Allograft replacement for infrarenal aortic graft infection: Early and late results in 179 patients

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Objectives: **We evaluated early and late results of allograft replacement to treat infrarenal aortic graft infection in a large number of patients and compared the results in patients who received fresh allografts versus patients who received cryopreserved allografts.**

Methods: **From 1988 to 2002 we operated on 179 consecutive patients (mean age, 64.6 ± 9.0 years; 88.8% men). One hundred twenty-five patients (69.8%) had primary graft infections, and 54 patients (30.2%) had secondary aortoenteric fistulas (AEFs). Fresh allografts were used in 111 patients (62.0%) until 1996, and cryopreserved allografts were used in 68 patients (38.0%) thereafter.**

Results: **Early postoperative mortality was 20.1% (36 patients), including four (2.2%) allograft-related deaths from rupture of the allograft (recurrent AEF, n = 3), all in patients with fresh allografts. Thirty-two deaths were not allograft related. Significant risk factors for early mortality were septic shock ($P < .001$), presence of AEF ($P = .04$), emergency operation ($P = .003$), emergency allograft replacement ($P = .0075$), surgical complication ($P = .003$) or medical complication ($P < .0001$), and need for repeat operation ($P = .04$). There were five (2.8%) nonlethal allograft complications (rupture, n = 2; thromboses, which were successfully treated at repeat operation, n = 2; and amputation, n = 1), all in patients with fresh allografts. Four patients (2.2%) were lost to follow-up. Mean follow-up was 46.0 ± 42.1 months (range, 1-148 months). Late mortality was 25.9% (37 patients). There were three (2.1%) allograft-related late deaths from rupture of the allograft, at 9, 10, and 27 months, respectively, all in patients with fresh allografts. Actuarial survival was 73.2% ± 6.8% at 1 year, 55.0% ± 8.8% at 5 years, and 49.4% ± 9.6% at 7 years. Late nonlethal aortic events occurred in 10 patients (7.2%; occlusion, n = 4; dilatation < 4 cm, n = 5; aneurysm, n = 1), at a mean of 28.3 ± 28.2 months, all but two in patients with fresh allografts. The only significant risk factor for late aortic events was use of an allograft obtained from the descending thoracic aorta ($P = .03$). Actuarial freedom from late aortic events was 96.6% ± 3.4% at 1 year, 89.3% ± 6.6% at 3 years, and 89.3% ± 6.6% at 5 years. There were 63 late, mostly occlusive, iliofemoral events, which occurred at a mean of 34.9 ± 33.7 months in 38 patients (26.6%), 28 of whom (73.7%) had received fresh allografts. The only significant risk factor for late iliofemoral events was use of fresh allografts versus cryopreserved allografts ($P = .03$). Actuarial freedom from late iliofemoral events was 84.6% ± 7.0% at 1 year, 72.5% ± 9.0% at 3 years, and 66.4% ± 10.2% at 5 years.**

Conclusions: **Early and long-term results of allograft replacement are at least similar to those of other methods to manage infrarenal aortic graft infections. Rare specific complications include early or late allograft rupture and late aortic dilatation. The more frequent late iliofemoral complications may be easily managed through the groin. These complications are significantly reduced by using cryopreserved allografts rather than fresh allografts and by not using allografts obtained from the descending thoracic aorta. (J Vasc Surg 2004;39:1009-17.)**

Although arterial allografts were used extensively for infrarenal aortic replacement during the first decade of reconstructive arterial surgery,[1-4] they were abandoned in the early 1960s because of difficulties with procurement and preservation, late deterioration, and availability of suitable arterial prosthetic grafts. In 1991 we reintroduced the use of arterial allografts for surgical management of infrarenal aortic prosthetic graft infection.[5] Since then, all patients seen at the Vascular Surgery Department, Pitié-Salpêtrière University Hospital in Paris, France, at first received fresh allografts, and since August 1996 cryopreserved allografts. The purpose of this study was to report our present experience in 179 patients and to compare the results obtained with fresh allografts versus cryopreserved allografts.

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PATIENTS AND METHODS

Population. From October 8, 1988, to November 30, 2002, all patients with infected infrarenal aortic prosthetic grafts seen at the Department of Vascular Surgery, Pitié-Salpêtrière University Hospital in Paris, France, underwent complete resection of the prosthesis, followed by in situ allograft replacement.

