Operative Techniques in Arterial Surgery

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Preface

This book is intended to provide a detailed account of the technical aspects of present day arterial surgery. It is hoped that the book will be more than just a manual of operative procedures and that it will provide the reader with an overall strategic plan when faced with a vascular problem. Emphasis has been placed throughout on careful preoperative assessment particularly at the sites of arterial involvement so that the most appropriate reconstruction technique can be selected. Meticulous attention to operative detail during each stage of a vascular reconstruction remains the basis for success. For this reason the various procedures in this book are described in step-by-step fashion aided by a large number of drawings. An updated bibliography citing the work of other authors is also included.

Vascular surgery by its very nature introduces changes in vessel compliance and geometry and these may be the starting point for subsequent complications. In addition, complications may occur as a result of progression of the underlying vascular disorder. An appreciable portion of this work has therefore been devoted to the prevention, recognition and treatment of these complications.

The last few years have seen important advances in non-invasive investigation which have widened the indications for surgery and improved the accuracy of preoperative and postoperative assessment. Parallel with this has been the advent of interventional radiology and in particular percutaneous transluminal angioplasty which may be integrated into the operative programme or used as the sole treatment modality. These and related topics are covered in appropriate sections of the book.

We hope that this book will be of value to the beginner in vascular surgery and that the more experienced surgeon also will find something of interest in these pages.

A.S.W. J.M.C.

Foreword

In the last decade a relatively large number of books dealing with vascular surgery has been published in increasing numbers, primarily on both sides of the Atlantic. The appearance of a new book on this subject such as *Operative Techniques in Arterial Surgery*, might then raise the question, why another one? There are several reasons which more than justify "another one". Indeed, the scope of each published book is not always identical, or even equal in the design of those in existence. Some of them indeed assume that all-encompassing textbook size, others limit themselves only to some aspects of this still never-ceasing to expand field of vascular surgery. Thus, in spite of over four decades since the advent of the renaissance of this speciality, newer and more sophisticated methods of diagnostic and operative procedures have made their appearance. To keep abreast with the newest advances one may consult without delay innumerable scientific journals. The accuracy and practical applications of the latest contributions are screened, evaluated, and ultimately filtered down to their essential facets to be found in a great variety of periodicals, and of course in the most updated textbooks on such subjects.

This book, that I am about to introduce to the reader by way of a brief Foreword, is a perfect example of what I was commenting upon above. First of all I would like to point out that this work is unique, not only in that it represents a collaboration between two prominent vascular surgeons across the English Channel, but in many other ways as well. As the result of a joint Anglo-French exercise, it is something of a new venture in vascular surgical circles. Furthermore, while contemporary textbooks in general, and vascular surgery ones in particular, are commonly written by a number of contributors usually selected for their expertise in special areas, this textbook was written and edited solely by the two authors. Whereas the method of multi-author writing of a textbook may offer a variety of opinions, it also has the disadvantage that it may suffer from a lack of uniformity of conceptual vision of the subject. In this respect, this book is also apart from others. Ward and Cormier, who have had a major interest in vascular surgery for many years and have contributed extensively to this speciality by many articles and textbooks, offer today the fruit of their personal experience, without however omitting some of the many contributions of others. This personal concept, authors without the usual list

of outside contributors, puts this textbook aside from most of those currently published.

Additionally, this book is remarkable by meeting four major criteria for excellence: (1) it deals in a logical sequence with the various arterial lesions and entities, (2) each chapter includes a succinct but comprehensive background of the vascular condition (an overview of the problem, the various clinico-pathologic forms) which precedes the operative techniques, the basic raison d'être of this textbook, (3) a large number of illustrations essential for the step-by-step repair of some of the more complex technical problems, and (4) not the least of this highly recommendable book, are the updated selected references at the end of each chapter.

It is well known that success or failure in vascular surgery are often not far apart, due to very narrow margins of safety of these procedures that are more challenging here than in any other surgical specialty. For that unique reason it is imperative that operative techniques dealing with vascular repairs be handled with utmost skill. The text and the detailed portrayal of *Operative Techniques in Arterial Surgery* by Ward and Cormier will be invaluable to all those dealing with these often complex cases. Residents in training, registrars, as well as experienced vascular and general surgeons should find in this combination of text and arterial techniques great help in the practice of vascular problems. This textbook is therefore highly recommended to all those interested in this difficult and sometimes complex field of surgery.

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Acknowledgements

It is a particular pleasure to acknowledge the collaboration of my friend and co-author Jean-Michel Cormier. Our professional association extends over a number of years and it has been a most enjoyable and rewarding task to combine our ideas and experience in this book. Although the chapter writing has been equally shared, the responsibility for the overall literary style, bibliography and construction of the drawings rests on the English side of the Channel and any criticism of these aspects should be pointed in this direction.

I am also grateful to David Cumberland, Consultant Radiologist at the Northern General Hospital, Sheffield, for his contribution in writing the chapter on angioplasty and for his helpful suggestions in the section on transcatheter embolization.

Pat Elliott of the Department of Medical Illustration, the Royal Hallamshire Hospital, Sheffield, has been responsible for all the completed art work – a monumental task. He and I spent many hours working on the final drawings and this book is a tribute to his patience, skill and attention to detail.

In addition, I should like to thank the staff of MTP Press and in particular David Bloomer and Martin Lister for their courtesy and forbearance during the preparation of the book. Mrs Eileen Murat and Mrs Doreen Miller kindly assisted in the preparation, typing and revision of the manuscript and my thanks are due to them.

Lastly, and in many ways the most important of all, I owe a very great debt of gratitude to my wife for devising and arranging the entire layout of the book, for checking page-proofs and galley-proofs, and for constant encouragement when completion seemed far from reality.

A.S.W.

1

Basic Techniques

Vascular surgery has expanded considerably in the last quarter of a century and now encompasses a great diversity of procedures. Despite this expansion the specialty rests on a relatively small number of basic techniques which when used alone or in combination will allow the successful correction of the great majority of vascular surgical problems. These techniques are described in this first chapter.

ARTERIAL MOBILIZATION

Major vessels are usually enclosed in an identifiable fascial sheath which must be opened before the artery can be exposed. After incising the sheath longitudinally the artery should first be freed on either side using the blunt tips of curved scissors. In some atherosclerotic vessels this plane may be obscured by a fibrotic reaction and dissection should then stay right on the surface of the artery. The deep aspect of the vessel is best freed by gentle spreading with the tips of a Mixter or other right-angled clamp. A Silastic loop passed around the vessel will then provide traction while an adequate segment of the artery is displayed. Exposure of a major branch is achieved by a similar technique of loop retraction on the main axis above and below the branch vessel (Figure **1.1**).

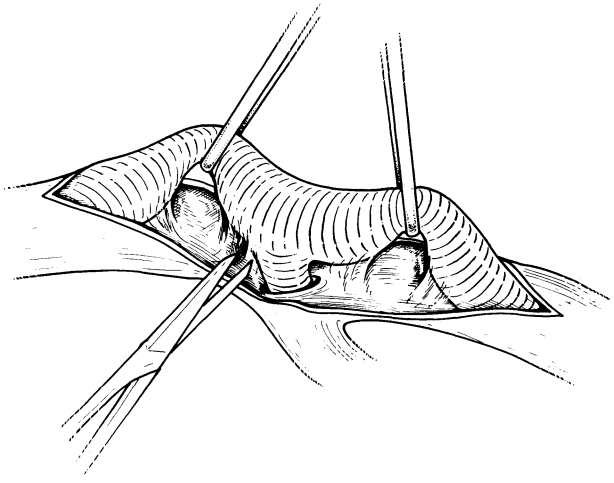


Figure 1.1 Exposure of a deeply placed branch, e.g. profunda femoris, with the aid of loop retraction

ARTERIAL CONTROL

A number of options are available for interrupting blood flow. These include tourniquets, arterial clamps, double-looped Silastic loops and endoluminal balloon catheters. Digital compression may also be useful as a temporary expedient.

Most modern vascular clamps achieve a non-crushing, non-slipping grip by virtue of longitudinal rows of interdigitating or opposing serrations. Although this design minimizes vessel damage, few clamps are completely atraumatic. If clamps are misapplied to atherosclerotic vessels, intimal fracture is very likely to occur, leading to immediate thrombosis or premature stenosis. In order to minimize this risk the artery wall should always be carefully palpated before clamping and the least diseased site selected. In addition, clamps should be applied once only and with the minimum degree of occlusion necessary to secure haemostasis. In this context once a proximal graft–artery anastomosis has been completed it is helpful to replace the proximal arterial clamp with a rubber-shod clamp placed across the graft adjacent to the suture line. This replacement clamp may then be released and reapplied as necessary during the rest of the procedure without risk of intimal damage.

Medium-sized vessels may be safely controlled by soft jaw Fogarty clamps or by double-looped Silastic loops. Smaller vessels may be secured by similar loops or by microvascular clips.

Endoluminal balloon catheters are useful when the vessels are particularly calcific. This type of control may also be necessary when arterial exposure is limited by fibrosis as a result of previous surgery or where rapid haemostasis is required in the midst of a haematoma, e.g. ruptured aneurysm, vascular trauma.

HEPARINIZATION

Most vascular procedures are conducted under heparinization though it may be preferable to omit heparin during particularly extensive aortic replacement[1]. Heparin will prevent distal thrombosis during the clamp period and this has been an important factor in reducing the incidence of postoperative leg ischaemia[2]. Regional heparinization is not really a practicality and a systemic bolus should be utilized. Although there is some individual variation in the response to heparin, a dose of 1 mg/kg body weight will achieve adequate protection in nearly all cases and this should be administered well ahead of clamping[3]. If prolonged clamping is required, additional heparin may be given according to the activated clotting time. If the clamp time is short, i.e. less than 1 hour, the heparin may be reversed with protamine on a 1:1 basis. However, in most cases the effect is merely allowed to wear off.

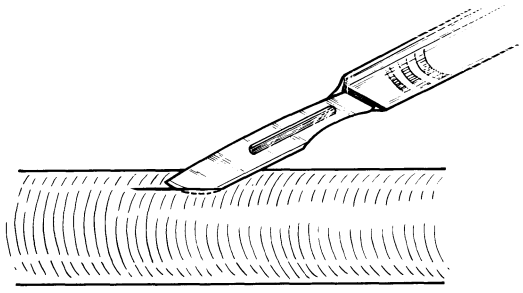
MAKING AND CLOSING AN ARTERIOTOMY

The initial incision into the artery should be made with the cutting edge rather than the point of the scalpel blade so as to avoid damaging the opposite wall. This opening is then extended with Pott's angled scissors (Figure **1.2a, b**). In most end-to-side anastomoses a linear arteriotomy will expand to form an adequate stoma and elliptical excision should be reserved for particularly sclerotic vessels, graft–graft anastomoses and some reimplantations. Excision of an ellipse of artery wall should be particularly avoided in vein graft–artery anastomoses as this may lead to splaying of the vein and a reduction in anastomotic circumference.

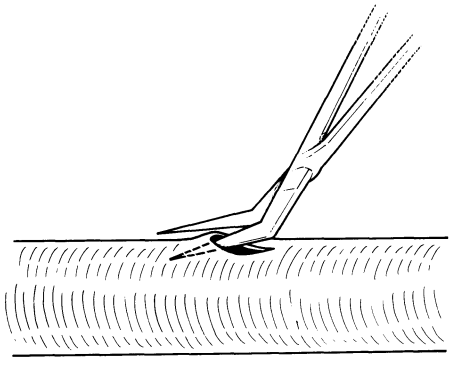
In large vessels a longitudinal arteriotomy may be closed directly using continuous or interrupted sutures. Longitudinal traction via a stitch at each end will appose the two edges and facilitate suture placement. In small or medium-sized vessels direct closure may lead to stenosis and a patch graft is usually required (see below).

Transverse arteriotomies are the standard method of access for catheterization or other instrumentation. The incision should extend over the anterior half of the vessel circumference since a more limited opening may risk intimal damage on catheter retrieval.

Transverse arteriotomies can usually be closed directly without inducing stenosis. Traction stitches are placed at each end of the incision and interrupted sutures inserted between these points. Sometimes the intima tends to slip beneath the cut edges and double-armed sutures inserted from within outwards may then be necessary to ensure that all layers of the artery are included in the suture line.



(a)



(b)

Figure 1.2(a, b) Making an arteriotomy

VASCULAR SUTURES

To some extent the selection of vascular suture material is a matter of individual preference. Most vascular sutures are swedged onto fine $\frac{1}{2}$ or $\frac{3}{8}$ circle round-bodied needles, though taper-cut needles may be required for a particularly sclerotic vessel. The finest suture capable of joining the two parts soundly should be chosen in order to limit the amount of suture material in contact with the bloodstream and to keep the size of the needle hole to a minimum. As a frame of reference 2/0 or 3/0 suture material may be used for the aorta, progressing down to 6/0 for vessels of popliteal or internal carotid size and 7/0–9/0 for anything smaller. Optical magnification and ophthalmic instruments are useful in the latter circumstances.

Monofilament sutures such as polypropylene (prolene) are widely used for arterial work. These have minimal drag so that a loosely looped or parachute technique can be employed at the critical heel and toe of an anastomosis, greatly facilitating accurate suture placement. Although there are obvious theoretical risks in relying on such sutures to maintain graft–artery union, in practice prolene does not appear to be associated with a significant risk of false aneurysm formation on medium to long-term follow-up[4]. It is of course absolutely essential to avoid any instrumental damage to this or any other suture material. In addition, very fine monofilament sutures are best avoided when the arterial wall is calcified because of the possible sawing effect exerted by the plaque with pulsation. Coated polyester sutures are preferable in these circumstances and also for graft–graft anastomoses.

BASIC SUTURE TECHNIQUE

The intima-to-intima everting stitch used in former times is in fact unnecessary and most arterial suture lines can be completed using a simple continuous over-and-over stitch. Interrupted sutures are essential for anastomoses in children where subsequent growth must be anticipated. They are also useful in adults when the vessels in question are particularly small or delicate.

In medium-sized vessels, sutures should be placed 1 mm apart and 1 mm from the arteriotomy edge, though if the artery wall is thickened with a tendency to split, deeper bites may be required. Appropriate adjustments in suture depth and spacing are also made in relation to vessel size. In all cases it is important to pass the needle through the artery wall in a smooth curve to avoid an unduly large stitch hole. In addition, it is usually recommended that the direction of suturing should be from within the arterial lumen outwards to avoid displacing atherosclerotic plaque (Figure **1.3a, b**). However, in some circumstances it is quicker and easier to sew in the reverse direction. With practice this is usually quite safe, particularly if a closed forceps is used to steady the artery wall (Figure **1.3c**), and during most anastomoses either direction of needle passage may be utilized according to convenience.

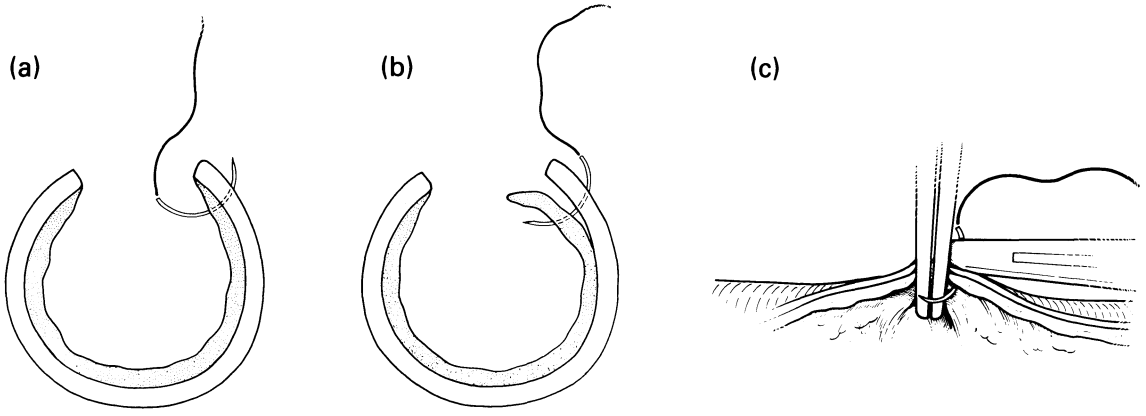


Figure 1.3(a–c) Suture technique in an atherosclerotic artery: (a) inside-out (b) outside-in with risk of intimal separation (c) a method of avoiding intimal separation

Before completing an anastomosis it is advisable to inspect as much as possible of the interior of the suture line. Any deficiencies or intimal fractures can then be corrected. Irrigation of the lumen with heparin-saline should also be employed as a routine to demonstrate any large leaks as it is clearly preferable to insert extra stitches at this stage, rather than after declamping. The controlling clamps are then flushed to evacuate any clots or debris and the lumen again irrigated before finally completing the suture line.

Once the clamps have been removed a little local pressure with gauze will help seal any small leaks. If significant leakage persists, extra stitches will be required. Attempts to insert these without reclamping are likely to enlarge the defect so that temporary arrest of flow is necessary either by digital compression of the graft or artery or by reapplication of the clamps. The latter carries at least a theoretical risk of thrombosis in the reconstruction zone and an alternative is to secure the suture line with pledget-backed sutures inserted into the unclamped vessel. (Figure **1.4**). This type of suture is also useful in buttressing side-arm grafts or reimplanted vessels when the artery wall is particularly friable. (Figure **1.5**).

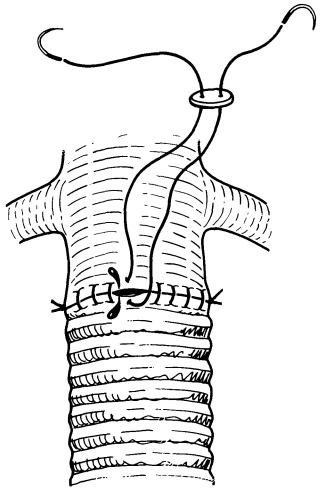


Figure 1.4 Anastomotic leak corrected by an additional stitch mounted on a Teflon or Dacron pledget

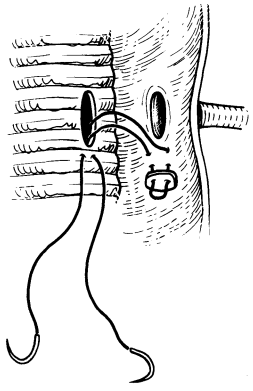


Figure 1.5 Pledget-backed sutures used to buttress an endosaccular anastomosis

END-TO-END ANASTOMOSIS

A technique for end-to-end anastomosis of large vessels is shown in Figure **1.6a–d**. The posterior wall is sewn in first, commencing intraluminally at the far corner (Figure **1.6a**). A double-armed suture is inserted without knotting and several loops are placed before the suture line is tightened to approximate the vessel and the graft. The posterior wall is then completed and the corner turned. The unused suture arm is then carried round anteriorly to meet its fellow (Figure **1.6c**). A rubber-shod or soft jaw clamp is placed immediately distal to the anastomosis and the initial controlling clamp released to distend the anas-

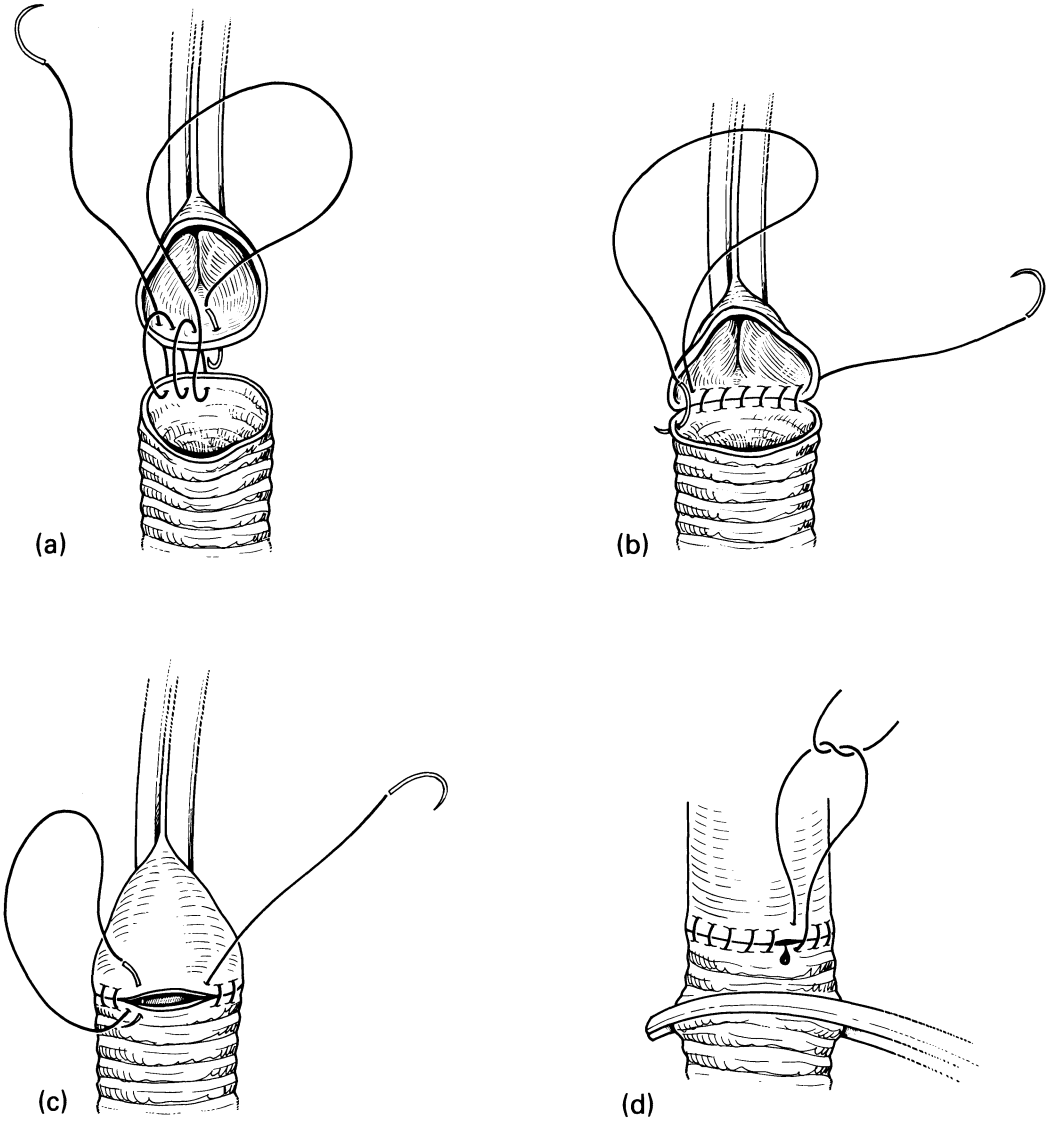


Figure 1.6(a–d) End-to-end large vessel anastomosis

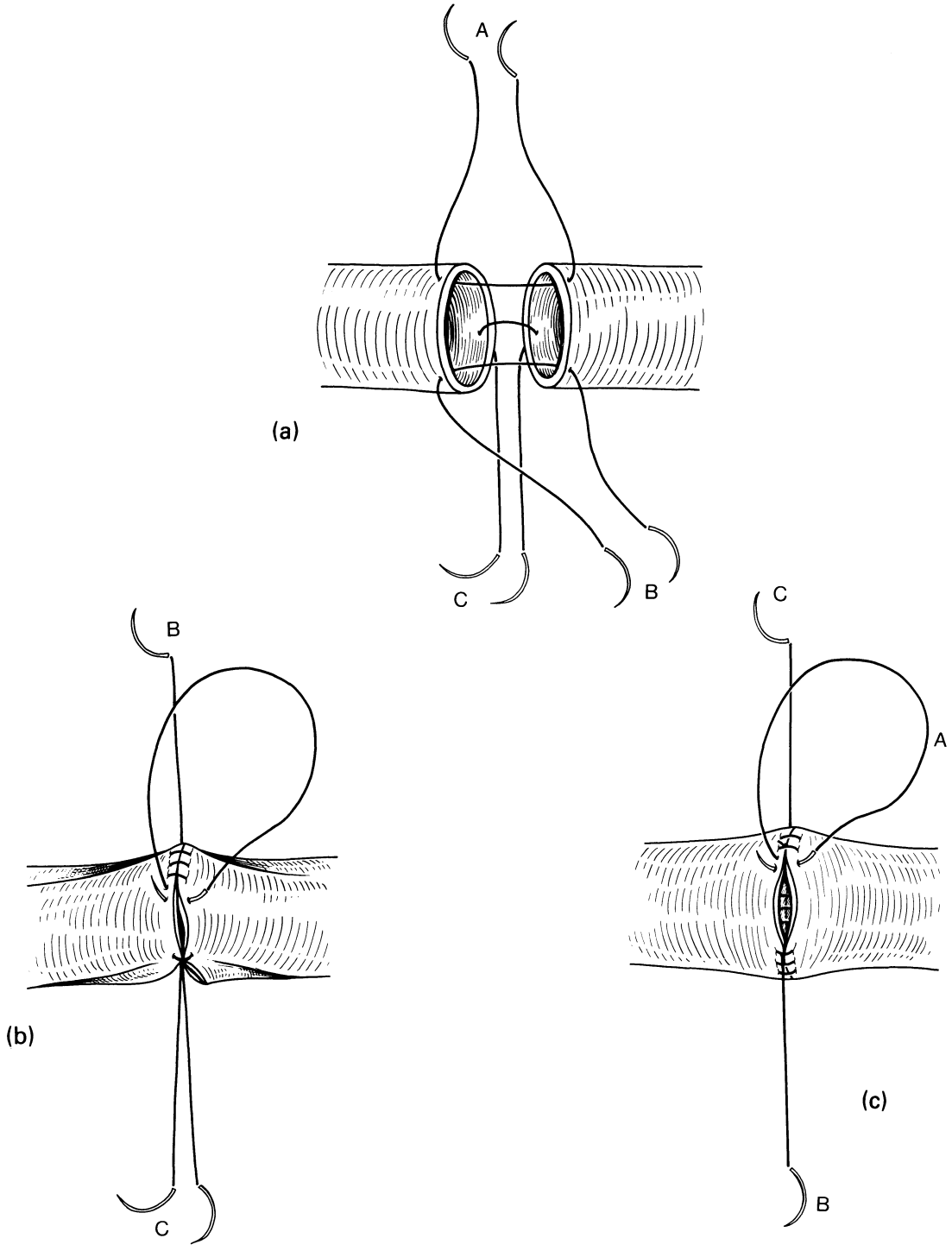


Figure 1.7(a–c) End-to-end anastomosis by triangulation

tomosis as the sutures are tied. This will avoid any possible purse-string effect.

An alternative technique for end-to-end anastomosis of large or medium-sized vessels is by triangulation (Figure **1.7a–c**). Double-armed sutures are placed 120° apart and the clamps then rotated or the initial stitches slung to allow each segment of the circumference to be closed from the outside. This method is only feasible when the mobility of the vessels is not impaired by adjacent branches.

End-to-end anastomosis between a graft and most medium or small arteries is best accomplished by spatulating the two extremities in order to enlarge the anastomotic circumference (Figure **1.8a–c**). Suturing is essentially the same as for an end-to-side anastomosis (see below).

Where the recipient artery is pathological this

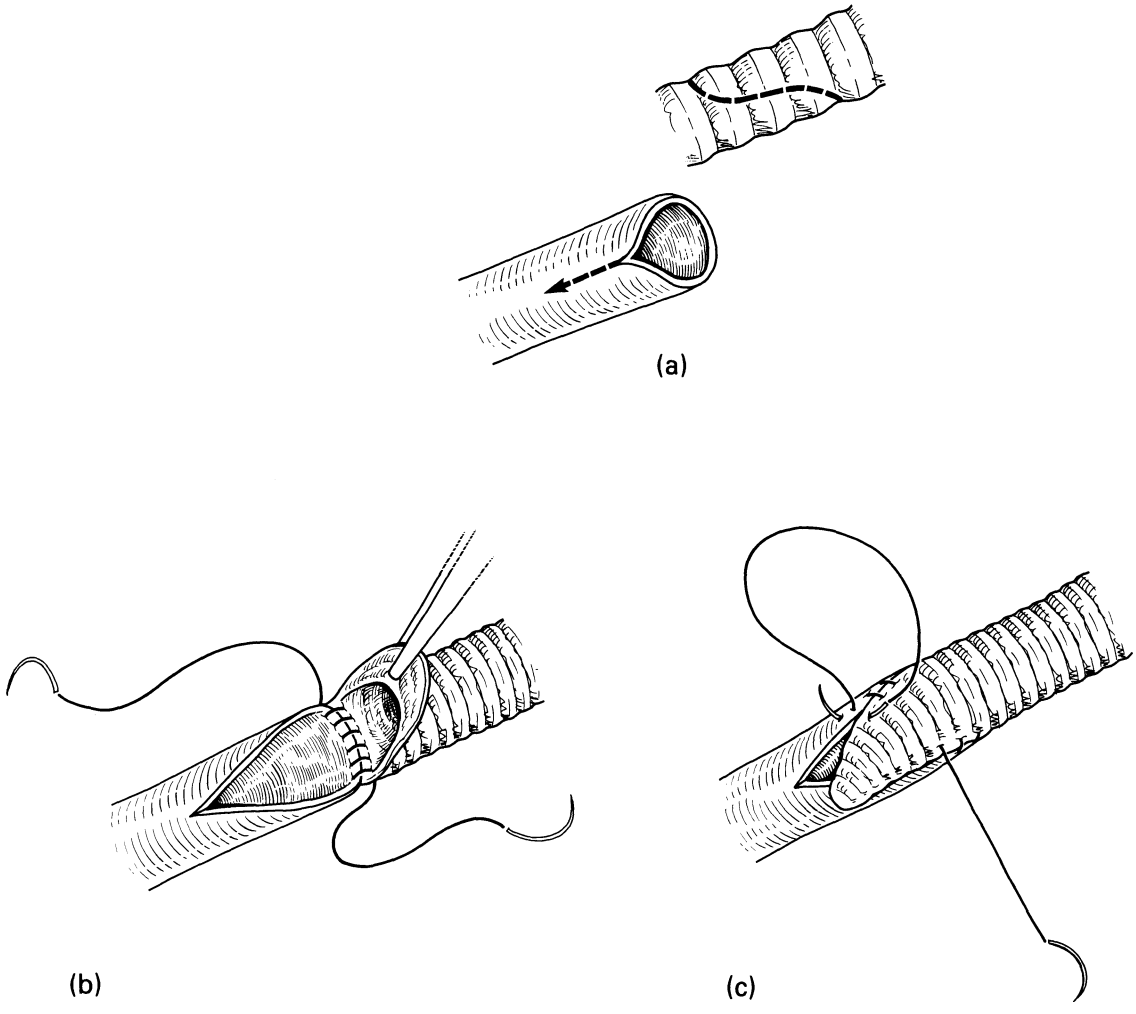


Figure 1.8(a–c) End-to-end spatulated anastomosis

method may risk intimal cut-out as there is inevitably a certain amount of tension when the ends of the graft and artery are pulled together (Figure **1.9a**). A safer technique in this situation is to anastomose the graft initially to the side of the artery at a site free from disease. A ligature is then placed close to the anastomotic heel, converting the anastomosis into an effective end-to-end configuration with consequent improvement in graft flow (Figure **1.9b,c**).

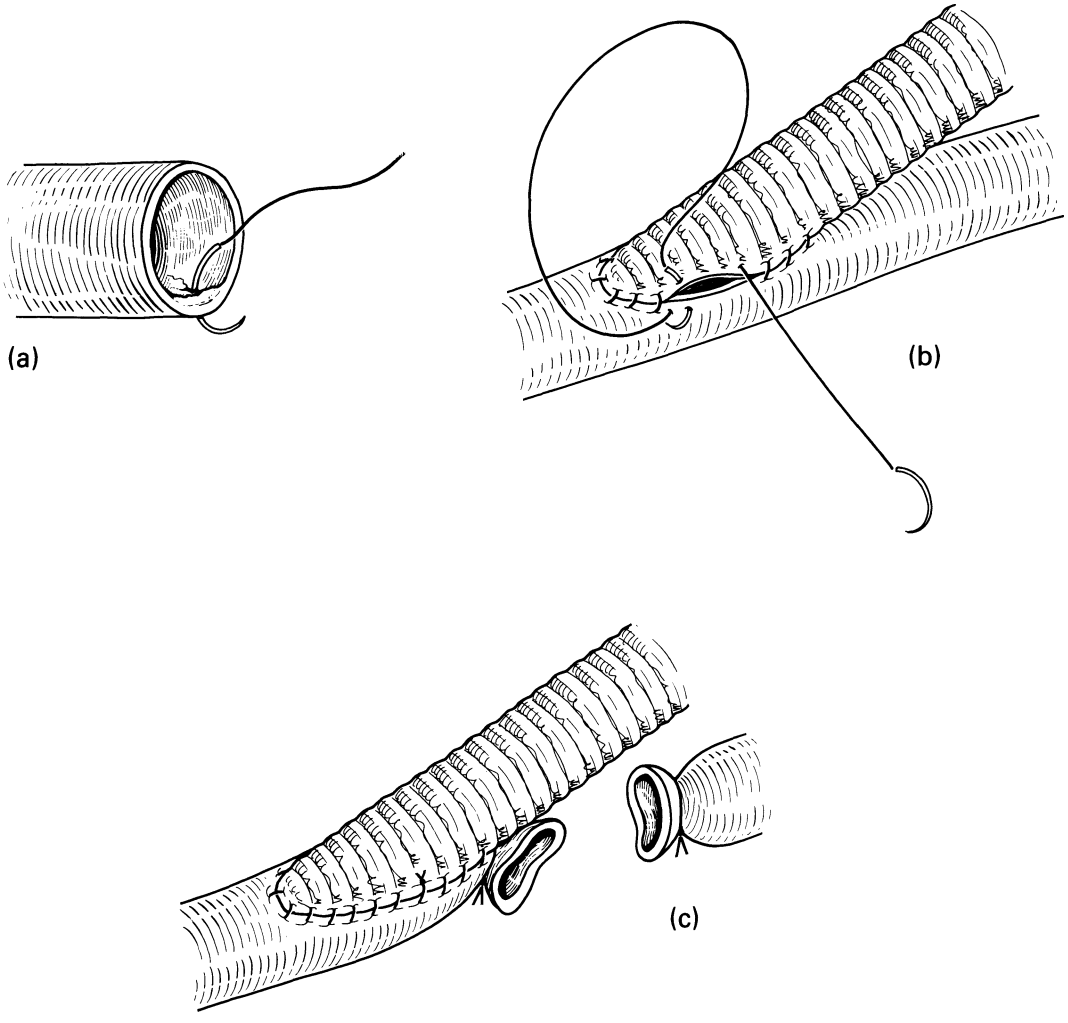


Figure 1.9(a–c) (a) Risk of intimal split or suture cut-out with direct end-to-end anastomosis in a pathological artery. (b), (c) risk avoided by end-to-side anastomosis with proximal ligation.

END-TO-SIDE ANASTOMOSIS

Prosthesis–artery

A technique for end-to-side attachment of a prosthetic graft is shown in Figures **1.10–12**. In general the length of the arteriotomy should be two to three times the diameter of the graft. Once the arteriotomy has been made, the end of the graft is cut to match. In the case of a Dacron graft, an S-shaped cut is used in order to produce a rounded contour at the heel and toe of the future anastomosis (Figure **1.10**). If the graft is merely cut obliquely in a straight line, it is difficult to avoid producing stenosis at these two points during suture placement. A suitable technique for cutting a polytetrafluoroethylene (PTFE) graft is shown in Figure **1.11**.

The anastomosis is commenced at the heel using a double-armed suture (Figure **1.12a**). Several loops are placed on either side of the extreme angle under direct vision with the graft and artery held apart. These loops are then carefully tightened, pulling the graft heel into place (Figure **1.12b**). Thereafter the far side is closed carrying the suture around the apex. Temporary use of a number of loose loops at the apex will again facilitate accurate suture placement at this critical point. Once the apex is passed the suture line is retightened. The unused suture arm is then taken along the nearside edge to meet its fellow (Figure **1.12d**). As with end-to-end anastomoses it is advisable to fully distend the suture line under arterial pressure before the sutures are finally tied, in order to avoid a purse-string effect.

This technique of anastomosis has the advantage of offering maximum control of suture placement at the heel and toe in contrast to the commonly advocated alternative in which an initial suture is placed and tied at each extremity.

