THE CAUSES OF FAILURES IN RECONSTRUCTIONS FOR OCCLUSIVE ARTERIAL DISEASE

C. J. MIENY, F.C.S. (S.A.), F.R.C.S. (ENG. AND EDIN.), *Department of Surgery, University of the Witwatersrand and Johannesburg Hospital*

For many years atherosclerosis obliterans, because of its generalized nature, was not regarded as suitable for direct arterial surgery.

In 1947 Dos Santos[5] described his operation of thromboendarterectomy for obstructive segments of atherosclerotic arteries. This was soon followed by Kunlin[16] who used venous autografts to bypass occluded arterial segments.

It was then recognized that although atherosclerosis is a generalized disease, it may manifest itself in localized segmental occlusions, suitable for reconstructive surgery.

Many reports have appeared in the literature in the last 15 years, describing the results of arterial reconstructions using various methods.

Although sporadic reports of the causes of failure in small numbers of cases appeared from time to time, a full analysis of large numbers of patients was mainly presented by three groups of workers.

Szilagyi *et al.*[25] studied the reasons for failures of 120 reconstructions in 80 patients. These cases in the large majority of instances were treated by arterial homografts.

Crawford *et al.*[4] analysed 1,225 cases consisting of both aorto-iliac and femoro-popliteal disease. All the forms of reconstructions were utilized in this very large group of patients.

Warren *et al.*[29] studied 202 arterial reconstructions. Methods of reconstruction included insertion of arterial homografts, venous autografts, thromboendarterectomy and plastic prosthesis.

The present report deals with the causes of failure of reconstructions in 43 patients with both aorto-iliac and femoro-popliteal atherosclerosis.

MATERIALS AND METHODS

The results of 102 reconstructions in 75 patients seen during a 5-year period have been reported by Gaylis *et al.*[9]

In 43 patients in whom the reconstructions have failed during this period, a study was made of the possible factors responsible for the failure. There were 39 male and 4 female patients. The average age was 50 years, with a range of 29 - 65 years.

Good clinical records were available in all the patients. This included pre-operative clinical descriptions of the extent of the disease, operation notes and follow-up in all the patients, until failure of the graft is reported. Failure was assessed either by clinical disappearance of pulses which were present previously, angiographically, or by findings at re-operation.

Pre-operative angiograms or reports were available in 37 patients. Follow-up angiograms or reports were available in 7 patients. Operative findings at amputation and relevant histology were studied in 19 patients. Valuable information was gleaned from the descriptions of operative findings in re-operations.

Occasionally, a conclusion regarding the probable reason for failure was based on clinical findings, the angiographical appearance before operation and the clinical findings at the time of occlusion of the reconstruction.

Group I. Failure due to Poor Inflow and Poor Flow-off

This group consists of 4 patients. The indication for surgery in every instance was an acute threat to the limb. In 2 patients a femoro-popliteal vein bypass was inserted. In the other 2 patients an ilio-femoral thromboendarterectomy was performed. Angiograms were available in all these patients showing no flow-off in the popliteal and its branches. In addition these 4 patients had stenoses present, proximal to the reconstruction.

All the reconstructions occluded in the immediate postoperative period. Three of these 4 patients came to an above-knee amputation. Patient no. 3 died 6 months after the failed reconstruction, from a carcinoma of the lung.

In this group failure was considered to be due to poor inflow as well as poor flow-off in the distal arterial tree.

Group II. Failure due to Poor Flow-off

This group consists of 4 patients. The indication for surgery was an acute threat to the limb in all cases. Angiograms were available and showed poor flow-off in every case. In 3 patients femoro-popliteal bypass grafts were inserted using vein graft, dacron graft and homograft respectively. In the fourth a thromboendarterectomy was performed. All 4 reconstructions occluded in the immediate postoperative period.