There were 179 consecutive patients, 159 men (88.8%) and 20 women (11.2%), with a mean age of 64.6 ± 9.0 years (range, 29-82 years). Fifty-four patients (30.2%) had

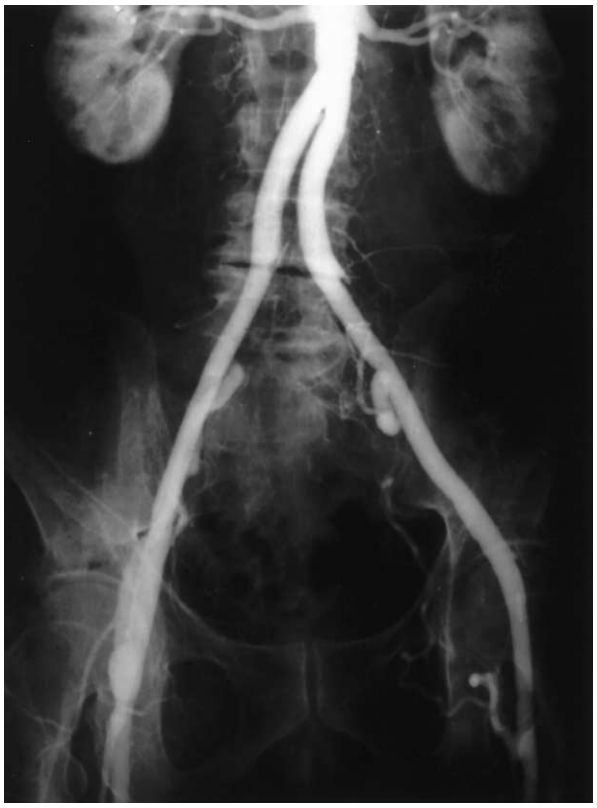

Fig 1. Postoperative aortogram shows bilateral hypogastric revascularization in a patient who underwent allograft replacement to treat infrarenal aortic graft infection.

secondary aortoenteric fistulas, and 125 (69.8%) had primary graft infections. Twenty-three patients (12.8%) had undergone initial operation in our center, and 156 patients (87.2%) were referred to us after an operation performed elsewhere. The initial operation was performed to treat atherosclerotic aortoiliac occlusive disease in 124 patients (69.3%), degenerative aortic aneurysm disease in 53 patients (29.6%), blunt aortic trauma in 1 patient (1.1%), and Takayasu arteritis in 1 patient (1.1%).

Mean interval between the initial operation and allograft replacement was 6.1 ± 5.4 years (1 month–24 years). Sixty-eight patients (38.0%) underwent no repeat operation between the initial operation and allograft replacement, whereas 111 patients (62.0%) underwent a mean of 2.8 ± 2.1 repeat operations (range, 1-16).

Clinical presentation. Sepsis, including fever and leukocytosis, was present in 125 patients (69.8%), an inguinal or retroperitoneal abcess in 67 patients (37.4%), draining inguinal sinus in 53 patients (29.6%), graft thrombosis in 44 patients (24.6%), femoral false aneurysm in 32 patients (17.9%), gastrointestinal bleeding in 24 patients (13.4%), aortic false aneurysm in 21 patients (11.7%), and septic emboli in 6 patients (3.4%). There were 58 secondary aortoenteric fistulas in 54 patients. The duodenum was involved in 40 patients, the small intestine in 12 patients, and the colon in 6 patients.

Procurement and preservation of allografts. Allografts were harvested from brain-dead donors as part of a program to retrieve multiorgan transplant tissue. Bacteriology and virology tests were routinely performed in donors. Matching of blood and tissue compatibility between recipient and donor was not attempted. The entire length of the descending thoracic aorta, aortic bifurcation, and iliac and femoral arteries was obtained. Small collateral branches were transected a few millimeters distal to their origin, to facilitate later ligation or suture. Hypogastric and deep femoral arteries were transected 2 to 3 cm distal to their origins, to enable revascularization of the corresponding arteries in the recipient.

Until August 1996, fresh allografts, stored from 48 hours to 37 days (mean, 13.0 ± 8.6 days) at 4°C in 500 mL of a preservation medium containing heparin and antibiotic agents,[6] were used in 111 patients (62.0%). After August 1996, because of changes in French health regulations, 68 patients (38.0%) received cryopreserved allografts prepared by the Tissue Bank of Paris Hospitals.

A segment of descending thoracic aorta was used in 10 patients (5.6%), a segment of infrarenal aorta and various lengths of the iliac and femoral arteries in 140 patients (78.2%), and both in 29 patients (16.2%).

