Factors Influencing the Long-term Results of Abdominal Aortic Aneurysm Repair

G. Geroulakos¹,², J.S.P. Lumley¹ and J.G. Wright²

¹Professorial Surgical Unit, St Bartholomew's Hospital, London, U.K. and
²Division of Vascular Surgery, Ohio State University Hospital, Columbus, OH, U.S.A.

REVIEW ARTICLE

The incidence of late graft complications such as para-anastomotic aneurysms, aortoenteric fistulas and graft infections following abdominal aortic aneurysm (AAA) repair is a major determinant of its overall benefit, yet most published reports of AAA repair have concentrated almost exclusively on the early postoperative mortality and morbidity. Accurate knowledge regarding the incidence of late complications is essential to making any decision regarding the operative vs non-operative management of AAAs. A similar analysis must be applied to endovascular repair of AAAs before this technique is accepted as an alternative method of treating AAAs. In this article we review the current knowledge and understanding on the late results following aortic aneurysm repair.

Introduction

Although the true benefit of abdominal aortic aneurysm (AAA) surgery depends almost entirely on its impact on the patient’s long-term survival, most reports on this subject have focused almost exclusively on the early postoperative mortality and morbidity. Accurate knowledge of late survival probabilities and complication rates is important for several reasons. It is essential to present patients with accurate predictions for long-term event-free survival in order to provide the patient with the opportunity to make a truly informed decision regarding the operative or non-operative management of his AAA. Knowledge of long-term, event-free survival probabilities is becoming even more important as some clinicians urge ultrasound screening programs for high-risk populations. These programs are likely to find significant numbers of older patients with small aneurysms less than 5 cm in transverse diameter.¹⁻³ Ongoing clinical trials in the United States and Europe are now investigating the short-term and long-term efficacy of surgical resection of smaller aneurysms.⁴

Furthermore, freedom from recurrent aneurysm formation and complications, such as aortoenteric fistulas and graft infections, should be considered as a major index of the durability and long-term success for the operation. Rutherford⁵ has suggested that para-anastomotic aneurysms, aneurysms in the graft itself and aneurysms in the immediate adjacent segments should all count as treatment failures, and we would agree with this perspective.

Taking all this into account, it seems to us that any decision regarding the operative vs non-operative management of AAAs will depend primarily on an accurate assessment and knowledge of the following six factors:

1. The natural history of untreated AAA.⁶
2. The expected late survival of the patient following successful AAA repair.
3. The expected incidence of late recurrence of clinically significant aneurysms.
4. The expected incidence of late graft infections.
5. The expected incidence of late graft-enteric fistula.
6. The influence of risk factors such as age or the presence of chronic disease on the previous five factors.⁷⁻⁹

Finally, if endovascular stents ever become a standard and accepted alternate method of treating...
AAAs, a similar analysis must be applied to the long-term results of endovascular repair of AAAs, to decide which patients will be the best candidates for this treatment. The aim of this article is to review the literature on the long-term results of aortic aneurysm repair.

**Late Complications**

**Para-anastomotic aneurysms**

The development of para-anastomotic aneurysms is a major complication of abdominal aneurysm repair: its incidence has not decreased despite refinement of surgical techniques, graft and suture material during the last two decades.