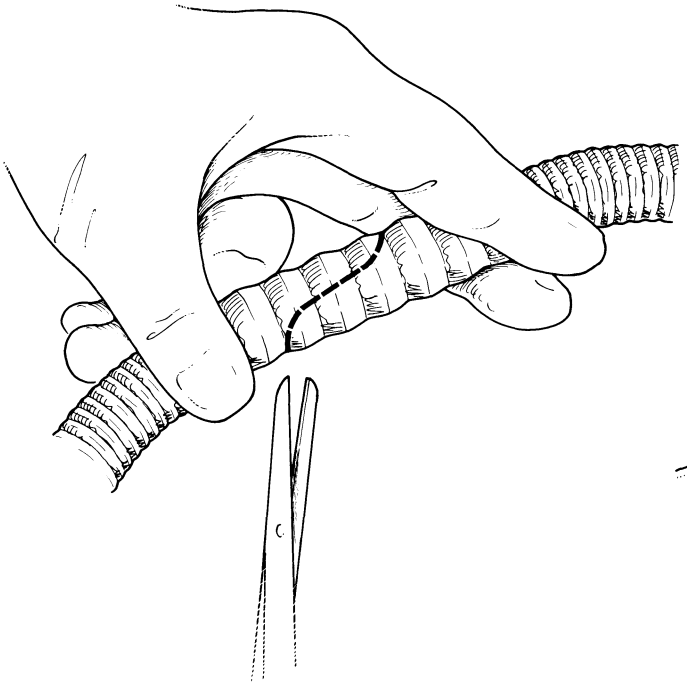


Figure 1.10 Method of cutting a Dacron graft

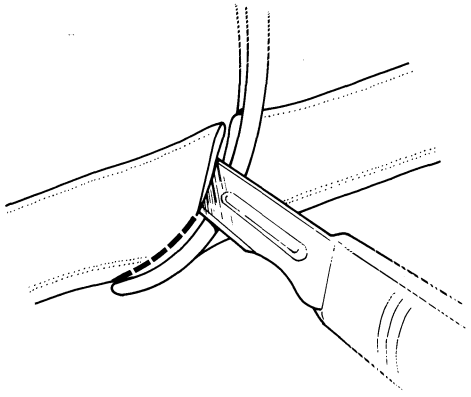


Figure 1.11 Method of cutting a PTFE graft

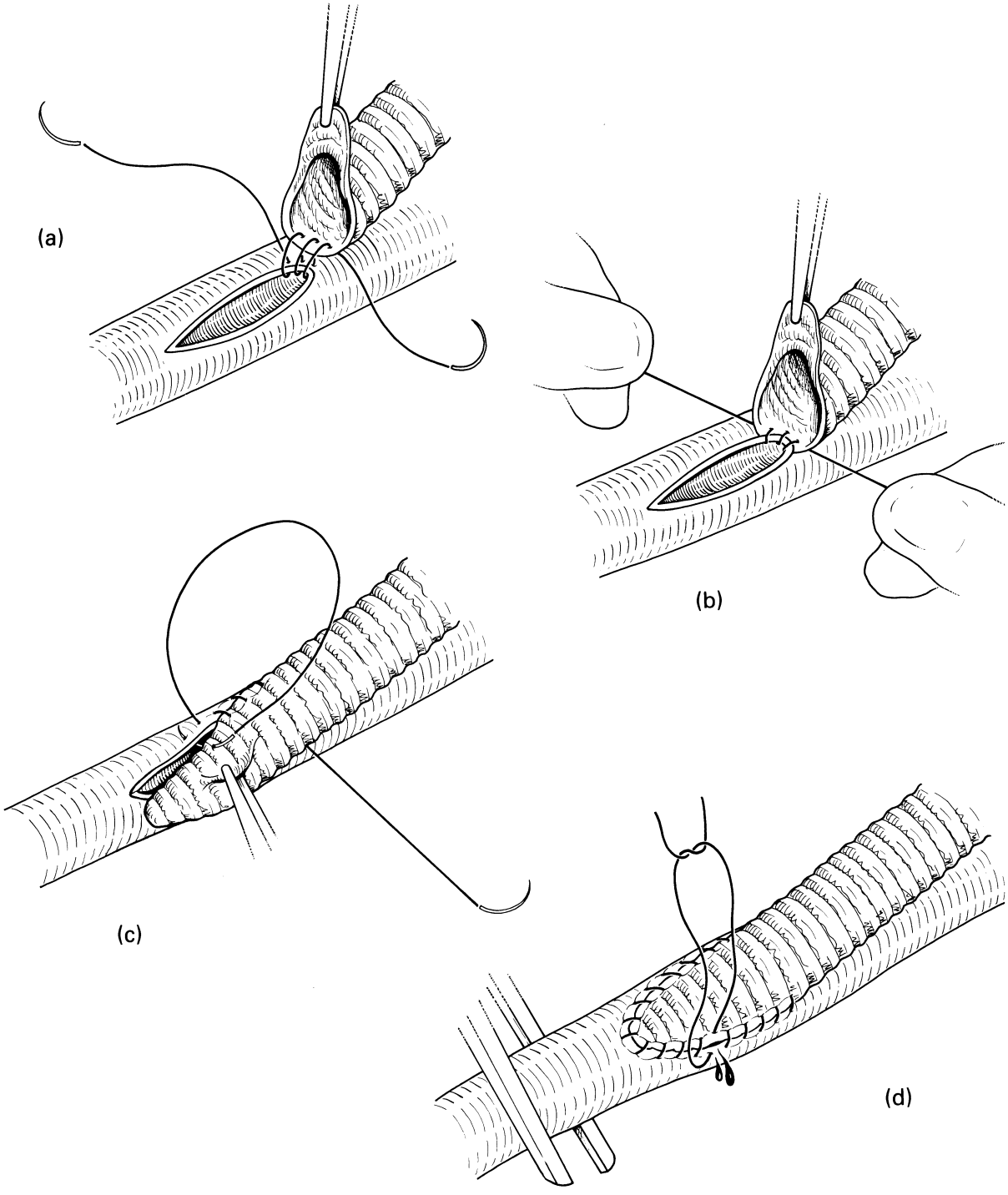


Figure 1.12(a–d) End-to-side prosthesis–artery anastomosis

Vein graft–artery

End-to-side anastomosis between a vein graft and artery requires a slightly modified technique. An initial arteriotomy is made extending for slightly more than twice the diameter of the vein. The vein extremity is then cut back for exactly the same distance and the corners trimmed (Figure **1.13a, b**). If there is a conveniently placed branch this should be used to produce a flange at the anastomotic heel (Figure **1.14a–d**).

The first essential is to secure each extremity of the anastomosis in order to place the edge of the vein under longitudinal tension. Without this provision the suturing may be uneven leading to wrinkling and turbulent flow. As before, a loosely looped suture technique is used with the vein and artery initially held apart (Figure **1.15a, b**). The second essential is to avoid a rolling-in effect of the vein on the artery. In this respect it may be helpful to secure the vein adventitia with forceps as each suture loop is pulled through (Figure **1.15c**). In addition the smallest possible suture bites should always be utilized, particularly at the proximal and distal angles of the anastomosis.

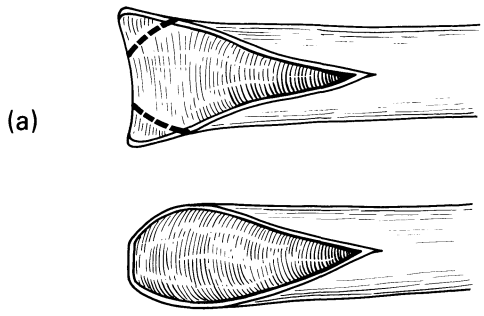


Figure 1.13(a, b) Preparation of saphenous vein for anastomosis

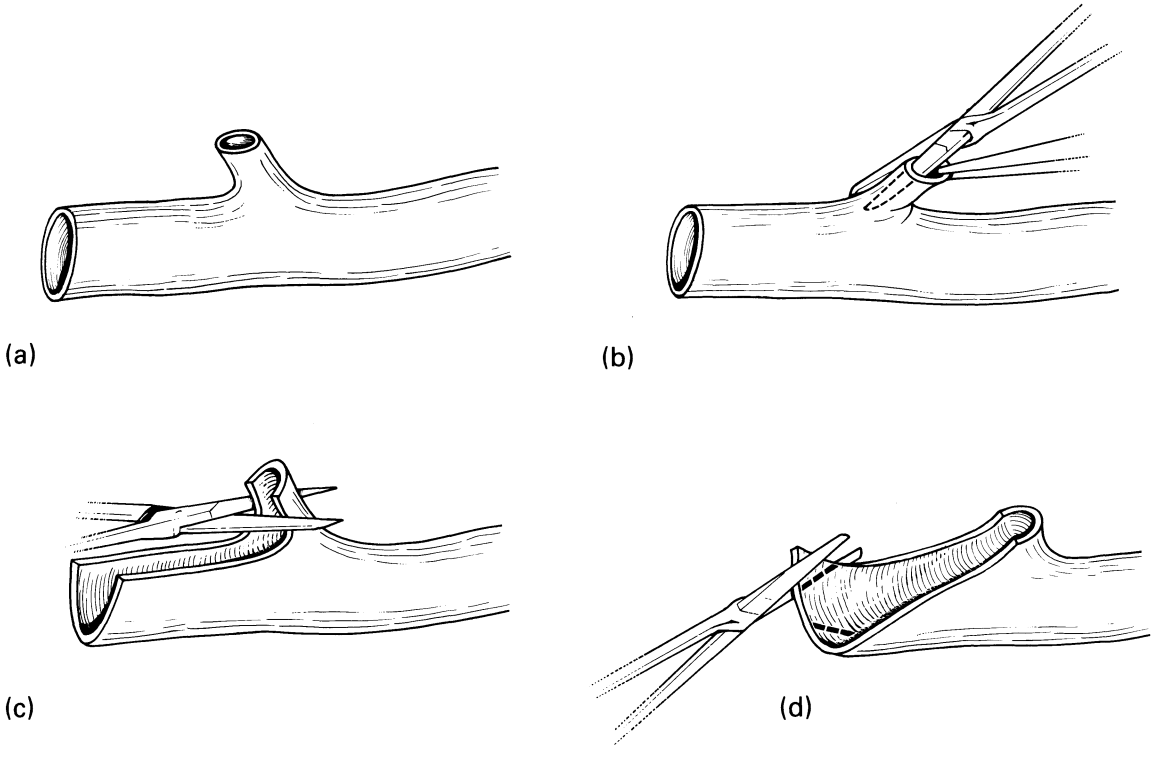


Figure 1.14(a–d) Use of a vein branch to enlarge the anastomotic heel

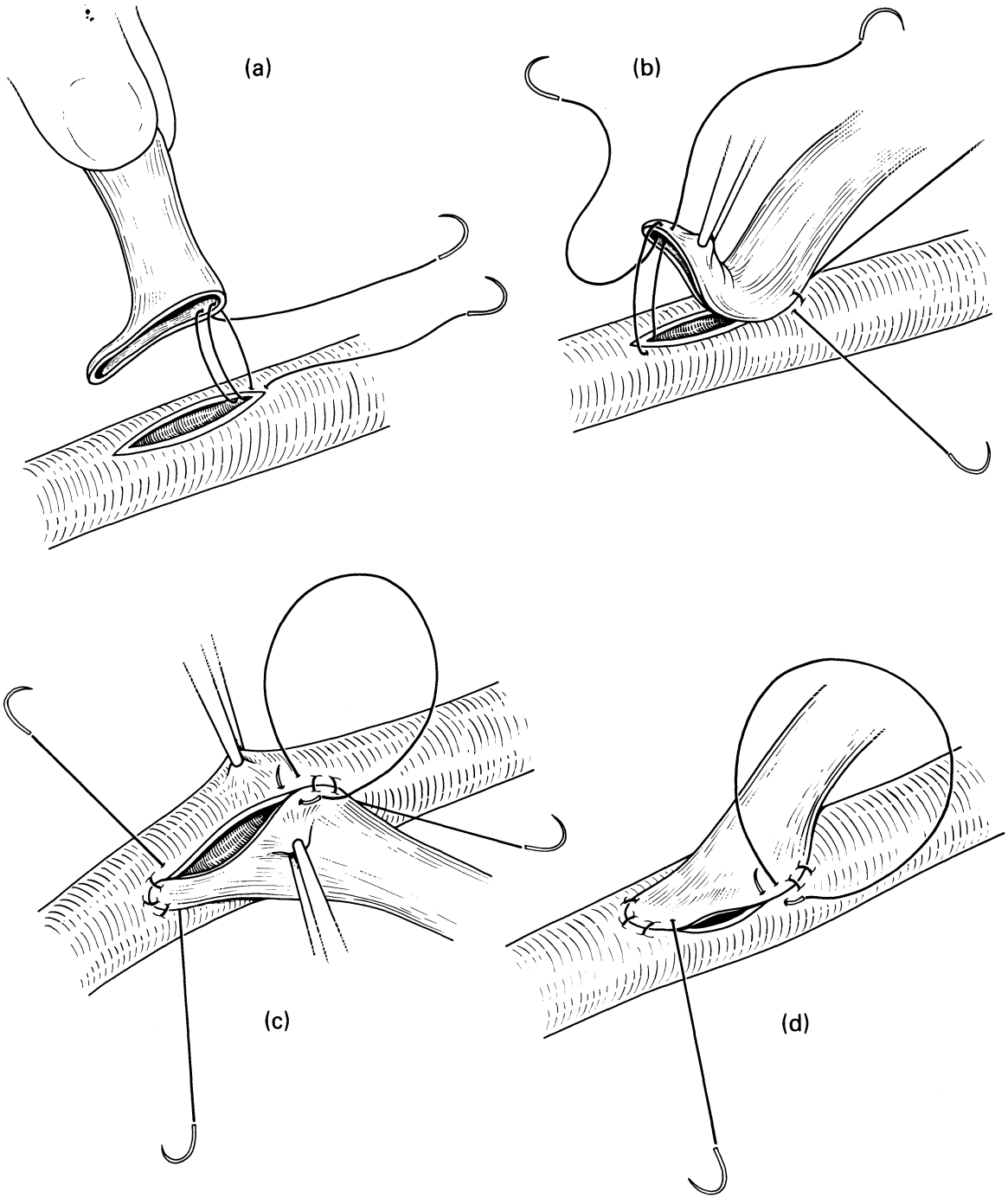


Figure 1.15(a–d) Vein graft–artery anastomosis: (a) suturing is commenced at the heel of the anastomosis. (b) the apex of the anastomosis is then similarly anchored. (c) needle passage from vein to artery with forceps holding the vein adventitia will avoid a rolling-in effect. (d) the nearside vein wall is then closed.

Intraluminal technique

Where access is limited e.g. vertebro-carotid reimplantation (see Chapter 10), end-to-side anastomosis may be achieved by sewing the least accessible side first using an intraluminal technique (Figure **1.16a**). Accurate suture placement may be facilitated by holding the two vessels (or graft and artery) apart initially and using a loosely looped running prolene suture along the back wall. This is then tightened and the two vessels approximated. Particular care is required when turning the corners to ensure that each suture loop falls correctly without inversion. (Figure **1.16b, c**). The anterior half of the anastomosis is then completed with either continuous or interrupted sutures according to vessel size.

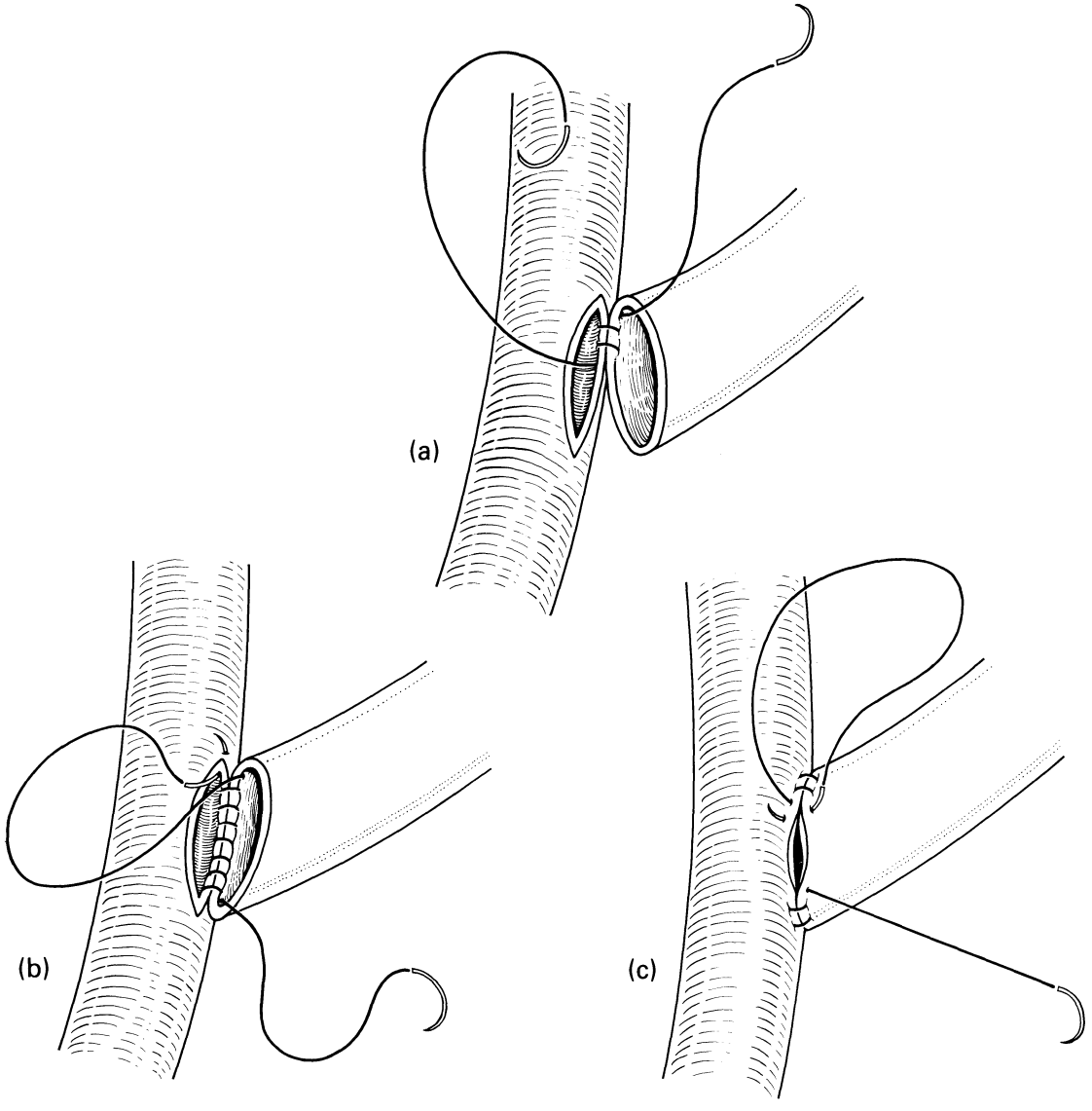


Figure 1.16(a–c) Intraluminal method of end-to-side anastomosis

PATCH GRAFT ANGIOPLASTY

Direct closure of a longitudinal arteriotomy inevitably produces some degree of constriction so that in most small or medium-sized vessels insertion of a patch graft is advisable (Figure **1,17a**). When the longitudinal arteriotomy has been used to perform a local endarterectomy, patching becomes particularly important both as a means of overpassing the distal intimal edge and as an aid in anchoring the intima[5] (Figure **1.17b**).

Patch graft angioplasty may also be used to correct an anastomotic or other local stenosis (Figure **1.17c**), to make good a deficiency in the artery wall, e.g. following trauma or excision of a saccular aneurysm (Figure **1.17d**), and to provide a platform for graft attachment to a sclerotic or degenerate artery wall[6] (Figure **1.17e**).

The Carrel button[7] used for reimplanting the inferior mesenteric and other arteries into the aorta or into an aortic prosthesis is a further specialized example of patch grafting. (See Chapters 2 and 14).

Autogenous saphenous vein is the preferred

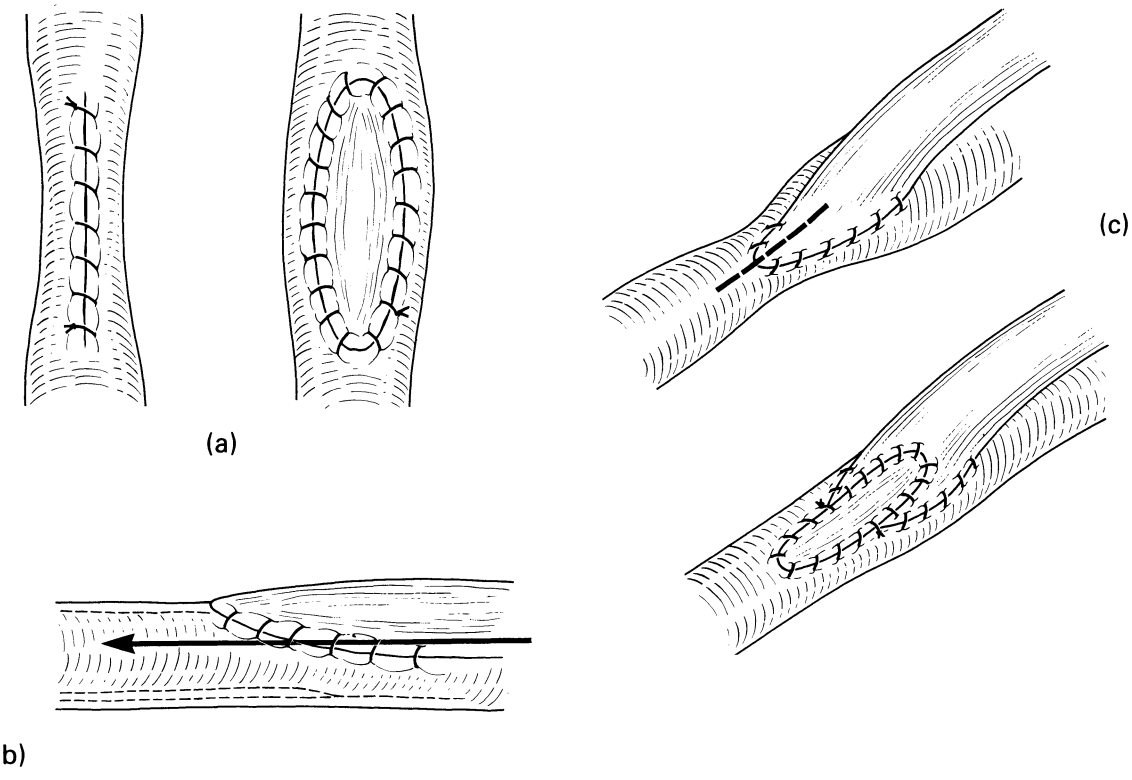


Figure 1.17(a–e) Some indications for patch graft angioplasty: (a) longitudinal arteriotomy closure. (b) overpass after endarterectomy. (c) anastomotic stenosis

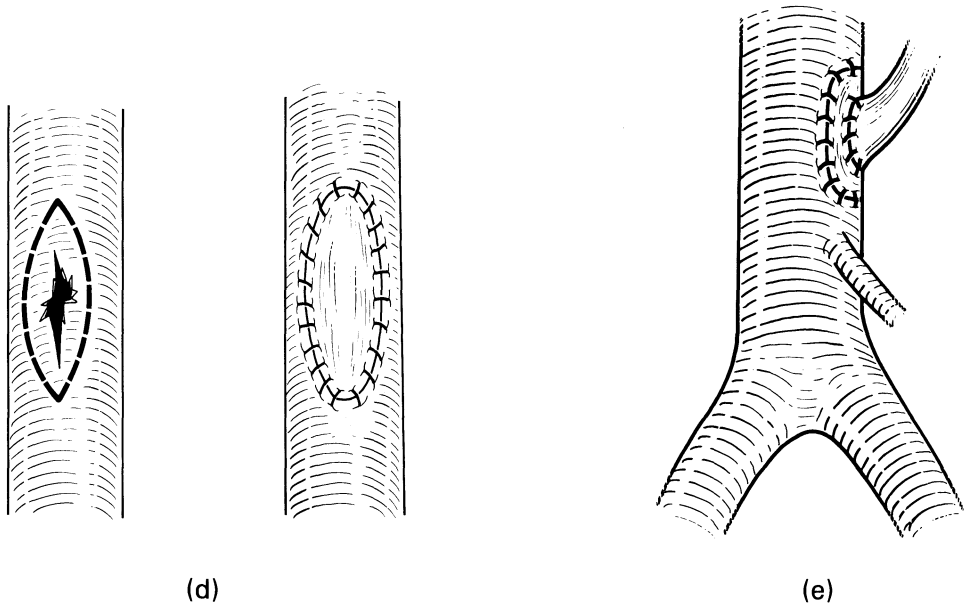


Figure 1.17 continued (d) partial excision of artery wall, e.g. for limited trauma. (e) platform for vein graft implantation, e.g. aortorenal bypass

material for patching most vessels. It should be harvested from the distal leg, thereby preserving the rest of the vein in case a subsequent coronary or femoro-distal bypass proves necessary. Usually only half the width of the vein is required. If too wide a patch is used dilatation will result, with a risk of mural thrombus deposition or frank aneurysmal change. Cephalic and jugular vein patches are in general not suitable, as they are less able to withstand arterial pressure on a long-term basis.

PTFE is an acceptable alternative patch material in most small or medium-sized vessels, while aortic patching can be accomplished with either Dacron or PTFE.

The technique of patch insertion is similar to that of an end-to-side vein graft anastomosis. Each end of the patch is anchored at the outset to produce sufficient longitudinal tension. The sides are then sewn in as previously described (Figure **1.18a–d**).

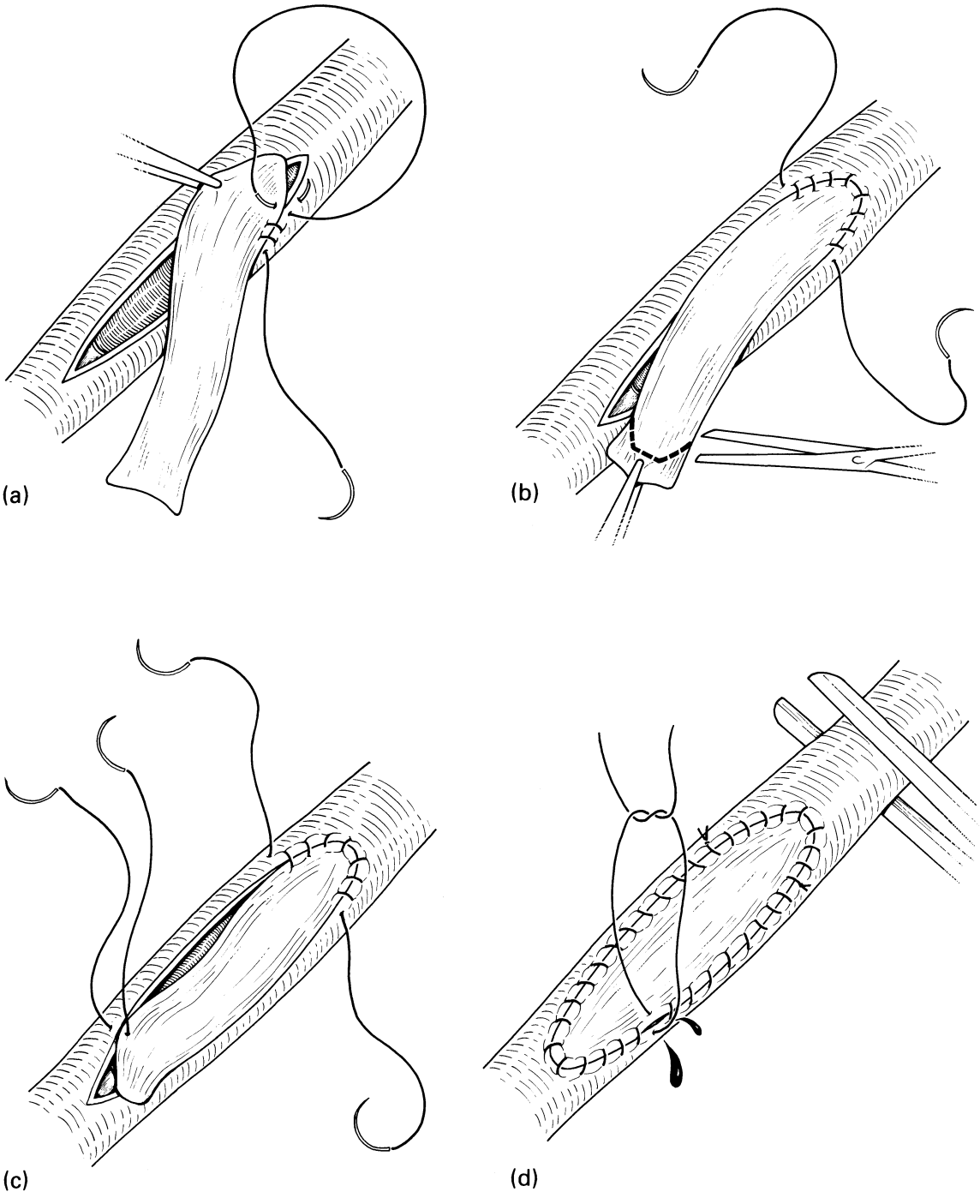


Figure 1.18(a–d) Technique of patch insertion

ENDARTERECTOMY

Atherosclerotic lesions involve the intima and a variable thickness of media and the principle of endarterectomy (or thromboendarterectomy if there is superimposed thrombus) is to develop a cleavage plane between the central atheromatous core and the undiseased outer part of the artery wall. The main possible cleavage planes are internal, trans-medial and external (Figure **1.19**). The first of these lies along the internal elastic membrane. In general this plane is insufficiently deep with a high incidence of recurrent atherosclerosis. The trans-medial plane leaves a smooth yellow, rather fragile interior surface. This is the preferred plane in the common–deep femoral segment, but it is a difficult plane to follow for any distance since the remaining fibres of the media are prone to scuffing and patchy erasure. The external plane removes all the media, leaving the adventitia lined by external elastic membrane. The resulting surface is smooth, shiny and reddish-brown in colour. In most vessels, e.g. the aorta, common iliac and internal carotid arteries, this is the best plane to follow. However, it is important not to stray into a deeper plane since this will result in a rough fragile surface which may leak or thrombose on restoring flow.

Advanced atherosclerotic lesions in elderly patients are usually the easiest to remove, since a satisfactory plane can almost always be developed even when the lesion is calcified. In contrast, the succulent adherent type of atheroma seen in younger patients and the inflammatory arteritides of the Takayasu's or Buerger's type are impossible to treat by endarterectomy since a satisfactory cleavage plane does not exist. Atherosclerotic degeneration with ectasia is an additional contraindication to endarterectomy since frank aneurysmal dilatation is likely on a long-term basis.

Several endarterectomy techniques are available:

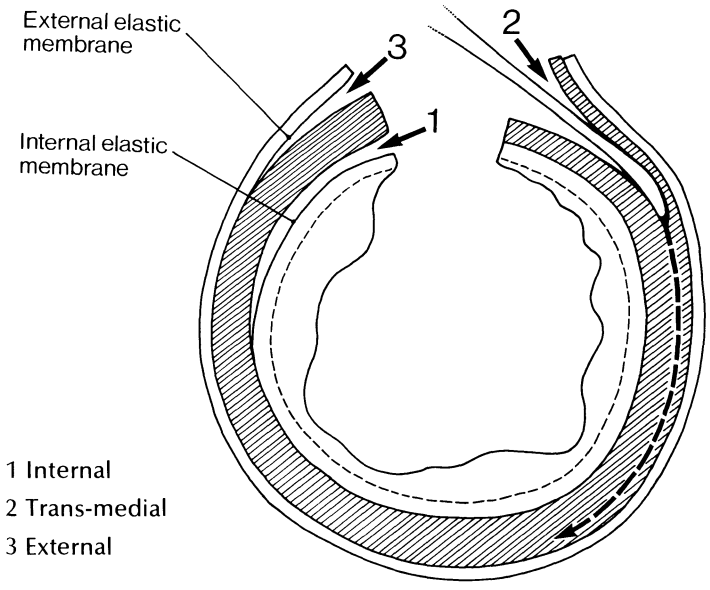


Figure 1.19 Endarterectomy planes

Open endarterectomy

This method is particularly suitable for dealing with short segmental lesions. A longitudinal arteriotomy is made over the diseased segment and a cleavage plane is developed using a spatula or blunt-nosed dissector (Figure **1.20a, b**). At the proximal limit, the lesion should be transected or, less preferably, fractured against the jaws of the controlling clamp (Figure **1.20c**).

The distal end point is best managed by gentle traction on the specimen and progressively more superficial use of the spatula. Ideally, the lesion should separate in a gently shelving tongue leaving an adherent intimal edge of minimal thickness. Where this cannot be achieved, the endarterectomy may be terminated by transecting with scissors and inserting tack-down stitches (Figure **1,20d, e**). Although these sutures should prevent a subsequent dissection, this is a less satisfactory method of termination. Commencing the endarterectomy at the distal limit rather than the mid-zone is an alternative which may avoid the problem of an overthick distal intimal edge (Figure **1.20f**).

Once the lesion and any additional debris have been removed, the lumen is well rinsed and the arteriotomy is closed with a patch (Figure **1.20g**).

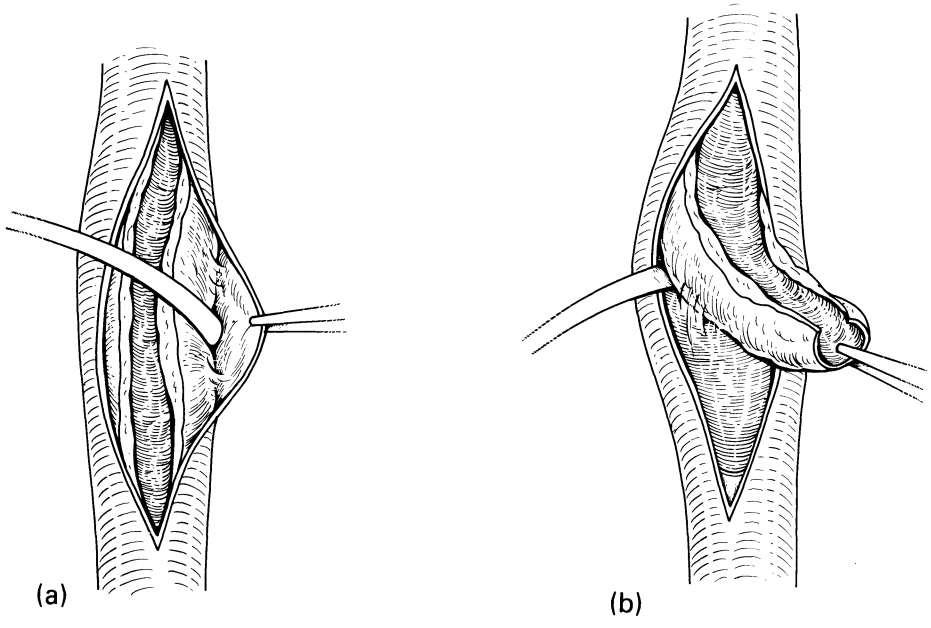


Figure 1.20(a–g) Technique of open endarterectomy

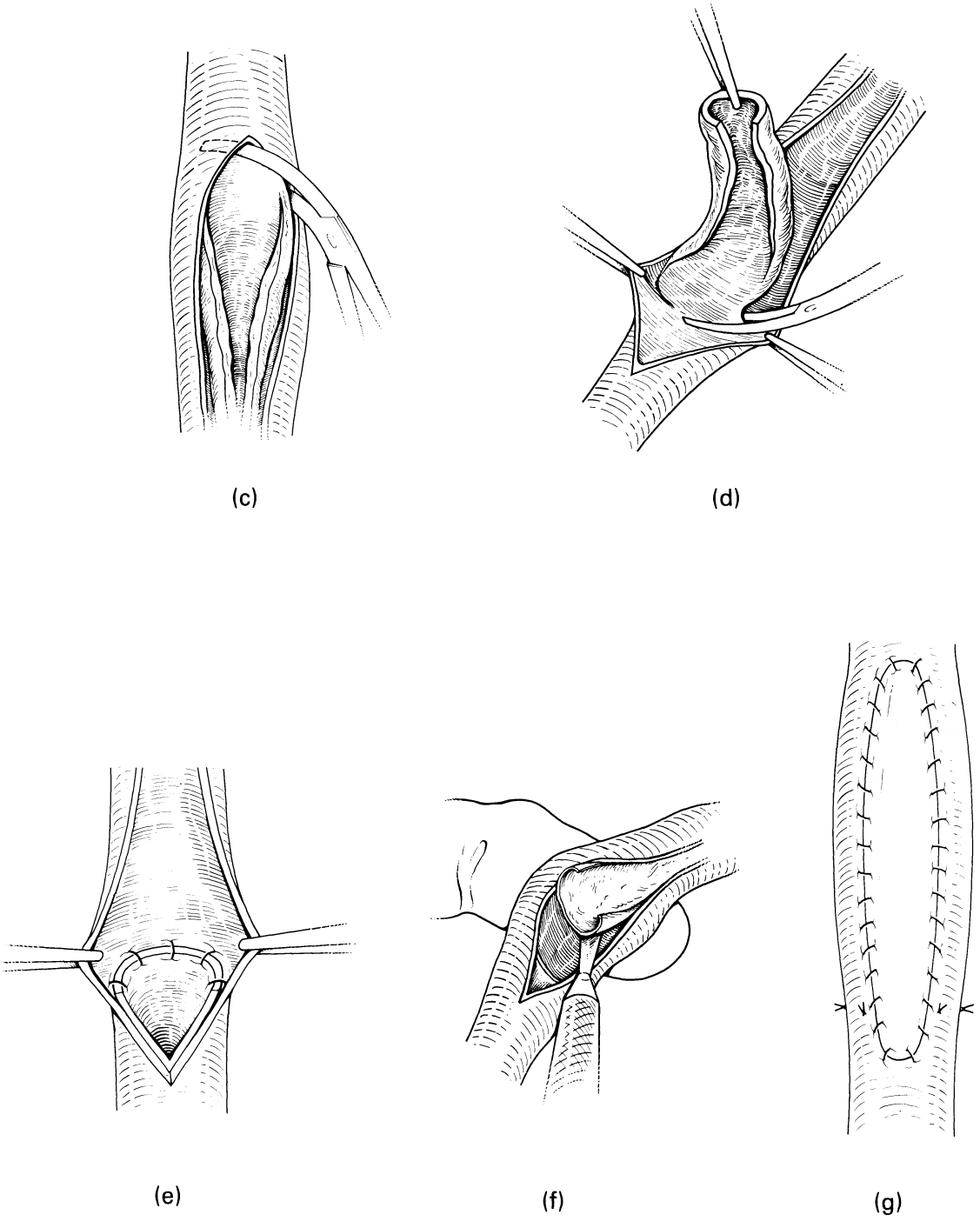


Figure 1.20 continued

Semi-closed endarterectomy

In this method the artery wall is left intact at the site of the occlusion and clearance is achieved by passing instruments between an arteriotomy at either end. At the distal arteriotomy the intima is transected and a core developed proximally. Heparin-saline injected between the core and the outer wall may assist this proximal separation (Figure **1.21a**). A Vollmar ring stripper of suitable size is then threaded over the distal end of the core and passed up the artery in a spiral manner while maintaining longitudinal tension on the artery (Figure **1.21b**). The dissected core is amputated at the proximal arteriotomy and retrieved along with the stripper from the distal end. The proximal and distal intimal edges are then trimmed and secured as necessary and the arteriotomies closed with a patch.

This semi-closed technique enables long arterial segments to be cleared rapidly. However, it is not without problems (Figure **1.22a–d**). Thus, the cleavage plane may be too superficial or too deep due to use of a wrong-sized stripper or there may be a change of plane induced by a calcific plaque. A cutting type of stripper may improve clearance of loose shreds of media but there may then be an increased risk of subadventitial dissection or perforation (Figure **1.22d**). Fogarty catheters and moist pledgets pulled through the endarterectomized segment may also be used to retrieve debris[8], but often doubts remain leading to the routine use of check endoscopy or arteriography in some centres[9]. An alternative, and in our view more certain, method of confirming adequacy of clearance is to turn the endarterectomized segment inside-out. Any retained fragments can then be removed under direct vision. (See Chapter 3.)