All 4 limbs came to an above-knee amputation. Patient no. 5 presented 2 years later with an acute threat to the opposite limb. He was treated conservatively with heparin and died of a cerebrovascular bleed.

Patient no. 7 died 5 months after reconstruction, from coronary thrombosis.

Group III. Failure due to Poor Head of Pressure

The indication for surgery in patient no. 9 was claudication, and an acute threat to the limb in the other 2 patients. Angiograms on patients nos. 9 and 10 showed disease in the popliteal artery and its branches, but flow-off was considered to be adequate. In case 9 a femoro-popliteal vein graft was inserted on the table with a good flow. The patient developed a coronary thrombosis during the operation with a profound drop in blood pressure in the postoperative period, followed by a thrombosis of the graft. This patient died 2 months after the operation from a further coronary thrombosis.

Case 10 also developed a coronary thrombosis during the operation with a fall in blood pressure. The reconstruction thrombosed in the immediate postoperative period. This limb came to an above-knee amputation.

Case 11 had an ilio-femoral thromboendarterectomy done for a threat to the limb. There was a palpable popliteal pulse present in the postoperative period.

The patient went into cardiac failure postoperatively and the segment rethrombosed.

The reason for failure in these 3 patients was considered to be due to decreased flow through the reconstruction caused by a diminished head of pressure.

Group IV. Failure due to Technical Error

This group consists of 3 patients. The indication for surgery was claudication in every instance. Pre-operative angiography showed good flow-off in the distal vessels.

In 2 cases a femoro-popliteal bypass graft was inserted using vein and teflon grafts respectively. In the third case an aorto-iliac thromboendarterectomy was performed. All 3 cases occluded in the immediate postoperative period.

Case 12 was re-operated on and the vein graft was found to be thrombosed but the popliteal artery and its branches were still patent. A dacron graft was then inserted which is functioning satisfactorily 8 months postoperatively. Both the other limbs had not deteriorated markedly at the time of occlusion of the reconstructions and were therefore thought to be segment thromboses.

Although no definite technical error could be pinpointed in the records, these cases have all had good distal flow-off but re-occluded in the immediate postoperative period. Failure was therefore regarded to be due probably to a technical error.

Group V. Failure due to Operation into Existing Disease

This group consists of 5 patients. The indications for surgery was threat to the limb in 4 patients and severe claudication in 1.

Pre-operative angiography showed patent, but severely diseased popliteal arteries with stenoses. The degree of ischaemia of 4 of these limbs bore out the limitation of blood flow caused by the severe involvement of the flow-off tracts.

The reconstructions all failed early with thrombosis of the popliteal artery, followed by an above-knee amputation in every instance. The failures in this group were thought to be due to thrombosis precipitated by operative trauma into severely diseased arteries.

Group VI. Failure due to Progression of the Disease at the Inflow Tract

This group consists of 2 patients. In case 20 a thromboendarterectomy of the adductor canal obstruction, stayed patent for 4 years and then re-occluded. At re-operation the popliteal artery was found to be patent but the disease had extended to involve the common femoral artery.

After thromboendarterectomy of the common femoral artery, a vein graft was inserted which is functioning satisfactorily. In case 21, a vein bypass graft was inserted around an adductor canal block. Angiography 7 months later showed progression of the disease both in the proximal and in the distal arteries. The vein graft occluded after 4½ years with disappearance of the femoral pulse.

Group VII. Failure due to Progression of the Disease in Flow-off Tract

Aorto-iliac reconstructions. This group consists of 5 patients. In all these patients the reconstruction stayed patent for at least 1 year. Occlusion of the reconstruction took place in every case due to progression of disease in the distal flow-off segments. Three of the patients lost their limbs after the occlusion.

Group VIII. Failure due to Progression of the Disease in the Flow-off Tract

Femoro-popliteal disease. This group consisted of 4 patients. The reconstructions remained patent for at least 11 months. Failure in every case in this group was due to progression of the disease in the distal segments. Three of the 4 patients lost their limbs after occlusion. Progression of the disease in the distal segments in both groups VII and VIII was proven either by follow-up angiography or exploration of the distal vessels in all but 2 cases.

