Part X

Factors influencing results

Late Complications in Aortoiliofemoral Surgery for Arterial Occlusive Disease

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Actually, it is possible to foretell excellent early and late results of direct aortoiliofemoral reconstructions for occlusive disease.
Perioperative mortality rates are under 3% in many centres and patency rates are close to 85% at 5 years and 75% at 10 years.
Nevertheless, other than graft failure several late complications of aortoiliofemoral surgery have been described, as summarized in Table I.

TABLE I

Surgical late complications of aortofemoral reconstructions
Late graft limb thrombosis
Graft infection
Anastomotic false aneurysm (pseudoaneurysm)
Aortoenteric fistula
Alterated sexual function
Chronic renal failure
Ureteral complications

Aorto-femoral graft limb occlusion

In some large series, the cumulative patency rates of aortofemoral bypass range from 83% to 92% at five years and 71% to 83% to ten years and 61% to 74% to fifteen years (1,2).

Late aortofemoral bypass occlusion, however, develops in 6% to 13% of patients after primary abdominal aortic reconstruction (1,3). Approximately 45% of patients with aortoiliac occlusive disease have associated distal occlusive disease at the femoral, popliteal or tibial vessels.

Progressive atherosclerotic involvement of the outflow and/or inflow tract, therefore, is the most common cause of late graft occlusion (4).
Graft infection is also a cause of late graft failure in a minor number of aortic reconstructions (Table II).

Graft surveillance is extremely important to detect a "failing graft" at the preocclusive stage.

Several document infact have underlined the improved results when intervention is performed at the time when the graft is still patent.

TABLE II

Causes of aortofemoral graft limb occlusion
Kinking of the graft through its retroperitoneal course
Stenosis at the level of the distal anastomosis
Pseudoaneurysm formation at the site of the distal anastomosis
Poor run-off
Graft infection

Open surgical procedures have been the traditional approach of bypass graft occlusion, directing procedures as thrombectomy and revision or replacement of the previously bypass graft (5,6).

Thrombolysis has been advocated as a less invasive, alternative treatment of restoring graft that also provides the detect stenotic lesions responsible for the obstruction and to reveal the run-off vessels (7).

The lesion is then treated with endovascular or operative procedure after successful thrombolysis.

The choice between endovascular or surgical treatment is based on the characteristics of the lesion.

The most appropriate treatment for a valve cusp stenosis is operative patching or resection and reconstruction.

The results of surgery are believed to be superior to dilatation and yield excellent secondary patency rates (7).
When the distal anastomosis is involved or there is evidence of the progression of disease, may be necessary to extend the bypass graft. Surgical reconstruction is indicated in delayed occlusion for more than 14 days' duration. Thrombolysis holds the advantages in term of mortality and amputation for less than 14 days occlusion (8,9).

If a bypass is occluded less than six months, the graft replacement is indicated (10) (Table III).

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TABLE III

Recommendation (95): Treatment of chronic critical leg ischemia due to bypass graft occlusion (11)
In patient with chronic critical leg ischemia , surgical revision or graft replacement is the preferred treatment for bypass graft occlusion. Thrombolysis may be considered as a treatment option in patients who present early after their bypass graft occlusion where the limb is not immediately threatened.

Aortofemoral graft infection

The management of an infected aortic prosthesis remains one of the major complications in vascular surgery. The traditional management of aortic graft infection requires infected graft removal and extraanatomic bypass grafting (12,13,14).

Coagulase-negative staphylococci are a significant cause of graft infections, because of their unique ability to adhere to an grow on implanted artificial surface and to secrete a biofilm or slime, which is a galactose-rich polysaccharide that provides protection from the immunity defence (15,16).

Staphylococcus epidermidis is the most frequent infecting organism (17).

Usually, vascular prosthesis infected with coagulase-negative staphylococci are associated with a less virulent clinical course compared with vascular graft infections caused by coagulase-positive staphylococci or gram-negative microorganisms (Table IV).

The high grade infections are usually caractherized by clinical symptoms of the sepsis and absence of tissue incorporation of the graft, with perigraft cavities and perigraft fluid, false aneurysms or graft-tissue sinues (Table V).

TABLE IV

Most common microbiology of graft infections (21)
Gram-positive
Staphylococcus epidermidis (most common)
Staphylococcus aureus
Streptococcus
Gram-negative
Eschierichia coli
Pseudomonas aeruginosa
Klebsiella pneumoniae
Enterococcus
Candida species

The low grade infections are not associated with fever, leukocytosis, or positive blood culture results (Table VI). Graft biofilm infections usually presents as a graft healing complication and absence of tissue incorporation, with or without perigraft cavities and perigraft fluid, false aneurysms, or graft-tissue sinues (Table VII,VIII).

