
Viability of the large bowel is difficult to judge from external appearance at the time of laparotomy and in general it is preferable to err on the side of resection in questionable circumstances, since maintaining large bowel length is not an absolute requirement for survival. Primary repair should not be undertaken after large bowel resection, diversion with secondary reconstruction is preferred.

When the small intestine is involved the problem becomes more complex. Every effort should be made to preserve as much small bowel as possible. Clearly necrotic segments of bowel and areas of perforation are resected or excluded immediately to prevent contamination during vascular reconstruction. Evaluation of the remainder of the small bowel is done after blood flow to the intestine is restored. Usually the bowel is observed for 15-20 minutes after revascularization and warm lap pads are applied to the intestines to reduce any vasospasm. External inspection, with attention to color and peristalsis, is more helpful than in the large bowel. Doppler interrogation of the antimesenteric border for arterial flow is useful when positive. Use of fluorescein (1 ampoule given intravenously) followed by inspection with a woods lamp, is the most sensitive means of determining perfusion. Viable bowel will be fluorescent yellow while non perfused bowel will appear dark purple. When the extent of resection is minimal and the remaining bowel is clearly viable, anastomosis and abdominal closure is appropriate. When there are large areas of questionable bowel that might mandate extensive resection an alternate approach is undertaken. Under these conditions, marginal segments of bowel are left in situ and their ends are simply closed over and returned to the abdomen. Plans for a second operation are made. Stomas are not performed at this stage to preserve intestinal length. Fluorescein is not used at this time but reserved for the second procedure. The abdomen is temporarily closed using a “Bogotá bag”, PTFE patch, or other temporary appliance (to minimize the chance of abdominal compartment syndrome) and the patient is returned to the Intensive Care Unit where resuscitation continues. A subsequent laparotomy is performed at 18-24 hours after the patient has been stabilized. At this point fluorescein is injected and non viable bowel is resected. Intestinal continuity is restored unless it is unsafe to do so. The abdomen often cannot be closed primarily at this point because of the danger of compartmental hypertension and an “open abdomen” approach with delayed closure may be needed. Any deterioration in the patient’s subsequent hospital course should suggest breakdown of an anastomosis and prompt the appropriate therapy.

Despite increased clinical awareness, advances in diagnostic modalities and perioperative care, management of intestinal ischemia remains a significant challenge to the most experienced surgeon with continued high mortality and morbidity.

**Management of Abdominal Vascular Trauma:**

Vascular injuries occur in 10 -15% of cases of penetrating trauma and about 3% of blunt trauma.(3REFS) Vascular injuries are highly lethal when they occur and remain the most common cause of death following penetrating abdominal trauma. Arterial and venous injuries occur with equal frequency. The most commonly injured vessels are the
vena cava, followed by the aorta, iliac arteries and veins and the superior mesenteric artery and vein. (REF) Multiple vascular injuries are common. This section will provide principles for management of injuries to the major arteries and veins of the abdomen and retroperitoneum. The reader is referred to the prior sections on vascular exposure for a description of how to obtain control of these vessels. The discussion here centers on management of specific injuries.


<table>
<thead>
<tr>
<th>Location</th>
<th>Arteries Injured</th>
<th>Veins Injured</th>
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<td>17 (33.3%)</td>
<td>34 (66.7%)</td>
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<td>24 (37.5%)</td>
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<tr>
<td>Extremity</td>
<td>123 (75.5%)</td>
<td>40 (24.5%)</td>
<td>163 (39.9%)</td>
</tr>
<tr>
<td>Total (%)</td>
<td>221 (54.2%)</td>
<td>187 (45.8%)</td>
<td>408 (100%)</td>
</tr>
</tbody>
</table>
Overall principles of trauma management are well established, and include initial resuscitation of the patient, rapid evaluation and triage, and expeditious operation when indicated. Stable patients, particularly those with blunt trauma may undergo one or more diagnostic tests, including peritoneal lavage, “FAST” ultrasound examination, and with increasing frequency CT scan. Many patients with penetrating trauma are taken directly to the operating room without further diagnostic evaluation. Consequently, in a

### Table 3. Location of Vascular Injuries

<table>
<thead>
<tr>
<th>Location</th>
<th>Artery</th>
<th>Vein</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Head and neck</td>
<td>Common carotid 8</td>
<td>Internal jugular 26</td>
<td>51 (12.5%)</td>
</tr>
<tr>
<td></td>
<td>External carotid 3</td>
<td>External jugular 7</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Thyrocervical trunk 3</td>
<td>Superior thyroid 1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Vertebral 1</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Superior thyroid 1</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Inferior thyroid 1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chest</td>
<td>Thoracic aorta 4</td>
<td>Superior vena cava 4</td>
<td>64 (15.7%)</td>
</tr>
<tr>
<td></td>
<td>Pulmonary 5</td>
<td>Inferior vena cava 7</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Innominate 5</td>
<td>Pulmonary 1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Subclavian 9</td>
<td>Innominate 1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Internal mammary 12</td>
<td>Subclavian 7</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Intercostal 5</td>
<td>Asygos 1</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Internal mammary 2</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Intercostal 1</td>
<td></td>
</tr>
<tr>
<td>Abdomen</td>
<td>Suprarenal aorta 8</td>
<td>Suprarenal vena cava 8</td>
<td>130 (31.9%)</td>
</tr>
<tr>
<td></td>
<td>Common hepatic 1</td>
<td>Hepatic 2</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Superior mesenteric 4</td>
<td>Portal 6</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Renal artery 2</td>
<td>Superior mesenteric 9</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Infrahepatic aorta 5</td>
<td>Spleenic 1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Inferior mesenteric 2</td>
<td>Renal 6</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Common iliac 4</td>
<td>Infrahepatic vena cava 23</td>
<td></td>
</tr>
<tr>
<td></td>
<td>External iliac 6</td>
<td>Inferior mesenteric 2</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Internal iliac 6</td>
<td>Colic 1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Epigastric 2</td>
<td>Common iliac 15</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Lumbar 1</td>
<td>External iliac 9</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Internal iliac 6</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Testicular 1</td>
<td></td>
</tr>
<tr>
<td>Peripheral</td>
<td>Axillary 10</td>
<td>Axillary 2</td>
<td>163 (39.9%)</td>
</tr>
<tr>
<td></td>
<td>Brachial 39</td>
<td>Brachial 9</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Radial 9</td>
<td>Common femoral 9</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Ulnar 6</td>
<td>Superficial femoral 11</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Common femoral 8</td>
<td>Profunda femoris 4</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Superficial femoral 23</td>
<td>Popliteal 4</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Profunda femoris 8</td>
<td>Tibiopeonal 1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Popliteal 14</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Tibiopeonal 3</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Anterior tibial 2</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Posterior tibial 1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>221 (54.2%)</td>
<td>187 (45.8%)</td>
<td>408 (100%)</td>
</tr>
</tbody>
</table>
significant proportion of cases the extent of vascular trauma is not known preoperatively and must be assessed by the surgeon in the operating room. Intraperitoneal hemorrhage is easily recognized and should be expeditiously controlled, by application of external pressure, vascular clamps or intravascular balloon occlusion catheters. Once active hemorrhage is controlled any visceral disruption is controlled by exclusion to prevent ongoing peritoneal contamination and any remaining solid organ injuries (i.e. liver, spleen, and pancreas) should be stabilized by packing. Definitive treatment of the vascular injuries should then receive priority over definitive visceral repair. The adaptation of “damage control” approach to abdominal trauma has improved outcomes in abdominal trauma. Vascular “damage control” involves the control of major venous injuries by ligation or packing and placement of temporary shunts to restore arterial continuity when arterial ligation will not be tolerated.

**Damage Control Resuscitation for Vascular Surgery in a Combat Support Hospital**

Charles J. Fox, MD, David L. Gillespie, MD, E. Darrin Cox, MD, John F. Kragh, Jr., MD, Sumera G. Mehta, MD, Jose Salinas, PhD, and John B. Holcomb, MD

**Background:** Hemorrhage from extremity wounds is a leading cause of potentially preventable death during modern combat operations. Optimal management involves rapid hemostasis and reversal of metabolic derangements utilizing damage control principles. The traditional practice of damage control surgery favors a life over limb approach and discourages elaborate, prolonged vascular reconstructions. We hypothesized that limb preservation could be successful when the damage control approach combines advanced resuscitative strategies and modern vascular techniques.