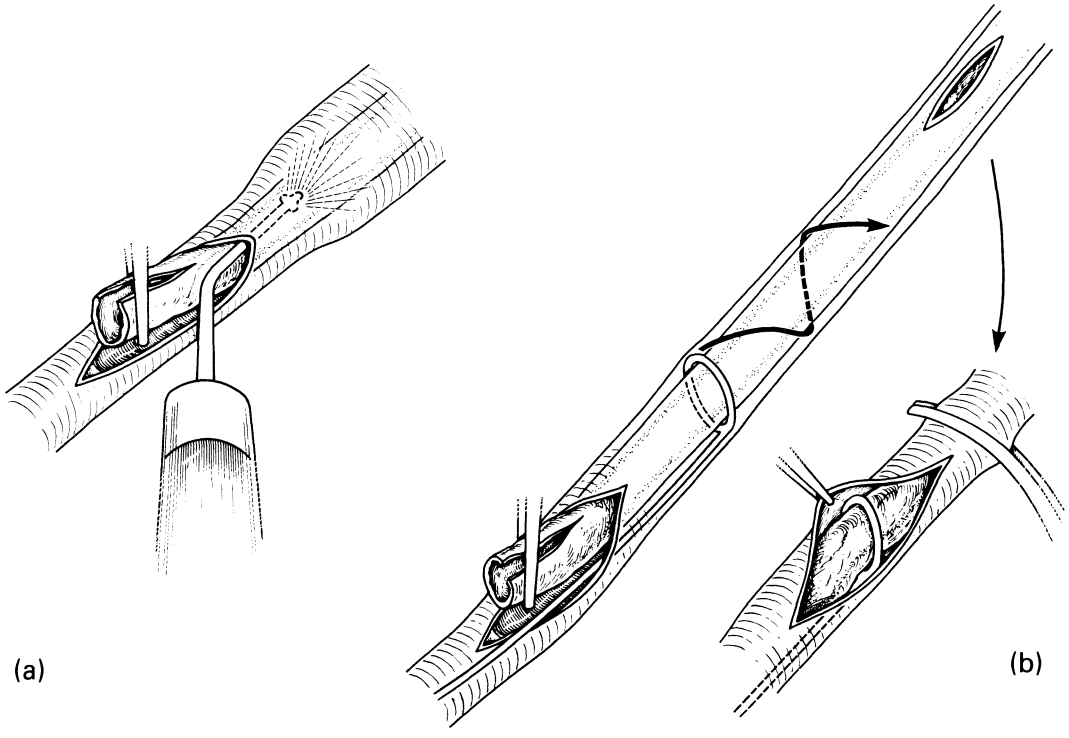


Figure 1.21(a, b) Semi-closed endarterectomy: (a) hydrodissection. (b) passage of a ring stripper with separation of the core up to the proximal arteriotomy

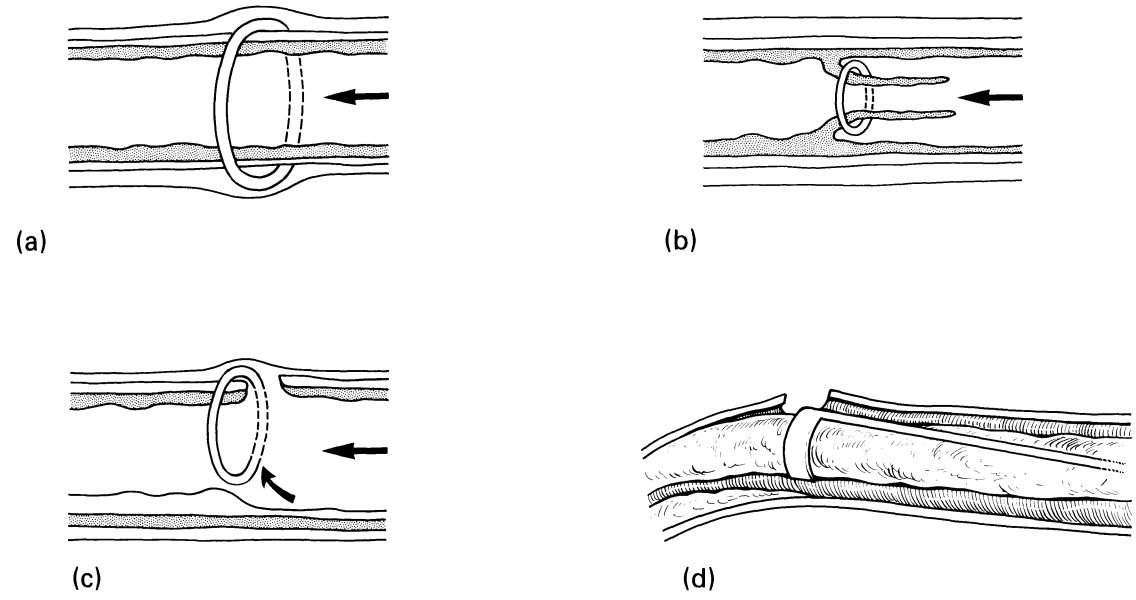


Figure 1.22(a–d) Some hazards of ring strippers: (a) cleavage plane too deep due to an overlarge stripper. (b) inadequate endarterectomy when the stripper is too small. (c) change of plane caused by an eccentric rigid plaque. (d) risk of perforation when using a cutting stripper.

Eversion endarterectomy

In the usual version of this method the artery is transected at the distal limit of the diseased segment and the outer layer of the artery wall is then peeled back from the central atheromatous core as far as the proximal clamp (Figure **1.23a**). The endarterectomy is terminated here with scissors or less preferably by fracturing the plaque against the clamp. The outer sleeve is then restored to its normal position and re-anastomosed distally (Figure **1.23b**). This re-anastomosis ensures good control of the distal intima as all layers of the wall are incorporated in the suture line. The main disadvantage of the above method relates to the proximal end point. Plaque removal may be incomplete here and rather than risk damage to the artery wall by further blind extraction a proximal arteriotomy may be required.

An eversion technique may also be used for dealing with an orifice lesion in a branch vessel. Examples include trans-subclavian vertebral endarterectomy (Figure **1.24a–c**) and transaortic visceral or renal artery endarterectomy (see Chapters 12 and 13). The main vessel is opened opposite the affected branch and an atheromatous core developed in a centripetal manner towards the branch orifice. By careful use of the spatula and gentle clamp approximation the branch vessel can be everted into the main artery lumen allowing separation of the plaque. Although control of the distal end point is not as good as with open endarterectomy, the method does avoid the necessity for an incision in the smaller branch vessel.

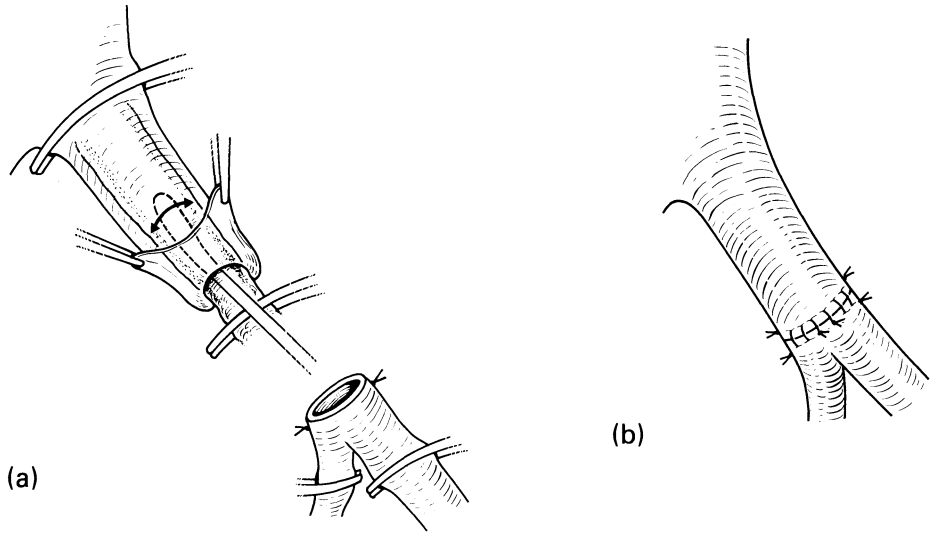


Figure 1.23(a–b) Eversion endarterectomy, e.g. left common iliac artery

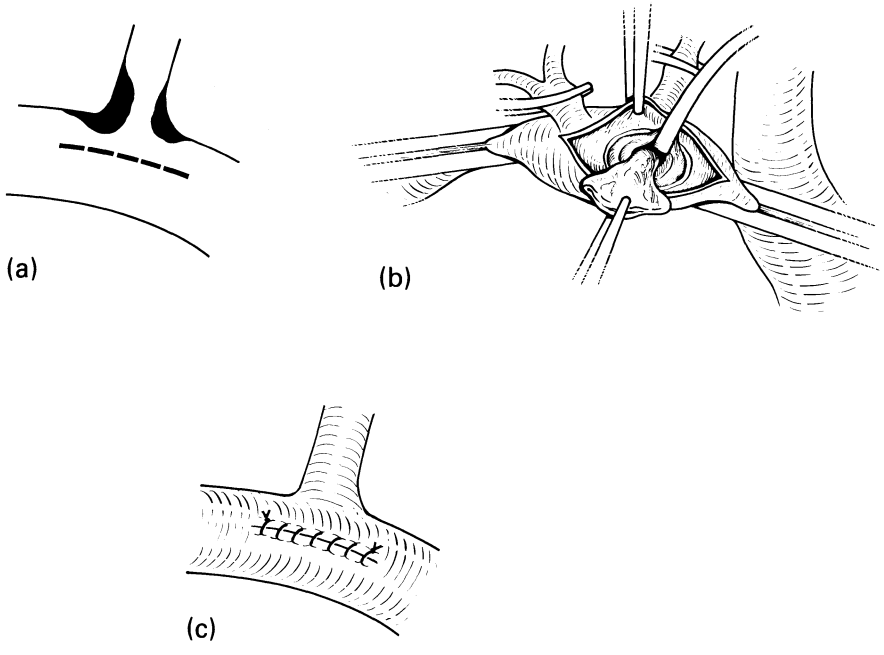


Figure 1.24(a–c) Trans-subclavian vertebral endarterectomy (right)

VASCULAR GRAFTS

There are several types of vascular substitute currently available. Dacron is the most widely used material for large vessel replacement particularly in the aortoiliac segment, supra-aortic trunks and visceral arteries, and in these locations it has proven long-term durability[10]. With the passage of time Dacron loses its flexibility and the pseudointima is prone to fracture at lines of stress across joints, so that is it not a suitable material for distal limb bypass. Difficulties may also arise in attempting to sew Dacron to a delicate vessel such as the endarterectomized profunda femoris artery and some form of supplementary autogenous reconstruction may be necessary at this level (see Chapter 3).

Dacron grafts may be either woven or knitted, the latter being available in jersey knit, warp knit and velour forms. Knitted velour grafts are generally preferable because of their superior incorporation into the tissues, their lack of fraying at the cut edge, better compliance and ease of suture passage. The main advantages of woven Dacron are that preclotting is unnecessary and blood loss through the graft is minimal. Similar low porosity may be achieved in the case of knitted grafts by collagen impregnation or gel coating.

Expanded polytetrafluoroethylene (PTFE) is a more recent alternative graft material. It appears to function well as a large vessel conduit and can be carried onto the profunda femoris with greater facility than Dacron. Results of above-knee femoropopliteal bypass have also been satisfactory though results of more distal bypass particularly at tibial level are inferior to autogenous vein[11]. PTFE grafts have no longitudinal elasticity and it is important to allow adequate length during implantation. In addition, fine sutures must be used to minimize stitch-hole bleeding.

Autogenous saphenous vein remains the material of choice for most small vessel replacement because of its pliability, ease of tailoring and amenability to fine suturing. Particular care is required in harvesting the vein when it is to be used as a free reversed graft (see Chapter 5). *In situ* usage is an alternative possibility in femoro–distal reconstructions. Although the saphenous vein is the best available small vessel conduit it is subject to structural change in a small but significant number of cases and some of these may lead to occlusion of the graft[12]. Arm veins have also been used in the past, but their thinner wall makes them generally unsuitable.

Autogenous artery may serve as a vascular conduit in special circumstances[13]. Examples include the use of the internal iliac artery for *ex vivo* or paediatric renal artery reconstruction (see Chapter 13). In the former case branch artery repair is facilitated by the natural branching arrangement of the internal iliac artery while in the latter group the autograft grows with the child and behaves as a normal artery.

Autogenous material is resistant to infection and an additional use for arterial autografts is in restoring vascular continuity in an infected field[14]. Many of these cases can be managed by extra-anatomic bypass, but as an alternative one or both previously occluded superficial femoral arteries can be harvested and used as an *in situ* conduit after eversion endarterectomy (see Chapter 14).

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2

Aneurysms of the Abdominal Aorta

Abdominal aortic aneurysms are associated with a high incidence of serious or fatal complications of which rupture is by far the most important[1,2]. The risk of rupture is difficult to predict on an individual basis for, although patients with large (>6 cm diameter) symptomatic aneurysms are at greatest risk[3], even small silent aneurysms have an appreciable rupture rate[4]. Hypertension increases the likelihood of rupture for any given size of aneurysm[3]. If operation can be undertaken electively the operative mortality rate is now very low indeed (2–3%), whereas once rupture has occurred this figure rises steeply and may exceed 80% for patients in shock[5]. In view of these features, operation is advisable in all cases. The only exceptions to this rule are patients with limited life expectancy due to widespread malignancy, severe cardiac or respiratory disease, renal failure etc. Even among poor risk patients it may be worth wiring or externally reinforcing the aneurysm[6] or inducing thrombosis by iliac ligation and then adding an axillobifemoral bypass[7,8]. It should be noted that age itself is not a contraindication to surgery, since many patients who survive into their seventh or eighth decades are in better physical condition than their younger counterparts.

PREOPERATIVE ASSESSMENT

Ultrasound or CT scanning may assist diagnosis when there is doubt whether or not an aneurysm is present. CT scanning in particular will provide additional information on the extent of the aneurysm and the presence of intraluminal thrombus and may help to distinguish between an intact symptomatic aneurysm and one in which localized leakage has already occurred[9,10]. Arteriography should also be undertaken in all elective cases to allow proper surgical planning. Features of surgical consequence which may be revealed by aortography include accessory renal arteries, renal artery stenosis, visceral arterial disease with secondary collateral routes in the mesentery, and additional occlusive or aneurysmal disease in the internal iliac and run-off vessels. Aortography may also reveal extensive intimal degeneration in the juxtarenal aorta precluding safe infrarenal clamping, and it may provide the first evidence of suprarenal extension of the aneurysmal process.

Coronary artery disease is a major determinant of early and late mortality after arterial reconstruction[11] and such disease should be carefully assessed before surgery. Routine coronary angiography has been adopted prior to aortic resection in some centres with prophylactic coronary artery bypass grafting when severe operable disease is encountered[12]. However, such a policy may have only a limited impact on immediate operative mortality, which has now been reduced to a very low level mainly as a result of improvements in perioperative monitoring and regulation of cardiac haemodynamics[13,14]. Indeed, the operative mortality for coronary artery revascularization

may exceed that for the aortic procedure, at least in the older age group[15,16]. At the present time preaortic coronary angiography would appear to be best reserved for patients with unstable angina or angina at rest and those with a markedly positive ECG stress test[17] or an abnormal radionuclide study[18,19]. If operable coronary artery lesions are then identified, coronary artery bypass grafting or percutaneous angioplasty may be undertaken prior to aortic resection unless the aneurysmal disease is symptomatic in which case, simultaneous correction of the coronary and aortic lesions is advisable[20,21].

Although coronary revascularization may be restricted to selected patients preoperatively, it is likely to have a much greater impact on long-term survival after aortic surgery and patients with clinically significant ischaemic heart disease should be carefully assessed post aortic resection with this in mind.

Extracranial cerebrovascular disease should also be evaluated prior to aortic surgery. This may include the use of digital subtraction angiography or duplex scanning of the carotid bifurcation (see Chapter 9). Those patients who have symptomatic carotid disease or positive screening data should undergo appropriate catheter studies at the time of aortography. Significant ulcerative or stenotic lesions are then corrected, usually before but occasionally at the time of aortic surgery[22].

All patients scheduled for aortic surgery undergo an additional battery of tests which include chest X-ray, ECG, full blood count, coagulation screen, blood urea, creatinine, fasting blood sugar, lipid profile and urinalysis. Pulmonary and renal function studies may also be undertaken in appropriate cases.

PREOPERATIVE PREPARATION

Medical risk factors, such as hypertension, diabetes or congestive failure should be controlled as far as possible prior to surgery. Patients with chronic obstructive airways disease may be improved by physiotherapy, antibiotics and bronchodilators. All patients should stop smoking well in advance of operation. Adequate preoperative hydration is essential in achieving good renal and cardiac function and 1 litre of Ringer's lactate with 500 ml colloid is usually given over a 12 hour period prior to surgery. Antibiotics active against staphylococci, coliforms and anaerobes are started as a routine with the premedication and continued until all lines and catheters have been removed. Subcutaneous heparin is also commenced preoperatively and is continued until the patient is fully mobile.

INTRAOPERATIVE SUPPORT MEASURES

After induction of anaesthesia, radial artery, central venous pressure and peripheral venous lines are inserted and an ECG monitor connected. A self-retaining catheter is inserted into the bladder and a nasogastric tube into the stomach. A Swan–Ganz catheter is employed in patients with angina, previous infarction or hypertension particularly if there is documented left ventricular dysfunction[19], and whenever supracoeliac clamping or additional visceral or renal artery revascularization is envisaged. Ideally this should be inserted on the day before surgery and myocardial performance curves constructed[13]. Intraoperative fluid therapy can then be adjusted to maintain the pulmonary capillary wedge pressure and cardiac index at optimal levels. Vasodilator therapy may also be needed to control the effects of aortic cross-clamping, though this is not usually a problem at infrarenal level. Apart from haemodynamic monitoring, transoesophageal echocardiography may also be used and may be a particularly sensitive index of intraoperative cardiac function[23].

Renal function may also be best protected by ensuring adequate left heart filling pressures[24] and these are a better guide than intraoperative urinary output[25]. If volume replacement is adequate and urinary output remains low, a renal-dose dopamine infusion may be utilized in preference to mannitol[26].

OPERATIVE TECHNIQUE

The patient is placed supine on a table furnished with a warming pad. Uprights for attaching a sternal retractor may be added at the head end of the table in case access to the supracoeliac aorta is required. The operative field is then prepped and draped to include both groins.

The abdomen is opened through a midline xiphopubic incision (Figure **2.1**). A retroperitoneal flank approach has been advocated by some authors[27], but this may provide inadequate exposure if the aneurysm is large or if an unanticipated problem is encountered. After preliminary exploration of the abdomen, the transverse colon and omentum are reflected superiorly and the small bowel displaced over the right-hand wound margin. Intestinal bags tend to promote fluid losses and it is usually preferable to cover the bowel with warm moist packs. A small tray abutting on the lateral abdominal wall adjacent to the wound may be used to support the displaced intestines and relieve undue drag on the small bowel mesentery.

The posterior peritoneum is incised at the left border of the fourth part of the duodenum and this incision is extended distally towards the aortic bifurcation (Figure **2.2**). The ligament of Treitz is divided and the duodenojejunal flexure displaced to the right with the rest of the small bowel.

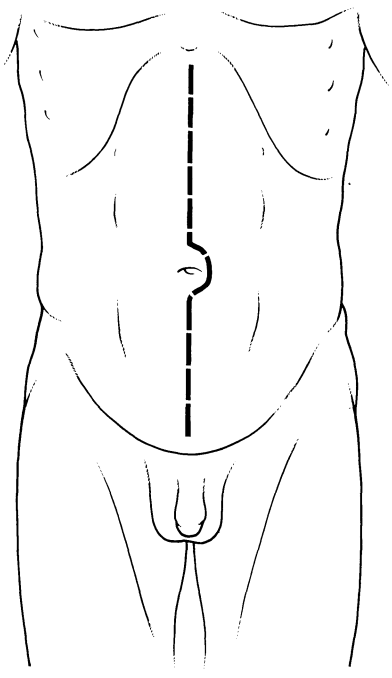


Figure 2.1 Midline xiphopubic approach

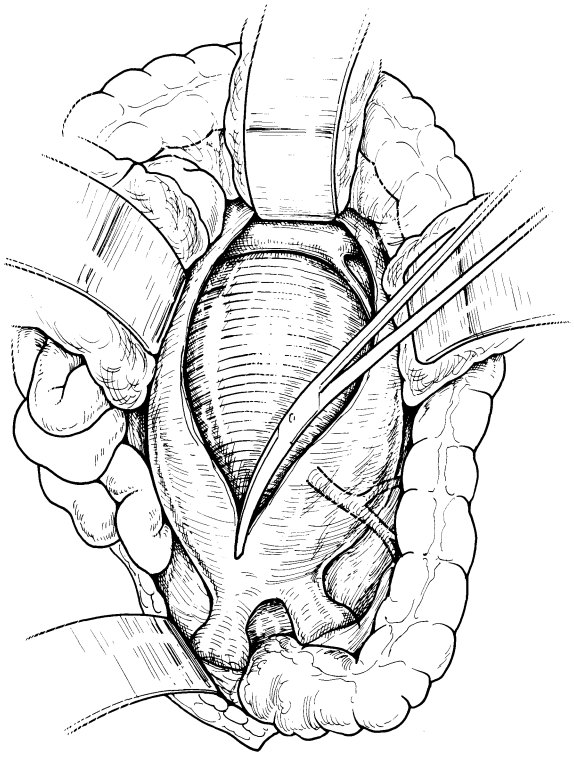


Figure 2.2 Incision of the posterior peritoneum to expose the aneurysm

The inferior mesenteric artery may then be palpated at the left border of the aneurysm and the presence or absence of pulsatile flow confirmed. If patent, the vessel is encircled with a Silastic loop distal to the aneurysm wall, leaving the proximal segment and its investing autonomic nerve fibres undisturbed.

A separate peritoneal incision is then made over the distal common iliac artery on either side. Access to the left common iliac bifurcation may require medial reflection of the sigmoid colon if the mesentery is short. In this case a tunnel is developed behind the root of the mesocolon and left ureter connecting the aortic and iliac exposures (Figure **2.3**). The external and internal iliac arteries are then cleared sufficiently to allow subsequent clamp placement. The proximal common iliac arteries should remain largely undissected in order to minimize the risk of iliac vein injury and to avoid disrupting the hypogastric nerve plexus. In the presence of a large common iliac aneurysm, only the external iliac artery should be mobilized. The graft is attached here and the internal iliac origin sewn off from within the aneurysmal sac. Similarly if the common iliac arteries are surrounded by a dense periarterial reaction, an external iliac graft attachment with oversewing of the common iliac origin will avoid a potentially hazardous dissection (see Figure **2.7c**).

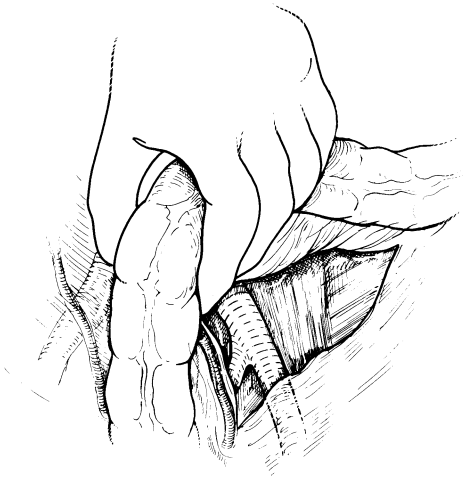


Figure 2.3 Left common iliac bifurcation exposed distal to the root of the sigmoid mesocolon

The final stage involves dissection of the proximal aorta up to the level of the renal arteries. The left edge of the initial peritoneal incision is retracted laterally and the transverse mesocolon displaced superiorly to bring the left renal vein into view. The inferior mesenteric vein adjacent to the cut peritoneal edge can usually be preserved, but if access is limited it should be divided. The left renal vein is then gently mobilized and retracted superiorly using a Silastic loop. If necessary its tributaries may be divided to improve mobility (see Chapter 13). Alternatively, if the aneurysm seems unduly high and the left renal vein is stretched tightly over the aneurysm neck, the vein may be transected to the right of the midline. This gives excellent access to the inter-renal aorta (see Figures **2.19, 20**). The vein can then be re-anastomosed at the end of the procedure. Although some authors have deemed this latter step unnecessary, restoration of renal vein patency may help avoid postoperative renal dysfunction[28].

Sometimes the aneurysm appears to involve the renal arteries. Nearly always this is not the case and the appearance is due to aortic tortuosity. Once the sides of the aorta have been cleared, gentle caudal traction on the sac will straighten out the curve and allow correct clamp placement (Figure **2.4**). During clearance of the front and sides of the proximal aorta several small branches including the gonadal arteries may be encountered and these should be ligated and divided. If a lower pole renal artery is identified arising from the aneurysm, it should be carefully preserved for subsequent attachment to the aortic prosthesis.

Complete encirclement of the proximal aorta is often advocated, followed by passage of a sling or tape. However, if the controlling clamp is applied vertically, only the front and sides of the aorta need to be mobilized. This technique is not only quicker, but avoids the risk of damaging a lumbar artery or vein and of embolizing loose debris from within the aortic lumen.

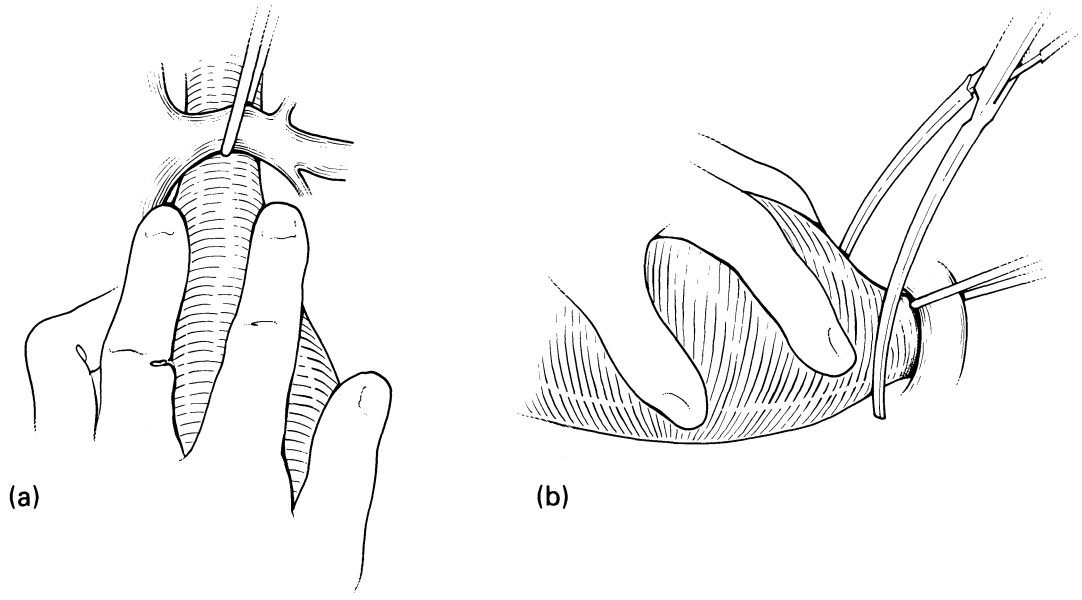


Figure 2.4(a, b) Digital dissection of aneurysm neck prior to clamping

Bifurcated graft insertion

The most common replacement technique in elective cases involves the use of a bifurcated graft. An appropriately sized knitted Dacron graft is selected and pre-clotted using blood withdrawn from the inferior vena cava. If a PTFE or collagen impregnated Dacron prosthesis is being utilized, preclotting is unnecessary. The graft should be cut with a short stem in order to avoid possible kinking of the iliac limbs. It is also important to avoid using a graft with overlarge limbs because these will become lined with mural thrombus which may detach or produce complete occlusion. Any discrepancy at the aortic end can be made up by cutting the graft stem in an S-shaped curve, thereby enlarging the anastomotic circumference.

Heparin (1 mg/kg body weight) is then given intravenously by the anaesthetist or by the operator directly into the vena cava and after a few minutes clamps are applied to the external and internal iliac arteries and then to the aorta above the aneurysm. Distal control is secured before occluding the aorta in order to reduce the risk of embolization[29]. After controlling the inferior mesenteric artery, the aneurysm is opened longitudinally along the right anterolateral wall (Figure **2.5a**). Distally the incision should stop short of the aortic bifurcation. Intraluminal thrombus is evacuated and any open lumbar arteries are sewn off from within the sac (Figure **2.5b**). This may require prior removal of rigid plaque from around the vessel origins.

Palpation within the proximal aortic lumen will usually reveal a ring marking the site at which the aneurysm commences. The sac should now be opened to this level and a T-shaped extension made on either side, leaving the posterior wall intact. Any loose intimal debris should be removed by thorough irrigation of the proximal stump.

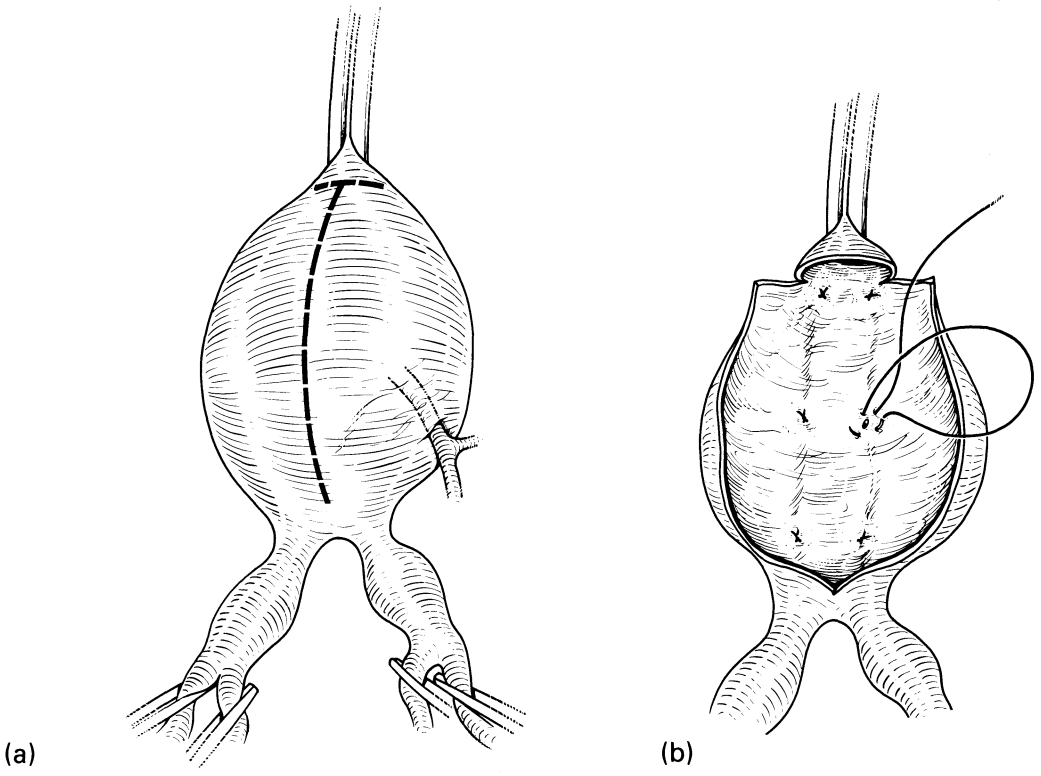


Figure 2.5(a, b) (a) Aortotomy. (b) oversewing of lumbar arteries

If the wall is unusually degenerate at this site it may be preferable to completely transect the aorta more proximally and perform a direct end-to-end anastomosis, if necessary under supracoeliac control. However, in most cases the intrasaccular method is possible and preferable.

Suturing commences at the far corner and the suture line is run towards the surgeon, taking double-thickness bites of the aortic wall (Figure **2.6a**). Double-armed 3/0 prolene on a taper cut needle is suitable for this purpose. The corners are turned and the anterior half of the anastomosis completed, again taking deep bites of the aortic wall. A rubber-shod clamp is then placed across the graft immediately distal to the anastomosis and after releasing the initial clamp the suture arms are tied together (Figure **2.6d**). Ideally once the aortic clamp has been released it should not be reapplied because of the risk of fragmenting the atherosclerotic intima. If supplementary sutures are required one solution is to insert them on Teflon or Dacron pledgets without reclamping (see Chapter 1).

Attention is now turned to distal implantation. If preservation of internal iliac perfusion appears problematical on one side because of aneurysmal extension into the common iliac bifurcation or atherosclerotic thickening, the easier side should be dealt with first. This will keep available the option of internal iliac ligation on the difficult side.

The appropriate graft limb is thoroughly aspirated and drawn down behind the peritoneum to the common iliac bifurcation. Distally the graft may be attached by a similar endosaccular technique, or by a direct end-to-end anastomosis to

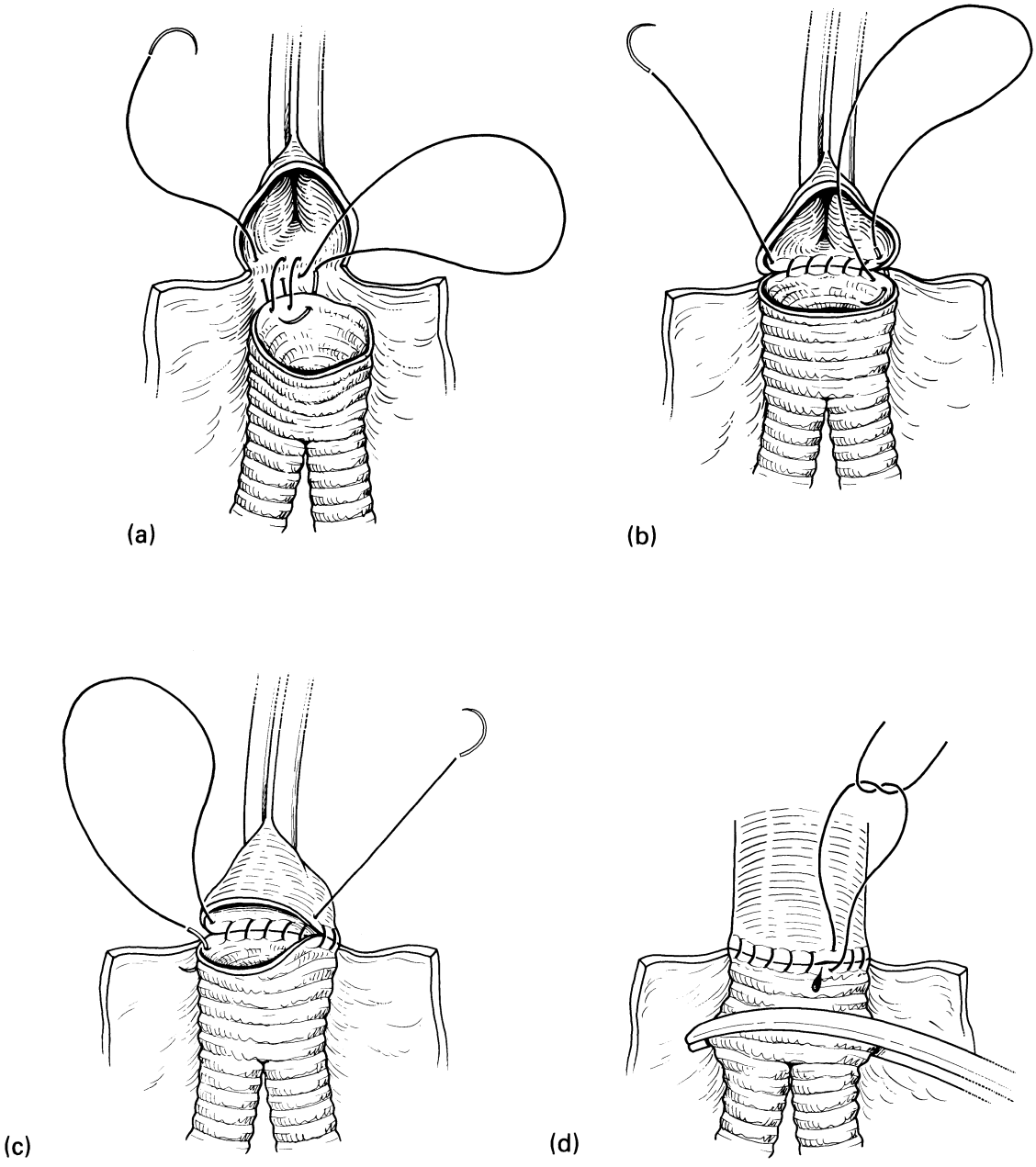


Figure 2.6(a–d) Proximal anastomosis using inlay technique

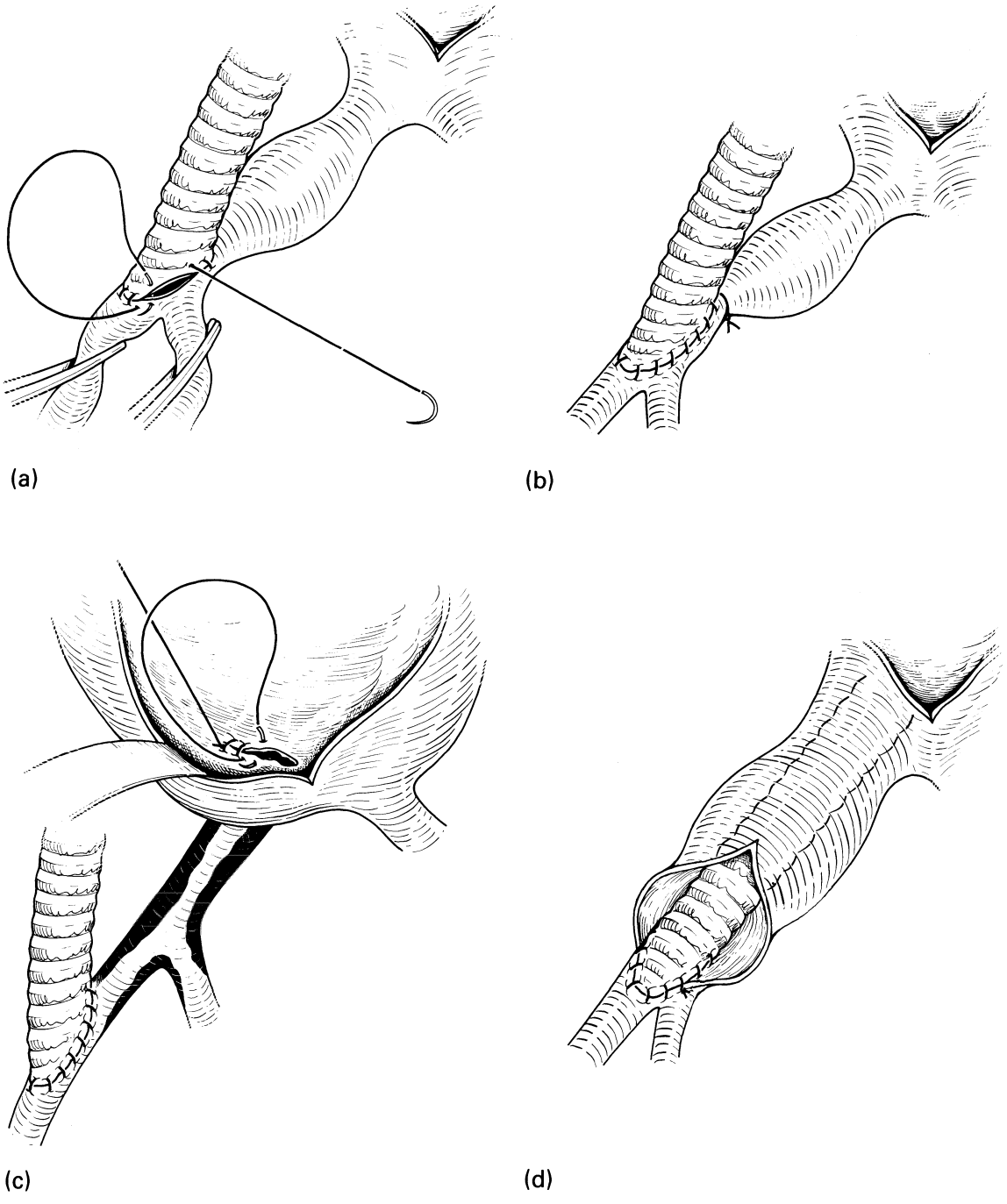


Figure 2.7(a–d) (a, b) Preferred method of distal anastomosis. (c) alternative when the common iliac artery is calcific or adherent. (d) graft limb tunnelled within iliac aneurysm with distal inlay anastomosis

the transected distal common iliac artery. However, the former requires laying open the iliac arteries which inevitably disrupts the hypogastric plexus, while the latter method may run into difficulties if the iliac artery is calcific or degenerate. In many patients, therefore, the most satisfactory method of attachment is by end-to-side anastomosis to the common iliac bifurcation or external iliac artery. A proximal ligature is then added, preserving internal iliac perfusion (Figure **2.7a, b**). If the distal common iliac artery is heavily calcified, it may be necessary to extract the rigid intima before this ligature can be tied. Alternatively, the common iliac origin may be oversewn from within the aorta (Figure **2.7c**).