TABLE V

Clinical manifestations in infected prosthetic grafts
Sepsis
Gastrointestinal bleeding (aorto enteric fistula)
Draining groin sinus
Groin abscess
Femoral false aneurysm
Aortic false aneurysm
Septic emboli
Hydronefrosis
Graft thrombosys

TABLE VI

Laboratory diagnosis in prosthetic graft infections
RBC count
WBC count
CRP (C reactive protein)
Blood colture
Fluid colture (groin fistula, intracavitary by needle aspiration)

TABLE VII

Instrumental diagnosis in prosthetic graft infection
Ultrasonography
Upper gastrointestinal endoscopy
CT scan with or without needle aspiration
Arteriography
Scintigraphy with labeled leukocyte

TABLE VIII

Classification of site of infections (21)
Intracavitary (aorto-iliofemoral graft infections)
Extracavitary (infrainguinal, axillofemoral, femorofemoral graft infections)

Because the bacteria are adherent to the prosthesis, they are seldom present in the periprosthetic tissue fluid. Microscopic examination of this perigraft fluid are routinely negative, and for many years investigators believed that grafts were not infected and that the lack of graft incorporation and development of perigraft fluid was a reactive process to the vascular prosthesis (18) .

Despite advances in critical care and surgical technique, the mortality and morbidity rates associated with aortic graft infections have remained high (10% to 25%) with major amputations required in 15% to 20% (17,19,20), particularly when an infected aortobifemoral graft is treated reflecting the magnitude of the procedures necessary to treat this complex problem and the inability to control aortic and retroperitoneal sepsis despite removal of the infected prosthesis (Table IX).

TABLE IX

Types of treatment of graft infection
Percutaneous drainage
Conservative Surgery
Graft removal and extraanatomic bypass grafting (axillofemoral –axillopopliteal)
Simultaneous aortic graft excision and in situ aortic graft replacement (synchronous treatment)

Percutaneous drainage

An appropriate drainage catheter must be chosen according to fluid collection charateristics, location and volume. Particular attention should be paid to side-hole numbers and dimension, because they should allow an effective liquid drainage despite its viscosity.

The catheter could be positioned following either the Seldinger or the Trocar technique. The Seldinger technique takes a little bit longer and is more expensive, due to the need of the use of guidewire, but is more accurate in tip positioning and permits an easy insertion also of wider catheters (up to 12-14 Fr). The Trocar technique is easier, quiker and cheaper but does not permit insertion of catheters exceeding 8 Fr.

The catheter tip should be accurately positioned in the fluid collection in the declive position and used for 3-4 weeks. In a series of 11 patients 22 treated with this procedure the average used time has been 18 days according to other series (23). The preliminary results are promising, although they must be confirmed by long-term outcome and results (24).

Conservative surgery

After a preliminary report in 1990 (25), Morris (26) reported in 1994 the results with this type of conservative surgical treatment of major aortic graft infection in ten cases with a mean follow-up at 61 months.

By an extraperitoneal approach, if feasible, the infected graft is exposed. The aneurismal sac is opened and the pus and nonaviable tissue are removed. Sample of pus and tissue are obtained for microbiological culture and the graft is irrigated with saline solution. Two silicone tube drains, with multiple side-holes, are placed inside the sac along the limbs of the graft. The tube are then brought out via the extraperitoneal plane to the skin. The native aneurismal sac is closed over the graft if possible.

The tubes are irrigated with antibiotic solution. Regular cultures of the drain effluent are made and the antibiotic therapy is changed according to the antibiogram. Irrigation is continued until three consecutive effluent cultures are sterile or as prompted by others events, such isolation of Candida Albicans. Drains are removed sequentially. Most patients manteined an oral antibiotics therapy for several week or months. This treatment isn't standardized.

Graft removal and extra-anatomic bypass grafting

An aortic stump complication after excision of an infected prosthesis continues to be a significant problem (19,27). Various methods have been proposed to deal with the aortic stump such as coverage with omentum, anterior longitudinal ligament, peritoneum, jejunal seromuscolar patch, or fibrous glue reinforcement (28,29).

Furthermore, although the use of externally supported grafts appears to have improved long term patency of extra-anatomic bypass grafts, long-term graft failure still occurs in 25% to 35% of patients after extra-anatomic bypass grafting and infected aortic graft excision. Therefore extra-anatomic bypass graft revision or replacement procedures must be done in a significant number of patients to achieve a good long-term limb salvage (30).