Allograft implantation. A median laparotomy was routinely used. Infrarenal aortic clamping was feasible in 111 patients (62.0%), whereas 68 patients (38.0%) had interrenal (n = 11), suprarenal (n = 25), supraceliac (n = 29), or thoracic (n = 3) aortic clamping. Complete removal of all prosthetic material, whether or not grossly infected, was routine. The aortic stump and periprosthetic tissues were aggressively debrided to obtain macroscopically normal tissues.

The allograft was implanted in situ with polypropylene running sutures for proximal and distal anastomoses. It was tunneled to the distal anastomotic site by way of either the previous pathway or a new pathway in close proximity to the previous pathway. There were 30 entirely intra-abdominal allograft replacements (16.8%), whereas 149 (83.2%) included at least one femoral anastomosis. The proximal allograft anastomosis was end-to-end in 151 patients (84.4%). Retroperitoneal and inguinal drainage was routine. The skin incisions were loosely approximated to avert superficial abscesses. In case of major fibrosis or infection of the inguinal region, the skin was left open, and the graft was covered with a myoplasty using the sartorius muscle.

Associated procedures. Ten patients (5.6 %) underwent associated reconstruction of the renal arteries (unilateral in 8 patients, bilateral in 2 patients). Seventeen patients (9.5%) underwent associated reconstruction of visceral arteries (inferior mesenteric artery in 10 patients, superior mesenteric artery in 6 patients, celiac and superior mesenteric arteries in 1 patient). Forty-four patients (24.6%) underwent associated reconstruction of hypogastric arteries (unilateral in 32 patients, bilateral in 12 patients; Fig 1). Twenty-six patients (20.1 %) underwent associated reconstruction of lower limb arteries, including femoropopliteal bypass in 23 patients (bilateral in 3 patients) and femoro-

distal bypass in 3 patients, with allograft or autogenous vein material. Three patients (1.7 %) with tight stenosis of an internal carotid artery underwent associated carotid endarterectomy.

The 40 duodenal fistulas were treated with suturing in 36 patients and resection-anastomosis in 4 patients. A feeding jejunostomy was performed in 23 patients (57.5%). The 12 intestinal fistulas were treated with suturing in 7 patients and resection-anastomosis in 5 patients. The six colonic fistulas were treated with colectomy in 5 patients (with proximal colostomy in 3 patients) and exteriorization in 1 patient.

Omentoplasty was performed in 104 patients (58.1 %) whenever technically feasible. Cholecystectomy was performed in 18 patients, ureteral repair or lysis in 6 patients, appendectomy in 5 patients, colectomy to treat colonic ischemia in 4 patients, nephrectomy in 3 patients, and splenectomy in 2 patients. Six patients underwent concomitant major amputation because of septic arthritis or established gangrene.

Bacteriology. Identification of the responsible organism or organisms was obtained either preoperatively from blood cultures, draining sinuses, or direct or computed tomography (CT)–guided needle aspiration or intraoperatively by culturing all available material, including periprosthetic fluid or tissue and part of the infected prosthetic graft. Identified organisms in 149 patients (83.2%) are shown in Table I (online only). One organism was identified in 68 patients (45.6%), two organisms in 40 patients (26.9%), three organisms in 31 patients (20.8%), and more than three organisms in 10 patients (6.7%). A mean of 1.92 $\pm$ 1.06 organisms was identified in the overall series. The most prevalent organisms were *Staphylococcus, Enterococcus, Escherichia coli, Pseudomonas aeruginosa,* and fungi. No organisms were identified in 30 patients (16.8%). This, together with the high incidence of fungal infection, probably attests to the long-standing preoperative antibiotic therapy in a significant number of patients.

All patients received broad-spectrum antibiotic agents before and during the operation, and then selective antibiotic agents after the responsible organisms were identified. Administration of antibiotic agents was continued for at least 6 weeks. No patients received long-term or indefinite antibiotic therapy.

Follow-up. All surviving patients underwent digital substraction aortography before or soon after discharge. Routine late follow-up included clinical examination and duplex scanning at 3-month intervals during the first year, and yearly thereafter. Late CT or aortography was performed depending on the results of duplex scanning.