In these 2 cases there was disease present in the distal flow-off segments on pre-operative angiography. At the time of occlusion of the reconstruction, the patient had severe ischaemia of the limbs followed by an above-knee amputation. In both these patients the distal vessels were found to be thrombosed.

Group IX. Failure due to Segment Occlusion

Aorto-iliac reconstructions. This group consists of 8 patients. In all these patients there was adequate distal flow-off present on angiography in the pre-operative films.

There was no excessive deterioration in the condition of the limbs in any of these patients at the time of the occlusion of the reconstruction. None of these patients lost their limbs when the segment failed. These failures are all due to occlusion of the reconstruction.

Group X. Failure due to Segment Thrombosis

Femoro-popliteal disease. This group consists of 5 patients. Pre-operative angiography showed localized lesions with patent distal vessels. The reconstruction thrombosed in 4 patients without severe deterioration in the condition of the leg. In 2 patients the distal vessels were shown to be still patent after the occlusion of the segment by angiography and exploration respectively. In patient no. 43 post-operative angiography showed stenosis of the distal suture line of the vein graft. The vein graft thrombosed after 3 months; in this case the failure may have been due to a technical error in fashioning the distal anastomosis.

RESULTS

Fig. 1 represents the reasons for failure in all cases. Eight cases (18·5%) failed in the immediate postoperative period, with loss of limb in 7 patients. These patients were all shown to have poor distal flow-off on angiography and reconstructions were performed as a desperate measure in an attempt to save an acutely threatened limb.

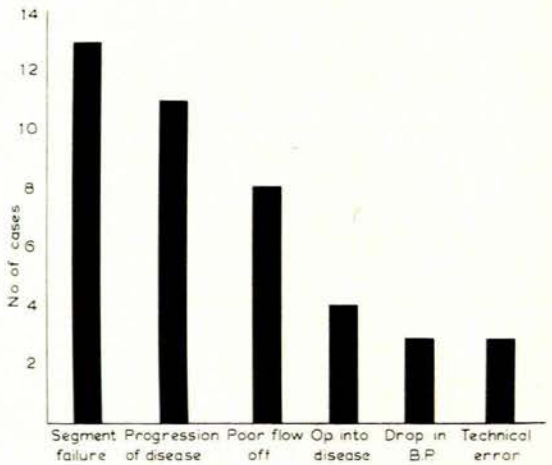

Fig. 1. Causes of failure in all cases.

The largest single group of failures was due to localized thrombosis of the reconstructed segment, namely 13 cases (31%).

In Fig. 2 the poorly selected group with bad distal flow-off was excluded. In the operable cases, the majority of failures (54%) was due to factors other than the underlying disease in the arteries.

Fig. 3 shows that when cases which have occluded before the elapse of one year are considered, it is found that 70%

failed because of factors other than the underlying disease.

Fig. 4 indicates that in late failures, however, the patient's disease is responsible for failure in 66% of cases.

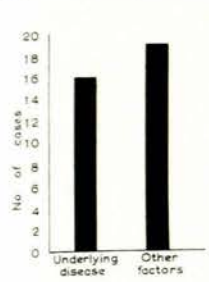

Fig. 2

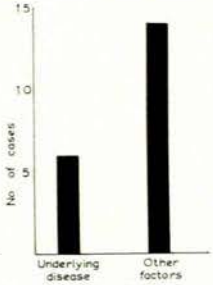

Fig. 3

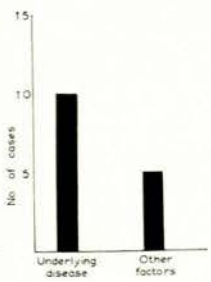

Fig. 4

Fig. 2. Causes of failure when those cases with poor distal flow-off are excluded. *Fig. 3.* Failures before the elapse of 1 year. *Fig. 4.* Late failures—after 1 year.