**Methods:** Trauma Registry records at a Combat Support Hospital from April to June 2006 were retrospectively reviewed. Patients with life-threatening hemorrhage (defined as >4 units of packed red blood cells) who underwent simultaneous revascularization for a pulseless extremity were included. Data collection included the initial physiologic parameters in the emergency department (ED), total and 24-hour blood product requirements, and admission physiology and laboratory values in the intensive care unit (ICU). Outcome measures were survival, graft patency, and amputation rate at 7 days.

**Results:** Sixteen patients underwent 28 vascular reconstructions for upper (3) or lower extremity (17) wounds. Patients were hypotensive (blood pressure 105/66 ± 29/18), acidic (pH 7.27 ± 0.1; BD -7.50 ± 5.8), and coagulopathic (international normalized ratio 1.3 ± 0.4) on arrival to the ED and essentially normal upon admission to the ICU, 4 hours later. Vein grafts (19/26, 95%) were used preferentially. Prosthetic grafts (1), shunting and delayed repair (4) or amputation (1) were infrequent. Heparin was not used or limited to a half dose (5/20, 25%). Tourniquets (12/16, 75%) and fasciotomies (13/16, 81%) were routine. Most (75%) received recombinant factor VIIa in the ED and in the operating room. All survived with normalized physiology on arrival in the ICU. Twenty-four-hour crystalloid use averaged 7.1 ± 3.2 L, whereas packed red blood cells averaged 23 ± 18 units, and 88% were massively transfused. Median operative time was 4.5 hours (range, 1.7–8.4 hours).

**Conclusions:** Aggressive damage control resuscitation maneuvers in critically injured casualties successfully permitted prolonged, complex extremity revascularization with excellent early limb salvage and graft patency. Recombinant VIIa and liberal resuscitation with fresh whole blood, plasma, platelets and cryoprecipitate, while minimizing crystalloid, allowed limb salvage and did not result in early graft failures.

**Key Words:** Vascular trauma, Management, Damage control resuscitation.


Shunts are most often used to temporarily restore flow to the lower extremities. We find them useful in subclavian/ax/brachial as well, which is supported by this reference. Also in the SMA. When you say “rarely useful in visceral injuries” I’m not sure if you are discouraging their use or just saying that it only rarely comes up.
but are rarely useful in management of visceral injuries. In general, visceral vessels are either repaired or ligated during the initial operation. The end organ will either tolerate ligation because of collateral circulation or be sacrificed. The “damage control” concept can combined with endovascular techniques may be of particular use when open vascular repair is exceedingly complex and associated with significant morbidity. This is particularly true of contained retroperitoneal or hepatic injuries. In these cases, definitive treatment can be deferred at initial laparotomy and attempted in an imaging suite using endovascular techniques after the patient is stabilized. Examples of this include embolization of intrahepatic arterial injury and treatment of some contained retroperitoneal hematomas. This approach is in evolution but holds significant promise.

There are a number of situations in which the surgeon must make a decision about whether to explore a contained hematoma. In these cases the risk of missing a major vascular injury is balanced against the morbidity of operative exploration. Classic trauma training requires exploration of all contained hematomas that result from penetrating injury. However, many experienced trauma surgeons forgo exploration of nonexpanding penetrating perirenal hematomas if they appear to be peripheral rather than hilar. This judgement may be based on preoperative imaging, or on intraoperative trajectory assessment. In the case of blunt trauma, central hematomas (Zone 1) (SEE FIGURE), are explored because of the risk of injury to the aorta or vena cava, while lateral and pelvic hematomas are explored only if there is active bleeding or expansion under observation. If exploration occurs it is important to obtain proximal, and whenever possible distal, arterial control outside the area of hematoma before proceeding. Venous control above and below the area of injury is desirable but may not always be obtainable. Approaches to vascular control, including endovascular techniques, at various areas have already been described. Intravascular occlusion catheters should be readily available for additional control as needed. Only after every attempt to control the arterial and venous ingress and egress to the hematoma has been made should it be entered.
The advent of endovascular techniques may be changing the classic paradigm of managing contained hematoma, from either blunt or penetrating cause. The mandate to explore non-expanding hematomas of any type was based on the concern for occult vascular or visceral injury. The advent of CT angiography and the existence of sophisticated intravascular imagining in the operating room can facilitate evaluation of non-expanding hematomas from both penetrating and blunt trauma without the need for operative exposure and its attendant blood loss. Furthermore, endovascular technique such as covered stents or coil embolization will allow treatment of many vascular injuries from remote access with reduced risk of blood loss. Such treatments are in fact preferred for trauma to branch vessels in the visceral, renal or pelvic circulations. This change in paradigm requires that the surgeon consider a form of vascular “damage control” in the case of contained hemorrhage, by considering an “endovascular first” approach for diagnosis and treatment of contained hematomas regardless of location. I think this is a neat idea. I’m a little concerned that for the casual Maingot reader, it should perhaps be either more explicitly speculative, or else more specific and practical. I don’t want to leave the reader thinking that there’s a new paradigm in place without clear explanation of it’s details or limits. With these general comments in mind, a discussion of specific vascular injuries and there management follows.

Injuries Of The Suprarenal Aorta And Vena Cava.

These injuries as a group are highly lethal and management is difficult. They should be suspected in any patient with a central hematoma from either blunt or penetrating trauma. In the stable patient, CT scan with intravenous contrast can help to identify the area of injury. If CT scan is not possible preoperatively a clear plan of exposure and management is crucial prior to commencing any attempt at repair. Because of the advances that endovascular techniques have made in this type of injury, patients should be treated in an operating room that has the capability of intraoperative fluoroscopic imaging and angiography whenever possible. If an injury to the aorta or vena cava is suspected the first step should be confirmation by angiography through the femoral artery or vein as appropriate unless the patient is exsanguinating. Again, this is a visionary approach. I may be wrong, but I’m not sure this has been actually done much. I don’t know if Maingot is the right forum to roll it out. Following this, proximal and distal control should be established. Open exposure of the aorta at the diaphragmatic hiatus, or endoluminal balloon control, both described previously, can be performed. Injuries of the Vena Cava can initially be controlled by balloon tamponade, although this may reduce venous return to the right side of the heart. The American trauma world has a standard spiel on caval injuries which commonly goes like this: Sponge sticks are often better than clamps for control. Some prefer to control long linear injuries with a string of Allis or Babcock clamps, then suture as you remove one at a time. Back wall injuries can be accessed by extending the anterior injury or by ligating tributaries and rolling the cava over.

Open repair of Suprarenal Aorta:
The visceral aorta is exposed by a left medial visceral rotation described previously. If access to the posterior aorta is required the left kidney should be elevated along with the other viscera, if access to the anterior aorta is needed it is left in its bed. Direct suture repair is undertaken whenever possible. Direct repair that does not narrow the lumen of the aorta more than 50% or impinge on a visceral vessel is well tolerated. Larger defects may require patch angioplastic using prosthetic material, arterial autograft or arterial homograft. In the absence of significant contamination prosthetic material provides a readily available, strong and durable material for repair. In the presence of gross fecal contamination biologic materials may be required. Arterial homograft, if available provides the most expeditious alternative both for size and durability. We have been hesitant to use saphenous vein in this circumstances due to concerns about strength and durability and would prefer superficial femoral artery as a readily accessible conduit. The removed artery can be bridged by a short prosthetic graft if necessary. If appropriate, the aortic repair can be buttressed by an apron of omentum of some paraspinnous muscle, to separate the suture line from any visceral vessels. This should be done in the presence of associated visceral, injury, particularly injury to the pancreas. Drainage is established as needed. If the damage involves the origins of one or more of the visceral vessels these are ligated. Revascularization of these vessels is described below. Might be worth mentioning that you can’t use a damage control shunt in the upper aorta because you’ll occlude too many important branches. Seems obvious, but I had to figure it out for myself.

**Endovascular Repair of the Aorta:**

This emerging alternative should be considered in selected circumstances. In a stable patient with a contained injury, placement of a suitable covered stent can be combined with extra anatomic debranching of one or two visceral vessels, as has been described for treatment of thoracoabdominal aneurysms (REF). This is most suitable when the single mesenteric vessels is involved, since the bowel will tolerate more prolonged ischemia than the kidney. Modification of the stent graft (“fenestrations”) to allow continued visceral perfusion, is possible. This is most feasible when the aortic defect is posterior and relatively remote from the visceral orifices. More precise fenestrations, as required in suprarenal aortic repair, are currently beyond the capability of most surgeons in an acute setting. If a stent graft is selected, its diameter should be 110 to 115% of the normal aorta to allow for fixation. A variety of off the shelf aortic cuffs are available and their successful use has been reported in conjunction with thoracic aortic transection.