RESULTS

Early results

Mortality. Thirty-six patients (20.1%) died during the early postoperative period or during hospitalization (Table II, online only). There were four allograft-related deaths (2.2%), from allograft rupture, including three recurrent aortoenteric fistulas. All of these patients had been operated on to treat aortoenteric fistulas and had received fresh allografts. Thirty-two deaths were not allograft related, but were due to septic shock in 12 patients, myocardial infarction in 5 patients, respiratory complications in 4 patients, recurrent duodenal fistula (without aortoenteric fistula) in 2 patients; coagulopathy, multiorgan failure, and intestinal infarction in 2 patients each; and adrenal insufficiency and acute pancreatitis in one patient each.

Univariate analysis identified seven risk factors for early mortality: presence of aortoenteric fistula ($P = .04$), occurrence of intraoperative or postoperative septic shock ($P < .0001$), emergency operation ($P = .003$), emergency allograft replacement ($P = .007$), surgical complication ($P = .004$) or medical complication ($P < .0001$), and need for a repeat operation ($P = .04$). The difference between fresh allografts and cryopreserved allografts approached statistical significance ($P = .07$).

Surgical complications. Forty-two surgical complications, either lethal or nonlethal, occurred in 36 patients (20.1%; Table III, online only). There were eight allograft-related early surgical complications, including three recurrent aortoenteric fistulas (two repeat operations, three deaths), three allograft ruptures without aortoenteric fistula (three repeat operations, one death), and two allograft thromboses (two repeat operations, no deaths). All of these patients had received fresh allografts. The most significant non-allograft-related surgical complications were ischemic colitis in six patients (six repeat operations, three deaths) and recurrent duodenal fistula without aortoenteric fistula in two patients (two repeat operations, two deaths). One patient (0.5%) underwent a major amputation after thrombosis of an associated femoropopliteal bypass and failure of thrombectomy.

Medical complications. Medical complications, either lethal or nonlethal, occurred in 81 patients (45.3%). The most significant medical complications were respiratory complications in 49 patients (four deaths), septic shock in 44 patients (12 deaths), acute renal failure (creatinine concentration >200 μmol/L) in 34 patients (no deaths), and myocardial infarction in 12 patients (five deaths).

Late results

Follow-up. Among the 143 survivors to the postoperative period, 4 patients (2.8%) were lost to follow-up at 6, 20, 24, and 46 months, respectively. One hundred thirty-nine patients (97.2%) were followed up until their death or the last 3 months of 2002. Mean follow-up was 46.0 $\pm$ 42.1 months for fresh allografts and 1 to 148 months (median, 34 months) for cryopreserved allografts.

Mortality. There were 37 late deaths (25.9%). Three deaths (2.1%) were allograft related, from rupture of the allograft at 9, 10, and 27 months, respectively. One of these ruptures was caused by persistent or recurrent infection; the other two were apparently mechanical in origin. Two late ruptures occurred in patients who received fresh allografts, and one occurred in a patient who received a cryopreserved allograft. There were 34 non-allograft-related late deaths.

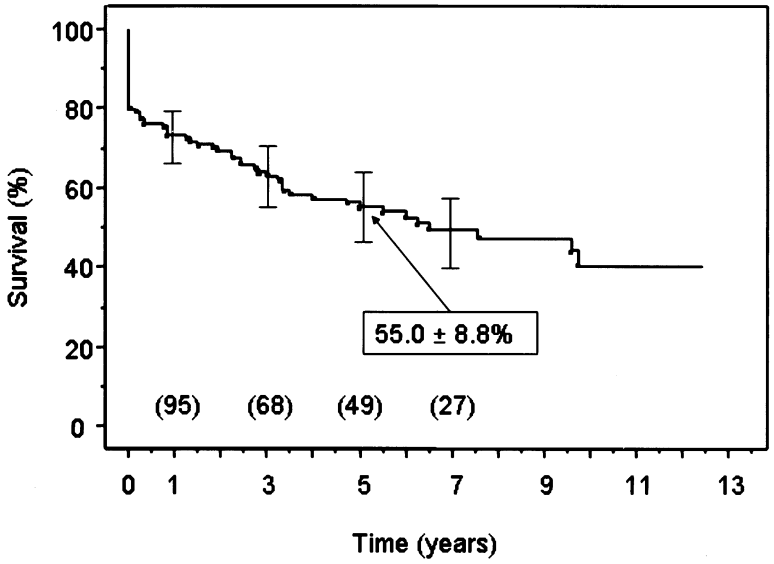

Fig 2. Cumulative Kaplan-Meier curve shows early and late survival in 179 patients after allograft replacement to treat infrarenal aortic graft infection.