Fig. 5 shows that when all failures due to segment thrombosis are broken down into types of reconstructions and expressed as percentages of examples available in the series, it is found that 30% of prostheses fail because of localized segment thrombosis, compared with 14% of endarterectomies and 6% of vein grafts.

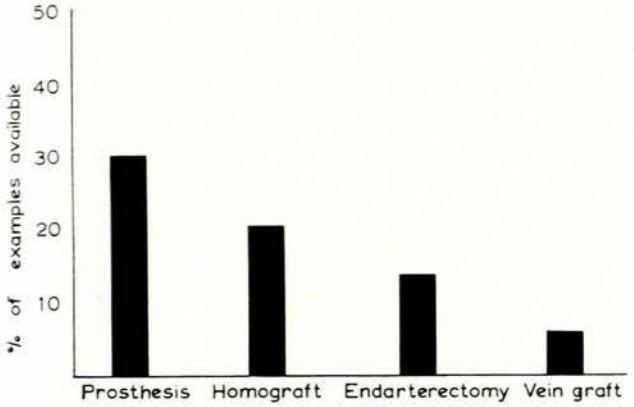

Fig. 5. Failures due to segment thrombosis.

There were only 5 homografts available for comparison. One of these failed because of localized segment thrombosis.

DISCUSSION

A. Immediate Failures

There is a 10 - 30% immediate failure rate irrespective of the method of reconstruction used.[1,17,22]

The basic cause of any arterial occlusion is decreased blood flow below a certain critical rate. The most important factors controlling the rate of flow are: (*i*) the central blood pressure; (*ii*) the inflow volume; (*iii*) the size of the reconstruction and (*iv*) the volume of the out-flow bed.

Selection of cases suitable for surgery. Selection of cases suitable for reconstruction is based on the interpretation of the angiogram.

The chances of a successful procedure are excellent if the flow-off tract below the point of reconstruction is adequate to carry away the volume of blood introduced through the reconstruction. In practice, however, reconstruction is often attempted in spite of inadequate flow-off, when a young man is going to lose his limb otherwise. In our experience such a reconstruction is doomed to failure in almost all cases.

When the reconstructions were attempted in limbs with considerable small artery disease beyond the popliteal bifurcation, or with lesions causing constriction of the popliteal vessel itself but without occlusion, failures within a few weeks to a few months were encountered. Irregularities of the flow-off tract may lessen flow to a surprising degree even if the total volume of the flow-off arterial tree remains high.

Constrictions in the popliteal artery itself decrease flow even more because of its proximity to the distal anastomosis. It is often impossible to avoid constructing the distal anastomosis into an area of disease in the popliteal artery. This trauma in an already diseased artery may predispose to thrombosis.

Prevention of embolization and thrombosis. Inadequate technique at the time of operation regarding construction of the suture line, injury to the intima or stagnation of blood in arterial segments for long periods of time, can be disastrous.

The judicious use of heparin, to prevent clotting in isolated segments of artery caused by the use of occlusive clamps, is of the utmost importance to prevent soft clots. The role that this factor plays in early failures is almost impossible to assess in a retrospective investigation. There is also the unknown factor of possible disturbance of blood coagulation in cases with an atheromatous diathesis. This is still rather hypothetical and has not been properly demonstrated or explained as yet.

Inflow volume. The best possible flow rate should be maintained by keeping the patient normotensive and normovolaemic. Proper blood replacement and anaesthetic handling is essential for success. Two early failures have been seen in this series due to a fall in the blood pressure as a result of coronary thrombosis during the operation. If there is an overlooked stenosis or complete thrombosis present proximal to the reconstruction, the inflow may be reduced to such an extent that thrombosis in the reconstruction is inevitable.