**Open Repair of the Suprarenal Inferior Vena Cava(IVC):**

Open repair of injuries of the suprarenal vena cava is one of the most difficult of all abdominal vascular operations. Exposure of the infrahepatic suprarenal IVC is achieved by an extended Kocher maneuver and right medial visceral rotation. One cannot overemphasize the use of intravascular balloon control in these cases to avoid hemorrhage. This control may need to be intermittent, since it will lead to reduced right
heart filling. Balloon control can be combined with external pressure and the application of partial occlusion clamps to provide hemostasis. Fine Allis clamps are useful in coapting and controlling the cut ends of the IVC and are preferable to more traumatic attempts at control. Wounds of the infrahepatic suprarenal IVC are usually managed by lateral venorraphy with running vascular suture. Narrowing the IVC 50-60% is often acceptable. Everyone asks if they should anticoagulate after these narrow repairs. They would welcome any advice, even if its just dealer’s choice. If lateral venorraphy is not possible then patch repair using prosthetic or biologic material is acceptable. Ligation of the suprarenal IVC should be avoided. Injuries to the retrohepatic vena cava, especially those that accompany blunt trauma, usually involve avulsion of the hepatic veins. Such injuries are highly lethal. Exposure of the retro hepatic IVC involves mobilization of the liver and anterior medial rotation of the right lobe as previously described. Repair of retrohepatic venous injuries may require hepatic isolation (control of the aorta at the hiatus as well as the vena cava above and below the injury and occlusion of the portal triad), placement of an intraluminal shunt between the right atrium and infrarenal IVC or veno venous bypass with hepatic isolation. Despite these techniques success is rare. In most cases, injuries in this area should initially be treated by packing, non expanding hematomas should not be opened, and the extent of injury should be defined and definitive repair planned after the patient has been stabilized.

**Endovascular Repair of the Suprarenal IVC:**

The complexities of, and poor results with, open injuries make an endovascular approach to suprarenal IVC injuries an attractive alternative to open surgery. Remote access and obviating a complex exposure, along with limited occlusion of the IVC are all points in favor of an endovascular approach. However at this point any such approach would be considered experimental. The size and distensibility of the IVC complicate the selection of an appropriate diameter endovascular graft. Patients with caval injury are often in shock and there may be external pressure on the vessel, both factors that cloud the estimation of caval diameter. No stent grafts have been made for caval use, and it is likely that aortic cuffs or short segment of grafts used for thoracic aortic repair would be most useful. Inadvertent coverage of the renal or hepatic veins represent a further complicating factor. Finally there have be no reports of endovascular treatment of hepatic vein injuries. Nonetheless, the potential treatment of these injuries by remote rather than direct access is appealing enough that it will undoubtedly be investigated in the future.

**Repair of the Infrarenal Aorta and Iliac Arteries:**

Injuries of the infrarenal aorta and iliac arteries can be managed by a combination of open and endovascular techniques. Use of an endovascular balloon to achieve proximal arterial control, described for ruptured aortic aneurysm, is an important component of
management. Still worried that this message, which applies to the minority of patients that aren’t actively bleeding, will be misused by the casual British general surgeon reader. I would be more comfortable with a message of “Most patients present with signs of hemorrhage and shock. They should have immediate lap with control of blah blah….Occasionally patients will present in stable condition. If equipment and expertise are available….blah blah. The balloon should be placed before celiotomy if possible, either through the femoral artery with a supporting sheath or the left brachial artery, as previously described. The balloon does not need to be inflated if the patient remains stable. Since concurrent visceral injury is common, laparotomy is almost universally required. After “damage control” of any gross intestinal spillage, attention is turned to the arterial injuries. Exposure of the aorta and iliac arteries has been described. In the presence of significant contamination, the infrarenal aorta and/or iliac vessels should either be repaired primarily, ligated, or a temporary shunt (commonly a nasogastric or thoracostomy tube) inserted as part of a “damage control “strategy. If ligation is required, extra-anatomic bypass with prosthetic material (e.g. axillofemoral bypass) can be used to restore perfusion to the lower extremities. If the aortic bifurcation is preserved a unifemoral pass is possible. In cases where the aortic bifurcation is not salvageable, primary end to end anastomosis of the proximal ends of the common iliac arteries can be performed, followed by axillo unifemoral bypass. If this is not possible, then axillo-bifemoral bypass may be required. Unilateral common iliac artery injuries may be ligated with subsequent cross femoral reconstruction using a prosthetic graft. Isolated external iliac artery injuries can be repaired in most cases with saphenous vein interposition I don’t understand. If its isolated (no contamination), then you advocate ptfé, right? Internal iliac artery injuries should be ligated. In the absence of significant contamination interposition graft replacement of the damaged vessel with a prosthetic graft is preferred.

As you know, most of these patients have simple vessel transections, and some bowel holes that have spilled a few tablespoons or cups of stuff. I think readers should get clear instructions about what to do with these bread-and-butter iliacs. I don’t know your biases, but my training and practice has been to rinse them out, sew in ptfé, and try to cover it with something. This practice is supported by the following paper (among others). My second choice, if you are not a ptfé fan in these circumstances, would be to say that you don’t like it, but that some do it with apparent success.
Endovascular repair of injured aorta and iliac vessels can be performed using techniques applied for repair of endovascular infrarenal aortic aneurysm repair. However one must remember that many of these patients are young and the durability of these repairs is unknown. In addition most patients will require laparotomy for associated injuries. These two factors suggest a limited role for stent grafts in the treatment of traumatic lesions of the aortoiliac system. However as previously noted, endovascular balloon tamponade is a valuable technique and endovascular coil embolization of difficult to access hypogastric artery branches can be employed with great success.

**Infrarenal IVC and Iliac veins:**

The principles of controlling venous injuries, including use of balloon tamponade and external pressure, has been previously described. The Infrarenal IVC, iliac confluence and right iliac vein are exposed through a right medial visceral rotation. The confluence of the iliac veins is obscured by the aortic bifurcation and right common iliac artery. If the aortic bifurcation cannot be sufficiently mobilized to provide exposure the right common iliac artery should be mobilized or even transected for additional exposure. This is often required in any event since concomitant arterial injury is common. The more distal left iliac vein is approached on either side of the descending/sigmoid colon depending on the location of the injury.

As with the suprarenal IVC, lateral venorraphy is the preferred approach, with autogenous vein patch or ligation as alternatives. If needed the infrarenal IVC and iliac veins can be ligated acutely, due to the rather extensive collateral network that can develop within hours. While this may cause fluid sequestration in the lower legs, it is usually tolerated in the short term and is preferable to an attempt at repair in an unstable...
In the rare case that ligation results in extreme distal venous hypertension, a bypass graft is indicated. In patients who are stable, we prefer venous repair, either with a vein patch, or when an interposition graft is required, a panel graft, or a ringed prosthetic conduit. Successful venous repair must use a conduit of equal or slightly greater diameter than the native vein and should avoid any tension. Saphenous vein is of insufficient diameter for replacement of the iliac vessels and must be modified to be useful ( "panel” grafts”). We find such panel grafts excessively time consuming to construct in these critically ill patients and prefer externally supported PTFE of suitable diameter and length. When short segments of prosthesis are used in the presence of distal venous hypertension, flow is usually sufficient to maintain patency without the need for anticoagulation or an adjunctive fistula. In our experience, when thrombosis of a prosthetic vein graft does occur, adequate collateral venous flow has been invariably present. Do you want to specify a role for extraanatomic venous bypass like we did the other week?