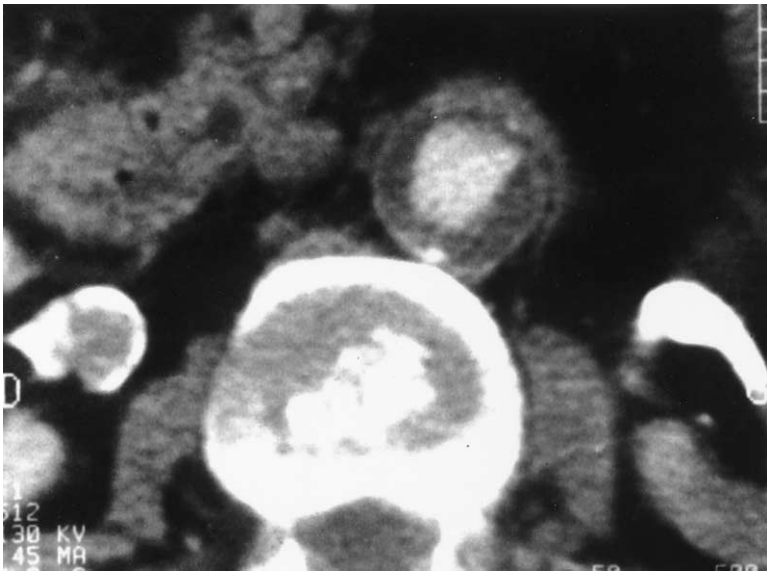

Fig 3. Computed tomography scan demonstrates mild dilatation of the aortic allograft, with mural thrombus, 35 months postoperatively.

The most significant causes of non-allograft-related death were cancer (11 patients) and myocardial infarction (8 patients). With the Kaplan-Meier method, and including early deaths, the probability for late survival after allograft replacement to treat infrarenal aortic prosthetic infection was 73.2% ± 6.8% at 1 year, 63.0% ± 8.0% at 3 years, 55.0% ± 8.8% at 5 years, and 49.4% ± 9.6% at 7 years (Fig 2).

Aortic events. There were 12 late aortic events in 12 patients (8.4%; Table IV, online only). Four thromboses occurred at 2, 4, 5, and 61 months, respectively. These were in two patients with fresh allografts and two patients with cryopreserved allografts. The four patients underwent successful repeat operation (repeat allografting, n = 2; thrombectomy, n = 1; axillobifemoral bypass, n = 1). Five dilatations involving the aortic portion of the allograft, defined as aortic diameter less than 40 mm (Fig 3), were diagnosed at 14, 14, 28, 30, and 35 months, respectively. Three patients died of unrelated causes without repeat

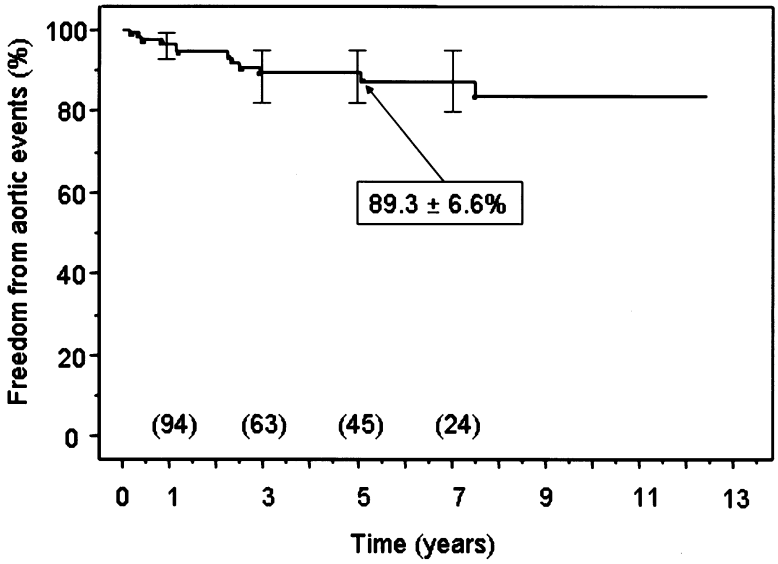

Fig 4. Cumulative Kaplan-Meier curve shows freedom from aortic events after allograft replacement to treat infrarenal aortic graft infection.

operation, and the other two patients are being followed up with periodic CT. All dilatations occurred in patients who received fresh allografts, including four allografts obtained from the descending thoracic aorta. In one patient (0.7% of the entire series) an aneurysm developed, defined as aortic diameter greater than 40 mm, and was diagnosed at 90 months. The patient underwent repeat operation with an endovascular prosthetic graft, and then a surgical conversion to treat type I endoleak. He had received a fresh allograft obtained from the descending thoracic aorta. Finally, there were two late lethal aortic ruptures, at 10 and 27 months, respectively. Both patients had received fresh allografts, including one that was obtained from the descending thoracic aorta.