However, this type of treatment remains the standard with which other treatment approaches must be compared (13) (Table X).

TABLE X

Graft removal and extra-anatomic bypass graft timing (14)	
Staged operation:	extra anatomic bypass followed by graft aortic removal
Single "traditional" operation:	graft removal preceding extraanatomic bypass
Single "sequential" operation:	extra anatomic bypass preceding graft removal

In situ replacement

The "in situ replacement" may be performed because is considered that in the "graft biofilm infections" the process is confined to the vascular conduit, therefore graft excision and in situ replacement is reasonable. The low virulence of coagulase-negative staphylococci and their unique characteristcs of colonization via surface biofilm permit treatment of infected grafts by excision of the involved graft segment, debridement of perigraft tissue and in situ replacement of another prosthesis (18) (Table XI). In this type of treatment is essential that the patient has the correct diagnosis.

TABLE XI

Types of aortic graft replacement: " in situ treatment"
Fresh homograft (hystorical - now abandoned)
Refrigerate allograft
Cryopreserved homograft
Prosthetic graft (PTFE - antibiotic-bonded Dacron graft)
Autogenous graft (superficial femoral - popliteal vein)

Infact, this procedure isn't recommended for other types of graft infection.

In situ replacement isn't indicated for gram-negative infection and for coagulase-positive Staphylococcus infections where the patient has systemic effects of infection and bacteria can be saw on gram stains of the perigraft fluid (18). This procedure may be considered in patients with low-grade aortic graft infection and negative blood and perigraft cultures (31) (Table XII).

TABLE XII

Successful criteria for selection of patients for in situ aortic graft replacement (31,32,33)
Sterile blood cultures
Absence of graft incorporation
Perigraft cavity fluid with no bacteria on gram's stain

The use of fresh allograft was abandoned years ago as a result of spontaneous rupture, thrombosis, and late aneurismal formations (34).

Kieffer (35) described in 1993 his experience using refrigerate allograft, from 1988 and 1992, in 43 consecutive patients with infected infrarenal prosthetic graft.

A fragment of the retrieved arterial allograft was routinely cultured for bacteriological control. After being flushed with eparinized saline solution to eliminate any residual intraarterial blood, allografts were stored at 4°C in 500 ml of a preservation medium that contained heparin and antibiotics.

Allograft were implanted after a minimum interval of 48 hours to decrease cellular antigenicity and a maximum interval of 21 days to avoid late degenerative changes (35).

The relatively high incidence of late complications observed with the use of refrigerate allograft, has not been observed with the use of cryopreserved allografts.

The arterial allografts are removed from brain-dead multiorgan donors or non-heart-breaking donors, aged 15 to 45. The ascending aorta, the thoracic arch and the entire descending aorta is removed in a sterile fashion from heart. Only great vessels with warm ischemia time less than 6 hours are removed. The time between the death of the donor and the beginning of the sterilization should be less than 18 hours (36,37).

Bacteriology and virology tests were routinely performed for donors (Table XIII). A fragment of the retrieved arterial allograft was routinely cultured for bacteriological control.

TABLE XIII

Microbiology tests of homografts (36)
Culture media
Aerobic bacteria
Anaerobic bacteria
Fungi
Mycobacterias (toxoplasma gondi, toxoplasma pallidum)
Sierology
HIV-1 and HIV-2 antibodies (Elisa,IMF,WB)
HIV-1-DNA
HTLV-1
HbsAG
Anti-HBc, if positive , anti-HBs
Anti-HCV
Anti HAV
VDRL
Q-fever-antibodies
EBV-Monospot IgG + IgM
CMV IgG + IgM

After harvesting, the great vessels are immediately stored at 4 °C in an ice-cold solution.

Then they are prepared, measured, and examinated by angioscopy for degenerative changes. Before and after decontamination by antibiotic solution, microbiology tests are performed. Finally, after cryopreservation in liquid nitrogen to – 100 °C, allografts are stored in

the vapor phase of liquid nitrogen at – 180 °C, and delivered in a cryogenic dry shipper, which maintained a temperature of – 130 °C. The grafts are washed immediately before the implantation (36,37).

However, resistance of allograft to infection cannot be considered complete, especially when dealing with highly virulent organisms and incompletely debrided infected tissues.

Secondary and late deterioration of allograft is probably partly immunologic in origin (38).

Cryopreserved homograft seems to be clinically resistant to bacterial infections (39,40). The reasons are hypothetical, involving immune cells, passive release of antibacterial substances by the graft, or physical and chemical homograft wall proprieties (41).