Treatment of Traumatic Arteriovenous Fistula:

Fistula between the major arteries and veins can occur at any level, since the vessels are in close proximity throughout their course. It is important to realize that, while this may occur acutely, such a fistula rarely represents a true vascular emergency. Exsanguinating hemorrhage does not occur, since the arterial blood is decompressed into the venous system. These patients may present with a continuous abdominal bruit, signs of lower extremity edema and high output cardiac failure. Management depends on an accurate history of trauma, including prior surgery (particularly lumbar disc surgery) or endovascular manipulation. Detailed vascular imaging is essential. These patients are rarely in extremis, and an effort to delineate the problem and develop a careful plan of correction is time well spent. Repair can usually be delayed until the patient is stabilized and other acute problems are corrected. Treatment is directed at repair of both the arterial and venous defect. This is most often done by primary suture closure, although patch closure is sometimes required. Proximal and distal arterial control is essential and is obtained using open or endovascular techniques as described above. Proximal and distal venous control should be obtained when possible before opening the fistula. This can be done by external dissection, compression or an intraluminal balloon. Central venous occlusion is important to prevent air embolization when the vein is opened. We generally avoid extensive venous dissection in close proximity to the fistula. On occasion venous control can be obtained by placing a balloon catheter through the fistula from within the artery and then closing the communication with interrupted or running sutures. In the acute circumstance the artery and vein may be separated, but this is more difficult in the case of a more chronic fistula and closure of the communication, by primary suture or patch, can be done from within the vessel. If this approach is chosen it is important to be sure that the communication has been completely interrupted at the end of the procedure by use of intraoperative ultrasound or angiography. Appropriate flushing of on both the arterial and
venous sides is important to avoid embolization of debris or air into the central venous circulation.

Arterial-venous communications can also be approached endovascularly using covered stents. The stent can be placed only on the arterial side of the defect if the site of injury is in a main artery and can be accurately identified. However it is important to remember that the arterial injury may be in a branch of one of the iliac vessels, in which case placement of a stent graft in the main artery will not correct the abnormality. Repair of these branch fistulae is complex and beyond the scope of this chapter. Suffice it to say that coil embolization is particularly dangerous in these cases due to the high flow in the venous system and chance of central venous embolization. A variety of techniques can be employed to reduce this possibility. Endovascular treatment of these lesions should only be undertaken by those with significant experience in endovascular techniques. As with open repair, it is important to be sure that complete interruption of the fistulous communications has occurred using completion angiography.

Trauma to the Mesenteric Arteries and Veins:

The origin of the celiac axis is exposed through the gastrohepatic ligament or by a left medical visceral rotation as described above. A short bypass from the aorta to the bifurcation of the splenic and hepatic arteries can be performed, usually with prosthetic material; however, the origin of the celiac artery can be ligated safely if necessary in most cases. This is preferable to attempting repair in a relatively small space. Collaterals through the pancreaticoduodenal and gastroduodenal are usually sufficient to preserve foregut flow. If there is any doubt, a bypass can be performed from the aorta to the common hepatic artery. The splenic artery can be ligated, as can the splenic vein. In the case of proximal injuries to these vessels the short gastric vessels provide adequate collateral flow. When the Splenic vessels are injured close to the hilum, a splenectomy is usually the best approach. Injuries of the common hepatic artery may be ligated because of collateral circulation, while injuries of the proper hepatic artery are more likely to require repair. Techniques in order of preference are primary repair, interposition vein graft and aortomesenteric graft using either saphenous vein or prosthetic. Two thirds or more of hepatic flow is supplied by the portal vein, and if this is intact, proper hepatic artery ligation becomes an acceptable option. Intrahepatic arterial lesions are generally treated with angiographically directed coil embolization unless massive exsanguination requires resection of the damaged area of the liver.

Injuries to the main trunk of the superior mesenteric artery should be repaired since significant loss of small bowel may result from sacrifice of the vessel. As noted below, ligation of proximal SMA aneurysms can be performed with acceptable results, due to the presence of collateral from the celiac and inferior mesenteric arteries. However, in the trauma setting, integrity of collateral pathways from the pancreaticoduodenal and middle colic vessels is not easily ascertained and repaired should be performed. Lesions at the
origin of the vessel are best repaired with a short bypass originating from the aorta. More distal lesions can be repaired by patch angioplasty, interposition graft using saphenous vein, or proximal ligation and distal bypass arising from the aorta. In the trauma setting, the infrarenal aorta is preferred as inflow for the more distal SMA since supraceliac exposure and control can be avoided in patients who may be unstable and have multiple injuries. Saphenous vein is the preferred conduit. The details of SMA bypass have been described, particularly the need for proper length and orientation to prevent kinking. Lesions of the branches of the SMA are usually ligated and any non viable bowel is resected. Attempts to repair the distal arterial and venous injuries in the mesentery are not rewarding.

Injuries of the inferior mesenteric artery can usually be ligated, since adequate collaterals will exist from the arc of Riolan, the marginal artery of Drummond and the hemorrhoidal vessels. If it appears that ligation will not be tolerated, reimplantation or a short bypass with saphenous vein is indicated.

Injuries to the splenic vein are treated by ligation, with or without splenectomy. There is often an accompanying injury of the splenic artery. In the rare instance of isolated splenic vein injury, consideration should be given to concomitant splenic artery ligation or splenectomy. Acute ligation of the splenic vein alone may result in sequestration of significant amounts of blood within the spleen and eventually left sided portal hypertension. This can be ameliorated by ligating the main arterial inflow to the spleen. Injuries to the main trunk of superior mesenteric vein should be repaired to avoid bowel ischemia secondary to mesenteric venous obstruction. If the vein cannot be repaired using a patch angioplasty or short interposition graft, a bypass from the superior mesenteric vein to the portal vein should be performed. This probably will require a large (6-8 mm conduit of either reinforced PTFE or deep vein (jugular or femoral). Injuries to the portal vein should be repaired if possible, by lateral venorraphy, patch angioplasty or interposition grafting if the patient is stable enough to undergo repair. The retropancreatic portal vein is best exposed by transection of the pancreas. (SEE FIGURE) Isolated injuries of the portal vein, with an intact hepatic artery, may be ligated if necessary to save the life of the patient, although significant hepatic dysfunction may ensue. Massive bowel edema with fluid sequestration can be anticipated, requiring volume repletion and limiting subsequent surgical access to the upper abdomen. Lesions of the hepatic artery and portal vein that are not immediately lethal should be repaired if possible.

Injuries to the Renal Artery and Vein:

Management of Renal artery lesions is dictated by the overall status of the patient, duration of ischemia and presence or absence of a contralateral kidney. It is important to remember that after 60 minutes of warm ischemia time most of the kidney’s excretory function is lost. While some authors advocate renal vascular repair within the first 3-6 hours after injury, preservation of long term renal function in these cases has been poor (REF). Therefore, situations in which there is non visualization of one kidney on a
preoperative CTA or IVP suggests that renal function will not be salvaged by revascularization. In most cases then ligation, with nephrectomy is indicated. We do not usually do a laparotomy to take out the kidney that is black on CT for two reasons: They have been reported to sometimes recannulate and return to function, and they don’t usually cause a problem. In circumstances where the status of the kidney is unknown, or when there is not a contralateral kidney, attempts at revascularization should be undertaken. The most expeditious approach is aortorenal bypass for lesions of the main renal artery, using saphenous vein with PTFE as a second choice. Lesions of the more distal renal artery, at or beyond branch points, are best ligated in the acute situation, unless they can be repaired with a simple vein patch, or the injury is to a solitary functioning kidney. In a typical young, healthy trauma patient a palpably normal contralateral kidney can be assumed to be functioning. If there is any doubt, the ureter of the injured kidney can be interrupted with a bulldog clamp and methylene blue or indigo carmine administered. Passage of dye-tinted urine into the urinary catheter bag confirms contralateral kidney function.

Lesions of the proximal renal veins may be ligated, as long as collateral flow through the gonadal, adrenal and hypogastric veins is preserved. While it is known that some transitory renal dysfunction will occur after renal vein ligation, it is generally well tolerated. I thought the dogma was that this works on the left because of the gonadal but not on the right. In any event we take the kidney pretty liberally these days, having been reassured by the safety of donor nephrectomy. If inadequate venous collaterals exist, or have been damaged during the course of the injury, a short bypass between the renal vein and the vena cava with 8-10 mm PTFE is indicated. Under rare circumstances of injuries to the renal hilum, e.g. with a solitary kidney, nephrectomy with ex vivo repair and autotransplantation may be indicated. However this extensive surgery is unwise in an unstable patient with a contralateral functioning kidney. I think you should make clear that this applies to a ptf e renal vein bypass as well. I think its too much time and surgery if there’s another one working.

TREATMENT OF RUPTURED ABDOMINAL AND VISCERAL ANEURYSMS.

In the patient presenting with abdominal pain, pathology of the abdominal aorta and its branches should always be included within the differential diagnosis. Because of their rapidly catastrophic potential, prompt diagnosis and timely treatment, or rupture abdominal aneurysms, is mandatory for patient survival and a successful outcome. While the most common aneurysms of the abdomen involve the abdominal aorta and iliac arteries, aneurysms of the visceral vessels may also rupture and present as abdominal emergencies.