A further option if the common iliac artery is particularly dilated is to tunnel the graft limb within the intact iliac segment for distal inlay anastomosis. This technique should also help avoid hypogastric nerve damage (Figure **2.7d**).

Before the iliac anastomosis is completed, the distal and proximal clamps are flushed, the latter via the unsutured limb of the graft. The suture line is then completed and flow released, first into the internal iliac artery and then after a minute or two into the external iliac artery. This staged declamping will avoid possible distal microembolization (trash foot), and if preceded by an intravenous fluid load will also prevent a fall in systemic arterial pressure.

An appropriate anastomosis site is then selected on the opposite side and the remaining graft limb aspirated, cut to the correct length and attached using one of the above techniques. A staged release of the distal clamps is again performed.

After the graft has been inserted bleeding sometimes starts up from previously dry lumbar or median sacral arteries or from the edges of the aneurysm sac and extra sutures should now be inserted. If operating time has been short, the heparin may be reversed with protamine, but usually its effect is allowed merely to wear off. Once haemostasis is secured the sac is wrapped around the graft to obliterate the dead space and prevent a peri-graft haematoma. Each iliac limb may be wrapped separately (Figure **2.8**), taking care to avoid placing sutures through the nerve plexus on the left anterolateral aortic wall.

The left edge of the peritoneal opening is then sutured to connective tissue at the right side of the aorta and prosthesis. The duodenum thus remains displaced away from the suture line as an intraperitoneal structure. If cover is deficient, a flap of omentum should be brought down over the graft and sac and extended into the pelvis. The small bowel and colon are replaced and the abdomen closed with suction drainage.

Postoperatively, the patient may be electively ventilated for 24–48 hours if the procedure has been unusually lengthy or complicated. In most cases this is unnecessary.

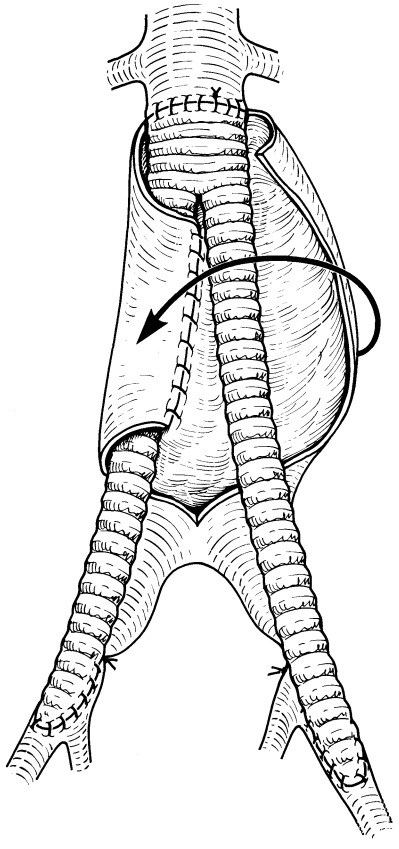


Figure 2.8 Closure of aneurysm sac around prosthesis

Tube graft insertion

Although a bifurcated graft is the most frequent replacement technique, a straight tube graft may be utilized when the common iliac arteries are free from gross aneurysmal or occlusive disease and the aortic bifurcation is suitable for graft attachment. This type of graft may have particular advantages in the ruptured case and in patients with a poor run-off, e.g. diffuse ilioprofunda disease and bilateral superficial femoral artery occlusion.

After applying clamps to the distal common iliac arteries and to the proximal aorta, the aneurysm is opened with a T-shaped extension at either end. A tube graft is then inserted using an inlay technique. At the distal end, graft attachment may be difficult due to calcification and reverse needle passage should be utilized to avoid elevating plaque (Figure **2.9a**). It is also helpful to use the needle point to probe the aortic wall in order to find a soft area that permits suture passage. Often the aorta is wider here than at the proximal anastomosis and a certain amount of gathering-in may be necessary during suture placement. If the aorta is particularly degenerate or calcific a Teflon or Dacron strip may be incorporated into the suture line (see Figure **3.7**).

Once the suture line is completed, flow is released, first into the internal iliac artery and then the external iliac artery on either side (Figure **2.9b**). Redundant aneurysm sac should not be excised but should be imbricated in two layers over the graft, thereby preserving the autonomic plexus (Figure **2.10a, b**).

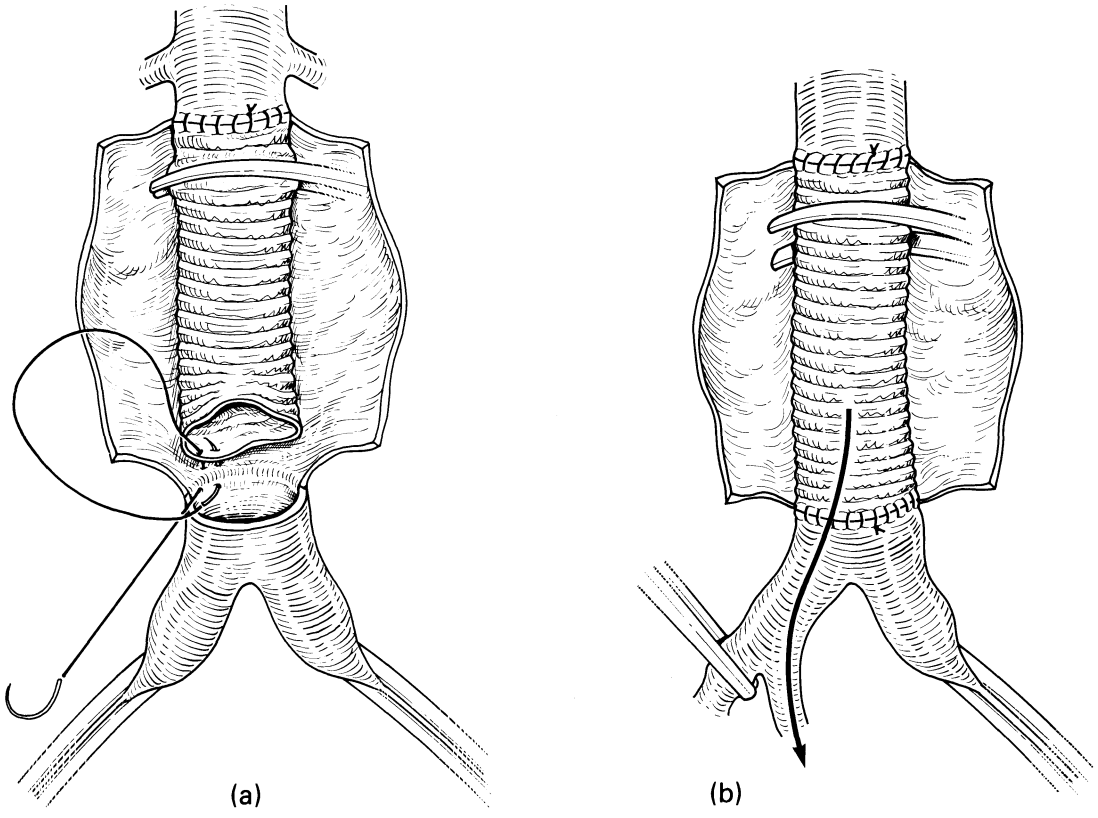
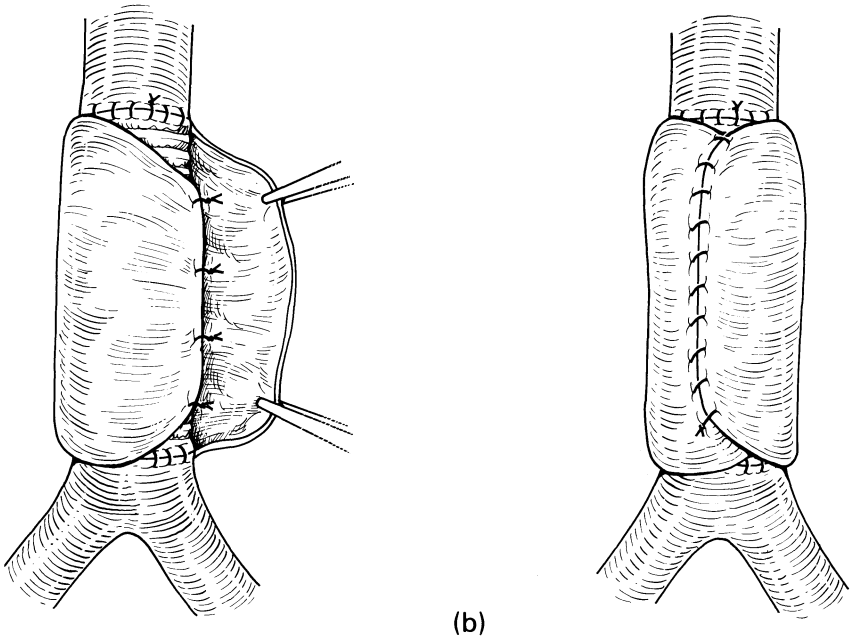


Figure 2.9(a, b) Technique of tube graft insertion: (a) inlay anastomoses. (b) staged distal declamping



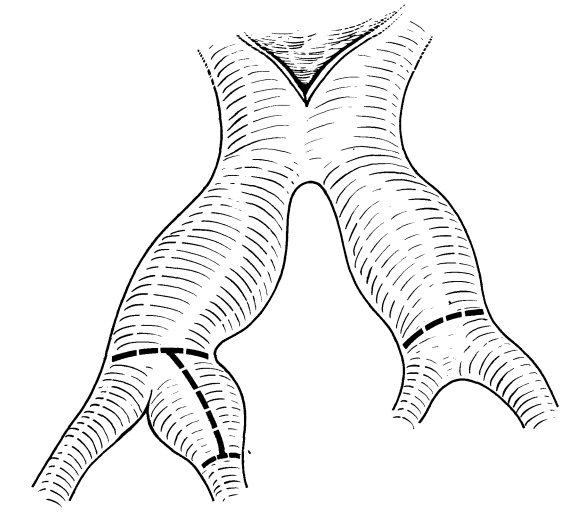
(a) (b)

Figure 2.10(a, b) Two-layer imbrication of redundant aneurysm sac around graft

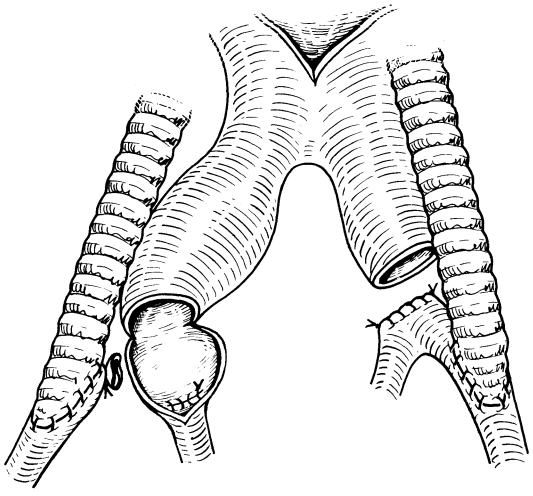
Internal iliac aneurysms

Aneurysms of the internal iliac arteries may occur in association with similar disease in the aortoiliac segment or very occasionally as an isolated event[30,31]. In the past, internal iliac aneurysms have been treated by ligation of the aneurysm neck. However this may not be sufficient since the sac may continue to enlarge and complete exclusion is advisable[30]. Distal control may be difficult, as these lesions lie deep within the pelvis, and the safest method of management is to open the sac and to sew off the outflow orifices from within, using digital or balloon control (Figure **2.11a, b**).

Internal iliac exclusion is quite safe if back-bleeding is profuse, as is usually the case if the opposite internal iliac artery has been preserved. Even when both internal iliac arteries are excluded and the inferior mesenteric artery has been sewn off or is occluded, the colon may still remain viable as a result of collateral flow from the superior mesenteric artery. However, this is not invariably the case, and if internal iliac back-bleeding is sparse, the inferior mesenteric artery should be reimplanted or an attempt made to revascularize one or other of the internal iliac arteries (Figure **2.12**).



(a)



(b)

Figure 2.11(a, b) Internal iliac aneurysm treated by endosaccular exclusion

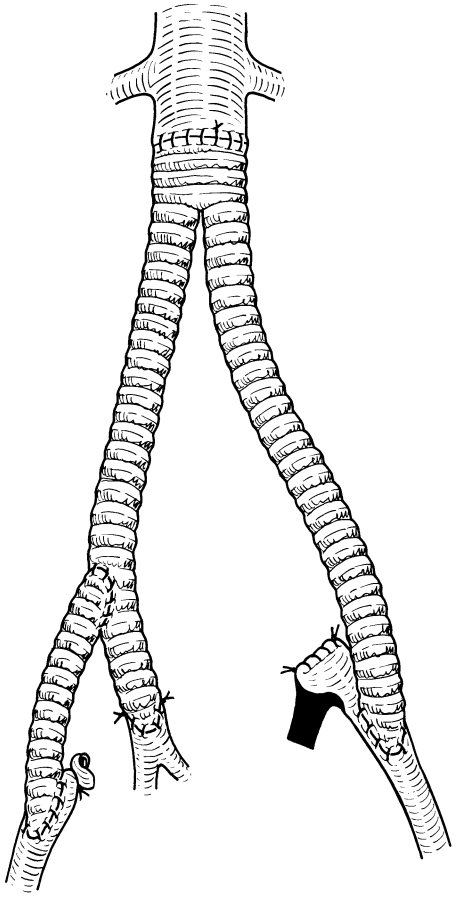


Figure 2.12 A method of revascularizing the internal iliac artery

Iliofemoral occlusive disease

Coexistent occlusive disease in the iliofemoral axis may necessitate extending one or both graft limbs to the groin (Figure **2.13a**). In most of these cases the superficial femoral artery will be occluded, so that the graft is usually attached to the common-deep femoral segment (see Chapter 3). Where retrograde filling of the internal iliac arteries cannot be assured, a compromise arrangement may be necessary in which some aneurysmal tissue remains in circuit (Figure **2.13b**).

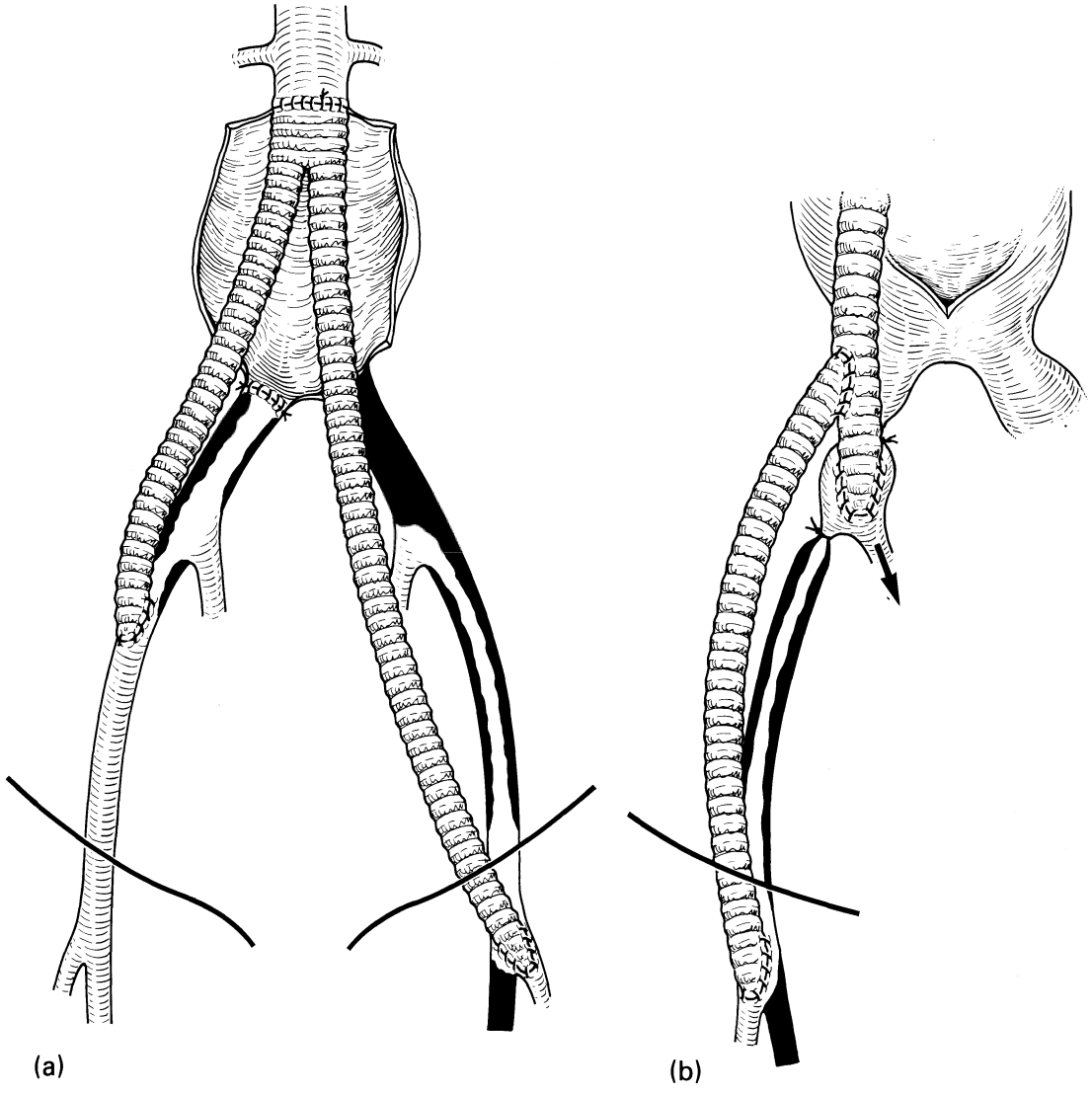


Figure 2.13(a, b) Coexistent outflow tract occlusive disease: (a) aortodistal bypass. (b) compromise arrangement preserving internal iliac perfusion

Reimplantation of the inferior mesenteric artery

In most cases the inferior mesenteric artery is either small or occluded and it may be sewn off from within the aneurysm without consequence. However, if the vessel is large with a prominent angiographic territory, it should be routinely reimplanted into the aortic prosthesis. This is particularly important when there is deficient collateral inflow from the superior mesenteric artery or the internal iliac arteries cannot be revascularized. Back-bleeding may be helpful in deciding on the degree of collateral supply and, if there is a brisk jet from the inferior mesenteric orifice on completing the distal anastomosis, reimplantation is probably unnecessary. Colon colour is less reliable, though persistent cyanosis or blanching on clamp release would clearly point to the need for immediate revascularization.

Less commonly, the inferior mesenteric artery is the main source of supply to the small bowel in patients with severe superior mesenteric artery occlusive disease. Inferior mesenteric reimplantation may then be necessary to avert postoperative small bowel infarction (see Chapter 14).

The most satisfactory method of reimplantation is via a Carrel patch (Figure **2.14**). A disc of aortic wall is excised around the inferior mesenteric origin and anastomosed to a matched opening in the main stem or left iliac limb of the prosthesis. Where the inferior mesenteric origin is narrowed by plaque, an eversion endarterectomy may be needed prior to reimplantation. If this is unsuccessful, the artery may be spatulated and attached to the graft directly or via a short PTFE or venous segment.

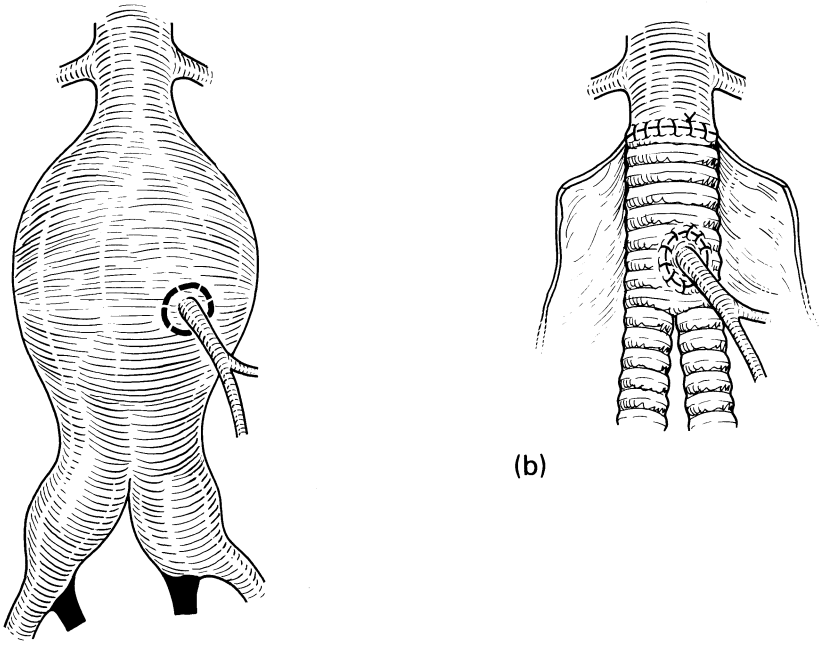


Figure 2.14(a, b) Reimplantation of the inferior mesenteric artery using a Carrel button

INFLAMMATORY ANEURYSM

In this unusual condition the aneurysm wall is affected by a dense inflammatory reaction which envelops adjacent structures, particularly the duodenum, ureters, inferior vena cava and the sigmoid colon. The condition is essentially a sterile chronic inflammation which is characterized by marked fibrosis and oedema of the aortic wall.

Clinical features which may suggest this entity include pain, tenderness and a high ESR[32]. Vomiting may occasionally occur from duodenal involvement. Others are discovered during investigation of hydronephrosis and/or medial ureteric displacement. The CT scan findings are specific and will allow accurate preoperative diagnosis in most cases[33].

At operation, the main technical problem is the densely adherent duodenum which may make access to the aneurysm neck impossible. Although endoluminal balloon control may be one solution, a preferable option is to leave the duodenum undisturbed and to clamp the aorta well above the aneurysm, if necessary in the supracoeliac segment. The iliac arteries are then clamped and the aneurysm opened, skirting the adherent duodenum. After the graft has been inserted, the thickened aortic wall is folded over the prosthesis with the duodenum still attached.

If the ureters are mildly dilated they may be left embedded in the aneurysm wall since resolution of the inflammatory reaction may follow graft replacement in most cases. However, if there is marked hydronephrosis with renal impairment or the patient has only one functional kidney, it may be safer to dissect out the ureters and place them within the peritoneal cavity.

MYCOTIC ANEURYSM

Colonization of the aortic wall during the course of a bacteraemia or septicaemia may result in softening of the wall and an eventual saccular aneurysm. This development is more likely if the aorta is atherosclerotic, or otherwise abnormal, with or without pre-existing aneurysmal change. Other cases arise by direct spread from an adjacent septic focus, e.g. vertebral osteomyelitis. Bacterial endocarditis is an infrequent cause nowadays. The commonest infecting organisms are salmonella and staphylococci[34,35].

The clinical presentation may be with fever, chills, abdominal or back pain and a pulsatile abdominal mass. Often the latter is difficult to feel and diagnosis may remain obscure until rupture occurs[36].

Prompt treatment is essential once the diagnosis is established because of the risk of early rupture. If the aortic lesion is well localized, *in situ* graft replacement and omentopexy may be feasible. However, where there is more extensive aortic involvement with overt sepsis this approach is not advisable and, instead, the aorta should be interrupted and an axillobifemoral graft inserted. In either circumstance, high dose antibiotic therapy should be continued for several weeks after surgery.

COEXISTENT RENAL OR VISCERAL ARTERY OCCLUSIVE DISEASE

Renal artery stenosis is a not infrequent finding in patients with an abdominal aortic aneurysm. High-grade stenoses should be corrected at the time of aortic surgery to avoid possible perioperative occlusion or the need for reoperation at a later date. Hypertension and impaired renal function may provide more specific indications. A separate Dacron or PTFE limb taking origin from the aortic prosthesis is the procedure of choice (see Chapter 13).

Stenosis or occlusion of the coeliac and superior mesenteric arteries should also be corrected at the time of aortic replacement. Failure to recognize and correct such additional disease has led to fatal postoperative gut infarction in our own experience and that of others[37]. Bypass grafts originating from the aortic prosthesis again form the basis of therapy (see Chapter 12).

ACCESSORY RENAL ARTERIES

If an accessory lower pole renal artery is encountered it should be reimplanted to avoid loss of renal tissue and possible secondary hypertension. A button of aortic wall is excised around the vessel origin and this is then implanted into the prosthesis once the aortic anastomosis has been completed (Figure **2.15a, b**).

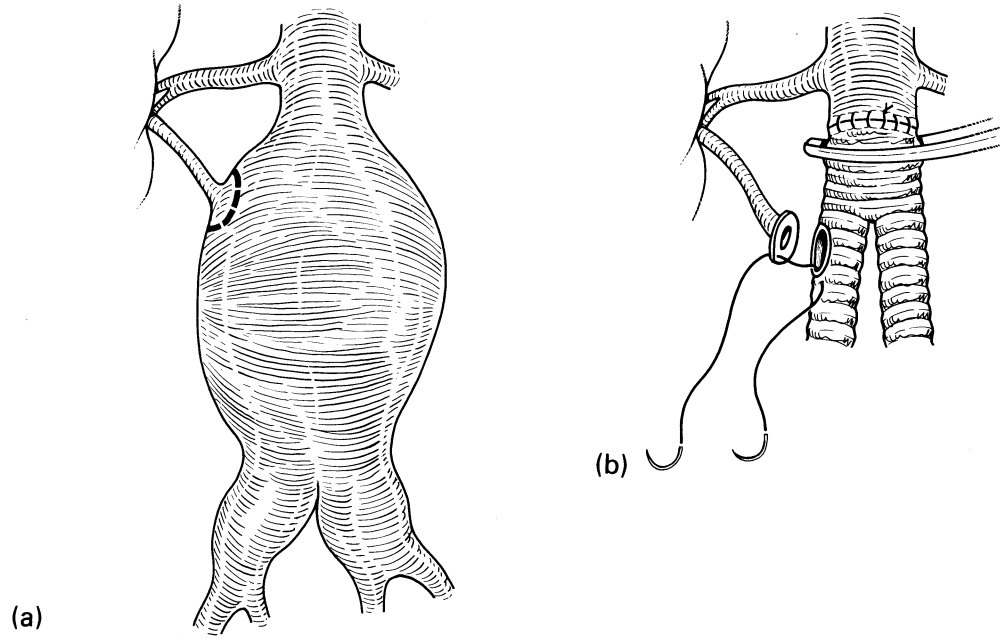


Figure 2.15(a, b) Reimplantation of accessory lower pole renal artery

HORSESHOE KIDNEY

Rarely, a horseshoe kidney may be found in association with an aortic aneurysm. Management is governed by the state of the renal blood supply and the nature of the isthmus.

If the renal vascular pattern is normal, the aorta may be replaced in the usual fashion by tunnelling the prosthesis behind the isthmus[38]. If the isthmus is thin and fibrous it may be divided for direct access to the aorta. Division of functional isthmic tissue should be avoided if possible, because of the risk of a urinary leak adjacent to the prosthesis.

In other cases there are single or paired accessory arteries arising from the aneurysm and supplying the isthmus. These vessels may be reimplanted into the aortic prosthesis using the button technique (Figure **2.16**). Multiple renal vessels arising from the aorta and common iliac arteries at several levels are likely to preclude aortic resection unless the origins are clustered sufficiently to allow reimplantation via an extended aortic strip.

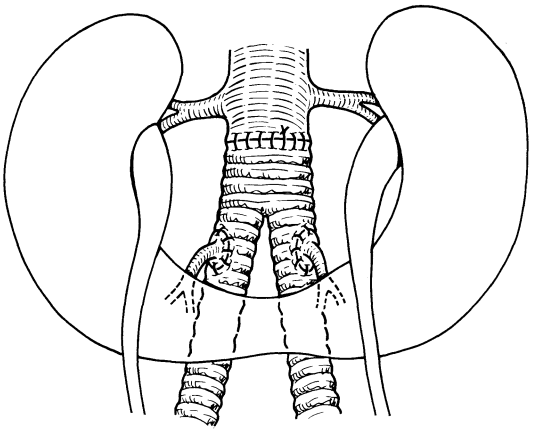


Figure 2.16 Horseshoe kidney. In this example two large accessory renal arteries have been reimplanted into the prosthesis

AORTIC ANEURYSMS INVOLVING THE RENAL ARTERIES

In some cases the aneurysmal process extends up to but not into the origin of the renal arteries (Figure **2.17a**). Proximal control for these juxtarenal aneurysms is best obtained at supracoeliac level. The left renal vein can often be retracted sufficiently but may need temporary division for access. The aneurysm is opened to the level of the renal arteries and a graft is inlaid (Figure **2.17b**). A rubber-shod clamp is then placed across the prosthesis immediately distal to the anastomosis and the supracoeliac clamp removed, restoring renal and visceral flow. The rest of the procedure follows the lines already described.

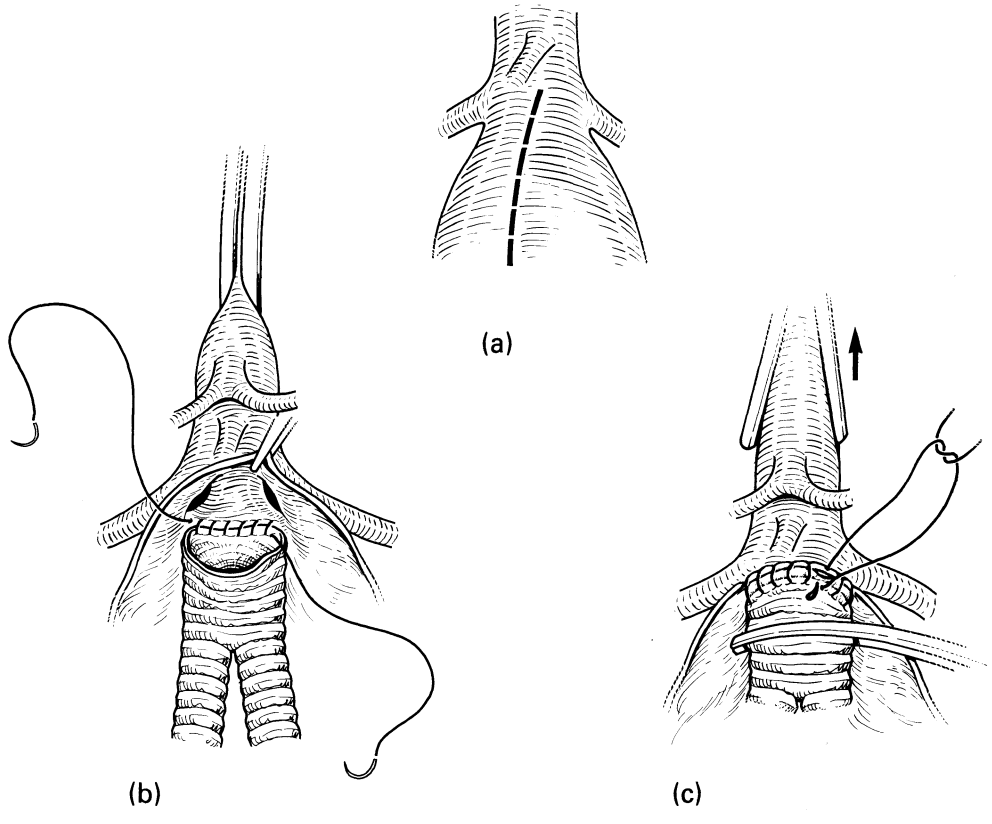


Figure 2.17(a–c) Method of managing a juxtarenal aneurysm

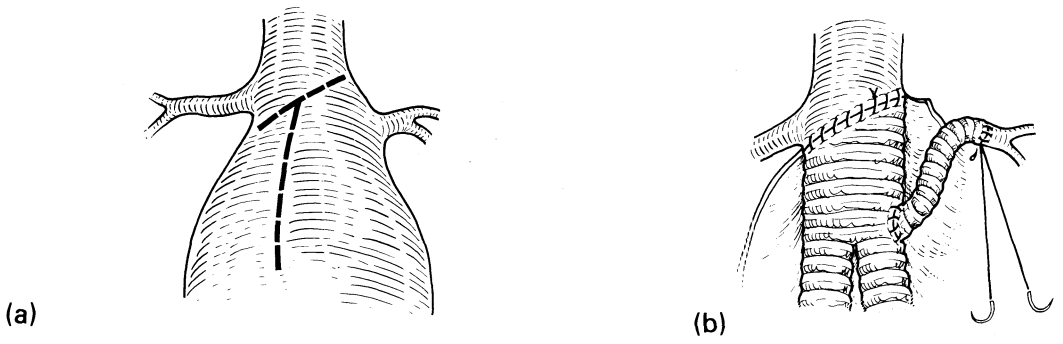


Figure 2.18(a, b) Aneurysm involving one renal artery

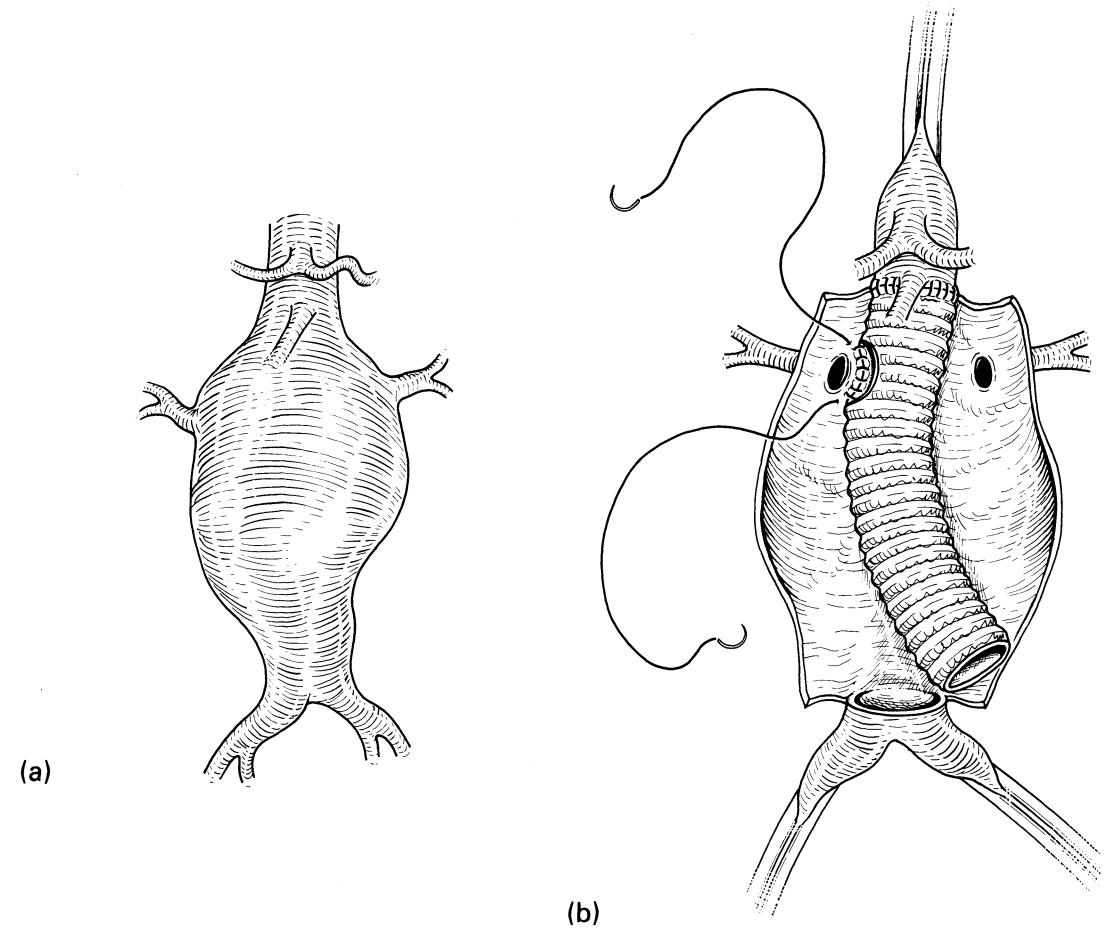
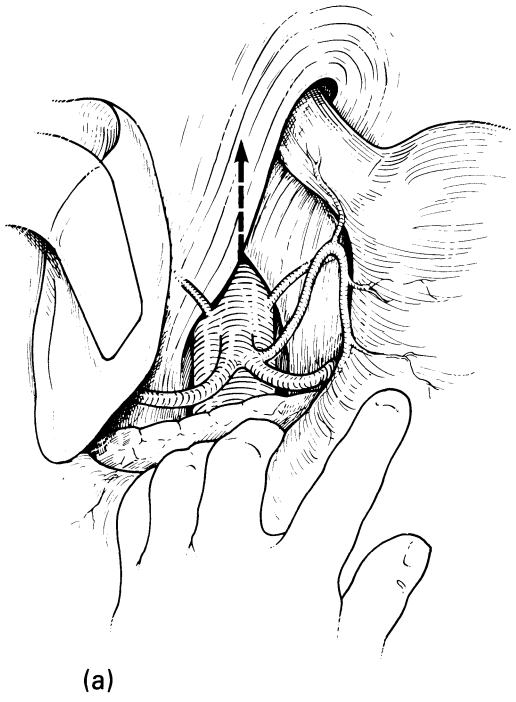
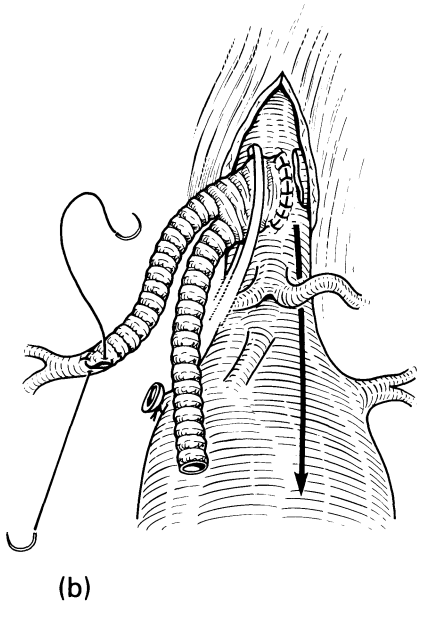


Figure 2.19(a, b) Suprarenal aortic aneurysm. Renal artery origins reattached by an inclusion technique

If the aneurysm extends above one of the renal arteries, the aortic prosthesis may be attached obliquely and the involved vessel either reimplanted by the button technique or revascularized using a bypass graft (Figure **2.18b**).