Univariate analysis showed that the use of an allograft obtained from the descending thoracic aorta is a significant risk factor for late aortic events ($P = .03$). The use of fresh allografts versus cryopreserved allografts almost reached significance ($P = .07$). With the Kaplan-Meier method, and including early aortic events, the probability of freedom from aortic events was 96.6% ± 3.4% at 1 year, 89.3% ± 6.6% at 3 years, 89.3% ± 6.6% at 5 years, and 87.3% ± 7.4% at 7 years (Fig 4).

Iliofemoral events. There were 63 late iliofemoral events in 38 patients (26.6%; Table IV, online only). These included 42 occlusive lesions, diagnosed at 2 to 89 months (mean, 25.8 months; Fig 5),; 20 dilatations or aneurysms, diagnosed at 2 to 113 months (mean, 52.7 months); and one recurrent infection, diagnosed at 62 months. Twenty-eight patients had received fresh allografts, and 10 had received cryopreserved allografts. Among these 38 patients, 30 (78.9%) have undergone repeat operation at least once. A total of 59 repeat interventions were performed, including 26 prosthetic (polytetrafluoroethylene) graft replacements, 14 repeat allograft replacements, 8 autogenous vein replacements, 6 endovascular procedures, and 5 thrombectomies.

Univariate analysis showed that the use of fresh allografts versus cryopreserved allografts was a significant factor for late iliofemoral events ($P = .03$) (Table V, online only,). With the Kaplan-Meier method, the probability of freedom from iliofemoral events was 84.6% ± 7.0% at 1 year, 72.5% ± 9.0% at 3 years, and 66.4% ± 10.2% at 5 years (Fig 6).

Patency and freedom from dilatation. Relevant data are provided in Table VI (online only).

DISCUSSION

Our reasons for embarking on allograft replacement to treat infrarenal aortic graft infection were as follows. First was dissatisfaction with conventional treatment, that is, graft removal and axillofemoral bypass grafting, in terms of both mortality and anatomic durability. Although contemporary series report significant improvement in mortality,[7,8] axillofemoral bypass grafting to treat infectious lesions is still laden with significant rates of reinfection and occlusion.[9] The second reason included low applicability of in situ repair with autogenous arterial material,[10] duration of operation for harvesting autogenous femoral veins,[11] and presumed high risk for reinfection after in situ replacement with prosthetic grafts, despite occasional reports of satisfactory results.[12,13] Third was the experience of cardiac surgeons with allograft replacement of aortic root in patients with infective endocarditis.[14,15] Fourth, there is experimental evidence of resistance to infection of fresh vascular allografts.[16,17] Fifth was availability of arterial allografts harvested from brain-dead donors as part of an active local multiorgan transplant retrieval program.