Ruptured Aneurysms Of The Aorta And Iliac Arteries:

Although historically called *atherosclerotic* aneurysms, the etiology of abdominal aortic aneurysms (AAAs) has come to be recognized as multifactorial. This complex interplay, which includes elastin degradation, increased proteolytic activity,
inflammation, matrix metalloproteinases, and other factors, leads to the ultimate development of aortic expansion and degeneration. It is for this reason that the term degenerative aneurysm better describes the pathophysiology of abdominal aortic aneurysms. Familial and sex-linked factors also likely contribute: the incidence is several times higher in men, and the relative risk for development of AAA among first-degree relatives of affected individuals is increased 11-fold. The infrarenal aorta is the most common intraabdominal location for aneurismal degeneration, Aneurismal degeneration of the suprarenal aorta is much less common.

Despite advances in treatment and early diagnosis, abdominal aortic aneurysms (AAAs), continue to be a significant cause of death. In the United States, AAAs are the 15th cause of death overall and the 10th leading cause among men over the age of 55. With improvements in the operative and perioperative management of elective AAAs, coupled with the introduction and refinement of endovascular techniques, ruptured AAAs overwhelmingly account for most of these deaths. Even among specialized centers, the operative mortality for ruptured AAAs is at least 40%, a number which has remained constant over the past three decades. When one also considers the proportion of patients who die without reaching the hospital, the mortality rate approaches 75%. Accordingly, and because AAAs are notoriously asymptomatic until ruptured, much clinical research has centered on the natural history of the disease, specifically focused toward identifiable risk factors for rupture.

The absolute diameter of the aneurysm is the principal determinant of rupture risk. As the diameter increases, the risk of rupture increases nonlinearly, such that larger aneurysms have a significantly higher rupture rate. For example, AAAs less than 5 cm have an annual rupture risk of less than 5%, whereas those 6 to 7 cm in diameter have a 10-15% annual risk of rupture. These “hinge points”, in which the rupture risk rises dramatically, are the basis for recommending elective repair for asymptomatic AAAs based on size alone (in general, greater than 5.5 cm in average risk patients). Several other factors also independently predict rupture risk. The strongest variables are hypertension, chronic obstructive pulmonary disease (COPD), and family history of AAA. Possible other risk factors include rapid expansion (more than 0.4 cm annually), female gender, and current smoking history.

The classical presentation for ruptured AAA is abdominal or back pain, pulsatile mass, and hypotension; however this complete triad is present in only a minority of patients. A large pannus or abdominal girth may preclude appreciation for a pulsatile mass; similarly, a blood pressure of 100 mm Hg systolic in an otherwise hypertensive individual may be mistakenly interpreted as “normotensive”. Pain is almost always a presenting symptom, and may include abdominal or back pain, groin pain, testicular pain, or flank pain. Less commonly, a patient with a large ruptured AAA may be obtunded and can present with hypotension only. The diagnosis of ruptured AAA must be included among the differential in every patient over the age of 50 years presenting with abdominal pain, abdominal pain and hypotension, or hypotension alone. When a pulsatile mass is also appreciated, the diagnosis of ruptured AAA is almost certain.

Much less commonly, an aorto-caval fistula may arise from rupture into the adjacent inferior vena cava; signs and symptoms may include a bruit, distended veins, and acute heart failure. In general these patients may be hypotensive but can usually be resuscitated. Since their treatment is different from that of a ruptured aneurysm, careful
examination of the abdomen, with an effort to identify a thrill or bruit will help in diagnosis.

**Diagnostic Imaging**

Ultimately, the decision for imaging should depend on the patient’s hemodynamic stability. In the patient with abdominal pain and hypotension and a pulsatile abdominal mass, immediate transport to the operating room without imaging is indicated. In the more stable patient, in whom the diagnosis is in question, abdominal ultrasound may be performed rapidly in the emergency room to identify AAAs. When performed expeditiously by an experienced ultrasonographer, the diagnosis of ruptured AAA may be rapidly confirmed. However, the technique is operator dependent and accuracy may be limited by excessive bowel gas and obesity.

CT scanning is the most accurate and useful radiographic method in the evaluation of ruptured AAA. (Figure1) The most common findings are retroperitoneal hematoma, aneurismal aorta, retroperitoneal stranding of blood. With a 100% specificity and a very high sensitivity\textsuperscript{xviii}, CT can reliably confirm or rule out the diagnosis of ruptured AAA and identify alternate on vascular causes of the patient’s symptoms. It also yields important anatomical information about adjacent structures (such as a retro-aortic left renal vein, horseshoe kidney, or concomitant iliac aneurysms) and about the aneurysm itself (such as an inflammatory AAA) CT scanning is particularly important if endovascular repair is contemplated. The newer generation of multi-slice scanners allow for complete chest and abdominal imaging to be completed in less than five minutes. Although intravenous contrast is very helpful in the planning for elective AAA repair, it is not required for diagnosis in the patient with suspected rupture, and may exacerbate postoperative renal dysfunction. Even with an endovascular approach, thin slice (2 mm) non contrast CT can provide sufficient information for repair.

**Preoperative Management**

Once the diagnosis of ruptured AAA is made, either by clinical presentation or radiographically, the patient should be taken immediately to the operating room. Large-bore intravenous access in the upper extremities (or central venous access), indwelling urinary catheter, type and cross match for at least 6 units of packed cells, and chemistry and coagulation studies should all be performed. Because elevated blood pressure may lead to frank rupture of an otherwise contained leak, a strategy of permissive preoperative hypotension with minimal fluid resuscitation has been recommended. Although no rigid blood pressure parameter exists, most vascular surgeons would favor a minimum systolic pressure to maintain consciousness (usually around 80 mm Hg systolic).

**Open Repair**

Open repair remains the most common and versatile approach to ruptured AAA. Since general anesthesia will lead to both generalized vasodilatation and relaxation of the
abdominal musculature, both of which can produce abrupt hypotension; the patient must 
be prepped and draped ("nipples to knees") and the surgical team scrubbed prior to 
induction. A cell saver device should be set up and utilized when possible. A midline 
incision is performed for rapid access to the supraceliac aorta. After induction, the 
abdomen is opened from xiphoid to pubis. The abdomen and retroperitoneum are 
inspected. If a small or moderate retroperitoneal hematoma is found without 
intraperitoneal blood, the supraceliac aorta is controlled, as described earlier, but the 
artery is not clamped. If the juxtarenal aorta is spared of hematoma, this area may be 
dissected and a clamp applied directly below the renal arteries. Should bleeding develop 
during the course of this dissection, the supraceliac clamp is applied.

If intraperitoneal blood is present, rapid supraceliac aortic control is obtained, 
usually by manual compression at the diaphragmatic hiatus while the anesthesiologist 
rapidly continues resuscitation. The supraceliac aorta is then exposed as previously 
described and occluded with a vascular clamp. Once the cross clamp is placed, the distal 
aorta is palpated to confirm obliteration of the pulse and attention is turned to the 
aneurysm. In patients with massive rupture, bleeding, or hypothermia, in which 
coagulopathy is almost certainly present, heparin is not given. In such cases, 
thrombectomy of the distal vessels and vigorous flushing of the graft is necessary prior to 
restoring flow. In all other cases, we give a small dose of heparin, 40 – 50 
units/kilogram.

There is an increasing tendency to obtain intravascular supraceliac balloon control 
of the aorta prior to celiotomy. This is performed by passing a wire and then a balloon in 
to the supraceliac aorta via either a retrograde transfemoral or a prograde transbrachial 
approach, as described earlier in the chapter, prior to induction of anesthesia. This 
requires intraoperative fluoroscopic capabilities and catheter/guidewire skills. This 
approach provides less invasive and more rapid control of the supraceliac aorta and can 
facilitate resuscitation of the patient in circumstances of profound shock.