The size of the reconstruction. The lumen of the reconstruction must be as large as possible. The results in thromboendarterectomies above the inguinal ligament are far superior to the results below the inguinal ligament. There is often a tendency to narrow the site of thromboendarterectomy after the clearance, in stitching up the arteriotomy. A vein patch graft can be utilized to try and maintain the normal diameter of the lumen.

In the blind method of thromboendarterectomy, loose atheromatous plaques or intimal flaps may be raised which may partially obstruct the arterial lumen.

In constructing anastomoses in vein or prosthesis grafts, the lumen of the anastomosis must be as large as possible. An end-to-side anastomosis probably offers the best reentry flow from a graft into the main artery.

B. Early Failures

When early failures are considered it seems apparent that there are different factors in operation in each method of reconstruction.

Vein grafts. It is usually contended that vein grafts for traumatic lesions, where the anastomosis is made in otherwise healthy arteries, will stay open in the large majority

of cases. That this is not altogether true, has been strikingly pin-pointed by the report of Jahnke.[14] He has found a 47% late-closure rate in battle casualties in the Korean war following autogenous vein grafts.

Although vein grafts are at present claimed by many authors to be the most satisfactory grafting material in atheroma,[2,6,17,18,20,21] there is no doubt that the failure rate is still disturbingly high. The most common cause for failure in the early period is suture-line stenosis, combined with fibrotic contracture of wound healing.[28]

The importance of suture-line stenosis has prompted some surgeons to lay great stress on the type of connection that is made. End-to-end anastomosis has been replaced by end-to-side anastomosis to try and attain a wider suture-line lumen. Although this precaution does improve the results, suture-line stenosis has still taken place. On the other hand some of the longest patencies were recorded in cases where the surgeon thought the anastomosis was not satisfactory.[32]

Homografts. Small homografts behave very poorly in the early period, giving problems with stenosis and thrombosis.[24,25] It has been observed in a number of cases that a lesion starts off with a small mural thrombus going on to complete thrombosis. These mural thrombi are seen as small irregularities on the arteriograms before a complete thrombosis.[25,29]

In other homografts there is an early stenosis followed by thrombosis. The great variability of the manner of behaviour of homografts suggests that this process is determined by a host-graft reaction that may be immunological in nature.

Plastic prosthesis. Results with plastic prostheses have turned out to be much worse than their protagonists have expected.

The pore size of the fabric appears to be a factor of great importance because it determines the ultimate nature of the lining of the graft. A tight weave inhibits vascularization and organization and consequently leads to degenerative changes followed by mural thrombosis and occlusions.[11]

Suture-line failure has also been seen in the use of plastic prostheses. Such suture-line separation probably represents failure of the graft to establish a sufficiently firm fibrous tissue bond with the host artery.[23] Kinking of the grafts and deformation of the anastomosis on flexion of the adjacent joints can cause thrombosis.

Thromboendarterectomy. The results of thromboendarterectomy are better in the larger vessels above the inguinal ligament than in the small vessels. A partial explanation for this difference in immediate results may be in the demonstration of manifold increases in the concentration of soluble thromboplastins in smaller muscular arteries as compared with the large arteries which contain only a little smooth muscle.[13]

Early failures commonly relate to sluggish blood flow through the reconstructed segment.[1,10] Flaps caused by dissection of the distal intima or an atheromatous plaque at the termination of the thromboendarterectomized segment has been known to obstruct the artery and predispose to thrombosis. The thromboendarterectomized segment may be significantly narrowed when the arteriotomy is sutured.

C. Late Occlusions

The high incidence of late failure in grafts done for occlusive disease compared with the almost non-existent incidence of thrombosis in grafts done for abdominal aneurysms, is strong evidence that the causes of failure are not inherent in the graft material or the basic techniques used in reconstructive arterial surgery.