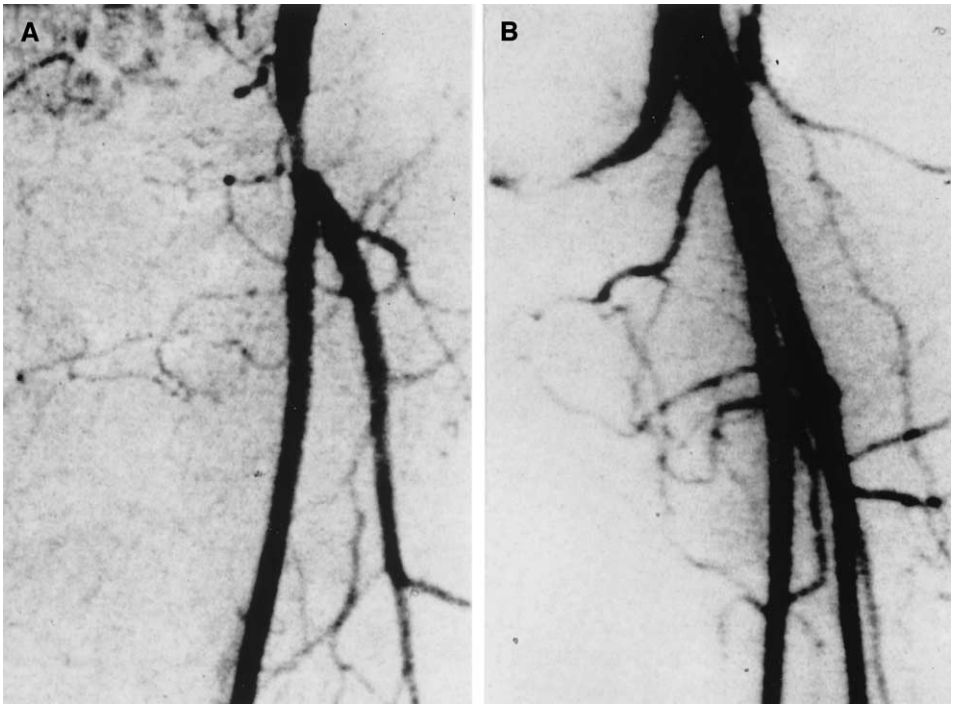

Fig 5. Preoperative **(A)** and postoperative **(B)** arteriograms show late femoral stenosis complicating an aortofemoral allograft treated with autologous vein replacement.

Being aware of the potential for late deterioration of aortic allografts, our initial intention was to use allografting as a bridge to late repeat prosthetic grafting[18] after the infection had subsided. Close follow-up of our patients has shown that allografts fared much better than we expected, with few indications for secondary prosthetic aortic grafting despite a significant number of late iliofemoral events.[6,19-21]

Allograft replacement, compared with conventional treatment with graft removal and axillofemoral bypass grafting,[7,8] has the distinct advantages of in situ reconstruction. It averts complications due to aortic stump disease, and occlusion or infection of prosthetic extra-anatomic bypass grafts.[9] It enables surgical reconstruction of hypogastric and deep femoral arteries as indicated, reducing the risk for postoperative ischemic colitis and lower limb ischemia. In our entire series there was only one major amputation after thrombosis of an associated femoropopliteal bypass. Autologous femoral vein reconstruction also has the advantage of in situ reconstruction.[11] However, although early and late results are good, vein harvesting significantly increases the duration of operation, and venous sequelae are not uncommon.

Early rupture of aortic allografts is a rare, though devastating, specific complication of allografting, and the cause may be either mechanical or infectious.[6,19,22-28] Although "cracking" from cryopreservation and thawing has been reported both clinically[22,25] and experimentally,[29] the incidence has been drastically reduced as a result of extensive worldwide experimental work on cryopreservation techniques.[30-33] Most early ruptures seem to be caused by infection, which raises the question of allograft infectability. Several mechanisms may account for the resistance of arterial allografts to infection, including the physicochemical properties of the cellular components of allograft, responses of the host immune system, and, above all, high antibiotic loading of the allograft.[34-36] These mechanisms are sufficient in most cases to prevent infectious necrosis of the allograft. However, continuous or recurrent exposure to an untreated infectious focus or infection with highly virulent organisms is a logical risk factor for allograft infection and rupture. This probably accounts, at least in part, for the significantly higher mortality after allograft replacement to treat secondary aortoenteric fistulas.[26-28,37]

This study further explores the early and late results of allografting to treat prosthetic aortic graft infection. Although this is not a randomized study, and follow-up was longer in patients who received fresh allografts, allograft-related complications were significantly less frequent in patients who received cryopreserved allografts than in those who received fresh allografts. There were no early late allograft ruptures and no early occlusion in patients with cryopreserved allografts. This in contrast to the series by Noel et al,[27] who reported five (9%) early cryopreserved allograft ruptures and five (9%) early graft limb occlusions. The difference may be explained by differences in cryopreservation techniques. Ten of 12 patients (83.3%) with late aortic events and 28 of 38 patients (73.7%) with late