The aneurysm is approached by evisceration of the transverse colon and omentum 
cephalad and the small bowel to the right. Care is taken not to injure the IVC or the 
inferior mesenteric, gonadal, or left renal veins. In most cases, the retroperitoneal 
hematoma facilitates the dissection. Efforts are made to identify an infrarenal neck of the 
aneurysm and place a clamp at this level. When there is a free rupture of the aorta, the 
surgeon can pass the fingers of one hand through the rupture into the aorta (after 
application of the supraceliac clamp) to help locate the proximal neck of the aneurysm. 
Bimanual palpation can facilitate the placement of a clamp above the aneurysm without 
extensive dissection. (NEED SOME DRAWINGS) Once the aortic neck is controlled, 
the iliac vessels are dissected to allow for clamping and control. Since the iliac veins 
often adhere to the artery, circumferential dissection around the iliac arteries should be 
avoided to prevent vein injury. In most cases, the iliac arteries may be readily clamped 
with minimal dissection. However, if the dissection is difficult, as with a large distal 
hematoma, endoluminal control may be obtained using a number 5 occlusion balloon, 
placed in each iliac artery after opening the sac. FIGURE Once the aneurysm has been 
isolated proximally and distally, the sac is opened longitudinally and thrombus 
evacuated. Bleeding from the lumbar vessels is controlled with direct suture ligation
using a mattress suture. *(FIGURE)* Venous bleeding encountered inside the sac suggests an aorto caval fistula. In those cases the patient should be placed in mild Trendelenberg position to reduce the chance of air embolus and the venous bleeding controlled by pressure. The defect is oversewn from within the aneurysm sac, with gentle digital compression of the cava proximally and distally *(FIGURE)*. No attempt is made to clamp or mobilize the cava.

Because of the significant risk of colon ischemia following ruptured AAA repair, reimplantation of the inferior mesenteric artery (IMA) should be considered in cases of ruptured AAA. Brisk back bleeding suggests adequate SMA collaterals and implantation is not required. If the IMA is patent and back bleeding is absent or sluggish reimplantation of the IMA should be planned after aortic repair. In these cases, the IMA is controlled just outside the aneurysm sac with a small bulldog clamp, and after the aortic repair, the IMA is reimplemented on the aortic graft using a Carrel spatulated patch. An IMA that is obviously occluded at its origin is not reimplanted.

With the aneurysm opened and bleeding controlled, the graft may be sewn in place. When possible this is done with an infrarenal clamp in place. It is absolutely mandatory that the proximal anastomosis be sewn meticulously into relatively healthy (nonaneurysmal) aorta. Poorly placed sutures in friable aorta will lead to proximal suture line bleeding once clamps are removed. If a secure anastomosis cannot be performed with an infrarenal clamp, the proximal anastomosis should be done with a suprarenal clamp in place. Tamponade of visceral back bleeding may be required while this is performed. Sutures must be placed in the aorta precisely and without tension or torsion of the needle. The proximal anastomosis may be reinforced with a Teflon felt pledget. *(FIGURE 2)*

Once the proximal anastomosis is completed and judged to be satisfactory, heparinized saline (5000 units/1000 ml saline) is flushed into the graft and the graft clamped. The distal anastomosis is then performed in similar fashion. If heparin had not been given, a number 4 balloon thrombectomy catheter is gently passed down each iliac artery to extract thrombus. The graft should also be flushed to ensure adequate forward flow and the anastomosis is then completed. *(FIGURE 3)*

The anesthesiologist should be notified prior to release of the distal clamps. One leg should be perfused gradually, once the pressure has stabilized, the contralateral leg may be perfused. Pulses are checked at the femoral level, and should be palpable; if not, thrombus or emboli are likely present and should be treated with thromboembolectomy. With the blood pressure stabilized and following a period of adequate perfusion, both feet should be assessed. Although palpable pulses may not be present, the feet should appear viable with reasonable capillary refill with Doppler flow.

Once adequate perfusion to the lower extremities has been achieved, the colon should be assessed. The colon should appear pink and Doppler flow should be present ideally at the antimesenteric border. If the colon appears ischemic, then IMA reimplantation should be performed if not already done.

Hemostasis should be assured as best possible prior to closure, and this may require infusion of additional clotting factors and protamine if heparin were given. The aneurysm sac is closed snugly around the graft with a running suture to obliterate the dead space and provide some hemostasis. The intestines should be excluded from contact with the graft as best possible, usually by closing the proximal retroperitoneum or occasionally with a mobilized segment of omentum.
If the abdomen can be closed without tension, then the linea alba is approximated and closed with a running suture. However, in many cases, the substantial hematoma precludes closure, and to prevent the development of abdominal compartment syndrome, the abdomen is left open with subsequent delayed closure several days later.

Endovascular Repair

The rationale for endovascular repair (EVAR) for ruptured AAAs is extrapolated from data showing less blood loss and improved outcomes in patients undergoing elective endovascular AAA repair\textsuperscript{xx} and from direct data from specialized centers demonstrating encouraging results with ruptured AAAs \textsuperscript{xx}. EVAR requires accurate assessment of aneurysm geometry using either CT scan or intraoperative calibrated angiography. Accepted anatomic criteria for EVAR include: 1) aortic neck diameter between 18 and 32 mm; 2) aortic neck length $\geq$ 10 mm; 3) proximal aortic neck angulation $\leq$ 60 degrees; 4) iliac artery fixation diameter of between 8 and 22 mm; 5) distal iliac artery fixation length of $\geq$ 10 mm (preferably $> 15$ mm); 6) access vessel diameter $\geq$ 7.5 mm. Other considerations include the degree of iliac tortuosity, circumferential thrombus or calcification, and the aortic length. Data suggest that 60% or so of ruptured AAA may be eligible for EVAR based on these criterion (NEED REF)

Successful application of EVAR technology in treatment of ruptured AAA requires an experienced surgical team, adequate endovascular imaging capabilities and an adequate supply of grafts, sheaths guidewires and balloons. The single most important consideration is the ability to expeditiously proceed with endovascular aortic control and suitable repair in the patient with a ruptured AAA before irreversible shock occurs. Multiple centers have described their techniques and operative strategy, and some variation exists; however, the fundamental principles are identical to our center’s technique. **WE NEED TO DISCUSS THIS** The preoperative management and anesthetic considerations are the same as for open repair. Either local or general anesthesia, the advantage of the former being that the fall in blood pressure with induction is avoided. This is most advantageous while balloon control is being obtained. In most cases the repair is completed under general anesthesia to facilitate control of the patients airway and minimize motion.

Access is obtained through both femoral arteries simultaneously. One artery may be accessed percutaneously with placement of a closure device. Once access is obtained by a Seldinger technique, bilateral 6 French sheaths are placed over floppy wires, subsequently exchanged for a stiff wire over a guiding catheter to the level of the proximal descending aorta. Contralateral to the side proposed for deploying the main body of the graft, the sheath is exchanged for a 12-20 French sheath and a compliant 45 mm aortic balloon introduced to the level of T12. If the patient is hemodynamically stable the procedure can proceed with the balloon in place but not inflated. Through the same 20 French sheath, a marking pigtail catheter is introduced over a second floppy wire, aortogram is performed, and the position of the renal arteries marked. The main body graft is then introduced through the opposite femoral artery over the stiff wire and deployed as is normally done for an elective EVAR. (Figure 5) The contralateral gate of the graft is then cannulated and the contralateral limb is placed through the sheath with the aortic occlusion balloon.
If the patient is unstable the balloon is inflated after the sheath has been brought into the suprarenal aorta to stabilize the inflated balloon. Angiography and deployment of the main body proceeds through the opposite femoral artery. After the main body is deployed the balloon is deflated and withdrawn below the main body of the graft. If the patient develops further hypotension, the balloon catheter may be reintroduced through the main body of the graft cannulated contralateral gate into the suprarenal aorta and re-inflated. The ipsilateral limb deployment is completed and over the same stiff wire, the aortic occlusion balloon catheter is reintroduced and inflated. The contralateral limb graft is introduced and deployed. The aortic occlusion balloon is again deflated (if not already), and an ipsilateral limb extension is introduced and deployed. Once the endografting has been performed, all fixation sites are molded with the compliant balloon, and a completion aortogram performed to document absence of endoleak. A Type I (attachment or perigraft leak) or Type III endoleak (modular disconnection) warrants further repair prior to leaving the operating room, whereas a Type II (branch endoleak) or Type IV (graft porosity) endoleak may be followed conservatively.

The femoral arteries are then closed primarily. If heparin had not been administered, inflow and back bleeding should be assessed prior to closure, and if judged to be poor, a thrombectomy catheter may be passed gently to retrieve thrombus.