When both renal arteries are involved, one approach is to insert the aortic graft at the proximal limit of the aneurysm. The renal arteries are then reattached by an inclusion technique (Figure **2.19b**) or by reimplantation or bypass grafting. These manoeuvres result in rather a long ischaemic time for the second kidney and although this may be perfectly safe, particularly if cold perfusion[39,40] and inosine[41] are used, an alternative is to revascularize the kidneys before dealing with the aneurysm. This can be achieved by attaching a small, e.g. 13×6·5 mm, bifurcated graft to the supracoeliac aorta (see Chapter 12). Each renal artery is then detached in turn from the aneurysm and anastomosed to one of the graft limbs. Thereafter the aneurysm is replaced in the usual way (Figure **2.20a–c**). Although more complicated, this method has the advantage that each kidney is ischaemic for little more than the time taken for one anastomosis.





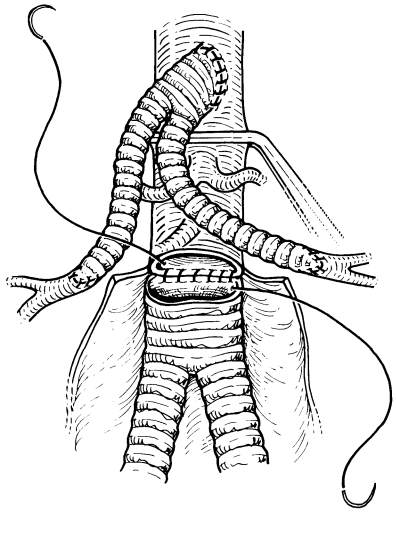


Figure 2.20(a–c) Alternative method of managing a suprarenal aneurysm: (a) supracoeliac aorta exposed by incising the aortic hiatus. (b) a bifurcated graft is attached to each renal artery in turn. (c) the aneurysm is then replaced

THORACOABDOMINAL AORTIC ANEURYSMS

Aneurysms involving the thoracic and abdominal aorta or the aortic segment from which the visceral arteries arise are uncommon. They may appear *de novo* or occur subsequent to graft replacement of the proximal thoracic or infrarenal aorta (see Figure **2.22a**). In the past these aneurysms have been treated by establishing a proximal–distal aortic bypass as a preliminary procedure and then connecting the visceral arteries in series using multiple side branches (Figure **2.21a, b**). Although this technique keeps the occlusion time for each vessel to a minimum, the operating time and blood loss may be considerable. The inclusion technique of Crawford[42] is superior in both respects and has led to a marked reduction in operative mortality. However, if there is associated visceral or renal arterial occlusive disease, separate side limbs may still be necessary to one or more vessels since orifice endarterectomy from within the aneurysm sac may not always be entirely satisfactory.

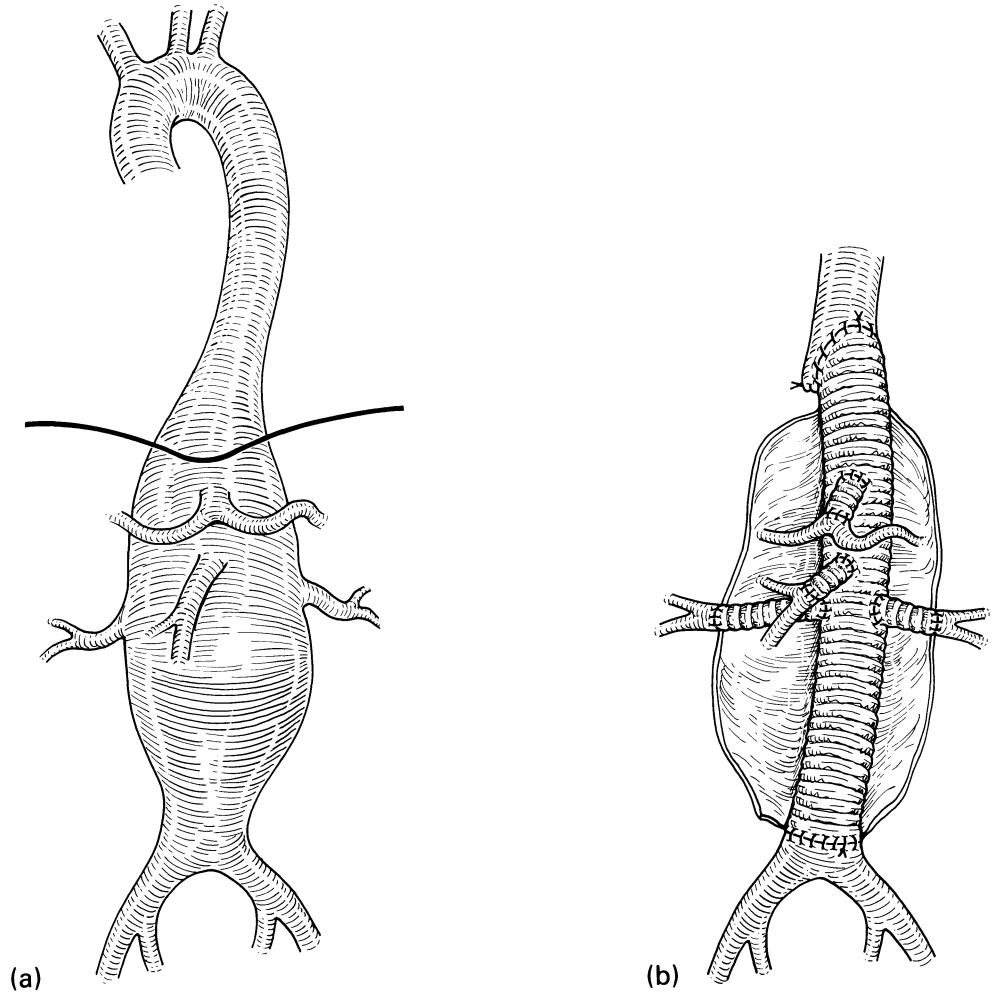


Figure 2.21(a, b) Thoracoabdominal aneurysm treated by preliminary aorto-aortic bypass and serial side limb grafts

Technique

A left thoracoabdominal incision is made and the diaphragm split to the aortic hiatus. The aneurysm is then displayed by a retroperitoneal approach in which the spleen, pancreas, colon and left kidney are all reflected to the right (Figure **2.22b**). The proximal and distal limits of the aneurysm are defined and clamps applied. Application of the proximal clamp may increase cardiac afterload considerably and this may require vasodilator infusion and adjustments in fluid therapy.

The aneurysm is then opened along the left posterolateral wall in a line behind the origin of the left renal artery (Figure **2.22b**). The renal arteries may be cannulated with an infant feeding tube or a coronary artery perfusion cannula and the kidneys temporarily cooled with heparinized Ringer's lactate at 4°C. Systemic or regional inosine may also help protect renal function[41]. A Fogarty balloon catheter may then be placed in each orifice and also in the visceral artery origins if back-bleeding is a problem. Sometimes large paired lumbar or intercostal arteries are encountered in the region of the hiatus and these may be similarly controlled with a view to subsequent inclusion in the graft. The other parietal vessel orifices are oversewn.

A Dacron tube graft is inlaid at the proximal limit of the aneurysm (Figure **2.22c**). The visceral arteries are then attached by means of a series of elliptical openings cut in the graft. Where the individual vessel origins are widely separated they can be sewn in singly. In some cases the coeliac and superior mesenteric artery origins may be sufficiently close together to use a common opening leaving the renal arteries to be implanted separately (Figure **2.22d, e**). When attaching the left renal artery it is important to cut the window well to the left side of the graft to ensure a satisfactory lie when the kidney is returned to its normal position.

After each anastomosis is completed the graft is allowed to fill by back-bleeding and the air is evacuated before the controlling clamp is moved down to the next site (Figure **2.22e**). Release of flow into the visceral arteries may produce precipitous hypotension and it is important to discontinue the vasodilator infusion well beforehand and to increase the intravenous fluid replacement. Additional bicarbonate infusion is also advisable prior to declamping as metabolic acidosis is inevitably more marked than with infrarenal aortic replacement.

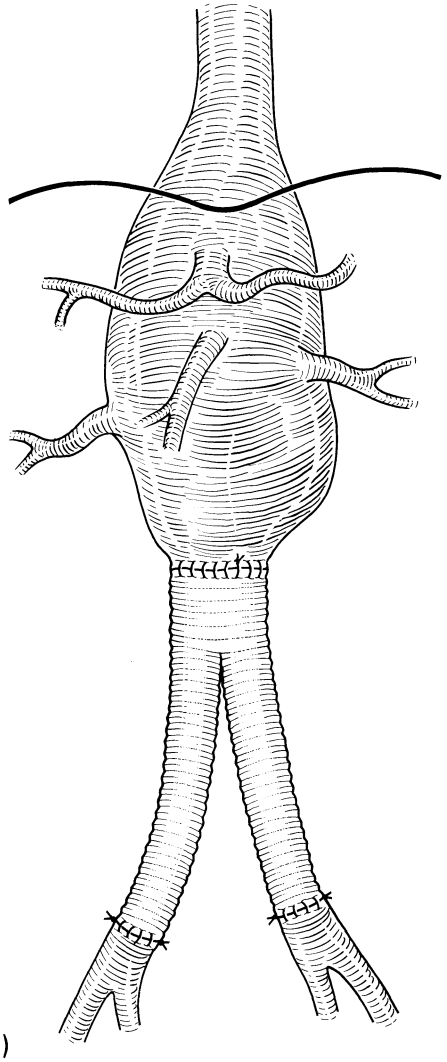


Figure 2.22(a–g) Thoracoabdominal aneurysm treated by inclusion technique: (a) aneurysm arising several years after infrarenal Teflon graft

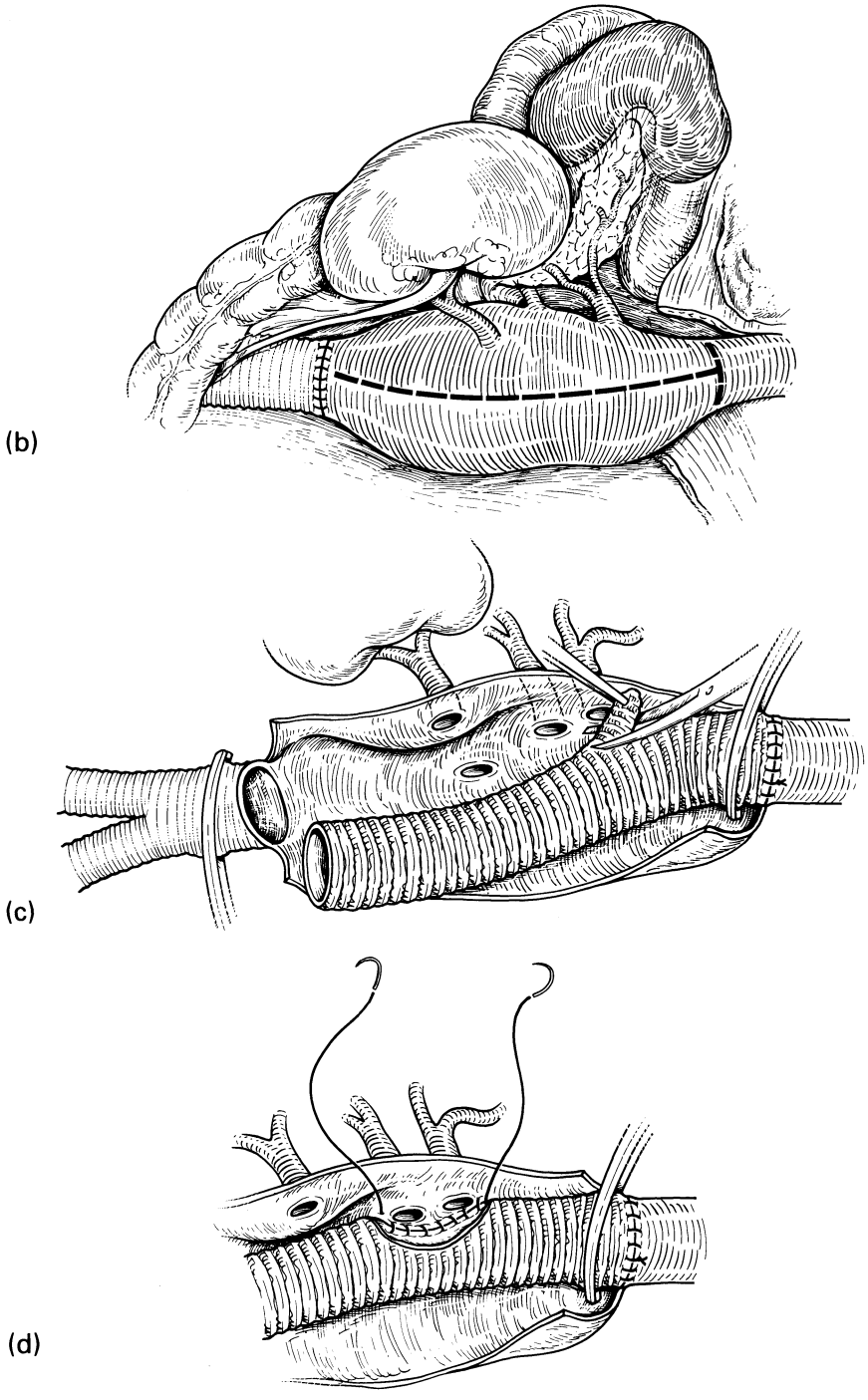
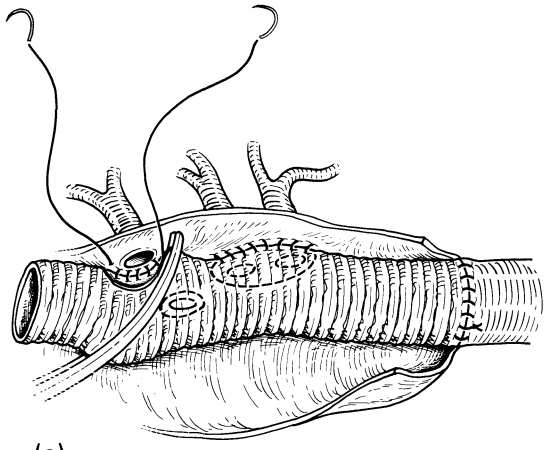
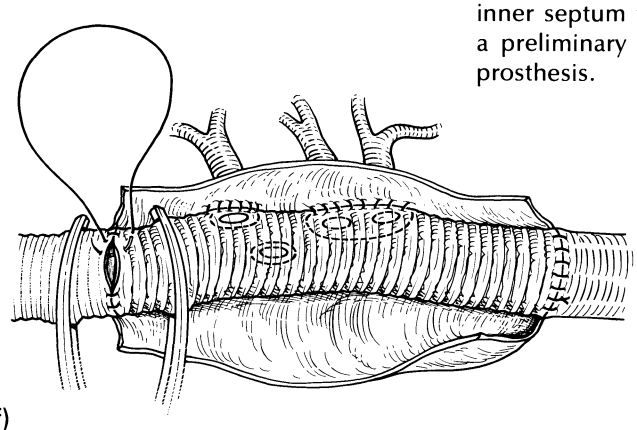


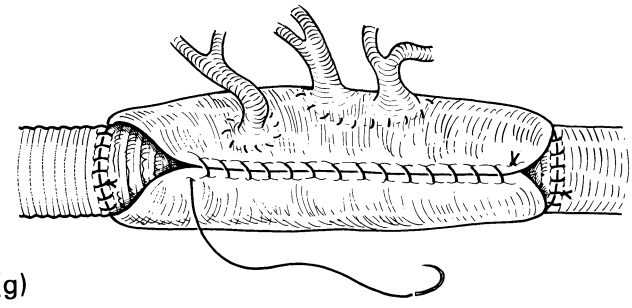
Figure 2.22 continued (b) thoracoabdominal exposure with reflection of all viscera to the right. (c) after completing the proximal aortic anastomosis an ellipse is cut from the graft. (d) the graft opening is sutured around the coeliac and superior mesenteric artery origins



(e)



(f)



(g)

Large lumbar or intercostal arteries may be reimplanted by a similar inclusion technique. However, this may only be necessary with particularly extensive aneurysms where most of the thoracic aorta is being, or has been replaced[43]. Once all the aortic branches have been revascularized the graft is anastomosed either to the stump of the previous infrarenal prosthesis if there is good distal patency (Figure **2.22f**) or in the unoperated case to the distal aorta. Coexistent aneurysmal or occlusive disease at the aortic bifurcation will require addition of a bifurcated extension to the main graft.

Chronic dissecting aneurysms which involve the thoracoabdominal aorta can also be managed by this technique. The dissection usually affects only part of the aortic circumference (most commonly the left side at this level) and the visceral artery orifices may or may not be involved. In the former case it is necessary to cut away the false inner septum from around the vessel origins as a preliminary to reimplantation into the aortic prosthesis.

Figure 2.22 continued (e) the right renal artery has been similarly attached to a second opening in the graft. Anastomosis of the left renal artery is proceeding. (f) distal anastomosis of aortic graft to infrarenal Teflon prosthesis. (g) aneurysm sac wrapped around the new graft

RUPTURED INFRARENAL AORTIC ANEURYSM

The first event in an aortic rupture is usually the escape of blood into the space between the wall of the aorta and the periaortic sheath. This is manifest clinically by sudden onset of pain and a transient drop in blood pressure. This periaortic haematoma then remains confined for a variable period of time – usually at least a few hours and sometimes much longer. If it is possible to get the patient into the operating room at this stage the chances of salvage are excellent. Often, however, this stage passes and the haematoma breaks through into the retroperitoneum and sometimes into the peritoneal cavity as well. The patient now becomes shocked and with continuing delay success is progressively less likely. Hypovolaemic shock is poorly tolerated, especially by the elderly and, though a technically successful operation may still be possible, many of these patients will die in the postoperative period from renal, respiratory or myocardial failure. It follows that the most crucial factor in managing patients with a ruptured aneurysm is to get them into the operating room and the aorta cross-clamped at the earliest possible moment. Meddlesome investigation of the apparently stable case, or futile attempts at resuscitating the patient with hypotension, in particular must be avoided.

On arrival the patient is taken directly to the operating room where preparations to open the abdomen and to anaesthetize the patient can rapidly proceed side by side. Blood is drawn for rapid cross-matching and two large-bore intravenous cannulae are inserted into the upper extremities for transfusion. If the patient is severely vasoconstricted, initial transfusion via a subclavian venous line may be necessary. At the same time, a Foley catheter is placed in the bladder, and the abdomen and groins are shaved, prepped and draped. In the presence of severe shock these latter measures will have to be abandoned and the abdomen opened immediately. Ventilation with a face mask and 100% oxygen is then used until the aorta is controlled by manual compression. Thereafter the patient can be intubated and anaesthetized and arterial, venous and bladder catheters inserted, as above. In all cases, intravenous antibiotics should be commenced as soon as practicable.

In the less severe case, anaesthetic induction is accomplished with the surgical team scrubbed and instruments ready for immediate laparotomy. Induction abolishes peripheral vasoconstriction and a previously stable but hypovolaemic patient may now become rapidly hypotensive. Vigorous volume replacement, preferably with blood, should therefore be commenced by the anaesthetist at this stage.

The abdomen is opened by a xiphopubic incision without wasting time on local haemostasis. A sternal retractor is rapidly secured at the upper end of the wound in case access to the supracoeliac aorta is required. The small bowel and colon are then displaced out of the wound as previously described. Atherosclerotic patients with hypotension have a marked tendency to distal arterial thrombosis during clamping and sytemic heparin (1 mg/kg body weight) should be given as soon as the abdomen is open.

If the rupture is still confined and the patient's condition stable, the iliac vessels may be dissected out before disturbing the aorta. This will minimize the risk of intraoperative embolization.

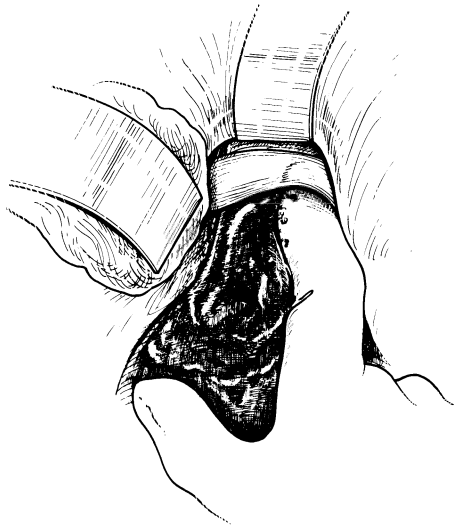
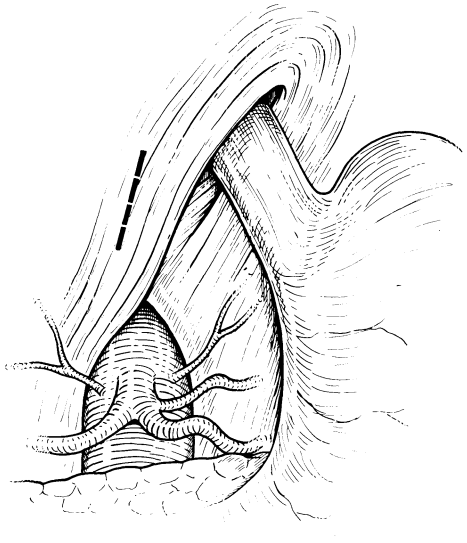


Figure 2.23 Infrarenal control in a ruptured aneurysm. The neck of the aneurysm is defined by digital dissection within the haematoma

In unstable cases an immediate approach should be made to the aneurysm neck. The posterior peritoneum is opened and a finger thrust into the haematoma along the left anterolateral surface of the aneurysm until normal width aorta is reached (Figure **2.23**). The sides of the aorta are then defined digitally and a clamp applied vertically at this point. By commencing digital dissection relatively low and to the left of the midline, damage to the left renal vein–caval junction should be avoided.

If the patient is severely hypotensive, or if infrarenal digital dissection dislodges clot from the rupture site with immediate fresh bleeding, the aorta should be either compressed manually at the hiatus or the supracoeliac segment definitively clamped. This elevates peripheral resistance maximally and conserves coronary and carotid perfusion. Access for supracoeliac control is achieved by opening the lesser omentum and displacing the oesophagus to the left and the liver to the right (Figure **2.24a**). The right crus is then split digitally or with scissors and the incision deepened to the sides of the distal thoracic aorta. At this level the aorta is well posterior and control may require insertion of the full length of the fingers. With the index and middle fingers of the left hand straddling the aorta (Figure **2.24b**) sufficient space is created to allow application of the aortic clamp (Figure **2.24c**). The infrarenal haematoma is then opened and the neck of the aneurysm located. A second clamp is applied here and the supracoeliac control carefully released. If the aorta is difficult to locate in the midst of the



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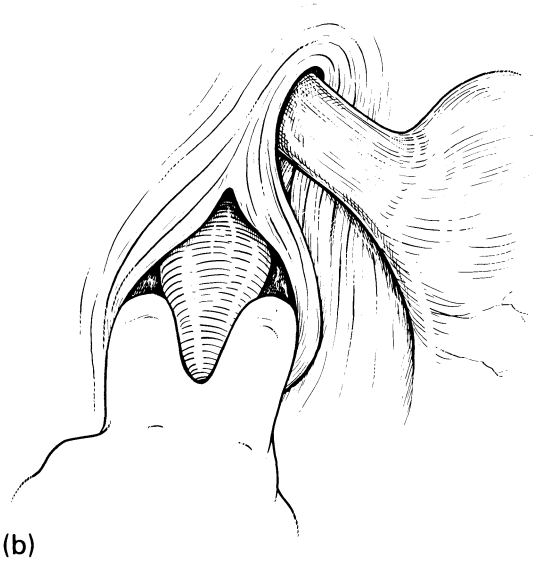

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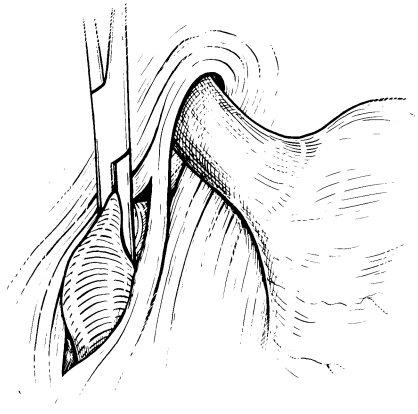

(c)

Figure 2.24(a–c) Technique of supracoeliac clamping

haematoma, the aneurysm may be opened and an index finger thrust upwards into the aortic lumen. The aorta is then lifted away from the lumbar spine and the infrarenal clamp applied.

Endoluminal balloon inflation is an alternative method of proximal control. However, there may be appreciable blood loss before effective control is achieved and there is at least a theoretical risk of displacing atherothrombotic debris into the renal arteries when the balloon is inflated; one of the above techniques is therefore generally preferred.

Once proximal control has been achieved the common iliac arteries are defined by digital dissection in the haematoma. If a tube graft seems possible the front and sides only of the common iliac arteries are cleared and clamps applied from in front. A tube graft has particular advantages in the ruptured case since operating time is shorter and blood loss less than with a bifurcated graft. However if the aortic bifurcation is degenerate or there is gross aneurysmal or occlusive iliac disease, a bifurcated graft will be needed and appropriate distal clamp control should therefore be obtained.

The aneurysm is then opened and replaced with a graft as previously described. Woven Dacron or PTFE prostheses may be preferred as these will limit blood loss. Tight closure of the sac around the graft and suture lines is particularly important in ruptured aneurysm patients since clotting is frequently impaired due to haemodilution, massive transfusion or disseminated intravascular coagulopathy from shock or hypothermia[44,45].

In view of the increased risk of graft infection in these patients, the graft area and retroperitoneum should be irrigated with antibiotic–saline or antiseptic solution and the operative site covered with omentum as a routine. Systemic antibiotics are continued into the postoperative period.

During the procedure various monitoring devices may have been added, including an ECG, intra-arterial and central venous pressure lines. A Swan–Ganz catheter is particularly useful and should be used whenever there is marked instability or an antecedent history of coronary artery disease or hypertension. Blood, colloid and electrolyte solutions are then given rapidly until normal pressures and cardiac action are restored. Blood replacement must be accompanied by adequate amounts of fresh frozen plasma, platelet concentrate and calcium. Cryoprecipitate or fresh blood transfusion may be helpful if there is a bleeding disorder on completion.

Renal function is a major concern in these cases and this may be best preserved by rapid correction of initial hypovolaemic shock, avoidance of subsequent hypotension from sudden declamping or blood loss, and by adjusting fluids to optimize left heart filling pressures throughout the perioperative period[24,25]. A renal-dose dopamine infusion may be an additional helpful measure.

Blood gas determinations are made at intervals throughout the procedure, particularly on declamping, and appropriate pH adjustments made. All cases are electively ventilated for 24–48 hours postoperatively to ensure adequate oxygenation of previously hypoxic tissues. In addition, cardiac support drugs or possibly intra-aortic balloon assist may be necessary if there is continuing poor cardiac output despite adequate volume replacement.

UNUSUAL TYPES OF RUPTURE

Aortocaval fistula

Occasionally an aortoiliac aneurysm may rupture into the inferior vena cava or an iliac vein[46]. This process may occur suddenly with abdominal or back pain and hypotension. However, most cases lack this acute onset, the fistula occurring as a result of more gradual erosion into the cava. Signs of an aortocaval fistula include high output cardiac failure, a continuous abdominal bruit or thrill, renal impairment, and pelvic or lower extremity venous hypertension. In many patients the fistula remains undiagnosed until operation[47,48].

In view of the marked haemodynamic disturbances which may occur during operative repair, insertion of a Swan–Ganz catheter is advisable prior to surgery. On opening the abdomen, the vena cava or iliac veins may be found to be grossly distended with a palpable thrill. Embolization of thrombus from the fistula site is one of the main

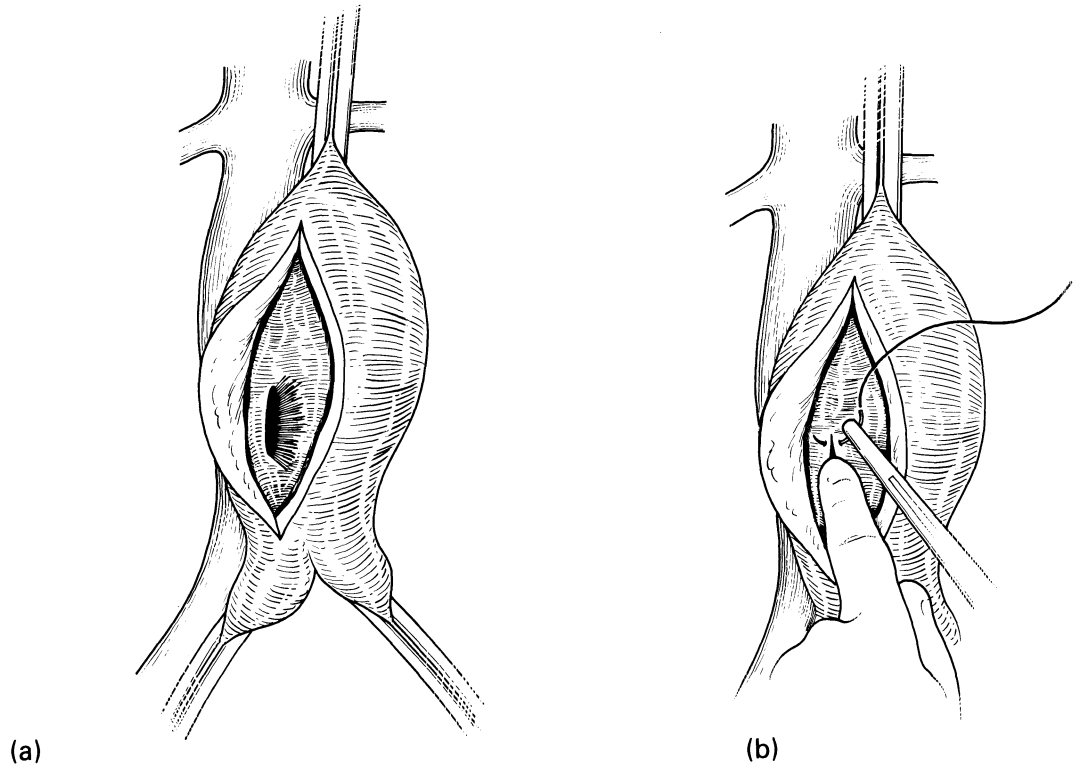

Figure 2.25(a, b) Aortocaval fistula closed from within the lumen of the aneurysm

operative hazards and it may be helpful to control the cava above the fistula before the aneurysm is opened. Heparin is given and clamps are applied above and below the aneurysm. On opening the latter, venous haemorrhage may be seen coming from the right hand wall at the site of the fistula (Figure **2.25a**). This can be controlled digitally while the opening is sewn off from within the aorta (Figure **2.25b**). The aneurysm is then grafted in the usual way. If there is thrombus lining the fistula it may be advisable to clip the cava below the renal veins to avoid postoperative pulmonary embolism.

Aorta–left renal vein fistula

This is a rare complication usually related to a retroaortic left renal vein[49]. It presents as loin pain, haematuria and a non-functioning kidney in association with an aortic aneurysm. Management is along similar lines to an aortocaval fistula, viz. transaortic closure of the fistula followed by graft replacement of the aneurysm.

Aortoenteric fistula

Primary aortoenteric fistulae are uncommon, most cases being secondary to previous graft insertion (see Chapter 14). The distal duodenum is the usual site, though occasionally the small bowel or colon may be involved. The diagnosis may be suspected whenever upper gastrointestinal bleeding is associated with a demonstrable aortic aneurysm. Intermittent self-limiting bleeds are typical of this condition and may continue for several weeks or even months before massive exsanguination occurs. Abdominal or back pain is present in about half of the cases.

In the absence of acute local sepsis, patients with a primary aortoduodenal fistula may be managed by enteric repair and *in situ* graft replacement of the aneurysm[50]. Omentum should be interposed between the graft and the duodenum. Where overt sepsis is present or the fistula involves the colon or terminal ileum, the aorta should be oversewn and an axillobifemoral graft inserted.

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3

Aortoiliac Occlusive Disease

Reconstruction for arterial occlusive disease is undertaken more frequently in the aortoiliac segment than at any other site. The vessels here are of large calibre and the flow rate high so that immediate and long-term surgical results are good. Moreover, in contrast to femoro–distal reconstruction, there is seldom any problem in finding a good distal run-off since the profunda femoris is usually disease-free or can be readily reconstructed to accommodate the new outflow.

Gangrene, rest pain and ischaemic ulceration form absolute surgical indications. Claudication is also an indication for surgery if it interferes markedly with the patient's life or if there is an embologenic lesion, such as an ulcerative plaque or local aneurysmal dilatation on aortography. Less severe claudicants may also be accepted for surgery providing the distal run-off is good and there are no significant operative risk factors.

Complete relief of ischaemic symptoms may be anticipated in patients with patent infra-inguinal vessels[1,2]. When the profunda femoris is the sole run-off vessel, aortoiliac reconstruction will relieve rest pain and allow healing of ischaemic ulceration or minor necrosis in nearly all cases[3,4], though more extensive foot necrosis usually requires synchronous femoro–distal reconstruction[5,6]. Among claudicants with multisegment occlusive disease, 20% or so may have incomplete relief and in about half of these cases a supplementary femoro–distal bypass will be required at a later date[1,6,7].

Additional physical disabilities are common in patients with aortoiliac occlusive disease and these may influence the therapeutic approach. Thus, respiratory or renal impairment, inoperable coronary artery disease or advanced age may contraindicate direct aortoiliac reconstruction. Some of these patients may be considered for extra-anatomic bypass if the ischaemia is sufficiently severe and distal run-off adequate. If the presenting complaint is claudication, abstention may be the wisest course.

ENDARTERECTOMY

Endarterectomy of the aorta and iliac arteries is the longest established method of treatment and in selected cases yields good long-term results[8,9]. One of the most important advantages of any endarterectomy technique is the relative immunity from sepsis. In addition collaterals are preserved or re-established and in consequence late failure of the reconstruction rarely produces severe limb ischaemia. Moreover, if late occlusion occurs it is relatively easy to treat compared with occlusion of a bypass graft.

On the debit side, endarterectomy is a more difficult technique than bypass and an inadequate or faulty clearance of a collateral vessel, such as the internal iliac artery, may lead to an overall result inferior to that of bypass grafting. Other

disadvantages include longer operating time and increased risk of sexual dysfunction as a result of damage to the autonomic nerve fibres in the region of the aortic bifurcation. However, it should be emphasized that when impotence is already present because of disease proximal to or within the internal iliac arteries, endarterectomy may offer the best prospects of restoring potency if coupled with a nerve-sparing dissection technique (see Chapter 14).

Aortoiliac endarterectomy is contraindicated where there is pre-aneurysmal degeneration in the aortic wall since the development of an overt aneurysm is likely. A second contraindication is extension of the atherosclerotic process into the external iliac arteries. Although complete clearance of the aorta and both iliofemoral axes is technically feasible, operating time is prolonged and a significant incidence of late closure in the external iliac segment is to be expected[10]. However, aortoiliofemoral endarterectomy remains a useful rescue approach in some cases of aortic graft sepsis (see Chapter 14).

BYPASS GRAFTING

This is now acknowledged as the best form of treatment for the majority of patients with aortoiliac occlusive disease[1,11]. The technique is relatively easy and quick, and the risk of technical error less than with endarterectomy. One of the disadvantages of bypass grafts is that they may lead to eventual closure of collaterals arising from the defunctioned segment between the two anastomoses. If the graft then occludes, severe ischaemia may be precipitated. Other risks of bypass insertion are sepsis and false aneurysm formation. Both of these complications relate predominantly to anastomoses in the groin and in order to reduce these hazards some surgeons have advocated aorta–external iliac bypass grafting. However, late failure may be anticipated with this type of reconstruction because of disease progression in the common femoral artery beyond the distal anastomosis[7,11]. In fact, with strict asepsis and a careful suture technique the risks involved in femoral anastomosis should be minimal and this is the preferred distal implantation zone for most bypass grafts.