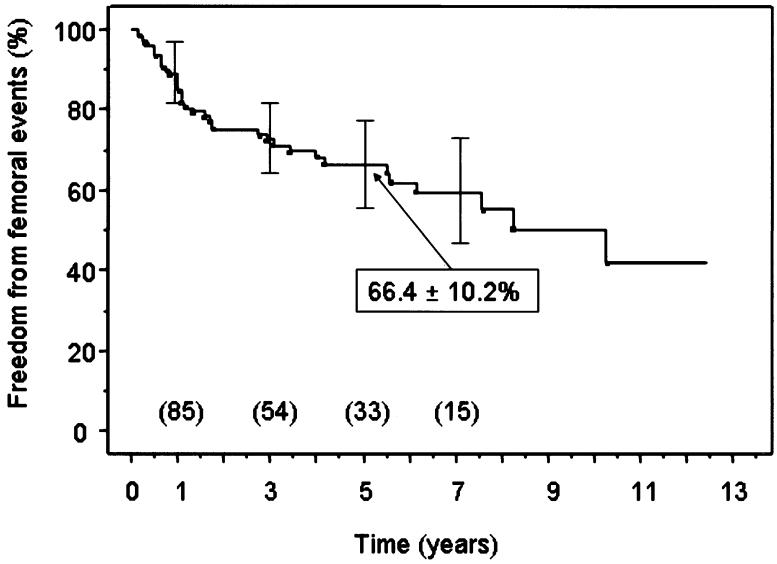

Fig 6. Cumulative Kaplan-Meier curve shows freedom from iliofemoral events after allograft replacement to treat infrarenal aortic graft infection.

iliofemoral events had received fresh allografts. Furthermore, we found that the use of an allograft obtained from the descending thoracic aorta is a significant risk factor for late aortic events. This is probably due to the lower number of elastic fibers in the descending thoracic aorta compared with the abdominal aorta, leading to late mechanical deterioration.

Late deterioration may be immune-mediated[32,33,38-40] or mechanical. Aneurysm dilatation, mural thrombosis (with or without dilatation), calcification, and secondary atherosclerotic disease have been reported in intermediate and long-term follow-up of many patients after early clinical experience with aortic allografts.[41-46] Our clinical experience, with a maximum follow-up of 12 years, confirms the observations of Szilagyi et al.[46] Deterioration of arterial allografts is much more likely to occur in the iliofemoral portion compared with the aortic portion of the allograft. This probably reflects major differences in the composition of the medial layer of the arterial wall. The media of iliac and femoral arteries is made predominantly of highly antigenic smooth muscle cells, accounting for frequent, early degeneration, probably due to chronic rejection. Conversely, the aortic media is made predominantly of elastic fibers, with few smooth muscle cells. This less potentially antigenic configuration probably accounts for late mechanical aortic deterioration.

Blood and tissue compatibility,[22,31] low-dose immunosuppression therapy,[31,33,39] and cell-free allografts[47] have been studied experimentally, with little clinical application thus far, and no demonstrable clinical benefit. Much to our surprise, late aortic deterioration has been rather uncommon and relatively stable, as opposed to the findings of Szilagyi et al.[46] Only one of our patients needed secondary aortic replacement, because of aneurysm dilatation, whereas in the series by Szilagyi et al, 9 of 14 patients with aortic dilatation required repeat operation, and 3 died of aortic complications. In the recent experience of aortic allografting, Desgranges et al[23] and Julia et al[48] reported the only two other secondary aortic replacements necessary because of aneurysm dilatation of the allograft. Different procurement and preservation techniques, influencing both the mechanical properties and the antigenicity of allografts, are responsible for such an important difference. It has been shown experimentally that cryopreservation protects against both early[49] and late[31] immune-mediated arterial allograft dilatation.

However, although our clinical results with cryopreserved allografts have been encouraging, compared with those obtained with fresh allografts, our cryopreservation techniques are probably far from optimal and have not completely eliminated late deterioration of allografts, especially in the iliofemoral segment. The use of glutaraldelyde-treated allografts to avert structural destruction has been suggested experimentally,[50] but has caused massive calcification and, to our knowledge, has had no clinical application thus far. Although in our experience late iliofemoral deterioration, including occlusion and aneurysm, has been much more common than aortic deterioration, it could usually be treated successfully with local procedures through the groin, without need for a repeat abdominal procedure. We hypothesize that most early iliofemoral deteriorations were due to chronic rejection, whereas late deteriorations were usually due to structural degradation of the medial layer.

CONCLUSION

Early and long-term results of allograft replacement are at least similar to those of other methods of management of

infrarenal aortic graft infections. Rare complications include early or late allograft rupture and late aortic dilatation. The more frequent late iliofemoral complications may be easily managed through the groin. These complications are significantly reduced by using cryopreserved allografts rather than fresh allografts, and not using allografts obtained from the descending thoracic aorta.

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