The long term fate of cryopreserved arterial homografts should be superior to freshly implanted homografts, as cryopreservation preserves a digestion resistant collagenous network similar to the glutaraldehyde pre-treatment of bioprosthetic hearth valves (42).

Even if the patients with cryopreserved homografts require late homograft-related procedures, the use of these conduits may help to eradicate the infection and may allow subsequent reconstruction with prosthetic graft (Table XIV).

Polytetrafluoroethylene material as replacement graft have demonstrated lesser bacterial adherence of slime-producing bacteria than Dacron grafts (43).

Rifampicin-bonded graft have been shown to be resistant to in vitro infection with Stafilococcus aureus for as long as 3 weeks (44).

TABLE XIV

Technical notes in cryopreserved arterial allografts (37)
√ Proper time of allograft thawing
√ Appropriate length and tension-free anastomosis
√ Proper ligature of allograft side branches by using a through-and-through polypropylene suture holding of the allograft wall
√ Enlargement of the anastomotic heel in allograft-to-prosthetic graft anastomosis after partial allograft replacement
√ Avoidance of any exudates collections around allografts by means of aggressive wound drainage
√ Circumferential anastomotic reinforcement with allograft strips
√ Gentamycin – impregnated fibrin – glue covering all allograft anastomosis
√ Prolongation of antifungal treatment to 3 months postoperatively

Initial attempts at in situ replacement with graft and antibiotic combination were unsuccessfull because of the rapid attenuation of drug concentrations around the replacement graft site (45). Coating the prosthesis with a material collagen or gelatin to provide a bond between the graft and the antibiotic has enabled such grafts to retain antimicrobial activity for prolonged periods.

Rifampicin is a particularly useful antibiotic in this situation, because it has a broad spectrum of activity against gram-negative and gram-positive organisms, especially Staphylococcus aureus (46). It is relatively hydrophobic and therefore does not dissolve into the circulation rapidly.

It is also used relatively infrequently in vascular surgery and is less likely to lead to bacterial resistance.

The use of great saphenous vein has reported short-term success in aortoiliac/femoral reconstruction (47,48,49,50). The failure is the result of focal and generalized hyperplasia and in addition the great saphenous vein in the aortoiliac-femoral position do not fare well because of his small size relative to the vessels that it is replacing (47).

On the contrary, the proximal end of a superficial–popliteal vein is 1.0 to 1.5 cm in diameter and this allows confortable anastomosis to the aorta without kinks, bends and other deformities that plague the great saphenous vein graft in this position. In addition, intimal thickening that may develop at valve sites, anastomoses, and other areas in superficial femoro-popliteal vein grafts, produce little or no hemodynamic disturbance because of the large luminal diameter.

In situ autogenous graft replacement may be most appropriate in younger, healthier patients who have a greater life-expectancy and, thus, a higher risk of long–term graft failure, whereas staged extra-anatomic bypass grafting and graft excision may be better in older, sicker patients in whom long-term graft failure may be less important (Table XV).

It is noteworthy, however, that the mean operative time for these complex aortic procedures was 7.9 hours in the Clagett' s experience and major postoperative morbidity occurred in 49% of the patients, including thrombosis in residual popliteal vein 12%, compartment syndrome in 12%, and limb paralysis in 7.5% (51).

TABLE XV

Indications for autogenous aortoiliofemoral reconstruction (51)
Infection of vascular prosthesis
Controindication to prosthetic reconstruction due to regional infection
Recurrent failure of standard procedures
Young patients with small aortoiliac-femoral vessels and extensive occlusive disease

After the traditional management of aortic graft infection by removal of infected graft and extra anatomic bypass procedure, because of the improved patient survival rates that accompany this treatment, an increased number of patients relys on the continued function of the extra-anatomic bypass graft for leg perfusion.

Extra-anatomic bypass graft are less durable than in situ aortic grafts (55,56).

Therefore, the limbs are at risk because the extra-anatomic bypass graft thrombosis, hemodynamic insufficiency or infection.

In 1983, Fulenwider (57) reported three cases who underwent placement of a retroperitoneal graft 2 years after septic graft removal and no reinfection was described.

Although the timing of the redo procedure may be important, the microbiologic features of the original infection is an important element of valuation. One year is not safe for all types of infections. Caution is necessary when planning a redo procedure in a case of previous aggressive gram-negative infection (58) (Table XVI,XVII,XVIII,XIX).