PERCUTANEOUS TRANSLUMINAL ANGIOPLASTY

The ideal lesion for transluminal angioplasty is a localized iliac stenosis or stenoses. Short iliac occlusions may also be successfully recanalized in the majority of cases (see Chapter 15). In general, angioplasty is more likely to benefit patients with claudication and is less successful in patients with severe limb ischaemia who usually have diffuse multisegment disease. Angioplasty may have particular merit in high risk cases, patients with short life expectancy or where surgery is limited by adverse local factors such as obesity, potential sepsis or previous operative treatment.

In some cases angioplasty may be used as an adjunct rather than an alternative to surgery, e.g. an iliac stenosis may be dilated prior to femorofemoral bypass for a contralateral iliac occlusion or before an ipsilateral femoro–distal bypass for a coexistent superficial femoral occlusion. Intraoperative dilatation has also been suggested as a means of dealing with stenoses beyond the operative field[12,13]. However, in the absence of fluoroscopic control or catheter expertise this is likely to have limited success.

PREOPERATIVE ASSESSMENT

Good quality biplanar or multiplanar aortography is essential. Lateral or oblique views are particularly important when an embologenic pathology is suspected or when there is coexistent disease in the visceral arteries. Adequate films of the profunda and below-knee vessels are also essential. In addition an ultrasound scan may be useful in assessing the state of the arterial wall, the presence or absence of mural thrombus, and luminal size. Coexistent ischaemic heart disease may merit full cardiological evaluation, including coronary angiography (see Chapter 2). Duplex scanning[14] or digital subtraction angiography[15] may be used to screen the carotid arteries and supra-aortic trunks, followed by conventional arteriography in symptomatic or selected cases. Surgical correction of significant coronary or extracranial arterial lesions is usually undertaken prior to aortic reconstruction.

AORTOFEMORAL BYPASS

The patient is positioned and draped as for aortic aneurysm surgery (see Chapter 2). If there is any doubt regarding the ability of the profunda to provide an effective run-off the leg should be prepped and draped for possible supplementary femoro–distal grafting.

The operation is begun by exploring the groins to assess the state of the vessels and in particular the profunda femoris (Figures **3.1, 3.2**). An approach lateral to the vascular axis is utilized with dissection of the profunda femoris down to the first perforating branch or beyond. This requires division of the lateral circumflex vein which crosses the main profunda trunk anteriorly (Figure **3.2a**). At the upper limit of the groin incision the inguinal ligament is partially incised lateral to the vascular axis to allow the graft to enter the groin without compression. The deep circumflex iliac vein should be identified and divided at this stage to avoid damage during subsequent tunnelling (Figure **3.2b**).

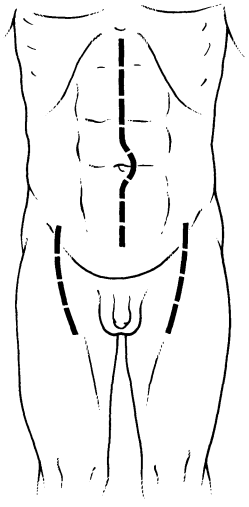
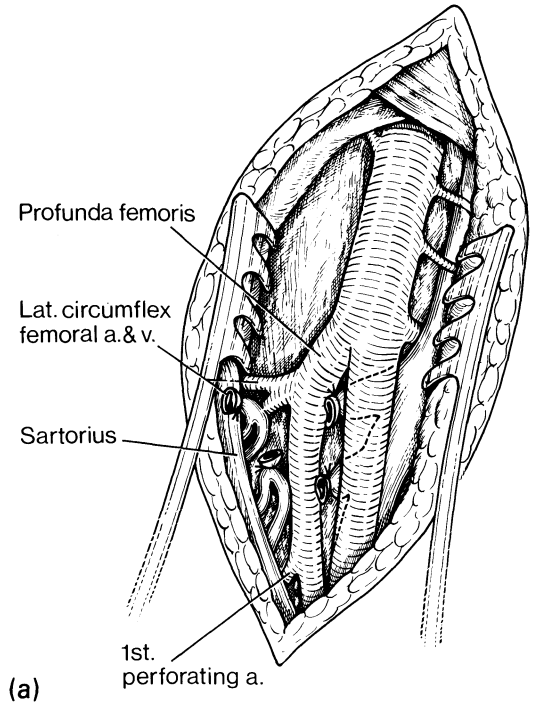


Figure 3.1 Incisions for aortofemoral bypass



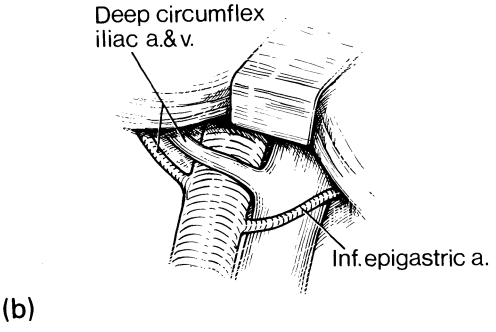


Figure 3.2(a, b) Groin exposure: (a) proximal profunda displayed by division of the lateral circumflex femoral vein. (b) relationship of vessels deep to the inguinal ligament. The deep circumflex iliac vein should be divided to avoid damage during graft tunnelling

The abdomen is opened by a xiphopubic incision and the aorta displayed as previously described. The preferred technique for attaching the prosthesis is by end-to-end anastomosis immediately below the renal arteries. The left renal vein is mobilized and a sufficient segment of the proximal aorta cleared to allow subsequent clamping and transection (Figure **3.3**). If posterior dissection of the aorta appears hazardous, only the front and side walls of the aorta should be cleared, the prosthesis then being inlaid onto the intact posterior aortic wall. Widespread peeling of the periaortic adventitia should be avoided because of possible damage to the autonomic nerve plexus.

After preparing the proximal implantation site, a retroperitoneal tunnel is developed on either side, lateral to the aortic bifurcation and behind the ureter and gonadal vessels (Figure **3.4**). Digital dissection from the groin will complete the tunnel. A rubber tube or tape may be left in place to provide a guide for subsequent passage of the graft.

A knitted Dacron or PTFE bifurcated graft of appropriate size is then selected and, in the former case, preclotted. The graft should be cut with a short stem since this configuration, together with a high implantation site, will ensure optimum haemodynamics and avoid kinking. After systemic heparinization, clamps are applied vertically to the aorta immediately below the left renal vein and a 2–3 cm segment of aorta is resected. The distal stump is then oversewn, preceded if necessary by a limited removal of local plaque (Figure **3.5**). If the infra-inguinal vessels are patent and the aorta particularly degenerate, it may be wise to temporarily clamp the external iliac artery before removing the distal aortic clamp to avoid peripheral atheroembolization. At a later stage when the bypass is functioning, this external iliac clamp can be removed.

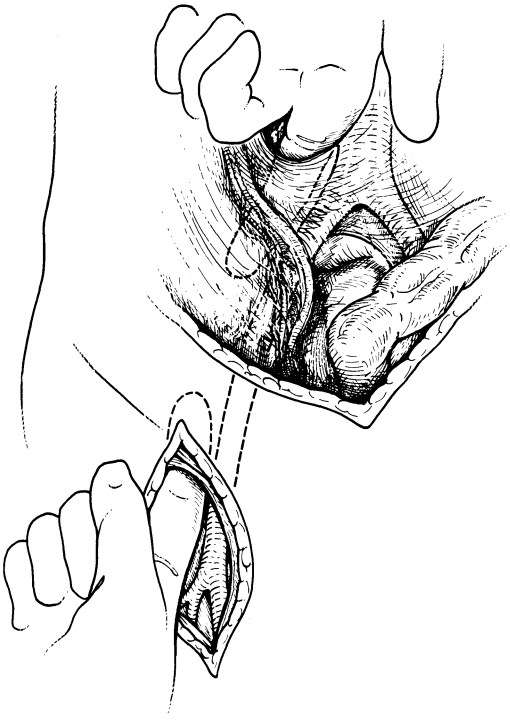


Figure 3.4 Development of a retroperitoneal tunnel by digital dissection behind the ureter and gonadal vessels

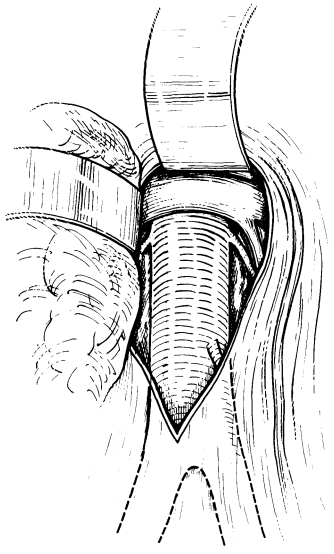


Figure 3.3 Exposure of the proximal infrarenal aorta

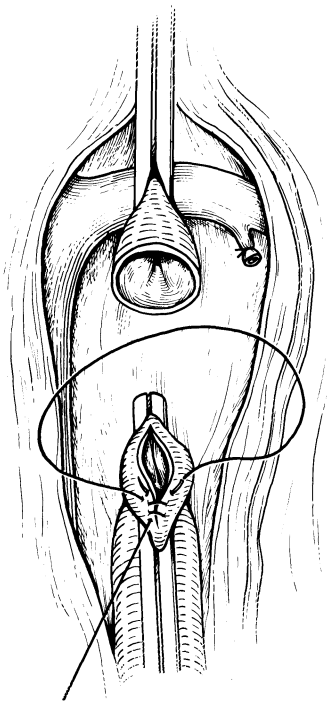


Figure 3.5 The aorta is transected and the distal stump oversewn

The proximal stump of the aorta is then prepared for anastomosis. A local endarterectomy may be needed if the intima is thickened or calcific but in most cases the graft can be anastomosed directly as the aorta is not usually severely diseased at this site. It may be helpful to place a slight clockwise rotation on the graft to allow the right limb to clear the duodenum adequately. An intraluminal suture technique is preferred for the posterior wall (Figure **3.6**). 4/0 prolene is a suitable suture material in most cases, though finer sutures may be adopted if the aortic stump has been endarterectomized. An additional useful measure in the latter circumstance is to incorporate a Dacron or Teflon strip in the suture line (Figure **3.7**). Alternatively if the aortic wall is unusually fragile, the graft may be attached by interrupted sutures each backed by a Teflon pledget[16].

Once the anastomosis is complete a rubber-shod clamp is placed immediately distal to the suture line and the aortic clamp removed. A Dacron collar is then slipped over the anastomosis to improve haemostasis and to distance the suture line from the duodenum (Figure **3.8**).

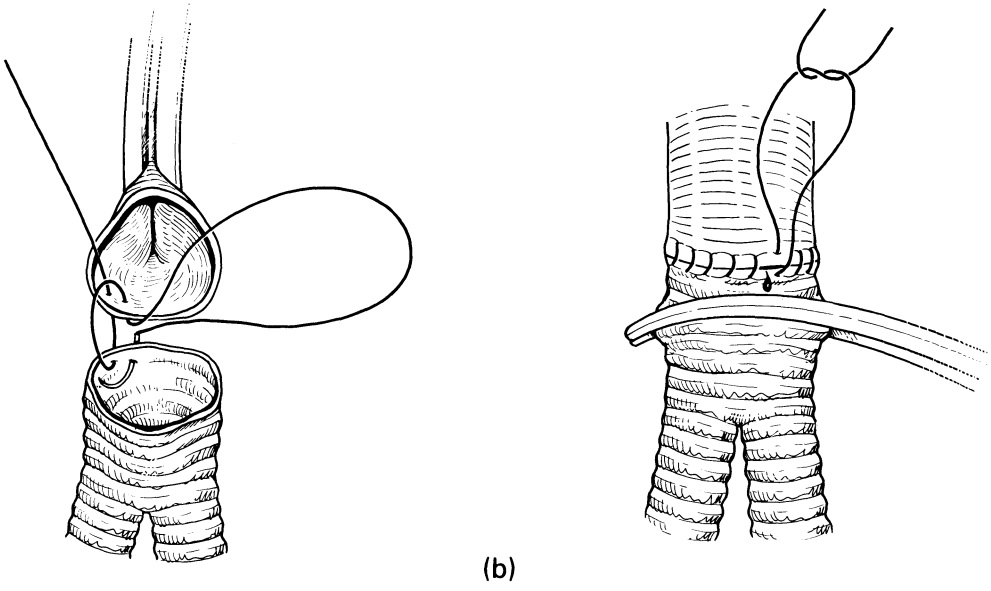


Figure 3.6(a, b) End-to-end anastomosis of the graft to the proximal infrarenal aorta

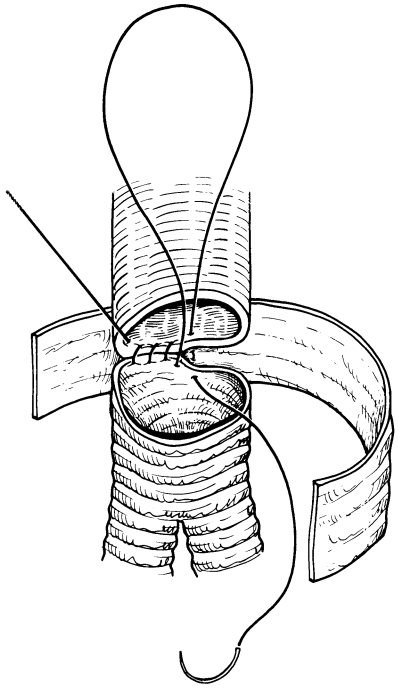


Figure 3.7 Incorporation of a Dacron or Teflon strip into the proximal suture line when the aortic wall is unduly fragile or calcific

The next stage concerns distal implantation. One of the limbs of the graft is drawn down the pre-formed tunnel into the groin with the aid of an aortic clamp (Figure **3.9**). Clamps are applied to the common, superficial and deep femoral vessels and an arteriotomy made in the distal common femoral artery. Most frequently this is extended across the profunda origin to produce an angioplasty effect. Unless there is gross local plaque or ulceration, a local thromboendarterectomy is not performed, as over-enthusiastic endarterectomy may favour false aneurysm formation.

The graft is cut to the correct length under tension and bevelled to produce a flange at the heel and at the apex (see Chapter 1).

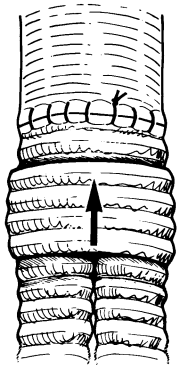


Figure 3.8 External Dacron collar

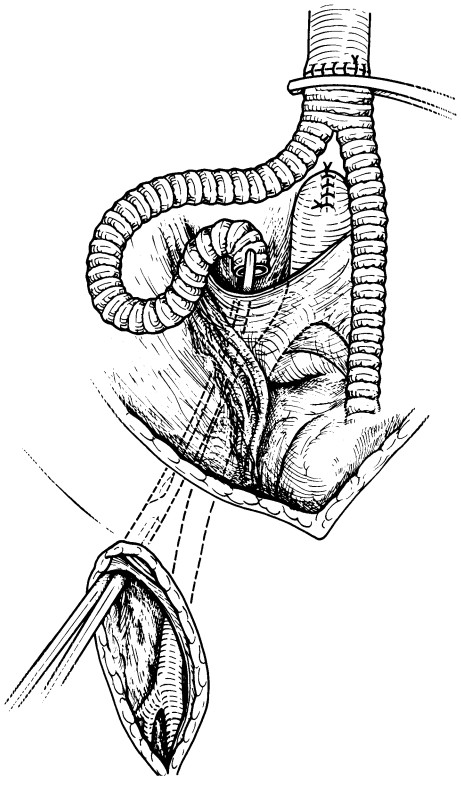


Figure 3.9 The right limb of the graft is drawn down the retroperitoneal tunnel lateral to the iliac vessels

After thorough aspiration of the graft limb, a conventional end-to-side anastomosis is constructed with a running 5/0 or 6/0 prolene suture according to the type of prosthesis (Figure **3.10**). Just before the suture line is completed, the distal and proximal clamps are flushed, the latter via the unsutured graft limb. The graft is then aspirated and after irrigation with heparin–saline the suture line is completed. The common femoral clamp is then removed and flow released retrogradely into the iliac axis (Figure **3.11**). The profunda and superficial femoral clamps are then removed in turn. This order of clamp removal is designed to minimize the risk of peripheral microembolization (trash foot) in patients with patent distal vessels.

The remaining graft limb is then carefully aspirated and drawn down the retroperitoneal tunnel to the opposite groin. A similar or modified anastomosis is then constructed on this side, depending on local pathology.

If the appearances are satisfactory the posterior peritoneum and pre-aortic fascia are closed over the graft, sewing the left peritoneal edge to the tissue behind the duodenum and base of the mesentery. If there is insufficient tissue to separate the duodenum adequately from the prosthesis, an omental flap is brought down over the graft. The abdomen and groins are then closed in layers with suction drainage.

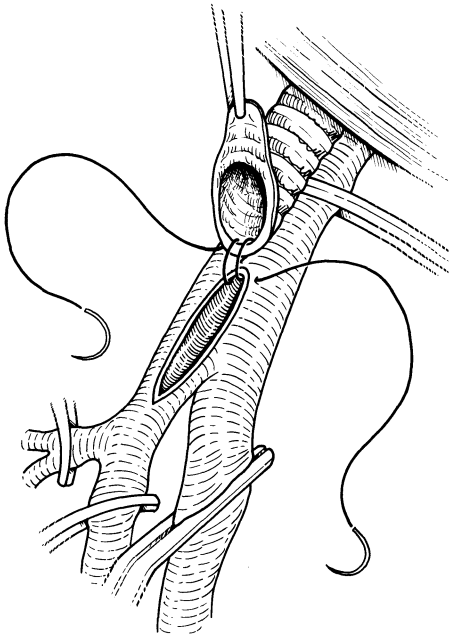


Figure 3.10 End-to-side anastomosis to the common–deep femoral segment

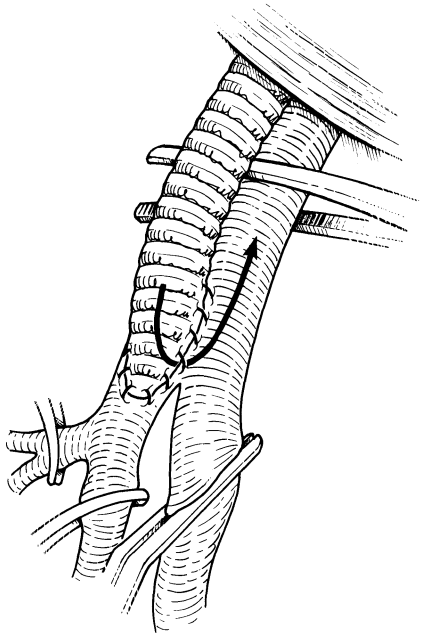


Figure 3.11 Preliminary retrograde release of graft flow

VARIATIONS

Proximal anastomosis

An alternative technique is to attach the graft to the aorta in end-to-side rather than end-to-end fashion. In general, the latter is preferable because the entire aortic flow is through the graft and haemodynamics are better than with an end-to-side configuration. More important, aortic clamping in the end-to-side technique risks distal embolization on clamp release, particularly if the aortic wall is significantly diseased or intraluminal debris is present at the clamp site. In addition soft tissue cover is more difficult to obtain with an end-to-side anastomosis and the risk of aortoduodenal fistula and graft sepsis consequently increased. However, an end-to-side anastomosis may be specifically indicated when there is severe bilateral external iliac occlusive disease or unilateral external iliac occlusion with contralateral internal iliac occlusion. In these cases aortic interruption may lead to inadequate internal iliac perfusion with at least a theoretical risk of colonic ischaemia or vasculogenic impotence. A large patent inferior mesenteric artery or accessory renal arteries may also be better preserved by a proximal end-to-side aortic anastomosis. However, aortic transection could still be utilized in these cases if combined with reimplantation of the relevant vessels into the aortic prosthesis (see Chapters 2 and 14).

A technique for end-to-side aortic anastomosis is shown in Figure **3.12a–c**. The graft should be inserted as high as possible on the aorta to minimize the risk of atheromatous progression. Partial occluding clamps provide restricted vision and it is safer to completely cross-clamp the aorta. An oblique disposition of the clamps may avoid the necessity for separate control of the posterior lumbar arteries (Figure **3.12a**).

An incision 3–4 cm long is made in the anterior wall of the aorta. It is not necessary to excise an ellipse from the aortic wall unless the aorta is excessively sclerotic or calcific. The temptation to perform a local endarterectomy to improve the implantation site should also be resisted. The graft is then cut with an S-shaped bevel to match the length of the aortotomy, leaving as short a stem as possible. Suturing is commenced at the heel and the far wall is closed first. Lateral traction

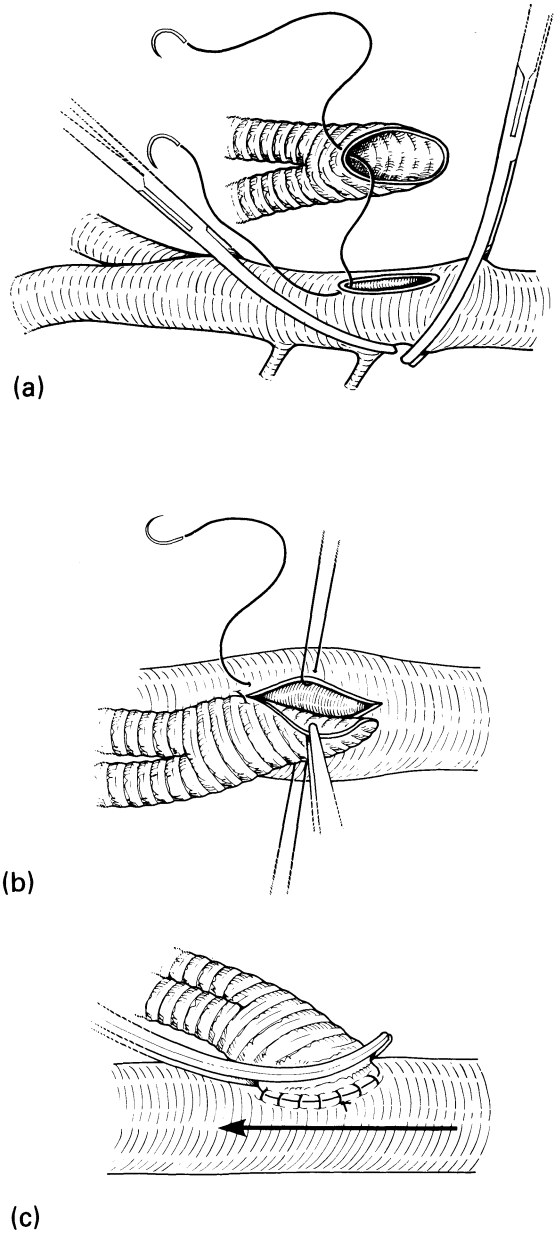


Figure 3.12(a–c) End-to-side anastomosis: (a) matching aortotomy. Note oblique disposition of the distal clamp to control the lumbar arteries. (b) anastomosis commenced at heel. Lateral traction sutures may be useful in holding the edges of the aortotomy apart. (c) anastomosis completed. A rubber-shod clamp is placed across the graft and flow restarted in the aorta

stitches may be helpful in keeping the aortotomy open during needle passage (Figure **3.12b**). Once the anastomosis is complete the proximal and distal clamps should be carefully flushed through the graft and a rubber-shod clamp placed across the prosthesis. Flow is then restarted in the aorta (Figure **3.12c**). The graft limbs are tunnelled to the groins and anastomosed as previously described.

Distal anastomosis

End-to-side anastomosis to the common femoral artery is a commonly advocated method of distal implantation. While this may be satisfactory initially, particularly in patients with a patent distal system, the development of progressive occlusive disease in the superficial femoral artery or around the profunda orifice may lead to premature graft failure. In most cases therefore it is preferable to carry the graft attachment across the profunda origin. However, if the profunda branches early (see Figure **4.4d**), common femoral implantation may become obligatory. This form of attachment may also be preferable when the superficial and profunda vessels are patent but affected by a smooth endarteritis. Sutures sufficiently heavy to secure the prosthesis (Dacron) may cut through the wall of the profunda, and a common femoral implantation, with or without venous patching of the outflow vessels, may be a more satisfactory option.

In many patients the superficial femoral artery is occluded and there is significant plaque extension from the common femoral artery into the profunda origin (Figure **3.13a**). Attachment of the aortofemoral graft across the stenosed profunda origin as a patch is not recommended as the sutures may need to be overdeep to avoid cut-out, thus favouring re-stenosis. A better solution is an initial endarterectomy of the common–deep femoral segment prior to graft attachment (Figure **3.13b**). Depending on the length of the profunda arteriotomy, a supplementary vein or PTFE patch may be used to close the profunda segment (Figure **3.13c**). Autogenous vein sews in well to the endarterectomized profunda and is generally preferable as patch material. However, there is a

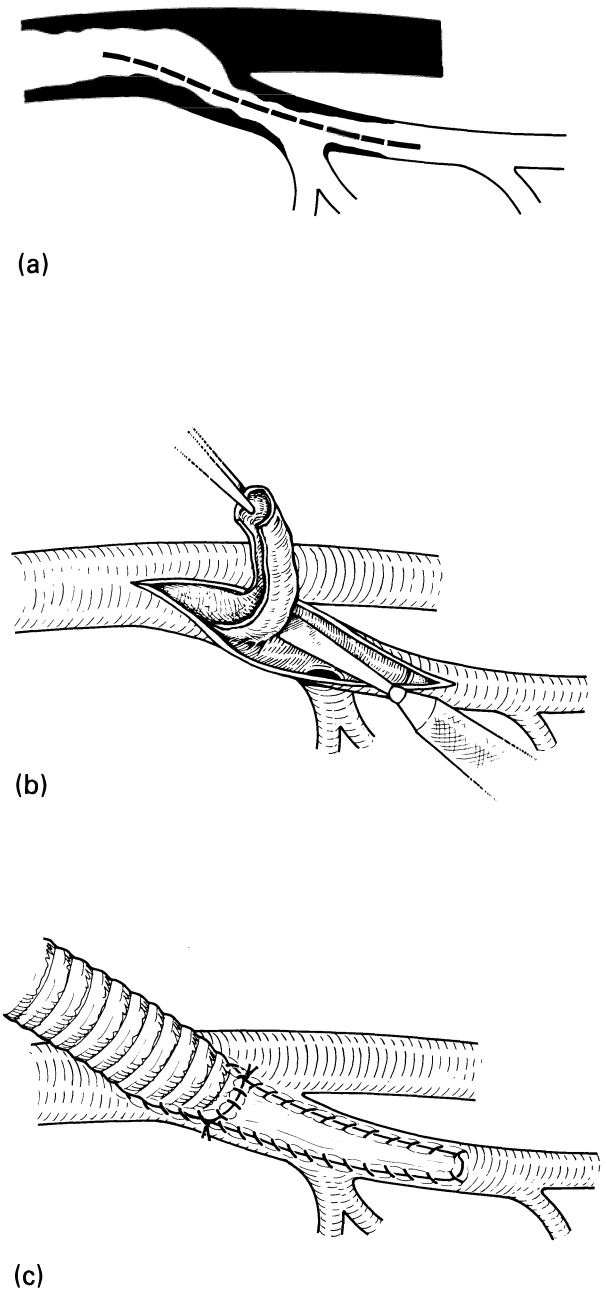


Figure 3.13(a–c) Distal anastomosis when there is profunda stenosis: (a) distribution of atheroma and arteriotomy. (b) endarterectomy of the common–deep femoral segment. (c) graft implanted with a supplementary vein patch

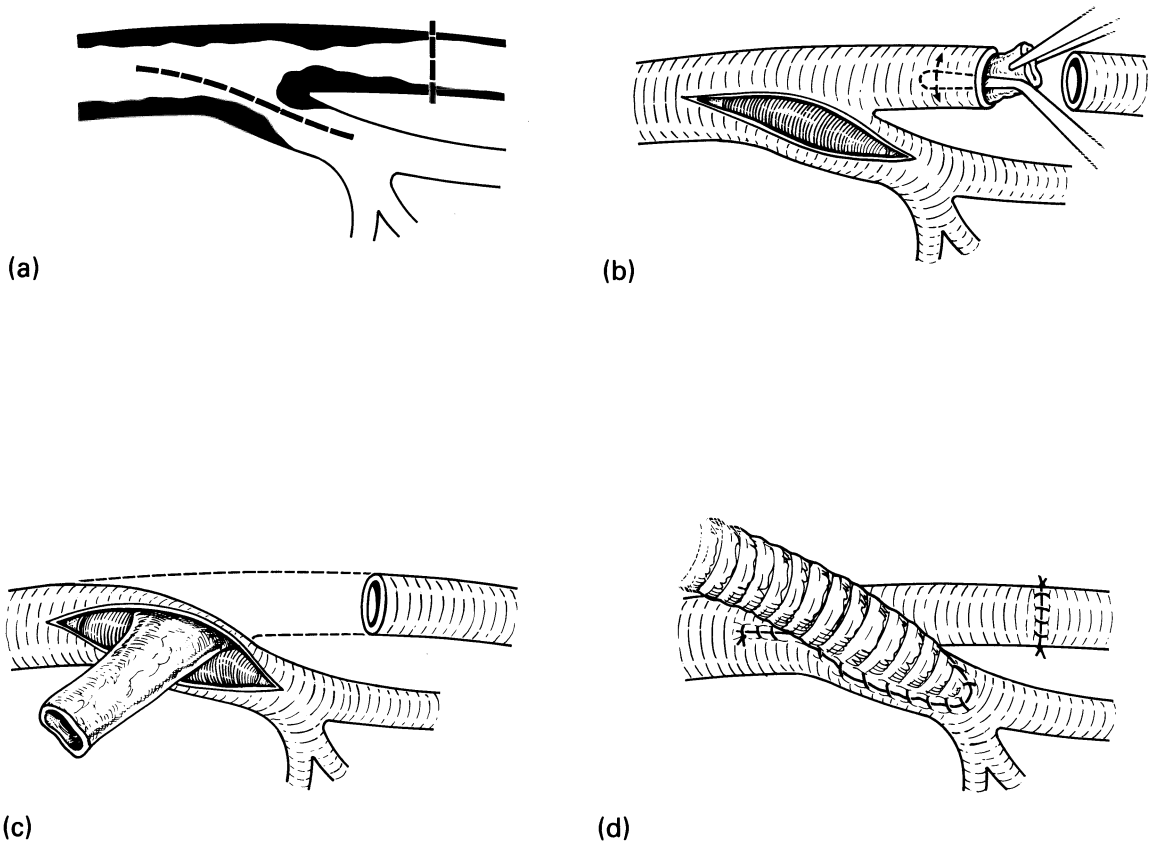


Figure 3.14(a–d) Method of aortic bypass attachment when the profunda is stenosed and the superficial femoral artery still patent: (a) pathology. (b) open common–deep femoral endarterectomy with section and eversion endarterectomy of the proximal superficial femoral artery. (c) superficial femoral artery turned inside-out to confirm clearance. (d) the superficial femoral artery has been re-anastomosed and the aortic bypass attached to the common–deep femoral segment

risk of false aneurysm formation at the Dacron-vein junction and a PTFE aortofemoral graft with a vein or PTFE profunda patch may be a better combination. Endarterectomized superficial femoral artery has also been used to patch the outflow of an aortic graft[17] but this material may be prone to premature degeneration and is no longer utilized.

In patients with retained superficial femoral patency, profunda endarterectomy may carry a risk of inducing acute superficial femoral occlusion due to an intimal flap at the origin. To avoid this possibility it may be advisable to control the superficial femoral intima by an additional section–eversion manoeuvre (Figure **3.14a–d**). If the superficial femoral artery is more grossly diseased but still patent, endarterectomy of the common femoral bifurcation is best avoided altogether. Instead the aortic bypass graft should be attached end-to-side to the profunda beyond the stenotic zone (Figure **3.15a, b**). Dacron is too rigid and thick to be attached satisfactorily to the perforator trunk and an aortic PTFE or a composite graft should be utilized for attachment at this level.

Occasionally the common femoral and superficial femoral arteries are occluded though patency is retained in the profunda due to refilling from the lateral or medial circumflex femoral vessels (Figure **3.16a**). A suitable method of attaching an aortofemoral bypass graft in these cases is by end-to-end anastomosis to the spatulated stump of the profunda (Figure **3.16b, c**).

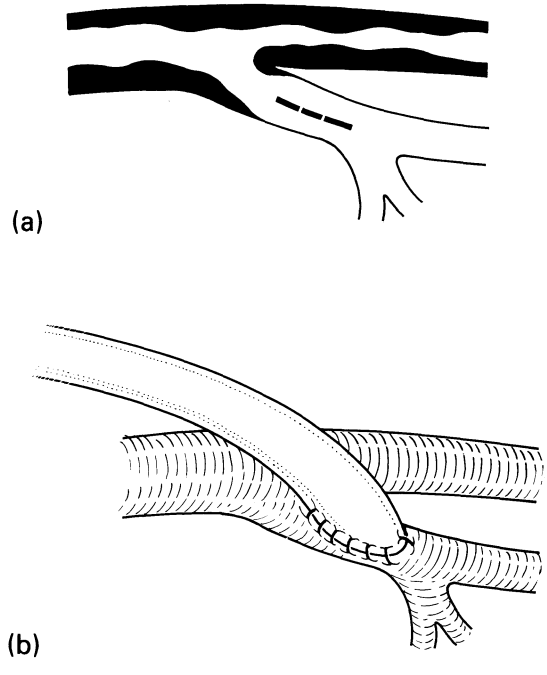


Figure 3.15(a, b) Alternative method of managing profunda ostial stenosis. A PTFE graft has been attached end-to-side to the profunda beyond the stenosis. Superficial femoral patency is preserved

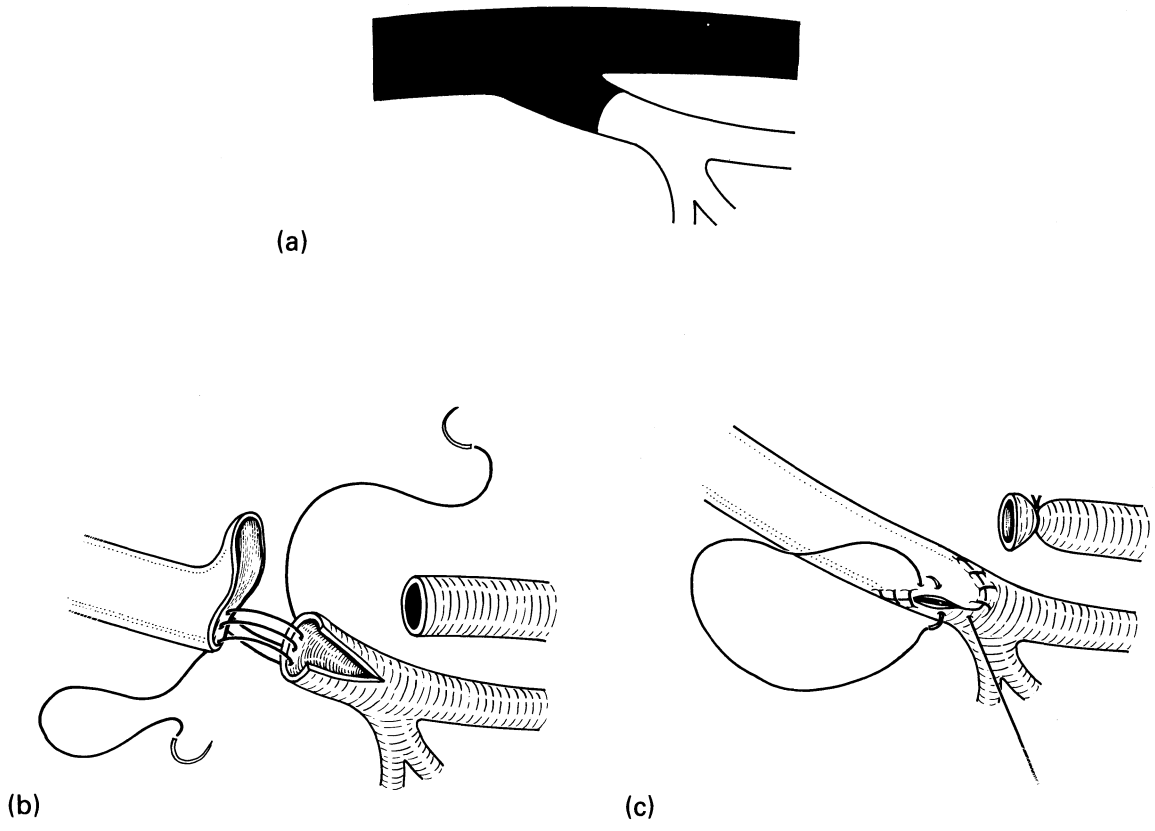


Figure 3.16(a–c) Distal anastomosis when the common femoral artery is occluded. The graft is attached to the spatulated profunda stump

In some patients it may be impossible to develop a satisfactory outflow tract in the profunda either because of diffuse disease (see Chapter 4) or because the profunda is small with poor thigh collateralization. An additional femoro–distal bypass may then be necessary to maintain the patency of the aortic reconstruction (Figure **3.17a**). However, it should be noted that when the profunda is significantly diseased beyond its proximal segment, the below-knee vessels are likely to be similarly affected. This is one of the factors contributing to the poor results of one-stage aortopopliteal reconstruction in previous reports[1,18].

A double reconstruction may also be needed where significant ischaemic symptoms persist or recur after aortofemoral bypass for multisegment disease. A convenient method for attaching a femoro–distal extension in these cases is shown in Figure **3.17b, c**.