In a consecutive series of 2126 aortic anastomoses Szilagyi et al. reported an incidence of para-anastomotic aneurysms of 0.2% (i.e. 4 cases). In the same study, in 1605 iliac anastomosis the incidence of iliac para-anastomotic aneurysms was 1.2%. This increased to 3% for femoral pseudoaneurysms in a total number of 4303 femoral anastomoses. However, this was a retrospective study and the identification of pseudoaneurysms was based mainly on the results of an annual clinical examination. Many of the intra-abdominal pseudoaneurysms, particularly in obese patients, may have been missed and therefore the reported incidence may have been an underestimate of the true prevalence. In a recent study, where annual ultrasound was used to monitor 111 patients with aortic grafts, 11 patients (10%) were found to have intra-abdominal para-anastomotic aneurysms, ranging in overall size from 4.1 to 6.2 cm. In this report para-anastomotic aneurysms were classified as either pseudoaneurysms, which were presumed suture line disruptions (n = 7) and appeared as discrete bulging of the lumen, or as true aneurysms of the adjoining artery (n = 4). It is interesting that true aneurysms were found exclusively in patients operated for aortoiliac aneurysm disease. By contrast pseudoaneurysms were found on both groups. Life-table analysis showed an increased predisposition for the development of intra-abdominal pseudoaneurysms with the passage of time. At 15 years the incidence was 20%. This high reported incidence of para-anastomotic aneurysms is a legitimate matter of concern to us regarding the long-term performance of endovascular AAA repair.

The aetiology of para-anastomotic aneurysms is multifactorial and finding a single cause at operation may not be possible. The break of a suture played an important role in the pathogenesis of this lesion in early reports where silk was used for the anastomosis. The type of prosthetic material can be a factor in the pathogenesis of false aneurysms as a result of fraying, compliance mismatch or dilatation. Degeneration of the artery wall at the level of the anastomosis has also been implicated.

Hypertension is the only clinical marker for which there is general agreement that it is associated with an increased incidence of para-anastomotic aneurysms. Good control of hypertension should be an important objective during the follow-up of patients who undergo aortic aneurysm repair.

As a result of their rarity, there are no studies on the natural history of para-anastomotic aneurysms. However, it is known that they do enlarge and rupture. Treiman et al. reported a series of 18 patients with non-infected para-anastomotic aneurysms of the abdominal aorta and iliac arteries. The operative mortality was 8% (1/12) for patients with intact aneurysms, whereas it was 67% (4/6) for patients who presented with a ruptured aneurysm. Similarly, in the series of Szilagyi et al., all three patients who presented with a ruptured para-anastomotic aneurysm of the aorta or iliac arteries died.

Elective operative repair should be carried out on all large and symptomatic para-anastomotic aneurysms. We favour the transperitoneal approach with medial visceral rotation. In this exposure the left kidney remains in its position and dissection is carried out behind the left colon, spleen, pancreas and stomach to allow displacement of these structures toward the midline. The entire intra-abdominal aorta can be exposed in this manner. The aneurysm is resected and replaced with an in situ graft.

**Aortoenteric fistulas**

Aortoenteric fistula is a rare and probably the most troublesome complication in aortic reconstructive surgery. Champion et al. has reported an incidence of 1.6% in a series of 1376 aortic reconstructions. This is comparable to an incidence of 0.6% reported by Elliot et al. group.

The pathogenesis of aortoenteric fistulas is unclear since both mechanical factors and infection are implicated. It has been suggested that a loop of the intestine adheres to the aortic prosthesis, most commonly to the proximal anastomosis, and dense adhesions are formed. Gradually the pulsation of the non-compliant aortic graft mechanically erodes into the bowel and results in contamination of the graft by the intestinal contents. If the proximal anastomosis is involved, a fistulous communication between the aortic graft and the bowel is formed, otherwise the communication is called graft-enteric erosion. According to an alter-
native mechanism which has been proposed, a low-grade infection in the anastomosis may result either in the formation of an abscess or in the development of a pseudoaneurysm, which subsequently erodes through the bowel wall.  

The majority of aortoenteric fistula are aortoduodenal. In a study of 256 cases cited in the literature, 221 (90%) were aortoduodenal, 19 were aortojejunal, 11 were aortoileal, and five involved other viscera.  

The time interval between aortic reconstructive surgery and the presentation of an aortoenteric fistula may range from a few days to 27 years, with a mean of 14.8–51 months. In practice this means that patients are at life-long risk.