TABLE XVI
Results in staged extra-anatomic bypass grafting and aortic graft excision

Author	Year	Mortality %	Amputation %	5yr Limb salvage %	5yr Primary patency %	5yr Secondary patency %
Yeager (30)	1999	13	10	82	73 / 93 *	-
Seeger (52)	2000	13	10	82	73	92

TABLE XVII
Results in one stage aortic graft excision and "in situ aortic replacement" with autogenous vein (superficial femoral -popliteal vein)

Author	Year	Mortality %	Amputation %	5yr Limb salvage %	5yr Primary patency %	5yr Secondary patency %
Claged (51)	1997	10	5	86	85	-
Nevelsteen (53)	1995	7	7	-	-	-

TABLE XVIII
Results in one stage aortic graft excision and aortic replacement with prosthetic graft

Author	Year	Mortality %	Amputation %	3yr Limb salvage %	5yr Primary patency %	5yr Secondary patency %
Towne (18) *	1994	0	0	100	100	-
Hayes (54) °	1999	18.2	0	-	-	-

** rifampicin bonded Dacron prosthesis*
° PTFE graft

TABLE XIX
Results in one stage aortic graft excision and aortic replacement with cryopreserved homograft

Author	Year	Mortality %	Amputation %	3yr Limb salvage %	3yr Primary patency %	3yr Secondary patency %
Lèseche (55)	2001	17.8	0	100	81	96
Vogt (37)	2002	6	0	-	-	-

Aortoenteric fistula

Aortoenteric fistula is associated with a high mortality risk. Primary and secondary forms of aortoenteric fistula are recognised as depending on previous aortic surgery at the site of fistula development (59). Secondary aortoenteric fistulas are usually related to a previous prosthetic graft reconstruction, but it may rarely involve a primarily repaired aorta without the use of a vascular prosthesis (60,61,62,63,64). Two types of secondary aortoenteric fistulas have been described, the aortoenteric fistula and aortic paraprosthetic fistula. In the latter condition, a sinus between the bowel lumen and the outer connective tissue capsule of the prosthetic graft develops giving rise to low grade or frank graft infection and sepsis which eventually leads to an aortoenteric fistula by eroding into the aorta (65,66).

The incidence in autopsy series is described from 0.04% to 0.07% (68) and may complicate aortic aneurysms in 0.1% to 0.8% of cases (69).

The occurrence of an aortoenteric fistula after abdominal aortic reconstruction has been reported in up to 4% of patients (70), but, nowadays, with the improvement of operative technique, it usually occurs in less than 1% of cases (71,72,73,74). Primary aortic reconstructions has as a higher risk for the development of secondary aortoenteric fistula (74,75).

The duodenum is the most common site for primary and secondary aortoenteric fistulization (82%), particularly in its third portion (51-55%) (76,77) because of its relative fixed position and intimate relationship with the aorta. The duodenum is also the most common site for development of secondary aortoenteric fistula (60,66,71,77).

Pathogenesis of aortoenteric fistula is still not clear but the development of a communication between the aorta and the bowel is likely due to local factors inducing erosion of both arterial and intestinal wall (59).

The pathogenesis of secondary aortoenteric fistulas has been suggested to be due to impairment of the blood supply of the posterior duodenal wall during the primary aortic procedure which may lead to its perforation (73,78,79). Inflammatory reaction to the prosthetic graft, which acts as a foreign body, has been suggested to cause adhesion between the duodenum or other intestinal segments to the anastomotic site or the midportion of the graft (74,80). Failure to cover the graft with retroperitoneal tissues, peritoneum or an omental flap during the original operation, in order to prevent direct contact between the prosthesis and the bowel, has been suggested as a major technical reason for the development of aortic- and graft-enteric adherence, and erosion leading to secondary aortoenteric fistula (73,75,81,82).

Mechanical injury of the posterior duodenal wall due to the pulsatile graft motion may lead to graft infection from the duodenal contents and disruption of the anastomotic suture line (66,83,84). However, an erosive and perforative event has been suggested to occur in absence of any associated infectious process (83).

The creation of a false aneurysm at the anastomosis site on the redundancy of the prosthetic graft, complicated operations, emergency aortic aneurysm repair, multiple operative procedures, graft infection and wound complications have been advocated as other factors potentially responsible for the occurrence of such a serious complication (66,73,74,79).

Upper gastrointestinal haemorrhage may occur as a chronic blood loss which is well tolerated by the patient or as an acute massive bleeding which may end up in fatal exanguination.

Hypotension after the initial bleeding episode, and clot formation are possible reasons for latent period (85). According to Dossa et al. (77), herald bleeding and haematemesis are the main clinical manifestations of aortoenteric fistulas 81% and 65%, respectively, as well as they are in secondary aortoenteric fistula.