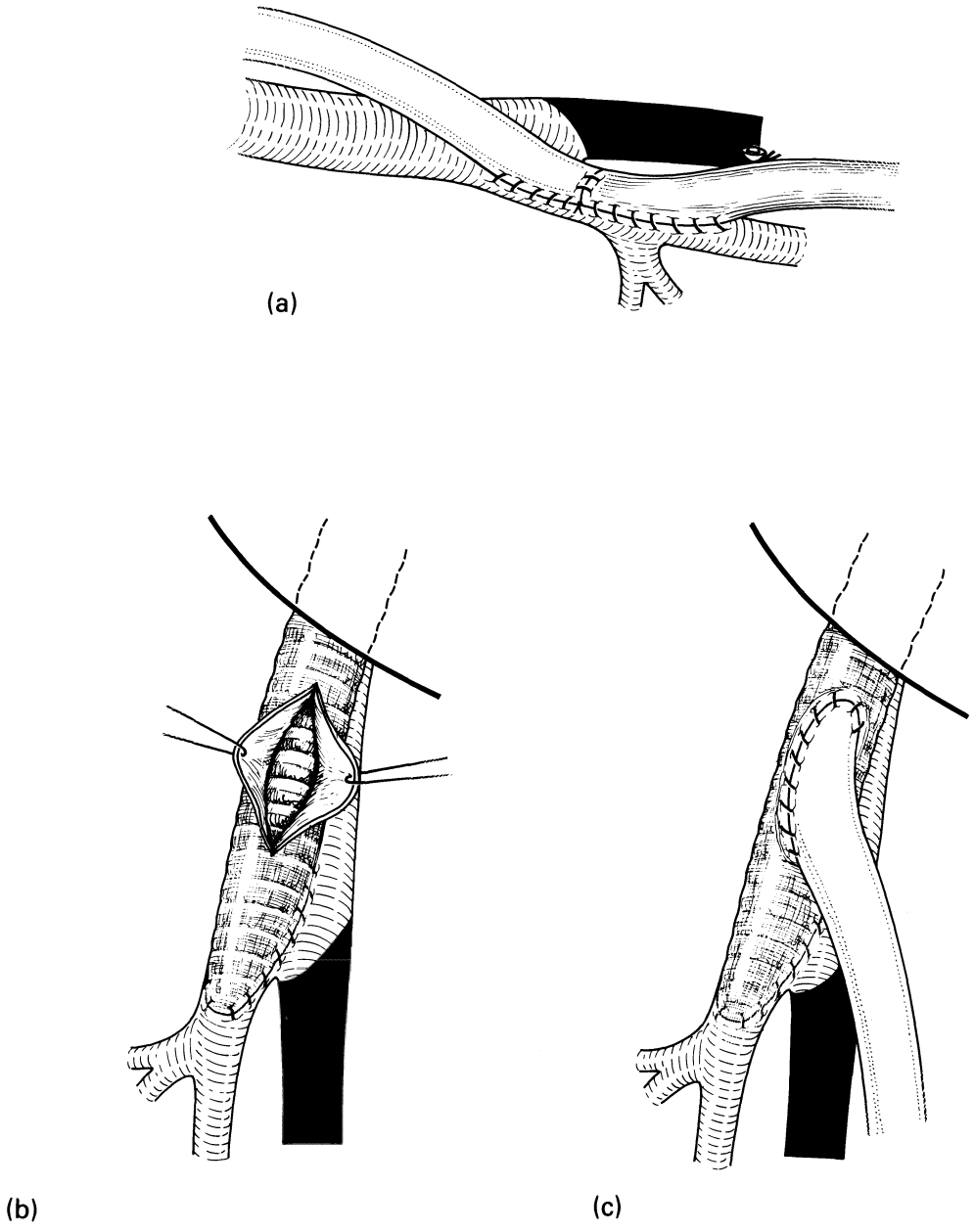


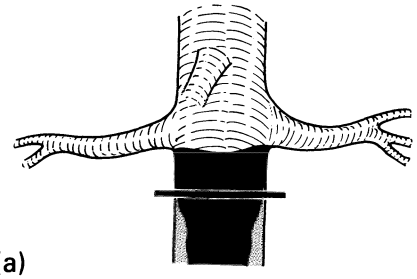
Figure 3.17(a–c) Combined aortofemoral and femoro–distal bypass: (a) one-stage reconstruction using PTFE and autogenous vein. The two grafts have been apposed so as to create a patch effect at the profunda origin. (b, c) alternative technique of supplementary femoro–distal bypass which is particularly suitable when some time has elapsed since the initial aortofemoral reconstruction

JUXTARENAL AORTIC OCCLUSION

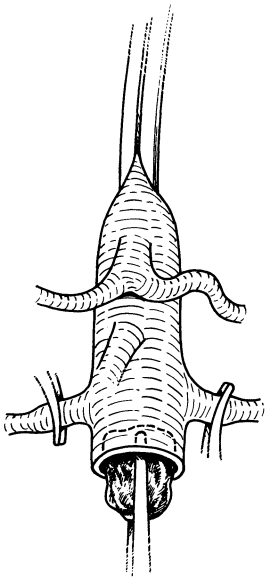
Atheroma in the distal aorta and common iliac arteries may uncommonly give rise to an ascending thrombosis which extends to the level of the renal arteries (Figure **3.18a**). Although these patients may be treated by remote bypass[19,20], there is a risk of continuing proximal extension of thrombus into the renal and visceral artery origins[21] and a direct operative approach is preferable whenever possible.

A standard exposure of the proximal infrarenal aorta is made with display of both renal arteries. The left renal vein will require retraction in either a caudal or cephalic direction after division of its adrenal, gonadal and lumbar branches. If access is still limited the vein may be divided between vascular clamps and re-anastomosed on completion.

Embolization of the renal arteries is a real hazard in this condition and after heparinization a soft clamp should be placed on each renal artery. The supracoeliac aorta is then clamped and the aorta transected immediately distal to the renal artery origins (Fig. **3.18b**). Usually there is minimal atheroma at this level, and the occlusion is due to soft thrombus which can be removed with forceps. Once the thrombus has been completely removed the aortic lumen is thoroughly irrigated and the renal clamps flushed in turn. An aorto-



(a)



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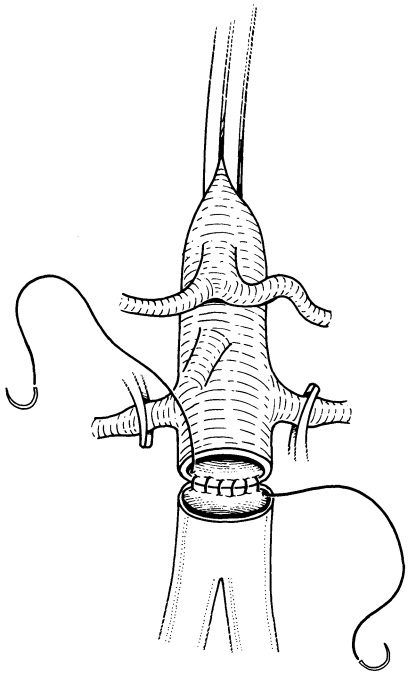


(c)

Figure 3.18(a–c) Juxtarenal aortic occlusion: (a) extent of thrombus. (b) the aorta is transected and the thrombus removed. (c) an aortofemoral PTFE graft is then inserted

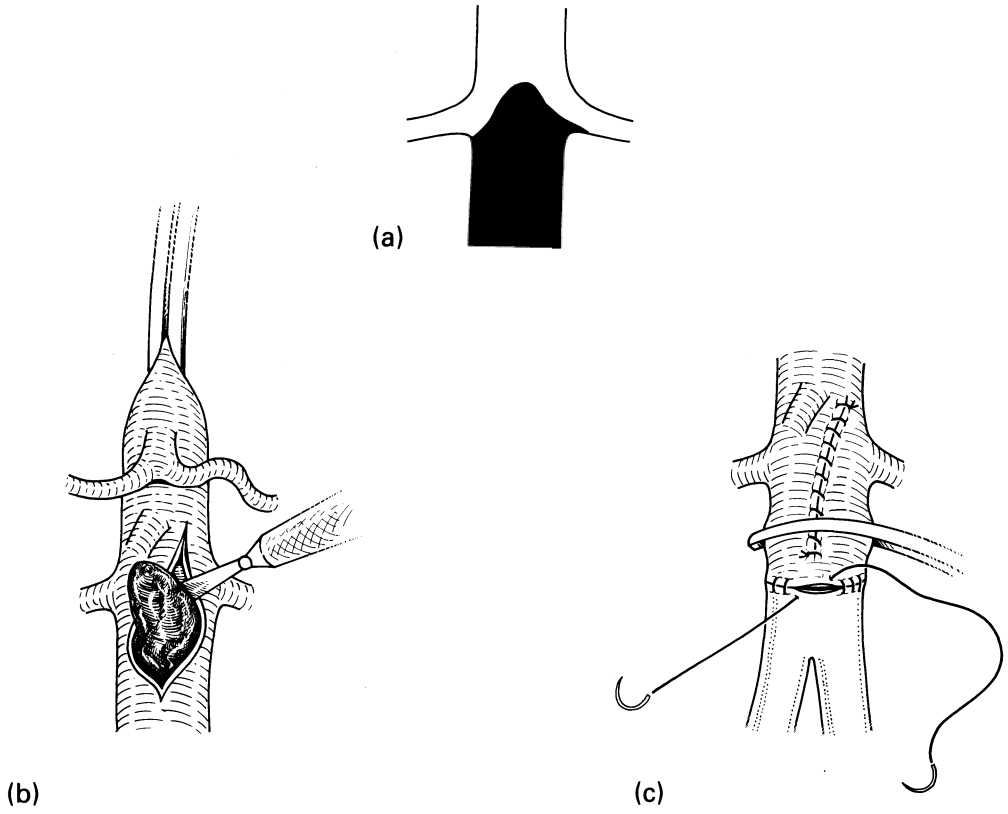


Figure 3.19(a–c) Inter-renal aortic thrombosis: (a) extent of thrombus. (b) thrombectomy via a longitudinal aortotomy. (c) after closing the aortotomy, a rubber-shod clamp is applied to the infrarenal aorta, thereby restoring renal perfusion. A PTFE bifurcated graft is then inserted

femoral bypass graft is then anastomosed end-to-end to the aortic stump (Figure **3.18c**). After the aorta has been flushed, the controlling clamp is transferred to an infrarenal level and flow is restored to the kidneys. The rest of the procedure follows the lines already described.

In cases where the thrombus has already extended partially above the renal artery origins (Figure **3.19a**) a longitudinal aortotomy placed between the renal arteries may provide better access. This approach is also preferable if a concomitant renal endarterectomy is required (see Chapter 13). Once adequate clearance has been achieved, the aorta is closed to below the level of the renal arteries allowing clamp transfer to an infrarenal position (Figure **3.19c**). Graft replacement then follows as above.

ASSOCIATED RENAL OR VISCERAL ARTERY OCCLUSIVE DISEASE

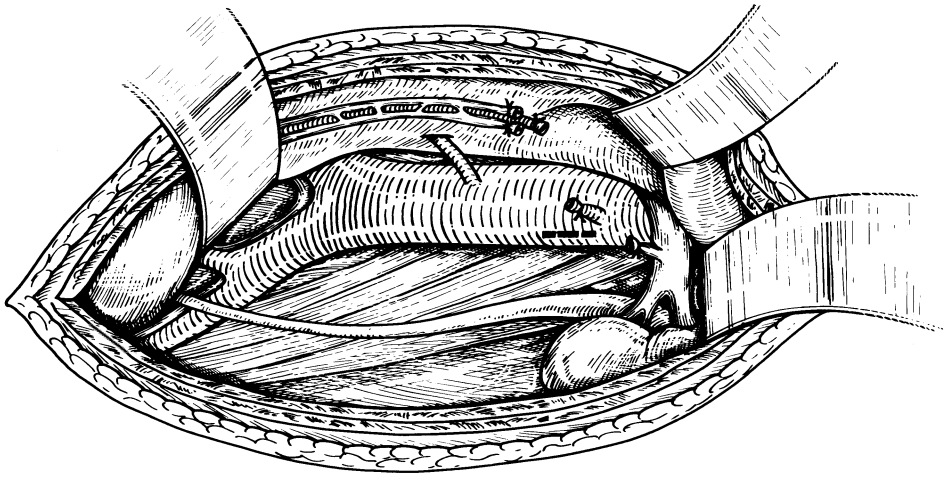
Significant additional renal or visceral artery occlusive lesions are usually dealt with at the time of aortic grafting. Separate Dacron or PTFE limbs attaching to the main prosthesis form the basis of therapy (for details see Chapters 12 and 13).

RETROPERITONEAL AORTOFEMORAL BYPASS

An aortofemoral bypass may be occasionally inserted via a retroperitoneal approach. This provides much less disturbance for the patient than a transperitoneal operation and may be particularly considered in patients with respiratory or cardiac impairment. It is also useful when there have been multiple previous abdominal procedures.

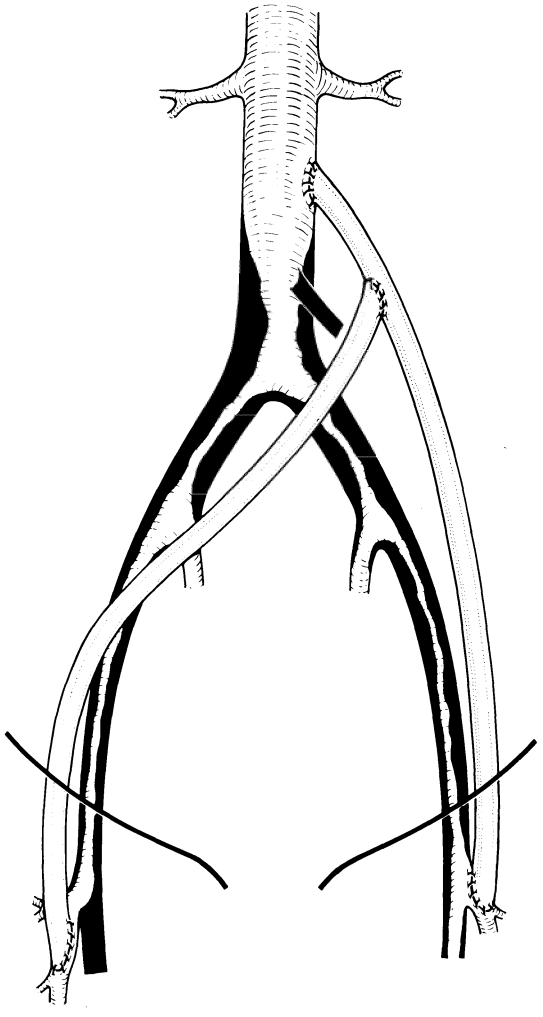
The abdominal wall muscles are cut in a line parallel with and lateral to the edge of the left rectus abdominis and the peritoneum then reflected medially. Access to the proximal aorta is facilitated if the ureter is left on the surface of psoas (a retro-uteric graft trajectory is subsequently adopted to avoid ureteric compression). Medial retraction of the peritoneal sac at the upper limit of the dissection is further helped by dividing the gonadal artery and vein (Figure **3.20a**).

The aorta is clamped close to the left renal vein and an 8 mm PTFE graft anastomosed to the left lateral aortic wall. An estimate is made of the length of graft required to reach the left groin and then a second PTFE graft is anastomosed to the medial border of the first prosthesis. This extension limb is clamped at its origin while the parent prosthesis is anastomosed in the left groin. Once flow has been restored to the left leg the crossover limb is tunnelled behind the peritoneum alongside the right iliac vessels to the right femoral exposure (Figure **3.20b**). Alternatively the crossover may take origin lower down the parent limb and is then routed through the space of Retzius (see Figure **3.23**).



(a)

Figure 3.20(a, b) Retroperitoneal aortofemoral bypass: (a) pre-ureteric approach to the aorta. Note division of the gonadal artery and vein.



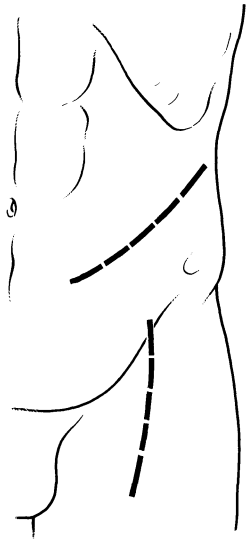
(b)

Figure 3.20 continued (b) PTFE graft anastomosed to the proximal aorta and then to the left common–deep femoral segment. In this example the crossover limb follows the line of the right iliac vessels to reach the right femoral exposure

ILIOFEMORAL BYPASS

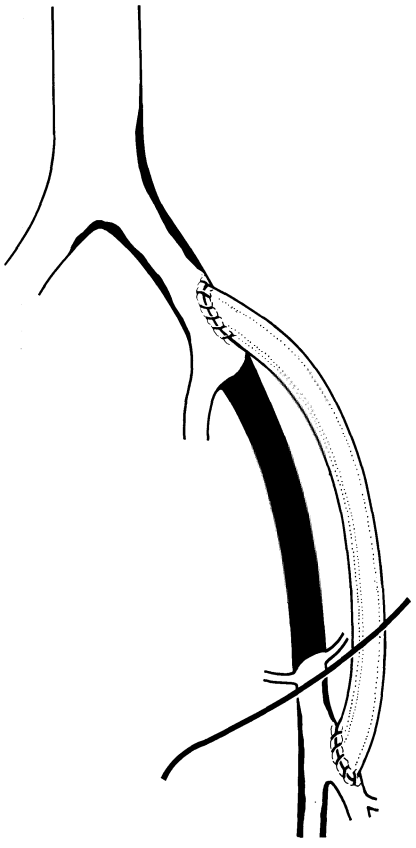
Patients with occlusive disease principally affecting the external iliac segment may be managed by placing a bypass graft between the common iliac and common femoral arteries.[22].

After the customary approach to the femoral vessels, the common iliac artery is exposed by a retroperitoneal flank incision (Figure **3.21a**). A 6 or 8 mm PTFE or Dacron graft is then anastomosed to the lateral wall of the common iliac artery (Figure **3.21b**). It is important to keep this anastomosis well to the lateral side of the vessel to avoid graft compression from the intraperitoneal



(a)

Figure 3.21(a–c) Iliofemoral bypass: (a) incisions



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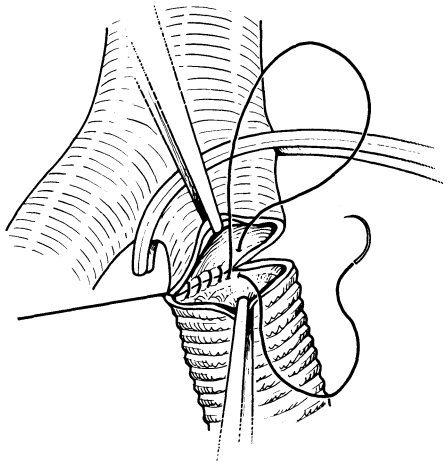


(c)

Figure 3.21 continued (b) PTFE graft bypassing an occluded external iliac artery. (c) end-to-end proximal attachment when the common iliac artery is also occluded

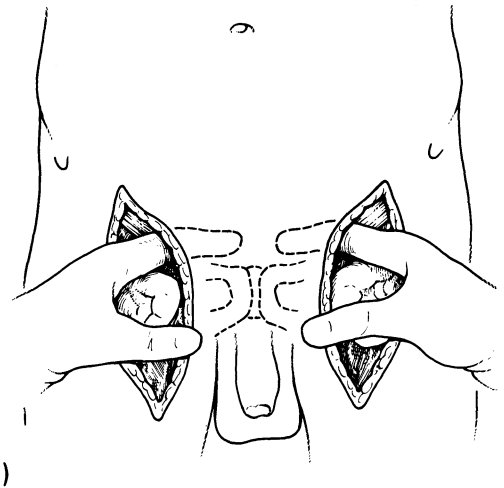
contents when the peritoneal sac is restored to its normal position. In cases where the entire iliac axis is occluded, the common iliac origin may be sectioned and the graft attached in end-to-end fashion with or without a local endarterectomy (Figure **3.21c**). Distally, the graft is tunnelled under the inguinal ligament and attached in the groin, using one of the options previously described.

Where there is additional common iliac stenosis a common iliac endarterectomy should be undertaken prior to iliofemoral graft attachment providing this is unlikely to compromise internal iliac patency (see Figures **3.35–37**). If there is more extensive proximal common iliac disease involving the distal aorta, an alternative method of reconstruction should be employed.

FEMOROFEMORAL BYPASS

Originally, femorofemoral bypass was restricted to elderly or poor-risk cases, or those where previous surgery, sepsis, or radiotherapy precluded a direct approach to the aortoiliac segment[23]. However, the long–term results have been good[24,25] and the procedure may now be considered for any patient with ischaemic symptoms due to unilateral iliac arterial occlusive disease. It is particularly useful for patients with unilateral occlusion of an aortoiliac or aortofemoral prosthesis when simple thrombectomy fails to restore patency. Femorofemoral bypass particularly via the transperineal route may also be used to manage arterial sepsis in the groin (see Chapter 14).

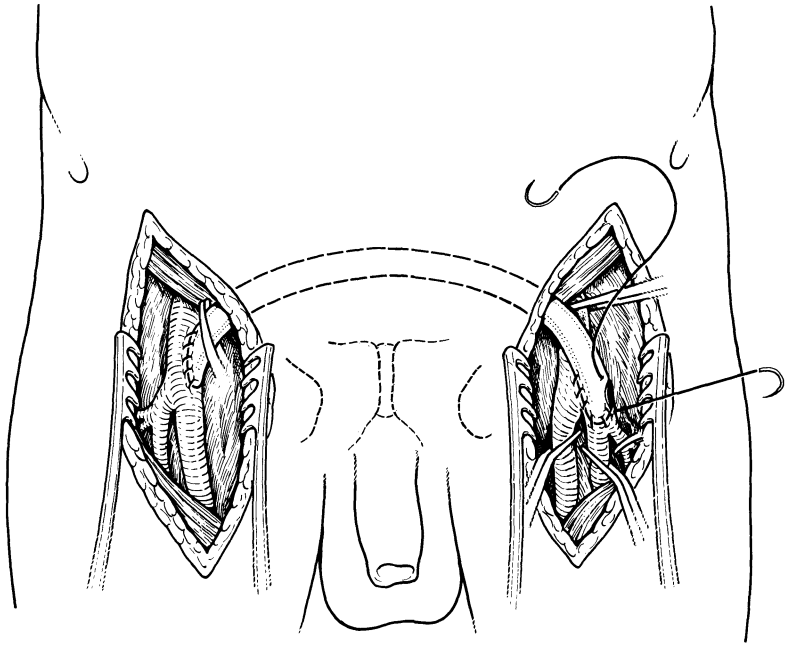
An essential prerequisite in crossover grafting is that the donor iliac system should be free from significant disease. However, preliminary transluminal dilatation of the donor axis may enable high risk patients with a degree of bilateral disease to also be accommodated. Additional distal disease in the donor limb need not contraindicate femorofemoral bypass, though it may be advisable to correct coexistent profunda ostial stenosis at the time of bypass insertion either by endarterectomy or by modifying the bypass attachment.



(a)

The simplest method of femorofemoral bypass is via the subcutaneous suprapubic route. The recipient groin is explored first to establish the adequacy of the run-off. The contralateral common femoral artery is then similarly exposed and a subcutaneous tunnel developed between the two incisions by digital dissection above the pubis (Figure **3.22a**). Although a high implantation site on the donor femoral artery with the tip of the graft pointing in a cephalic direction may have theoretical haemodynamic advantages, in practice the reverse configuration has proved quite satisfactory. A 6 or 8 mm knitted Dacron or PTFE graft is suitable in most cases.

After heparinization a longitudinal arteriotomy is made on the anteromedial wall of the donor



(b)

Figure 3.22(a, b) Femorofemoral bypass: (a) development of a subcutaneous suprapubic tunnel. (b) PTFE graft attached on the donor side, passed through the tunnel and anastomosed to the common–deep femoral segment on the recipient side

common femoral artery and the graft anastomosed in end-to-side fashion. Anastomosis may be facilitated by using the controlling clamps to rotate the medial aspect of the artery anteriorly and sewing intraluminally along the back wall. A rubber-shod or soft jaw clamp is then placed across the graft close to the anastomosis and flow restored to the donor limb. The graft is then passed through the subcutaneous tunnel for attachment on the opposite side.

An alternative method of crossover grafting is to use the contralateral external iliac artery as source, the graft being routed retroperitoneally through the space of Retzius (Figure **3.23**). This affords a greater degree of protection for the graft than the subcutaneous femorofemoral route, and is particularly advantageous in re-do cases as it avoids the need to open the previously dissected groin.

The donor external iliac artery is exposed via a short retroperitoneal incision above the inguinal ligament. A tunnel is then developed digitally through the space of Retzius and the inguinal ligament is partially divided to allow the graft to exit on the recipient side. Sufficient length should be allowed when cutting the graft to ensure a smooth curve at its take-off and at the attachment to the femoral vessels.

A third possible method of crossover grafting utilizes the transperineal route. This approach may be necessary when there is arterial sepsis in the groin and is described in Chapter 14.

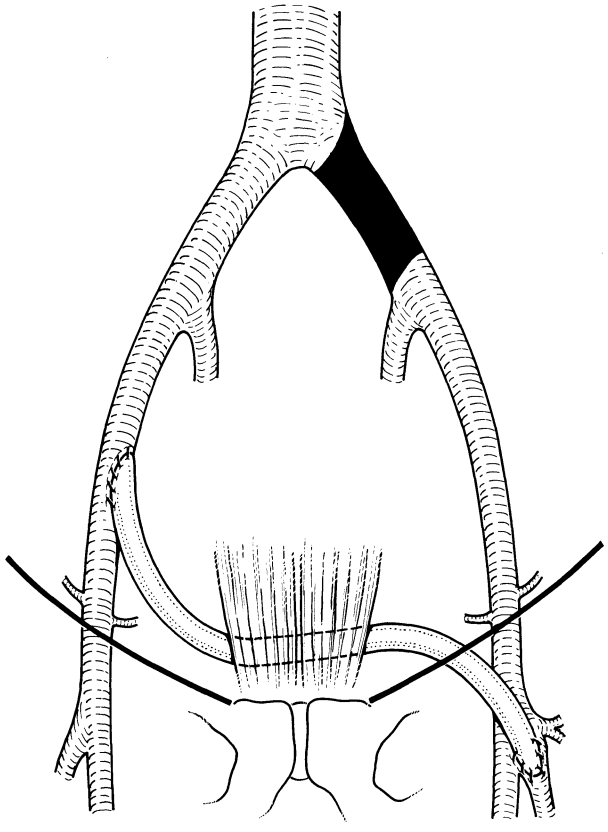
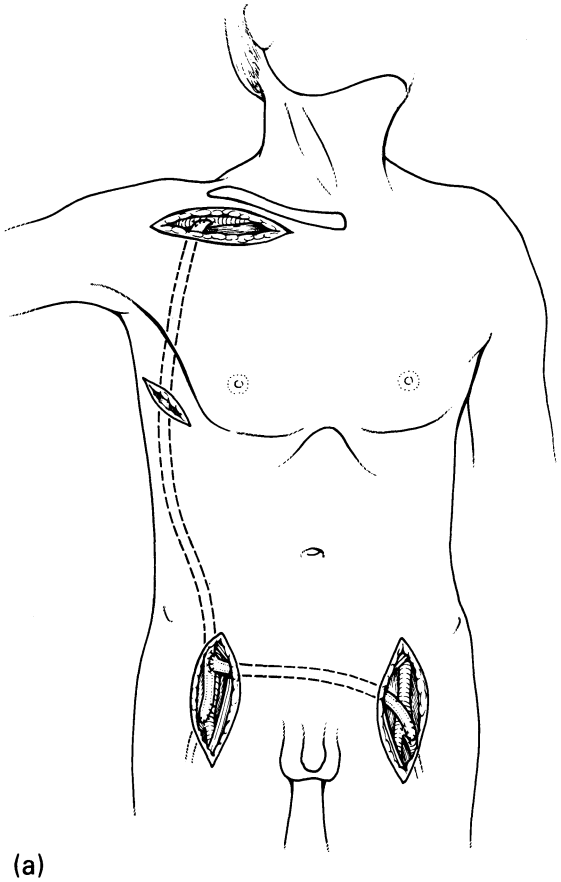


Figure 3.23 Iliac–femoral crossover graft via the retroperitoneal (Retzius) route

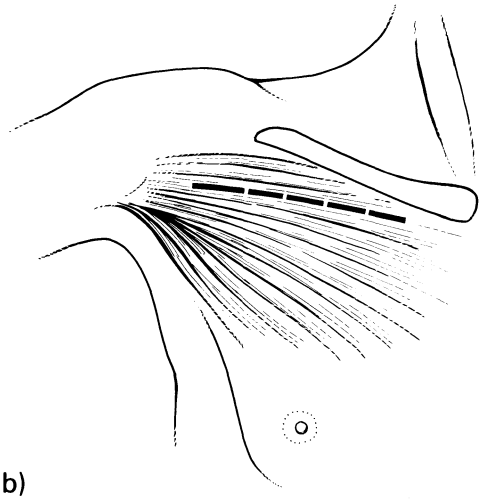
AXILLOFEMORAL BYPASS

This method is an alternative to direct aortoiliac reconstruction in poor risk cases or whenever abdominal surgery is contraindicated by local factors such as sepsis, radiotherapy or previous operation[26]. It requires superficial tissue dissection only and may even be done under local anaesthesia[27], so that its potential in poor risk cases is obvious. However, durability is less than in-line aortic grafting[28,29] though some centres have reported reasonable long-term results[30,31]. Our experience is in agreement with the former view and we have restricted axillofemoral bypass to patients with limb-threatening ischaemia who cannot be managed by any other means. Axillofemoral bypass may also be considered in some high risk patients with aortic aneurysmal disease, though its role here is strictly limited[32]. A remaining indication for axillofemoral bypass is in the management of aortic graft sepsis or secondary aortoenteric fistulae (see Chapter 14).

Most commonly one axillary artery acts as donor source for both lower extremities (Figure **3.24a**). Graft flow is thereby increased compared with unilateral bypass and patency may be enhanced[33]. Ordinarily the donor arm is on the same side as the leg with the worst run-off so as to produce a high flow rate in the crossover section. In septic cases ease of tunnelling around the groin or around other sites of potential contamination may dictate the choice of axillary artery. If there are no clear pointers, the right axillary artery is preferred as this is less likely to be affected by proximal atherosclerotic progression in the subclavian artery. In all cases an adequate subclavian-axillary inflow is essential and this may be determined by clinical and Doppler examination. If doubt remains, digital subtraction or conventional arch aortography should be performed before proceeding.



(a)



(b)

Figure 3.24a–g Axillofemoral bypass: (a) graft arrangement. (b) infraclavicular approach, splitting pectoralis major

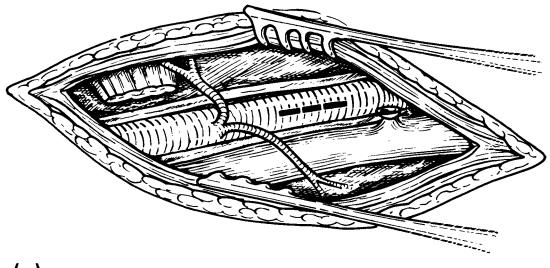
The patient is placed in the supine position with the trunk slightly tilted towards the contralateral side and the donor arm abducted at something less than 90° from the trunk. The operative field is prepped to include the base of the neck, the shoulder and upper arm, the ipsilateral hemithorax, both lower quadrants and the upper thighs. If a supplementary femoro-distal bypass or an extension to the popliteal–tibial segment is likely to be necessary, the entire lower limb should also be prepped. An adhesive plastic drape is particularly useful in this operation to avoid contact between the prosthesis and the large area of skin.

The femoral vessels are first exposed to establish the status of the run-off on either side. An approach is then made to the proximal segment of the axillary artery. This is the preferred site for anastomosis, as the graft then pursues a gentle curve to the chest wall and traction on the anastomosis as a result of arm movement is avoided. A skin incision is made from below the medial end of the clavicle in the direction of the edge of the deltoid muscle (Figure **3.24b**). The pectoralis major fibres are split to expose the clavipectoral fascia. The latter is then incised and the artery identified by palpation. The vessel is progressively cleared with division of the branches of the acromiothoracic artery and accompanying veins as necessary. Usually the tendon of pectoralis minor is also divided to obtain sufficient space. A Silastic loop is passed around the artery, and used to apply gentle traction as the vessel is mobilized back towards its emergence from beneath the clavicle (Figure **3.24c**). The only branch arising from this most proximal segment is the superior thoracic artery which should be divided.

In patients of short stature it may be possible to create a tunnel all the way from the axilla to the groin, using a tunnelling instrument. However, in most patients a relay incision is utilized on the lateral chest wall. This incision should be placed obliquely over the sixth or seventh intercostal space in the midaxillary line. It is important not to place the graft anterior to the midaxillary line because it may kink when the patient bends or sits down. The graft course should also be offset from the relay incision in case of postoperative wound complications.

A tunnel is then developed by digital dissection between the axillary exposure and the relay incision. This tunnel usually passes in front of the axillary vein and then deep to pectoralis major and the deep fascia of the chest wall. Distally the tunnel is extended to the groin either in a subcutaneous plane or deep to external oblique aponeurosis[26]. A tape may then be drawn into the tunnel as a guide for subsequent graft passage. Finally a suprapubic tunnel is made for the femorofemoral extension to the opposite groin.

After heparinization, clamps are applied to the proximal axillary artery and a longitudinal arteriotomy is made on the antero-inferior aspect. An 8mm PTFE graft is selected and after suitable bevelling is anastomosed in end-to-side fashion with 5/0 prolene (Figure **3.24d**). The graft is then



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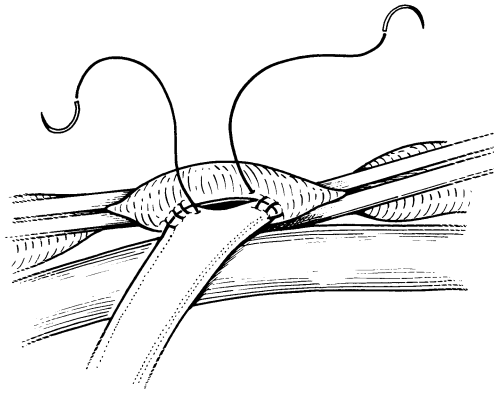


(d)

Figure 3.24 continued (c) axillary artery exposed by division of pectoralis minor and the clavipectoral fascia. (d) PTFE graft attached to the axillary artery

filled with blood under arterial tension and drawn down the pre-formed tunnel to the groin (Figure **3.24e**). Prior distension of the graft in this manner aids assessment of graft length and avoids the risk of torsion.

Once a decision has been made on the future site of the distal anastomosis the graft is clamped at its origin and emptied of blood. A 6 mm PTFE graft is then attached end-to-side to the medial wall of the axillofemoral prosthesis close to the distal end (Figure **3.24f**). An alternative option if the common femoral artery is of reasonable quality is to attach the crossover limb directly to the arterial axis[33].

The distal anastomosis of the axillofemoral limb is then completed after carefully flushing any air or debris via the unattached end of the crossover graft. The latter is then clamped at its origin and flow released firstly into the iliac axis and then distally into the leg.

The crossover limb is then passed through the suprapubic tunnel to the opposite groin. The graft is cut to length and attached to the common-deep femoral segment or to the profunda as appropriate (Figure **3.24g**). The proximal common femoral artery on this side may then be ligated to reduce competitive flow. However, in the presence of severe common iliac occlusive disease this step may be omitted in order to preserve retrograde internal iliac perfusion.

After a final check on haemostasis the wounds are irrigated with antibiotic–saline or antiseptic solution and the incisions closed with suction drainage.

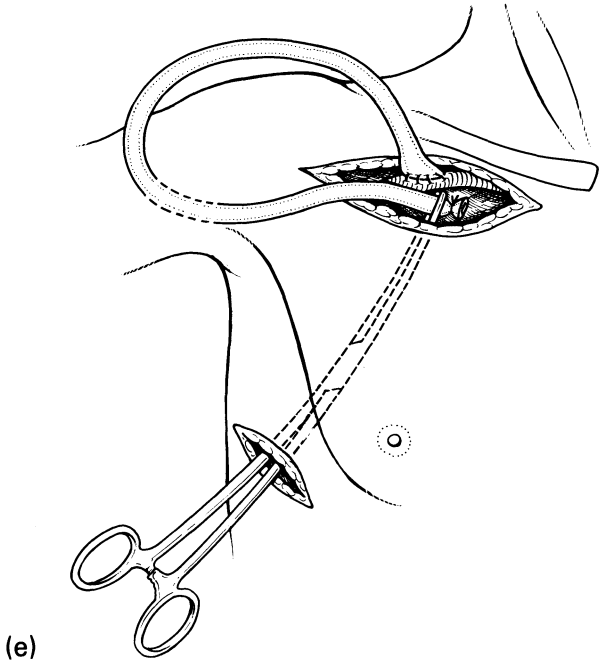


Figure 3.24 continued (e) graft tunnelled via the relay incision to the groin

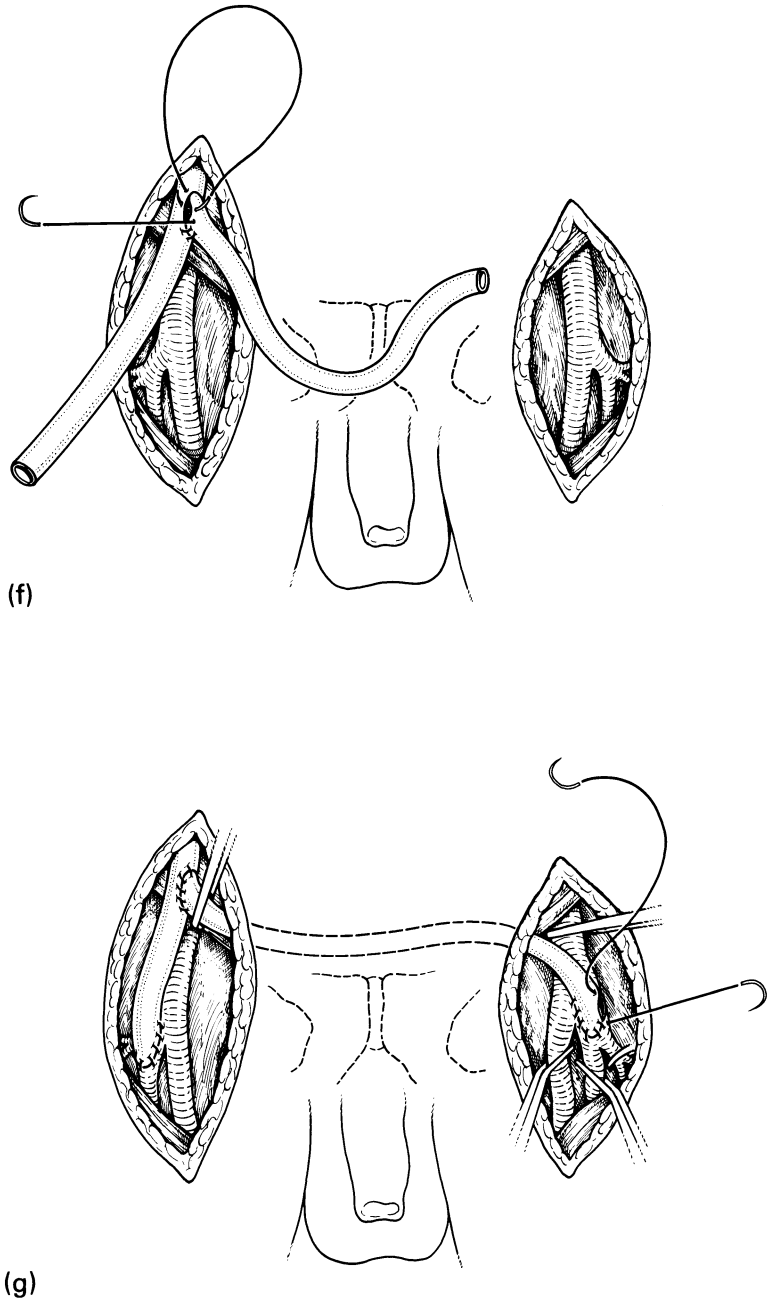


Figure 3.24 continued (f) crossover limb attached. (g) the ipsilateral groin anastomosis has been completed and the crossover graft is tunnelled to the opposite side for distal attachment

ENDARTERECTOMY OF THE AORTA AND COMMON ILIAC ARTERIES

In a minority of patients, perhaps no more than 10%, the occlusive process is localized to the terminal aorta and common iliac arteries with relatively normal vessels beyond. Endarterectomy is a suitable method of treatment in these cases. This may be accomplished using open or semi-closed techniques in various combinations[34,35] or by the section–eversion procedure described below (Figures **3.25–28**).