Although the above describes one approach for ruptured EVAR, multiple options exist, and the surgeon should be well acquainted with the options based on anatomic criteria, should an endovascular approach be undertaken. These may include conversion to an aorto-uniliac device with a femoral-femoral crossover graft or a proximal aortic extension in the case of a Type I endoleak. It is anticipated that the future generation of endografts, along with greater surgeon experience, will lead to greater use of EVAR for ruptured AAA.

Results

Although some variation exists among individual series, pooled data suggests an overall perioperative mortality of approximately 50% after open repair for ruptured AAA. Attempts have been made to correlate both preoperative and postoperative variables with the probability of survival. Poor prognostic preoperative predictors include hypotension on induction (systolic blood pressure < 90), age over 80 years, preoperative cardiac arrest, and low hematocrit (27). Similar logistic regression analysis has identified postoperative myocardial infarction, respiratory failure, coagulopathy, and renal dysfunction as strong predictors of postoperative mortality; the probability of survival decreases dramatically with two or more complications or with the need for dialysis.

Encouraging survival results have been reported following EVAR for ruptured AAA, with perioperative mortality of less than 20% and decreased renal, cardiac, and respiratory complications when compared to historical (open repair) controls. Clearly, these admirable results cannot be applied universally, given the small number of
specialized centers routinely performing ruptured EVAR, and whether this will lead to a global decrease in the mortality of ruptured AAA is unknown.

**Visceral Artery Aneurysms**

Aneurysms of the visceral arteries are uncommon, seen in 0.01 to 0.02 percent of autopsy studies. However, the increased utilization of routine body imaging has resulted in greater recognition and discovery of asymptomatic visceral artery aneurysms, and thus their true prevalence is likely higher. The elective treatment of visceral aneurysms is beyond the scope of this chapter. The major complications of these aneurysms are rupture or distal embolization and prevention of these complications is the rationale for elective treatment. Table XXX summarizes the relative frequency of these aneurysms, their estimated risk of rupture and recommended treatment. Approximately 20-25% of splanchnic artery aneurysms are ruptured at the time of presentation (REF) and about 1/3 are associated with aneurysms elsewhere in the arterial tree (REF) This chapter will not concern itself with the elective management of visceral aneurysms, but rather the proper surgical approach once rupture has occurred.

**Splenic Artery Aneurysms** are the most frequent visceral aneurysms (60%) of visceral artery aneurysms are the only aneurysms with a female predominance (3:1) and have the lowest risk of rupture. Splenic artery aneurysms have the lowest risk of rupture, perhaps no more than 10% overall and less than 2% in low risk patients. However, the risk of rupture rises dramatically among pregnant patients, with maternal and fetal mortality rates of over 70%, and after liver transplantation, which is the rationale for recommending repair of asymptomatic aneurysms in these groups. Both arterial medial dysplasia (more common in females) and the underlying vascular effects of multiple pregnancies (both hormonal and hemodynamic) have been proposed as contributing factors. Other possible etiologies include portal hypertension and splenomegaly, pancreatitis or pseudocyst-associated local inflammation, and trauma. Ruptured splenic artery aneurysm initially presents with abdominal pain referable to hemorrhage in the lesser sac without abdominal distention or shock. These signs may become apparent later after continued hemorrhage spills into the peritoneal cavity through the Foramen of Winslow (“double rupture”).

In most cases splenic artery aneurysms are treated by laparotomy and ligation. Restoration of arterial continuity is rarely necessary because of the collateral supply to the spleen, and therefore either open or endovascular obliteration of the aneurysmal segment is appropriate. Operative repair of proximal and mid splenic artery aneurysms entails exposure through the lesser sac, proximal and distal control, and simple ligation of the aneurysm without arterial reconstruction. It is important to ligate all feeding vessels; this may require opening the aneurysm and ligation from within the sac. Aneurysms of the splenic hilum require mobilization of the spleen and may be treated by ligation of all branches or splenectomy if necessary. As in trauma, early control of the proximal splenic artery is important or the treatment of hilar aneurysms. While laparoscopic techniques have been reported for the elective resection of splenic aneurysms, they have no place in the acute setting. Endovascular approaches are generally reserved for patients at high
operative risk such as those whose aneurysms are associated with pancreatitis, advanced portal hypertension or liver transplantation. In these cases, if the patient is stable, vascular access to the splenic artery is obtained through the celiac artery from a femoral or brachial approach. Using guiding sheaths and micro catheters the splenic artery is engaged and coils are placed distal to the aneurysm, in the aneurysm sac and then proximal to the aneurysm. There is a 10-15% risk of re-bleeding (REF) using endovascular techniques, as well as a risk of splenic infarction when hilar aneurysms are treated. However the difficulties of open surgery in patients with pancreatitis or advanced liver disease justify attempts at endovascular treatment as a first effort. Endovascular stent graft placement has also been described, and may be particularly useful in certain subsets, such as patients in whom preservation of splenic blood flow need be maintained (as for portal-systemic shunts) or in high-risk patients with pancreatitis associated aneurysms and severe inflammation.

**Hepatic Artery Aneurysms:**

Hepatic artery aneurysms, unlike splenic artery aneurysms, occur more frequently in men. Etiologies include medial degeneration, atherosclerosis, trauma (up to 20% of cases), infection (usually secondary to illicit drug use), vasculitis, and as a consequence of orthotopic liver transplantation. Approximately 80% of hepatic artery aneurysms are extrahepatic (REF). Hepatic artery aneurysms have a rupture risk of no less than 14% and possibly higher. About half the ruptured hepatic artery aneurysms present with signs and symptoms of intraperitoneal hemorrhage; while the other half will rupture into the biliary tract, manifesting as either hemobilia or gastrointestinal hemorrhage.

A variety of treatment options exist for hepatic artery aneurysms, including ligation, excision, repair with arterial grafting and reconstruction, hepatic resection, and endovascular approaches. Treatment of ruptured hepatic artery aneurysms generally depends on their location and the status of hepatic blood flow. When feasible, preoperative arteriography is helpful in planning operative approach. Arteriography can provide information on the collateral flow to the liver, demonstrate anomalies such as a replaced right or left hepatic artery, and identify multiple aneurysms, especially in the case of intrahepatic lesions.

Ruptured common hepatic artery aneurysms are treated by simple ligation and exclusion, unless the liver appears ischemic after clamping. Collaterals from the right gastric and gastroduodenal arteries will maintain hepatic artery flow in most cases. Arterial reconstruction is indicated for most aneurysms of the proper hepatic artery and its extra hepatic branches unless the patient is to unstable to tolerate attempts at bypass. In most instances, this requires interposition grafting (preferably with autologous saphenous vein) aneurysmectomy or endo aneurysmorrhaphy. Due to their proximity to the bile duct and portal vein, dissection of the more distal hepatic or extrahepatic branch arterial aneurysmal segments may be tedious, and proximal and distal control may be easier from within the aneurysm itself. Ruptured aneurysms may require concomitant control at the supraceliac aorta level. If an interposition graft is not possible (as with distal common or proximal proper hepatic artery aneurysms), an aortohepatic bypass can be performed by exposing the right anterolateral border of the aorta through an extended...
Kocher maneuver and medial visceral rotation. The aortic anastomosis is performed first, the graft is tunneled retro-duodenal to the porta hepatis and anastomosed to the hepatic artery after opening the aneurysm. If the patient is unstable, ligation of the hepatic artery, at any level is acceptable as long as the portal vein is patent; the risk of hepatic infarction is low and is less than the risk of an extended procedure in a compromised patient.

Intrahepatic aneurysms are best treated by catheter based embolization unless they are large. Options for endovascular treatment of hepatic artery aneurysms include both coil embolization and stent graft placement. Embolization has been most useful for small, saccular intrahepatic pseudo-aneurysms, as may be seen following trauma or percutaneous biliary procedures with iatrogenic arterial injury. Large intrahepatic aneurysms may require liver resection. Endovascular approaches have also been described for extrahepatic aneurysms, including both coil embolization and the placement of endovascular covered stents.