Gastrointestinal bleeding is the most common manifestation of aortoenteric fistula. Haematemeses or melaena may occur. Acute, severe, life-threatening bleeding is relatively uncommon. The classical presentation is with an acute haemorrhage, often accompanied by hypotension that stops spontaneously, the so called ‘herald bleed’. This is followed by recurrent bleeding after a period of hours or at most days. This pattern is seen in 37–80% of cases. Chronic intermittent bleeding leading to anaemia occurs in 18–25%. About half of the patients have some abdominal and or back pain that may occur for several weeks prior to admission. Aortoenteric fistula must be considered in any patient who presents with gastrointestinal bleeding after abdominal aortic aneurysm repair. Endoscopy is often felt to be the first step in diagnosis in patients who are haemodynamically stable and are not having exsanguinating haemorrhage, as it is the most likely to show the diagnostic features of the fistula.

A portion of bile stained graft eroding in the bowel lumen, or suture material seen protruding into the duodenal lumen, are pathognomonic signs. However, normal findings on endoscopy do not exclude aorto-duodenal fistula and a sensitivity of approximately 50% has been reported. Computed tomography (CT) has assumed a central role in the diagnosis of aortoenteric fistula. Although it cannot reliably distinguish this condition from perigraft infection it can help detect either complication with a high degree of sensitivity (94%) and specificity (85%). In a series of 23 patients the most common CT findings were perigraft soft tissue present in 20 patients 87%, focal bowel wall thickening in 11 cases (48%) and ectopic gas in 11 cases (48%). Vertebral osteomyelitis was identified in one case. Ultrasound can show retroperitoneal fluid collections and false aneurysms but not ectopic gas and the area of interest is often obscured by overlying bowel gas. For these reasons, ultrasound is only recommended if CT is not available. Magnetic resonance imaging is as accurate as CT in the demonstration of fluid or soft tissue but is unable to detect ectopic gas. Aortography will only demonstrate a fistula if active bleeding occurs at the time of the examination. Its value mainly is in planning surgical treatment, by demonstrating the distal vasculature and the proximity of the renal arteries to the top anastomosis.

Survival depends on the degree of haemorrhage and sepsis, and the severity and the length of time of lower limb ischaemia. Resuscitation, control of haemorrhage and the eradication of infection are general principles in the management of these patients. An antibiotic regime for at least 4 weeks is required for every patient. Diametrically opposing options in the surgical literature make it difficult to lay down strict guidelines on the surgical management of this complication. However we and others treat most patients with total graft excision, closure or segmental excision of the defect in the gut, debridement of grossly infected retroperitoneal tissues, closure of the aortic stump and buttressing with omentum, and extra-anatomic bypass. It is desirable to fashion the extra-anatomic bypass as a staged procedure a few days prior to the laparotomy. A reduced incidence of amputation and graft reinfection has been reported for the staged procedures and a reduced mortality rate. This principle may not be feasible in the presence of severe gastrointestinal haemorrhage through the fistula. Bacteraemic seeding of the axillo-femoral graft placed prior to graft excision has been a theoretic concern but has not been reported.

**Primary abdominal aortic graft infection**

Primary graft infection is a most dreaded complication because of the high mortality and amputation rate. In early reports, the mortality rate was in excess of 40%. Only recently some investigators in specialised centres have reported improved perioperative mortalities of 9.5–17%. Even though it would appear that the management of this complication is improving, questions remain on the durability of the surgical techniques and the quality of life of the survivors. A perioperative amputation rate of approximately 20% has been reported. Since graft infections may become apparent many years after aortic reconstruction the true incidence...
may be an underestimate of the prevalence reported in various series. A survey of recent literature comprising more than 4000 patients indicates that approximately 0.3% of all aortic procedures are performed for aortic graft infection. Factors which have been reported to increase the incidence of graft infection include surgical revision, emergency aortic aneurysm repair (as opposed to elective) and the use of the femoral artery for the distal anastomosis. Jamieson reported a graft infection rate of 3.2% for operations where the distal anastomosis was in the femoral artery, whereas only 0.9% for those with no inguinal incision.