The single important factor in late failures again is decrease in rate of flow, predisposing to thrombosis. The impedance to flow may occur again (*i*) in the reconstruction and (*ii*) in the inflow and outflow tracts, due to progression of the underlying disease.

All authors are not agreed on the importance of the progression of the underlying disease as a cause of failure. Linton[26] states that in his own patients, failure in the great majority of cases is due to changes in the grafts themselves and only rarely to the advance of atherosclerotic disease in the patient's arteries. This was also the view of Crawford *et al.*[4] and Shepherd and Warren.[22]

On the other hand some authors[15,25] feel that late closures are related, in a considerable number of cases, to progression of the underlying disease. In our own cases, late failure was considered to be due to the progression of the disease in 66% of patients.

If one bears in mind that atherosclerosis is a progressive systemic disease and that even in its most sharply localized forms it involves to some degree the entire arterial system, this is not a surprising finding.

The Reconstruction

The methods of reconstruction are considered under the following headings:

Vein grafts. Narrowing at the suture lines, wound fibrosis and fibrotic contracture followed by thrombosis are all problems related to vein grafting. The grafted segment of vein also develops atheroma.

Homografts. The patency of initially successful homografts falls to less than 50% after 18 months.[24,26] Thrombosis follows earlier stenosis and degeneration of the graft due to mural fibrosis or slowly progressive atheromatosis.[29] Aneurysms begin to appear after the first year and continue to crop up at fairly regular intervals in subsequent years. These complications probably occur as a result of failure of vascularization and incorporation of grafts into the host tissue, followed by degenerative changes.

Aneurysms are fortunately rarer in the large homografts, probably because of their tendency to early calcification.[8] However, reports of late false aneurysms of the aortic homografts rupturing into the gastro-intestinal tract are numerous.[3,19,27]

Plastic prosthesis. Apart from failure to form a firm bond between the graft and the host artery, predisposing to ruptures and aneurysm formation, an important problem is the fact that the plastic material is firmly encased in fibrous tissue leading to kinking at adjacent joints.[7] Mural thrombi form on the intima of plastic prostheses followed by complete occlusion. It has also been shown experimentally that the new intima of the plastic prosthesis can develop atheroma quite readily.

Thromboendarterectomy. In many of the cases of thromboendarterectomy which have remained patent for several months, a gradual but progressive narrowing of

the segment has been seen. This narrowing is due to perivascular and mural fibrosis, and may predispose to thrombosis.[29]

The process may stabilize after 1 year and some of the narrow channels are compatible with patency for long periods of time before finally occluding.[25]

The Underlying Disease

The generalized nature of atherosclerosis makes it impossible to bypass or resect diseased artery completely. For this reason the distal anastomosis is often made in a segment with pre-existing disease, because a healthy area cannot be reached for technical reasons or in fact none exists.

In many instances a block is not necessarily due to the development of new lesions, but to the progression of already existing ones. This may be the explanation for the relatively early occlusion distal to reconstructions in some cases.

Warren and Villavicencio[25] described 15 cases in which late failures are attributed to progression of the disease process. Szilagyi *et al.*[23] claim that in 16 of 21 late failures out of 71 grafts, a role may be assigned to the disease process. They conclude that the factor of the greatest importance in the genesis of late failures is the extent and degree of severity of the occlusive disease.

SUMMARY

Forty-three failures after arterial reconstructions have been studied.

When failures due to poor flow-off (bad selection) are excluded, the majority of re-occlusions (54%) were due to factors other than the underlying disease of the patient. In those cases which have occluded after 1 year and longer, the underlying disease is responsible for failure in 66% of cases. When segment failures were considered, prostheses fared worse with 30% failures and vein grafts fared best with only 6% failures.

I should like to express my thanks to Prof. D. J. du Plessis under whose care all these patients were admitted, and to Messrs. H. Gaylis and L. Stein, who operated on the large majority, for permission to study their cases. Mr. A. Veenstra prepared the figures used in this paper.

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