Signs of infection are the second most common manifestation of primary, secondary aortoenteric and paraprosthetic fistulas, and each of them in a patient previously undergone vascular surgery should highly rise the suspicion of such a catastrophic condition. Therefore, sepsis calls on for an immediate, throughout clinical evaluation.

In the haemodynamically stable patient, several diagnostic tools are nowadays available for preoperative diagnosis of aortoenteric fistula, but none of them has a high sensitivity and specificity (86).

Esophagogastroduodenoscopy is the preferred initial diagnostic method, either to evaluate more common causes of bleeding or to diagnose an aortic fistula (65,74). The endoscopic diagnosis of lesions other than aortenteric fistula, does not exclude the presence of the latter. A definitive, preoperative diagnosis was obtained by esophagogastroduodenoscopy and 10% of cases of primary aortoenteric fistulas, and in 25-33% of cases of secondary aortoenteric fistula (71,75).

Although angiography is not considered useful in evaluating a patient with an aortoenteric fistula, it is of value in planning arterial reconstruction (73). It can reveal bulging dilatation at anastomotic site or leakage of contrast agent at emergency aortography or during routine angiography (86). It detected an aortic fistula in 17% of primary aortoenteric fistula, but only rarely was diagnostic of primar and secondary aortenteric fistula (71,77,86).

Contrast-enhanced computed tomography may show periaortic and perigraft tissue changes related to an aortic fistula. Collection of fluid or gas surrounding the graft strongly suggests the presence of an aortoenteric erosion or fistula (59). It was considered to be dia-

gnostic of primary aortoenteric fistula in 22% of cases and of secondary aortoenteric fistula in 20% of cases (77,78,86).

However, in a large number of patients, no diagnostic procedures can be performed because of the life-threatening gastrointestinal bleeding or preoperative diagnostic methods are negative, thus diagnosis is made during emergency surgery or at autopsy (87,88).

With regard to the surgical treatment, a median laparotomy or a left retroperitoneal incision is done, and the aorta and the bowel involved by the aortoenteric fistula are exposed. In the case of bleeding from inaccessible locations, an intra-arterial balloon control method can be successfully used to control the bleeding and offer a clear field for adequate exposure of the involved structures (89). After control of bleeding is achieved, a temporary or definitive repair of the bowel defect is carried out in order to avoid contamination of the operative field by intestinal contents. Primary intestinal closure is usually feasible. A duodenal defect may be closed by a transverse two-layer suture or, eventually, when nonviable bowel is present, resection of the third and fourth portions of the duodenum with end-to-end or end-to-side anastomosis is required.

Transverse suture is usually indicated also when other intestinal segments are involved (90), but a segmental resection followed by end-to-end anastomosis should be carried out in the case of extensive intestinal bowel lesions.

Because of the rarity of primary AEFs, no treatment method for vascular repair has been yet standardised. However, clinical data showed that primary AEF may differ from the secondary one in permitting, in almost all cases, a definitive repair by an in situ standard aortic reconstruction (79,69). In fact, it seems that bacterial infection associated with primary AEF is confined to tissues surrounding the fistula or may even be absent (73) rendering aortic aneurysmectomy, duodenorrhaphy and standard aortic reconstruction a feasible, safe procedure with operative mortality rate of less than 30% and long-term survival of about 50% (67,77).

Occasionally, a successful local repair have been performed for secondary aortenteric fistula not associated with signs of local sepsis (73,66). Two of the three patients treated by Higgins et al. (66) with a direct repair of the suture line for aortenteric fistula survived at 4-month and 2-year follow-up. Although it is not clearly described, Bergqvist et al. (60) reported three of five patients (60%) who survived after closure of the fistula. Other 4 patients underwent closure of the fistula and axillobifemoral grafting, and three of them survived (75%).

Patients with an end prosthesis to side proximal aortic anastomosis, thrombosed graft, or those in whom aortoiliac occlusive disease with good collateral vessels was the indication for the original aortic reconstruction, may be treated by excision of the graft and aortorrhaphy alone since pre-existing collateral vessels may provide adequate blood flow for leg salvage (65,71). Doppler ultrasonography may be useful to identify those patients who may tolerate aortic graft removal without lower limb revascularisation.

However, poor results have been achieved by this method with mortality rates approaching 80% (71).

If a preoperative diagnosis of aortoenteric fistula is made and the patient is not bleeding seriously, an extra-anatomic bypass can be performed before aortic graft removal, in order to provide lower limb viability.