Superior Mesenteric Artery Aneurysms:

Superior mesenteric artery (SMA) aneurysms have been associated with an infectious etiology, dating back to DeBakey and Cooley’s 1953 report of successful resection of a mycotic aneurysm, and systemic infection (usually associated with endocarditis) continues to be a significant factor in their development. Other less common causes of SMA aneurysms include atherosclerosis, connective tissue disorders, vasculitis and trauma. The risk of rupture of SMA aneurysms is in the range of 40-50%. The majority of SMA aneurysms occur in the proximal 5 cm of the vessel. SMA aneurysms are usually symptomatic, presenting with abdominal pain and sometimes signs of intestinal angina. Treatment of ruptured superior mesenteric artery aneurysms is complicated by their frequent infectious etiology, and difficulty with arterial reconstruction. Unlike the situation with trauma to the SMA, resection and reconstruction of aneurysms is often more difficult since the lesion is more extensive. While early teaching mandated proximal SMA reconstruction, larger, contemporary series suggest that ligation without revascularization can be considered in most patients. In these cases, test occlusion of the vessel to assess the extent of intestinal ischemia is critical prior to decision on the need for reconstruction. When collateral circulation from the celiac and inferior mesenteric arteries, through the pancreaticoduodenal middle colic vessels respectively, is sufficient to maintain intestinal viability after test occlusion of the SMA, ligation can be performed. If extensive intestinal ischemia is present after test occlusion, bypass grafting is required. This is usually performed as an interposition graft or a bypass from the infrarenal aorta, using autogenous vein. More distal aneurysms of the SMA can often be treated by ligation with resection of the compromised small bowel as needed. Access to the origin of the SMA is obtained by left medial visceral rotation. The more distal segments of the SMA are exposed by elevating the mesocolon and dissecting through the small bowel mesentery, using the middle colic artery as a guide. Transcatheter embolization is usually reserved for multiple small bleeding aneurysms in a hemodynamically stable patient. Assessment of bowel viability by angiographic determination of collateral flow and celiotomy is mandatory.

Celiac Artery Aneurysms:
Medial degeneration is the most common etiology of celiac artery aneurysms. This is particularly true in those cases associated with anatomic anomalies such as a common celiomesenteric trunk. On occasion, aneurismal dilation occurs distal to compression by the median arcuate ligament, although the incidence of rupture in these cases is unknown. Atherosclerosis is also associated with celiac aneurysms. Ruptured celiac artery aneurysms are usually treated by ligation, which is generally well tolerated.

In saccular or very focal aneurysms, aneurysmectomy and arterial reconstruction may be considered. In the patient with pre-existing liver disease or evidence of portal hypertension, reconstruction is indicated to maximally preserve hepatic nutrient flow. When necessary, arterial continuity may be established using either an aorto-celiac bypass, originating from the supraceliac aorta or, less commonly, with an interposition graft. In some cases, the aneurysm may be confined to a portion arterial wall; aneurysmorhaphy may be accomplished with excision of that portion of aneurismal wall provided the remaining wall is healthy. Exposure and control of the celiac artery is best obtained through a transabdominal incision and medial visceral rotation, allowing for visualization and subsequent division of the crura and median arcuate ligament. Alternatively, a direct approach through the lesser sac may be used.

**Gastric, Gastroepiploic, Gastroduodenal, Pancreatic and Pancreatoco Duodenal Aneurysms:**

Gastric and gastroepiploic aneurysms represent 4% of splanchnic aneurysms, the majority of which are solitary and involve the gastric artery. The etiology is undefined but likely results from either medial degeneration an associated inflammatory process. These aneurysms have a very high incidence of rupture, either into the peritoneum or the gastrointestinal tract and 70% present with gastrointestinal bleeding. These aneurysms are best treated by ligation, including resection of involved organs as necessary. The excellent collateral supply of the stomach and the urgent nature of the operation make reconstruction inadvisable.

Aneurysms of the gastroduodenal, pancreatic and pancreaticoduodenal arteries are usually associated with either acute or chronic pancreatitis. Occasionally these aneurysms are seen after liver transplantation or pancreaticoduodenectomy, particularly when complicated by postoperative pancreatic fistula. Most are symptomatic; rupture and gastrointestinal hemorrhage are common occurrences. Because of their association with pancreatic inflammation, gastroduodenal and pancreaticoduodenal aneurysms are best managed with transcatheter embolization and obliteration, especially in the setting of active hemorrhage.

**Aneurysms Of Mesenteric Branches, Aneurysms Of The Inferior Mesenteric Artery:**

Jejunal, ileal and colic branch aneurysms are usually small and often solitary. These aneurysms are often identified during angiography to investigate gastrointestinal bleeding or on CT scans for evaluation of abdominal pain. Presence of multiple mesenteric aneurysms suggests a systemic pathology such as polyarteritis nodosa, septic emboli from bacterial endocarditis or a connective tissue disorder. Rupture is most commonly seen in
aneurysms involving colonic branches. Rupture most often occurs into the mesentery, although free intraperitoneal rupture can occur. Management is operative ligation, with resection of involved bowel as necessary. Transcatheter embolization has a very limited role, since laparotomy is required in any case to assess intestinal viability.

Aneurysms of the inferior mesenteric artery are exceedingly rare and little is known about their etiology or natural history. These aneurysms can usually be managed by ligation, with revascularization using autogenous vein if collateral circulation is inadequate.

**COMPLICATIONS AFTER Ruptured ABDOMINAL Aneurysm**

Local and systemic complications are frequent after rupture of an abdominal aortic or visceral aneurysm. A high index of suspicion, prompt recognition, with early treatment of complications is mandatory for survival. Mortality rates range from 10-60% for ruptured visceral artery aneurysm and 40-75% ruptured aortoiliac aneurysms. Postoperative bleeding may occur as the result of ongoing coagulopathy (“medical bleeding”) or from a technical defect (“surgical bleeding”). Correction of hypothermia and coagulopathy (using blood component therapy) should be prompt and abdominal re-exploration if bleeding continues is mandatory. In the face of extensive blood loss and resuscitation, abdominal compartment syndrome may occur and should be promptly recognized. Abdominal compartment syndrome results in increased peak airway pressures, progressive hypoxemia, renal dysfunction and visceral ischemia from direct compression of mesenteric and hepatic capillary flow and venous compression, reduced cardiac output, and increased intracranial pressure.\(^{xxxviii}\) The diagnosis is suspected on clinical grounds and confirmed by bladder manometry. Bladder pressures that exceed 20 mm Hg should be treated with decompressive celiotomy. (REF). Once the edema has resolved (usually within 7 days) the abdomen is closed, either primarily or with mesh.

Residual visceral ischemia may occur after resection of aortic or visceral aneurysms. Patients who have persistent fever, leukocytosis or ileus after surgery should be evaluated for residual visceral ischemia, pancreatitis or intraabdominal abscess. This is particularly true when resection of abdominal organs has been performed. Colon ischemia occurs in up to 30% of patients after ruptured AAA repair, with an associated mortality of more than 50%.\(^{xxxix}\) It occurs unpredictably, when antegrade flow in the internal iliac arteries and IMA is preserved, and can present with a range of signs and symptoms. Diarrhea, which may or may not be bloody, that occurs within 24 hours of AAA resection should raise suspicion of colonic ischemia; flexible sigmoidoscopy should be promptly performed in questionable cases. If the diagnosis of colonic ischemia is confirmed, differentiation between transmural and mucosal ischemia may be difficult, and the decision between nonoperative treatment (with broad spectrum antibiotics, fluids, and bowel rest and repeat colonoscopy) or celiotomy and resection should be based on the patients clinical course. In questionable cases, it is better to err on the side of operative intervention and colon resection.
Rupture of the aorta or a major visceral vessel often results in shock and multi-system organ failure. Cardiac (myocardial infarction, heart failure, arrhythmias) and respiratory (respiratory failure, adult respiratory distress syndrome) problems predominate. Renal dysfunction occurs in about one-third of patients undergoing ruptured AAA repair; the need for dialysis portends a poor prognosis, with mortality rates of greater than 75%. Gastrointestinal and infectious complications may also occur, usually in the later stages of protracted convalescence. Finally, the culmination of these manifests as multisystem organ failure, which is the most common cause of death beyond 48 hours in patients with ruptured AAA.

Limb ischemia may be seen in patients after resection of ruptured AAA and is caused by distal embolization of aortic debris. If femoral or popliteal pulses are absent at the conclusion of surgery, prompt vascular exploration, usually by a groin incision is indicated. In most cases the offending thrombus can be removed with an embolectomy catheter. If femoral and popliteal pulses are present, but pedal Doppler signals are diminished or absent, more distal embolization has occurred. This sometimes manifests as “blue toes”, and may be associated with microembolization of atherosclerotic debris to the buttocks, spinal cord and sometimes abdominal and pelvic viscera. Treatment of this condition is generally supportive, since retrieval of microemboli is not feasible. Outcome depends on the severity and location of embolization and attendant ischemia and may range from full recovery to amputation and death.
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