In 1967 Fry et al. concluded that infection beginning in the groin seemed able to ascend a thrombosed but not a patent aortobifemoral graft. This was subsequently proved in 1976 when Weber demonstrated that percutaneous distal infection of a patent aortofemoral graft in the dog could ascend to the proximal anastomosis if the graft was not well incorporated into the surrounding tissues.

Graft infection may present as a sinus tract, local purulence, local haemorrhage, pseudoaneurysm, graft thrombosis, ill-defined abdominal pain, septic embolisation usually manifested in the lower limbs as clusters of petechia, fever of unknown cause, aortenteric fistula and as systemic sepsis.

Staphylococcal organisms (both S. aureus and S. epidermidis) and pseudomonas account for a large number of isolated micro-organisms in several series. It should be noted however that cultures of perigraft fluid frequently show no growth and a Gram-stain may show only leukocytes.

CT is the investigation of choice for aortoiliac graft infection. Endoscopy should be reserved for the evaluation of cases of gastrointestinal bleeding; it should also be employed even if the gastrointestinal bleeding is manifest solely by guaiac positive stools. Arteriography may show the presence of pseudoaneurysms but cannot differentiate between sterile and infected pseudoaneurysms. If a draining sinus is present, a sinogram may help in the assessment of the extent of the infection. Some preliminary evidence suggests that the use of technetium 99m-hexametazine white blood cell scanning may help in the detection of low-grade graft infection.

Once the diagnosis of infection of a straight aortic graft or aortoiliac graft is made, we recommend placement of an axillofemoral graft and excision of the infected aortic graft along the principles outlined in the previous section. A reinfection rate of 20% has been reported for these procedures, associated with a 67% amputation rate.

The role of in situ replacement of aortic prosthetic graft infection is not yet well established. The selection of patients is one of the key questions that must be answered. Theoretical advantages of this technique include improved graft patency, shorter operative time, less surgical stress and reduced perioperative complications. The major problem is recurrent graft infections. Jacobs et al. reported a series of 21 patients with aortic graft infections. In 18 patients in situ reconstruction was performed following total graft excision, debridement and irrigation. Of these, 12 had a low-grade graft infection with negative blood cultures and sterile cultures of the perigraft fluid. In this group all patients were alive at a mean follow-up of 8 years with only one reinfection treated successfully with an axillofemoral bypass. By contrast in six patients with severe graft infection, positive blood cultures, perigraft fluid containing pus and cultures revealing one or more bacteria, the reinfection and death were respectively 100% and 83%. It is unclear in this report whether the perigraft cultures were taken intraoperatively. In this case, the results would be available at least 24 h later and would be of no assistance to the surgeon at the time of the operation.

If the graft infection is localised in the femoral anastomosis of an aortobifemoral graft this limb of the graft can be removed. Through a suprainguinal retroperitoneal approach the proximal graft limb is exposed and divided. The distal segment of the graft is removed through an inguinal incision and the femoral arteriotomy closed. If revascularisation of the extremity is required an axillofemoral or obturator foramen bypass is performed to the distal femoral or popliteal artery. More recently the use of rotational muscle flaps to cover exposed grafts in the groin, promises to improve the clinical outcome of this condition. Turnipseed reported a series of 40 patients with locally infected or exposed bypass grafts in the groin, treated with rotational flaps. Of these only one had graft failure due to persistent infection and there were no deaths. Again selection was an important contributor to the high success rate.39 Grafts compromised by suture line breakdown, long pieces of graft bathed in purulent material, and infected grafts presenting with recurrent episodes of bleeding were excluded.

**Long-term Survival**

The remarkable decrease in the perioperative mortality for AAA repair seen in recent decades has not been associated with a concomitant decline in the long-term mortality rate. Most investigators attribute this to undetected and untreated coronary artery disease. Johnson et al. observed a perioperative mor-
tality rate of 0.8% for those patients without, and 6.2% for those with, coronary artery disease in a series of 666 patients who had elective AAA repair. The same author, in an extended prospective analysis of 680 patients undergoing surgery for non-ruptured AAA repair, reported a 6 year survival rate of 90.2%, significantly less than the 79.2% expected for an age and sex matched normal Canadian population. For patients after AAA repair cardiac complications accounted for 44.4% of the late deaths.