A significant higher mortality rate has been reported in patients who underwent excision of the graft followed by revascularisation as compared to mortality rate observed among patients treated by lower limb revascularization before graft removal (91). This is likely due to the unstable haemodynamic conditions of patients belonging to the former group who required an emergent operation for control of haemorrhage (92).

Since aortic stump dehiscence may heavily contribute to postoperative deaths after aortenteric fistula repair (82,87), a careful closure of the aortic stump with two rows of nonabsorbable synthetic suture on viable aortic tissue is done. Coverage of the stump with pedicled omental grafts, muscle flaps, anterior spinal ligament, serosal patches or fibrin glue has been suggested (65,78,84). In a review of 93 cases treated before 1982, the mortality rates after graft removal and extra-anatomic bypass was 36% (81). A more recent review of series reporting on graft removal and extra-anatomic bypass surgery for aortoenteric fistula identified an average perioperative mortality of 33%, while a stump disruption occurred in 17% of these cases (85). Recently, perioperative mortality rates lower than 20% have been reported (71).

Since an aortic stump blow-out may occur in a large number of patients resulting in high mortality rates, a recurrent fistula may occur between the aortic stump and the bowel and extra-anatomic bypasses may subsequently become infected or have a low patency rate (74), several authors have supported an in situ bypass grafting after excision of the original graft and extensive debridement of all infected tissue (36,66,93,94). Despite they achieved a 56% survival rate after in situ graft replacement, Peck (74) outlined the technical problems of sewing a new prosthetic graft to an old proximal aortic stump, and the occurrence of sepsis and recurrent fistulas during the postoperative period.

Fiorani et al. (94) reported excellent results in 8 patients with secondary aortoenteric fistula treated by in situ PTFE graft replacement. Only one patient (13%)

TABLE XX
Endovascular repair of aortoenteric fistula

Author	Year	Patients (N°)	Type of fistula	Follow-up	Redo
Deshpande (95)	1999	1	Aorto-enteric	-	-
Chuter (96)	2000	1	Aorto-duodenal	8 months	Staged operation
Curti (97)	2000	1	Aorto-ilioenteric	-	-
Grabs (98)	2000	1	Aorto-enteric	18 months	-
Kinney (99)	2000	1	Aorto-enteric	-	-
Burks (100)	2001	7	Aorto-enteric	27 months*	-

** average follow-up (11-66 months)*

died postoperatively of myocardial infarction, while the others had both survival and leg salvage at 34-month follow-up. Other series have reported mortality rates ranging from 30% to 83% (77,86). A large review of reported cases of in situ graft replacement for AEF identified an average perioperative mortality rate of 30%, a reinfection rate of 6%, while the average rate of stump disruption was 25% (87).

Although endovascular management of aortoenteric fistulas is a new technique, it is very interesting because it may provide rapid control of bleeding by a minimally invasive technique in patients which are elderly and physiologically compromised, often with multiple comorbidity conditions and a limited life expectancy.

The review of the literature shows that the endovascular aortic-stent graft placement seems to provide an effective method of rapidly controlling an acutely haemorrhaging aortoenteric fistula (95-100) (Table XX).

However, it is clear that without debridement of contaminated prosthetic and retroperitoneal tissue, it can not be considered as definitive therapy. The new placement endograft is contaminated by gut bacterial flora. Complete eradication of infection by this procedure seems probably impossible (100).

Adjuntive treatments such as percutaneous drainage and proximal bowel diversion may be necessary to contain sepsis in the setting of gross aortic graft infection. Life – long antibiotic treatment long term suppression may be feasible.

In patients in whom these procedures fail must be converted to surgical repair on elective basis and in optimised clinical state.

False aneurysms

Anastomotic femoral false aneurysm remains a complication of vascular prosthesis reconstruction, occurring with an incidence of 2% to 5% (101,102).

The most common surgical tecnique is aneurysmectomy with interposition prosthetic graft replacement.

Because of the propensity of femoral anastomotic aneurysm to produce serious life and limb threatening complications early, elective operative correction has become the mainstay of management.

Technical error in the construction of the anastomosis is a likely cause of false aneurysm occurring in the early postoperative period. Failure to incorporate all layers of the artery wall or include an adequate amount of prosthetic tissue in the anastomosis will results in anastomotic dehiscence similar to a traumatic false aneurysm.

Structural defects in the native artery is a possible etiology for false aneurysm formation. False aneurysm occur at arterial anastomoses of patients having procedures for aneurismal or occlusive disesase, in the same way.

Some study has demonstrated that graft material from an anastomotic femoral false aneurysm is frequently colonized with bacteria in the absence of clinical signs of graft infections (103).

Prosthetic colonization by non virulent micro-organisms at the time of implantation may result in a infectious process that does not manifest until months or years after the first procedure (Table XXI).

However, excision of false aneurysm and in situ replacement is a safe vascular procedure in this complication.

Alterated sexual functions

The incidence of erectile impotence after aortic reconstruction may approach 25% (11).

The impotence implies inadequate preservation of the hypogastric artery and pelvic circulation.

TABLE XXI

Advocated causes of false anastomotic aneurysm degeneration (102-108)
Structural defects in the native artery
Hypertension
Mechanical stress resulting in tension on the anastomosis
Forces resulting from motion of the adjacent join
Excessive physical activity of the patient
Defects in the structure of the graft material
Healing complications after surgery (seroma, hematoma, wound infection, skin edge necrosis)
Previous infectious process
Technical error in the construction of the anastomosis

Retrograde ejaculation is also a frequent occurrence and is attributable to disturbance of autonomic nerve fibers that course along the left wall of the aorta and cross the common iliac arteries.

The study of pelvic circulation is performed by penile brachial index (PBI), Penile Dynamic Colour Doppler Ultrasonography and arteriography.

Nevelsteen (109) in 1990 reported his study about the influence of aorto-femoral reconstruction on sexual function and pelvic circulation in 62 male patients with aortoiliac occlusive disease.

Erectile function remained unchanged in 77% of the cases.

Improvement or deterioration was seen in 11.3% and 20.5% of the patients respectively.

Impotence occurred in 31% of patients that presented preoperatively impaired erection but only in one patient with normal preoperatively function.

Pelvic circulation, as reflected by the penile brachial index was not affected in 74%, increased in 11% and decreased in 15% of the procedures.

Pelvic circulation was preserved in 90% after end-to-side proximal anastomosis and in 82% after end-to-end aanastomosis.

Since fluctuations in sexual function could be related to changes in penile brachial index in only 36% of the cases, it is concluded that alterated sexual functions after aortofemoral reconstruction are due to a combination of pelvic circulation and interruption of autonomic plexus (109).

On the other hand, reduce penile arterial inflow and carvenovenous leakage are equally important in the pathophysiology of erectile disfunction in patients with artoiliac occlusive disease, suggesting that atherosclerosis may also compromise the penile veno-occlusive mechanisms (110).

A nerve sparing approach to the infrarenal aorta is helpful, and preservation of the hypogastric artery flow is also essential (11).

Ureteral late complications

Ureteral complications occur infrequently during management of aortoiliac vascular reconstructions. In addition the incidence of hydronephrosis caused by ureteral obstruction after aortoiliac reconstruction is also low, ranging from 2% to 14% in several prospective studies (111,112).

Postoperative ureteral obstruction, first described by Jacobson in 1962 (130), has usually documented by case reports or limited series. Since Shaw in 1963 (114).

First suggests a relationship between graft and ureteral complications, Schubert (115) have analysed a group of 19 patients with postoperative hydronephrosis and the implications of this complication. He reported an 89% incidence of graft complication associated with hydronephrosis.

Initial reports of postoperative ureteral obstuction implicated placement of the graft anterior to the ureter producing ureteral entrapment between the graft and host iliac vessels (116,117).

Subsequent reports have noted entrapment in less than half of the cases of postoperative ureteral obstruction (118).

These findings suggest that other mechanisms in addition to entrapment are responsible for ureteral obstruction. Prosthetic graft complications may also determine ureteral complications, which in turn may signal an underlying graft complication (119). Anastomotic aneurysms may obstruct or rupture into the ureter (116,119,120).

The inflammatory process accompanying graft infection may obstruct the ureter, which in turn may be a marker for such graft complications.

Aside from the obvious causes of the ureteral obstruction as a result of graft complications, another cause is the retroperitoneal reaction that appears to be stimulated by implanted graft (118).

This fibrotic process has been found to be either localized to the site of graft-ureteral contact or as generalized retroperitoneal process. However, it is most likely a combination of retroperitoneal dissection, hematoma resolution and host response to the prosthesis that produces a fibrousus reaction.

If ureteral involvement is documented, precautions should be taken to avoid ureteral injury during graft revision such as insertion of stents to aid in ureteral identification.

Isolated ureteral operations after aortic reconstruction are rarely required. Hydronephrosis after aortic reconstruction will often spontaneously resolve. With progression operative correction should be considered. Operative intervention is mandatory when obstruction compromises the renal fuction.

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