Further information on the influence of coronary artery disease on the long-term survival comes from a recent retrospective study of 327 patients operated on for asymptomatic AAA. Their expected 10 year survival rate was 52% and the observed was only 38%. Thirty-four percent of the patients had known cardiac disease prior to the aneurysm repair and in this group the 10 year survival rate was 24%. In contrast, long-term survival for patients without recognised cardiac disease did not differ from the expected in the normal population.

It has been suggested that the presence of an AAA is a marker of severe atherosclerosis and an indicator of extensive coronary artery disease. Indeed, in a series of 263 consecutive Cleveland Clinic patients with AAA who had preoperative coronary angiogram, severe surgically-correctable coronary artery disease was documented in 44% of patients clinically suspected to have coronary artery disease and in 14% of those without previous indications of cardiac disease. Based on the knowledge that coronary artery bypass grafts prolong life in patients with left main stem stenosis and triple vessel disease, one must ask whether prophylactic coronary artery bypass should be performed prior to AAA repair in selected cases. So far there have been no randomised studies to ascertain the value of prophylactic coronary artery bypass graft for enhancing life expectancy after AAA repair.

Cerebrovascular disease is another emerging factor which is contributing to an increased mortality among survivors after AAA repair. In the Canadian aneurysm study, cerebrovascular causes accounted for 8.3% of late deaths vs 5.8% in the normal population. This is not only explained by a high coexistence of haemodynamically significant carotid disease and AAA as recently reported, but also by an increased prevalence of hypertension among the population of patients with aneurysms. In the Olmsted County population-based AAA study, the incidence of hypertension approached 50%. A 5–6 mm Hg decrease of the mean diastolic pressure by antihypertensive treatment, is associated with a 42% reduction in stroke rate.

In addition, the multicentre VA study recently published by Feinglass concludes that only four discrete risk factors were found to be significantly associated with long-term mortality at $p < 0.05$ in the multivariate regression model. These were greater than 69 years of age, chronic obstructive pulmonary disease, left ventricular hypertrophy, and a history of cerebrovascular disease. Multiple aneurysms in patients with aneurysmal disease of the aorta at the time of diagnosis of an AAA, or the development of additional true aneurysms during the follow-up, represents a significant contributor to the late morbidity and mortality. In a series of 1510 patients treated for aortic aneurysms, Crawford and Cohen found that 138 (10.9%) had multiple aneurysms occurring simultaneously. They suggested that aortic aneurysmal disease is multifocal and total screening for diagnosis and complete replacement of all disease would give the best results. At the Mayo Clinic in a series of 1087 patients who underwent AAA repair, Plate et al. found new arterial aneurysms in 59 patients at a median interval of 5.2 years after the initial operation. Forty-nine of these were true aneurysms. Twenty-four of these aneurysms were in the thoracic aorta and caused the death in 20 patients, five thoracoabdominal, seven aortic, six iliac, 10 femoral, four popliteal and one renal were also detected. The risk of recurrent aneurysms was significantly higher in patients with hypertension.

Conclusions

Most late deaths following AAA repair are the result of systemic complications of atherosclerosis. The diagnosis of an AAA is an early warning sign that underlying coronary artery disease may be a greater threat. Patients should be screened at presentation for accompanying ischaemic heart disease and this should be treated on its merits. Monitoring of the blood pressure and control of hypertension should be an important objective of the follow-up of all patients undergoing AAA repair. This may reduce the incidence of stroke and the development of new aneurysms. Follow-up examination must include careful palpation of the popliteal fossa, groin and abdomen to identify the presence of new aneurysms. Since it is difficult to detect recurrent aneurysms in the abdomen and the almost impossible in the thorax, a chest X-ray and an abdominal ultrasound should be considered for screening hypertensive patients who have undergone AAA repair.

References