A xiphopubic transperitoneal approach is made to the aorta. It is important to palpate the external iliac arteries at the outset, because in many cases the disease is more extensive than may be apparent from the preoperative angiogram. If scattered or lengthy lesions are found in the external iliac arteries an aortofemoral bypass should be utilized in preference to an extended endarterectomy.

If endarterectomy seems appropriate, the peritoneum over the aorta is incised and this incision is then extended down the right iliac axis to the common iliac bifurcation. A separate peritoneal incision is usually required to display the left common iliac bifurcation beyond the root of the sigmoid mesocolon (see Figure **2.3**).

The peritoneal incision is then deepened through the periaortic fascia and the aorta progressively mobilized within this plane. By staying deep to the periaortic sheath, damage to the autonomic nerve plexus may be kept to a minimum. Great care must be taken in freeing the aorta because there is usually a certain amount of loose friable debris within the lumen and distal embolization is a constant threat. Successive pairs of lumbar arteries may be identified by cautious digital dissection posteriorly. Proximally the aorta should be freed up to the level of the left renal vein. The region of the inferior mesenteric artery origin should be left undisturbed and this vessel subsequently controlled by a distally placed clamp or a double-looped Silastic loop.

Attention is then paid to the proximal segment of each common iliac artery. On either side, but particularly on the left, the hypogastric nerves may be identified as a bundle passing over the anterior surface of the artery to join the presacral hypogastric plexus (see Figure **14.20**). This bundle should be carefully freed from the underlying

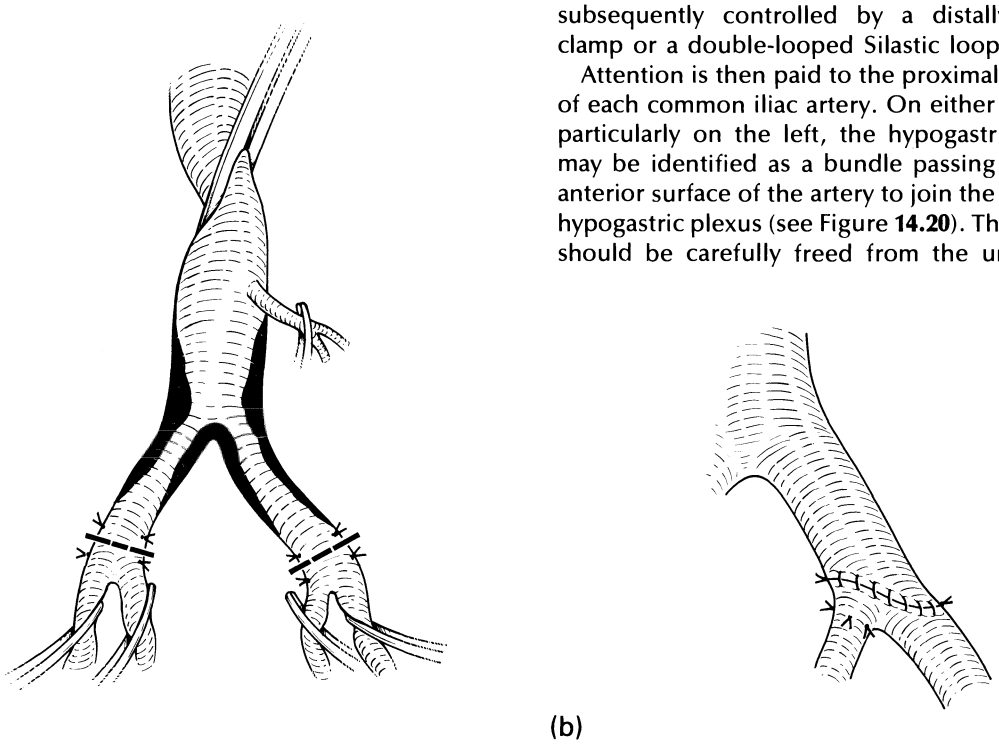


Figure 3.25(a, b) Endarterectomy of the distal aorta and common iliac arteries: (a) extent of atherosclerosis and common iliac transection sites indicated. Note marker stitches for realignment. (b) variant using oblique iliac transection. In this example the intima at the internal iliac origin has been controlled with tack-down stitches

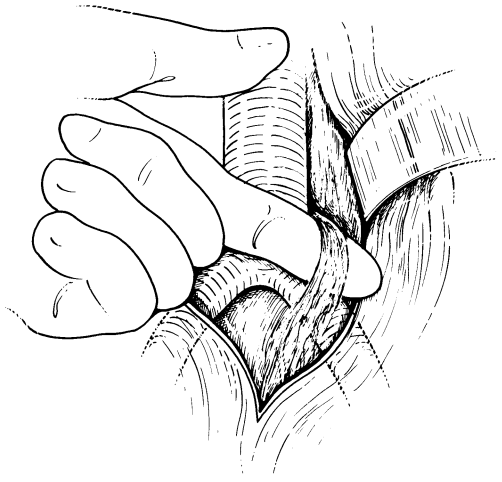


Figure 3.26 Hypogastric nerve fibres separated from the common iliac artery

common iliac artery so that at a later stage the artery can be divided at its distal end and delivered to an anterior position. The common iliac bifurcation and its branches are then cleared and after heparinization, clamps are applied to the internal and external iliac arteries and to the proximal infrarenal aorta. Additional bulldog clamps may be required to control the lumbar and median sacral vessels.

Marker stitches are placed on either side of the proposed line of transection in the distal common iliac arteries. These stitches will facilitate correct alignment during re-anastomosis. Each common iliac artery is then transected 1 cm or so proximal to its bifurcation. An oblique line of section (Figure **3.25b**) may give improved access in case a limited endarterectomy of the distal stump is also required, and will produce a larger anastomotic circumference. However, re-anastomosis is more difficult than in the case of a transverse section.

The left common iliac artery is then delivered from under the sigmoid mesocolon and transposed anteriorly to the hypogastric nerve fibres (Figures **3.26, 27a**). The artery is held under slight longitudinal tension and a central atheromatous core developed proximally with a spatula. The remainder of the vessel wall is then turned back on itself and the core developed into the aorta (Figure **3.27b**). The same procedure is then undertaken on the right side. Continued use of the

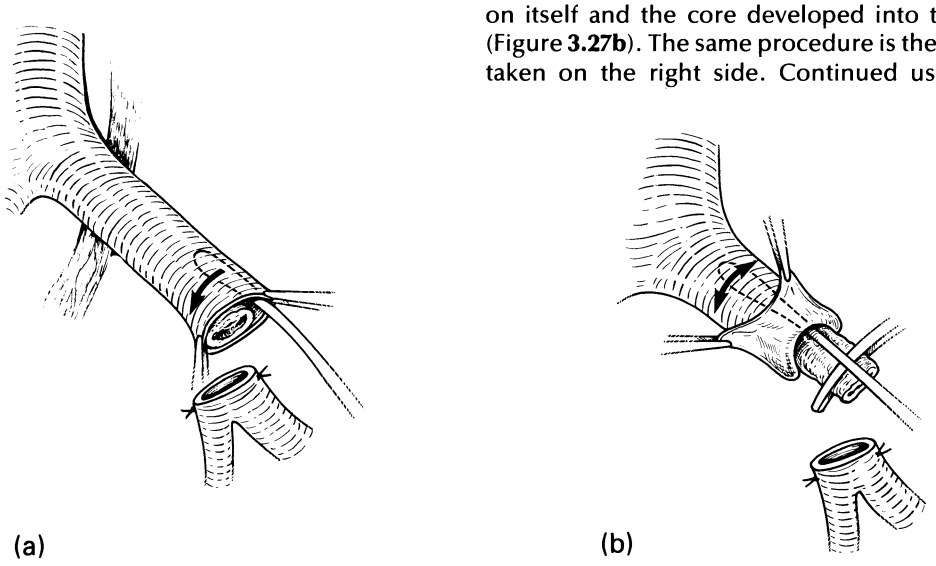


Figure 3.27(a–h) Endarterectomy by section–eversion: (a) the distal common iliac artery is transected and transposed anterior to the hypogastric nerve bundle. A central atheromatous core is then developed proximally. (b) the outer wall of artery is turned back on itself as the core is progressively separated

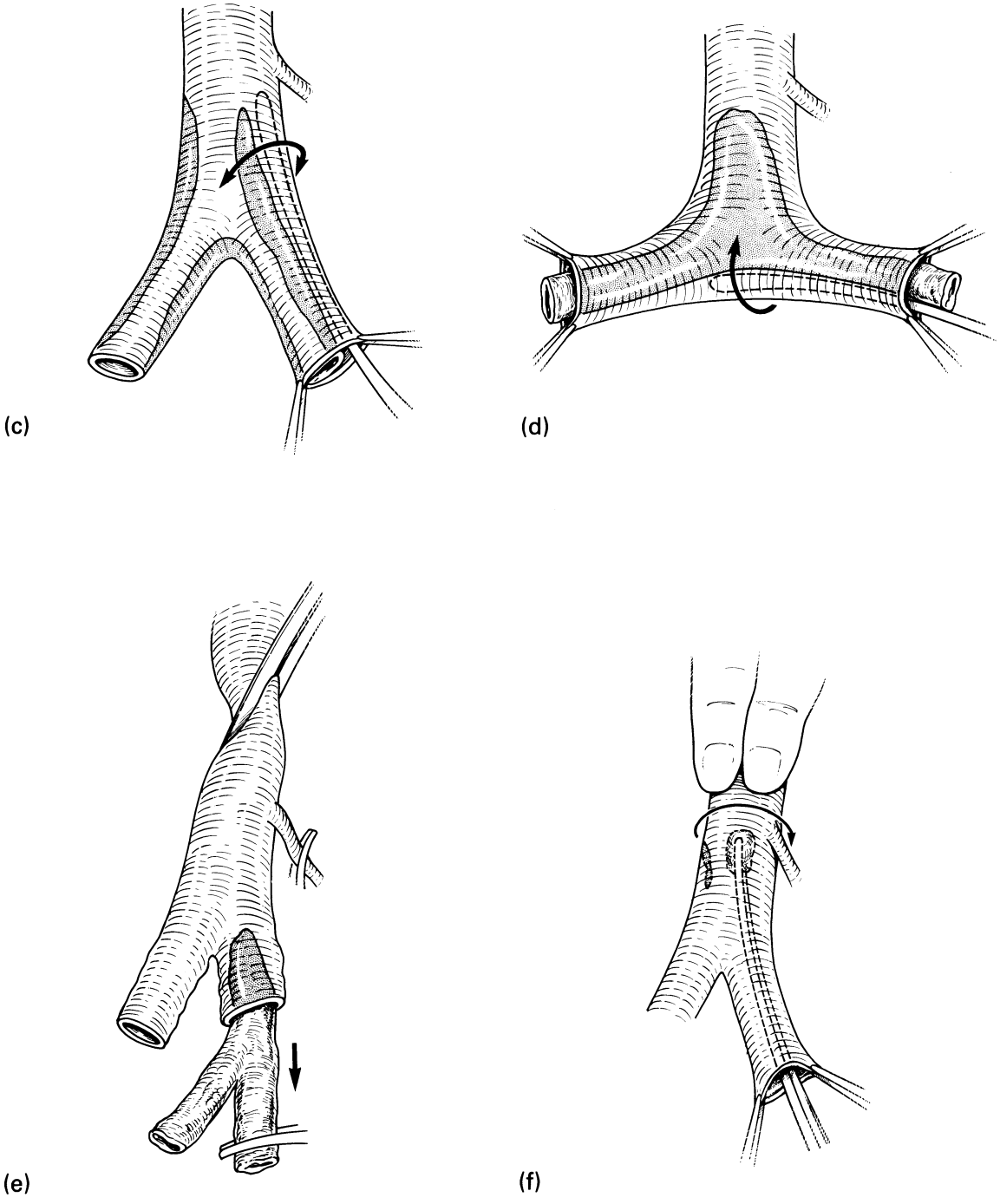
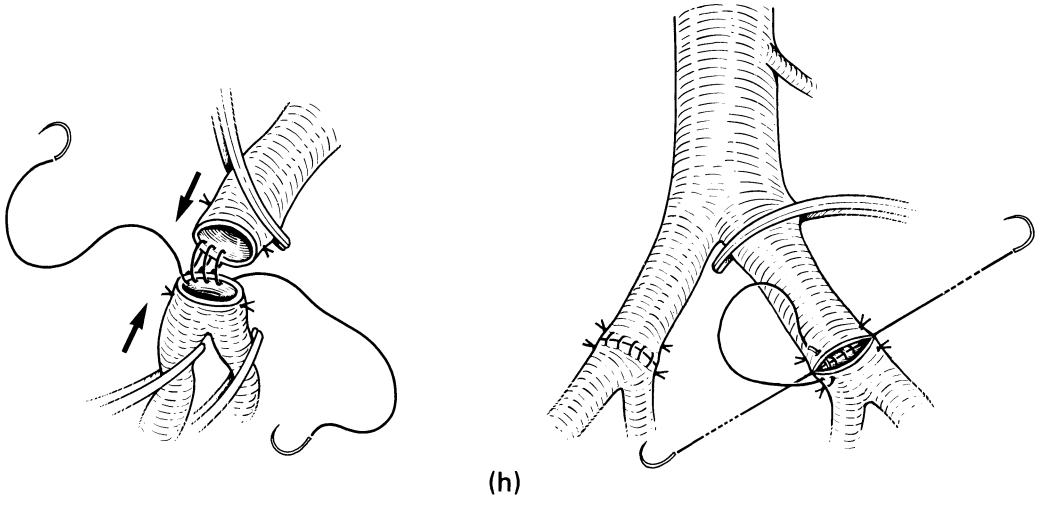


Figure 3.27 continued (c) the aortic atheroma is separated with a spatula. (d) freeing the bifurcation. (e) the atheromatous core is extracted. (f) the remaining debris is removed by wiping out with a moist pledget



(g) (h)

Figure 3.27 continued (g) re-anastomosis of the right common iliac artery. The clamps are approximated to take tension off the suture line. (h) right iliac re-anastomosis completed. Left side in progress

spatula will detach the core from the front, back and side walls of the aorta as far as the proximal limit of the gross disease. Only the extreme angle of the aortic bifurcation then remains to be freed. The common iliac arteries are held apart to abolish this angle and the spatula passed along the inferior surface of the bifurcation to separate the remaining plaque (Figure **3.27d**). The atheromatous core is then fractured against the aortic clamp and removed via one of the iliac arteries, if necessary in a piecemeal fashion.

Any residual loose fragments within the aorta may be detached by irrigation or by wiping out with a moist pledget (Figure **3.27f**) after which the aortic clamp is briefly released. Clearance may be judged to be complete by palpation and by the appearances on distension with heparin–saline. If doubt remains the aorta should be opened and the lumen inspected directly (see Figure **3.28a–d**).

If case selection has been correct, the transection site in the common iliac artery should be distal to the main atherosclerotic zone. The distal common iliac stump should then require little attention. However, in some patients a tongue of atheroma may extend into or just beyond the common iliac bifurcation. This plaque may be removed using a Cannon spatula or Beaver blade in retrograde fashion. If this manoeuvre results in an unsatisfactory distal edge, a supplementary transverse arteriotomy should be made in the proximal external iliac artery and the intima secured. Further measures may also be necessary to clear the internal iliac artery origin (see Figures **3.35–37**).

Once the endarterectomy is complete, vascular continuity is restored by end-to-end iliac re-anastomosis. On the left side this will necessitate passing the artery back under the root of the sigmoid mesocolon to its former position. Re-anastomosis is facilitated by approximating the two controlling clamps so that the sutures can be inserted without tension (Figure **3.27g**). The posterior wall is then completed using an intraluminal running stitch starting at either the inner or outer border. A second suture is inserted and tied to the first opposite the starting point to provide lateral traction (Figure **3.27h**). The midpoint anteriorly is then marked off and the anterior wall closed in two halves. On completion the clamps are flushed through the contralateral iliac axis and flow restored to the limb. A similar anastomosis is then undertaken on the opposite side.

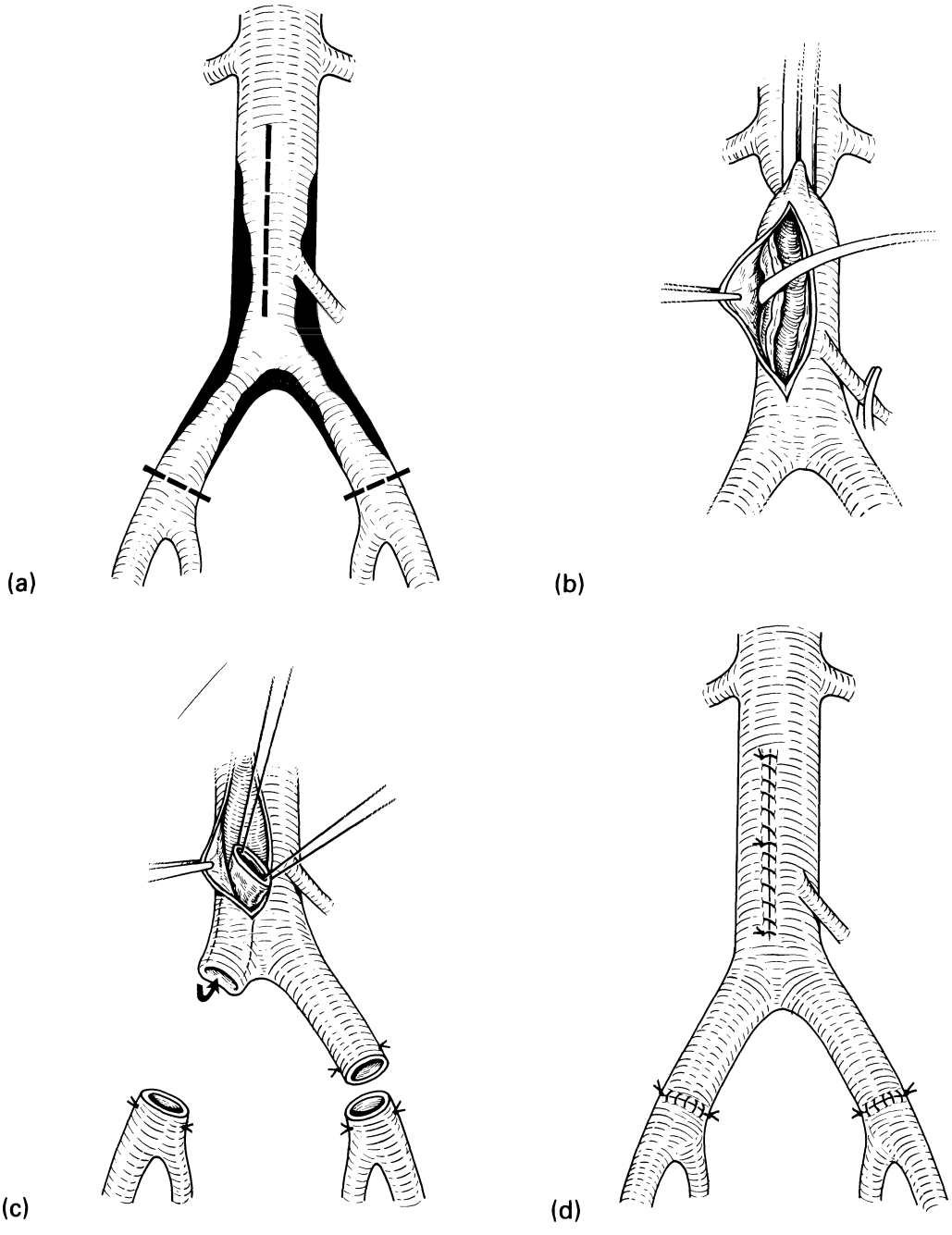


Figure 3.28(a–d) Aortoiliac endarterectomy when there is significant aortic atherosclerosis proximal to the bifurcation: (a) distribution of atheroma. (b) open endarterectomy of the infrarenal aorta. (c) after section–eversion, each common iliac artery is turned inside-out via the aortotomy to allow complete inspection of the interior. Any remaining intimal fragments are then removed. (d) the aortotomy is closed and the common iliac arteries are re-anastomosed

The above technique is satisfactory for dealing with disease which is strictly localized to the distal aorta and common iliac arteries. When the proximal infrarenal aorta is also significantly involved, an additional aortotomy is required to ensure adequate proximal clearance. An open endarterectomy of the infrarenal aorta, including the inferior mesenteric origin, is first accomplished (Figure **3.28a, b**). The bulk of the atheroma is then cleared from the common iliac arteries by section–eversion as described above. The right common iliac artery is then turned inside-out on itself via the proximal aortotomy (Figure **3.28c**). This manoeuvre allows the interior of the entire vessel to be inspected and any residual debris can be completely removed. The vessel is then restored to normal alignment before dealing with the left side in similar fashion. Finally the aortotomy is closed, usually in two sections starting from each end. After flushing and rinsing the arterial lumen, the common iliac arteries are then re-anastomosed (Figure **3.28d**).

This method affords the same degree of control as a completely open endarterectomy without the need to incise the aortoiliac junction. This is an important advantage as a longitudinal arteriotomy across the common iliac origin is difficult to close without inducing some degree of stenosis. In addition open endarterectomy is more likely to result in hypogastric nerve damage and postoperative sexual dysfunction.

COMBINED ENDARTERECTOMY AND BYPASS

Occasionally endarterectomy of the aorta–common iliac arteries may be combined with iliofemoral bypass. One indication for this combined procedure is when difficulty has been encountered in terminating an aortoiliac endarterectomy due to underestimated external iliac disease. A second indication may arise in younger male patients with lower limb ischaemia and impotence due to severe occlusive disease in the aorta and both iliofemoral axes (Figure **3.29a**). An aortofemoral bypass is the most straightforward method of revascularizing the legs but there may be little improvement in internal iliac perfusion and impotence may be unrelieved.

The first stage in a combined procedure is to endarterectomize the aorta. The common iliac arteries are then cleared by retrograde stripper passage from a distal longitudinal arteriotomy. This particular method of endarterectomizing the

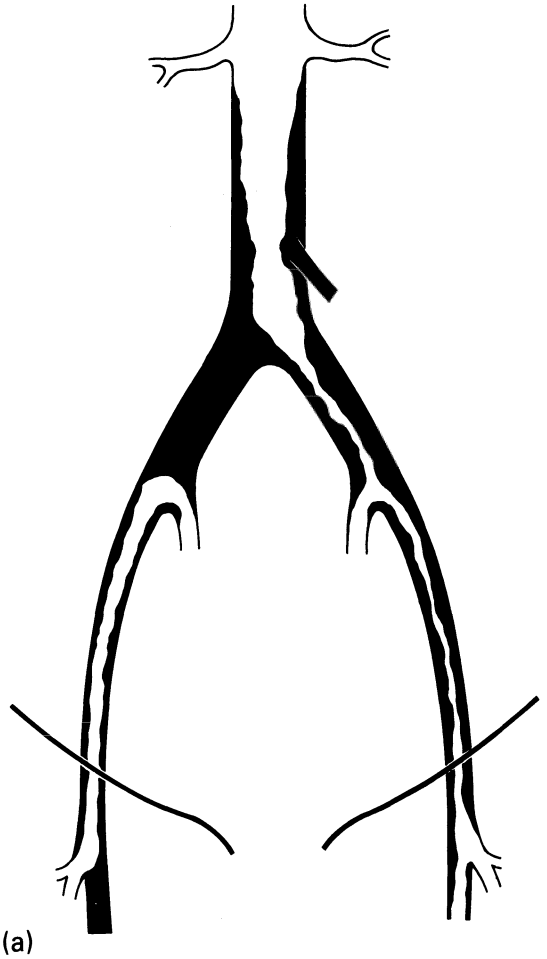
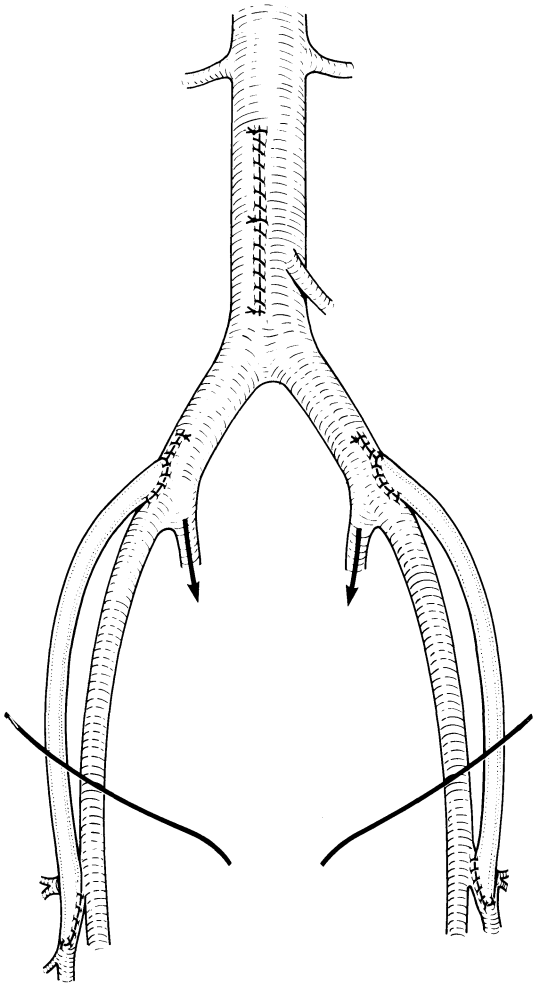


Figure 3.29(a, b) Combined aortoiliac endarterectomy and bilateral iliofemoral bypass: (a) distribution of atheroma in a younger male patient with leg ischaemia and impotence

common iliac arteries is chosen to maximally safeguard the hypogastric nerve plexus. Particular attention is then paid to clearing the internal iliac artery on either side (see Figures **3.35–37**). Finally each common iliac arteriotomy is closed using the lower part as attachment for an iliofemoral PTFE or vein graft (Figure **3.29b**).



(b)

Figure 3.29 continued (b) initial endarterectomy of the aorta–common iliac arteries will restore antegrade internal iliac perfusion. A PTFE iliofemoral bypass is then inserted on either side

ILIOFEMORAL ENDARTERECTOMY

Severe occlusive disease may occur in the common and external iliac arteries without comparable aortic involvement. Often, symptoms are confined to one extremity only, despite bilateral angiographic changes. One method of treating these cases is by aortobifemoral bypass in the likely expectation of eventual disease progression. In other cases, preliminary iliac dilatation and crossover grafting or in-line iliofemoral bypass may be appropriate. However, a third option is to perform a unilateral iliofemoral endarterectomy on the symptomatic side. This can be readily achieved by a retroperitoneal approach which provides direct access to the iliac axis with minimum morbidity[36,37]. In the event of late failure or disease progression on the opposite side, correction by a transperitoneal aortic bypass operation is relatively easy.

An important advantage of iliofemoral endarterectomy is that it has the potential for reopening the internal iliac territory, a feature which may not be afforded by bypass grafting. The external iliac artery is a difficult vessel to endarterectomize by conventional methods due to the adherent nature of its medial layer and the eccentric distribution of the atheroma. An eversion manoeuvre is probably the best method of ensuring complete clearance of this vessel and this may be achieved by several methods in addition to that described below[9,36,38]. Early results have been excellent though there is a continuing attrition rate with time, as the endarterectomized external iliac artery seems particularly prone to progressive intimal thickening and atheromatous recurrence (see Chapter 14).

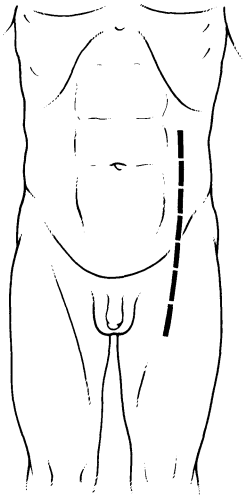
Technique

Operation is commenced in the groin to confirm the adequacy of the profunda femoris. A longitudinal muscle-cutting incision is then made lateral to the edge of rectus abdominis and in continuity with the groin exposure (Figure **3.30a**). The inguinal ligament should be divided to fully expose the iliofemoral junction which is a critical zone in the endarterectomy. Careful subsequent re-suture of the inguinal ligament will avoid any risk of postoperative herniation. The peritoneum,

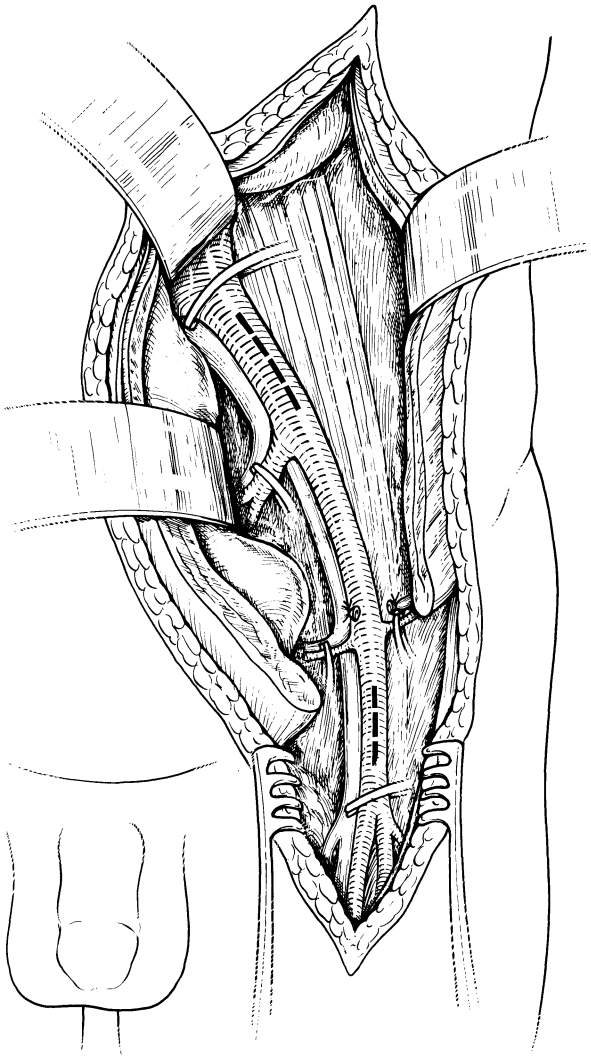
including the ureter, is then swept medially off iliopsoas to expose the iliac vessels (Figure **3.30b**).

The common iliac artery is mobilized sufficiently to allow a curved clamp to be subsequently placed at its origin. This dissection may necessitate interrupting some of the hypogastric nerve fibres. Other fibres passing posteriorly from the lumbar sympathetic chain can usually be preserved, and of course the nerve fibres on the contralateral side remain intact. Adherence to the common iliac vein may be a major problem, particularly on the right side. If venous injury occurs it may be more prudent to change to a bypass method of revascularization (see Chapter 14). The internal and external iliac arteries are then mobilized, the latter by dividing the deep circumflex iliac vein at its distal end. However, the inferior epigastric and deep circumflex iliac arteries should be carefully preserved as these may be an important source of subsequent collateral flow.

After heparinization, clamps are applied to the common and internal iliac arteries, to the com-



(a)



(b)

Figure 3.30(a, b) Exposure for iliofemoral endarterectomy: (a) skin incision. (b) muscle-cutting approach with division of the inguinal ligament and medial reflection of the peritoneum

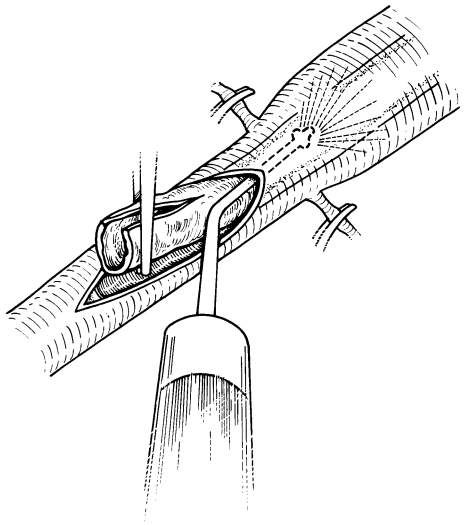
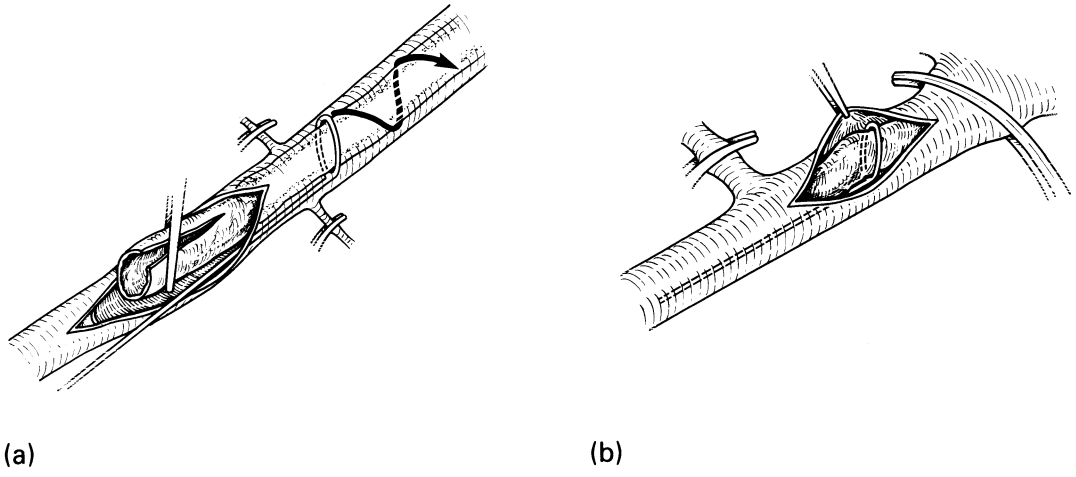


Figure 3.31 The common femoral artery is opened and an atheromatous core developed proximally by hydrodissection

mon femoral bifurcation and to all intervening branches (Figure **3.30b**). Endarterectomy is commenced in the common femoral artery, leaving clearance of the profunda femoris until a later stage. A central atheromatous core is developed proximally, first with a spatula and then by hydrodissection (Figure **3.31**). Separation is completed with a Vollmar ring stripper of suitable size (Figure **3.32a, b**). A longitudinal incision is then made in the common iliac artery and the dissected atheromatous core amputated and subsequently withdrawn from the femoral arteriotomy along with the ring stripper. In some cases the cleavage plane initiated at femoral level is trans–medial (see Chapter 1). If this is the case the additional sleeve of media should be removed from the common iliac artery since endarterectomy in the external plane is preferable here (Figure **3.33a, b**).



(a) (b)

Figure 3.32(a, b) A Vollmar ring stripper is passed up to the common iliac clamp and the core removed

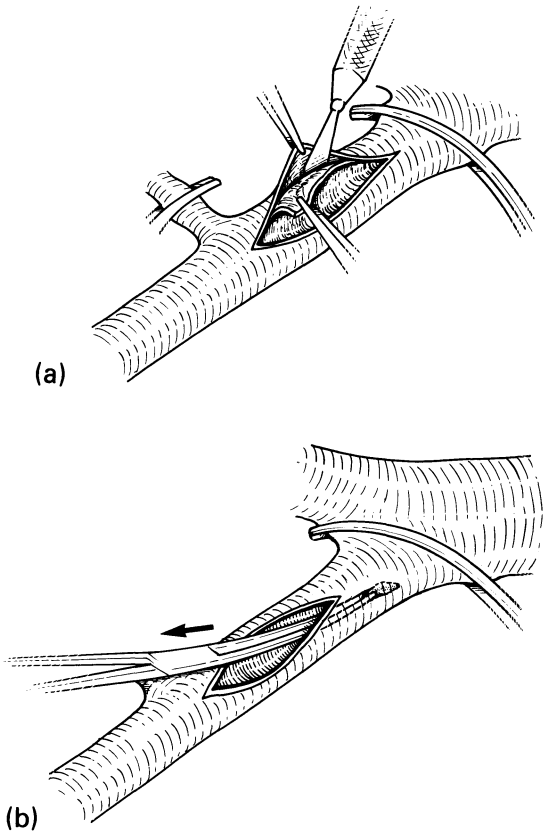


Figure 3.33 (a) sleeve of media removed from the common iliac artery when the initial cleavage plane has been too superficial. (b) residual atheroma removed from the origin of the common iliac artery. Note oblique clamp position to facilitate full clearance

The next stage is to turn the external iliac artery inside-out via the common iliac arteriotomy. The external iliac artery is accordingly transected immediately proximal to the inferior epigastric and deep circumflex iliac branches after placing marker stitches for subsequent realignment (Figure **3.34a**). Several 6/0 prolene sutures are inserted in the proximal cut edge of the vessel and these are tied to a small ring stripper passed down the vessel from above. Proximal withdrawal of the stripper then turns the artery inside-out (Figure **3.34b**). If an external dissection plane has been followed, there may be only a few remaining shreds of media to be removed from the internal aspect of the external iliac artery (Figure **3.34c**). If the trans–medial plane has been employed cleavage is not usually uniform and patches where the media has been scuffed may be evident throughout its length. In this case, the remainder of the media should be carefully peeled away. Once clearance has been completed, the external iliac artery is restored to normal alignment (Figure **3.34d**).

Attention is then paid to clearing the internal

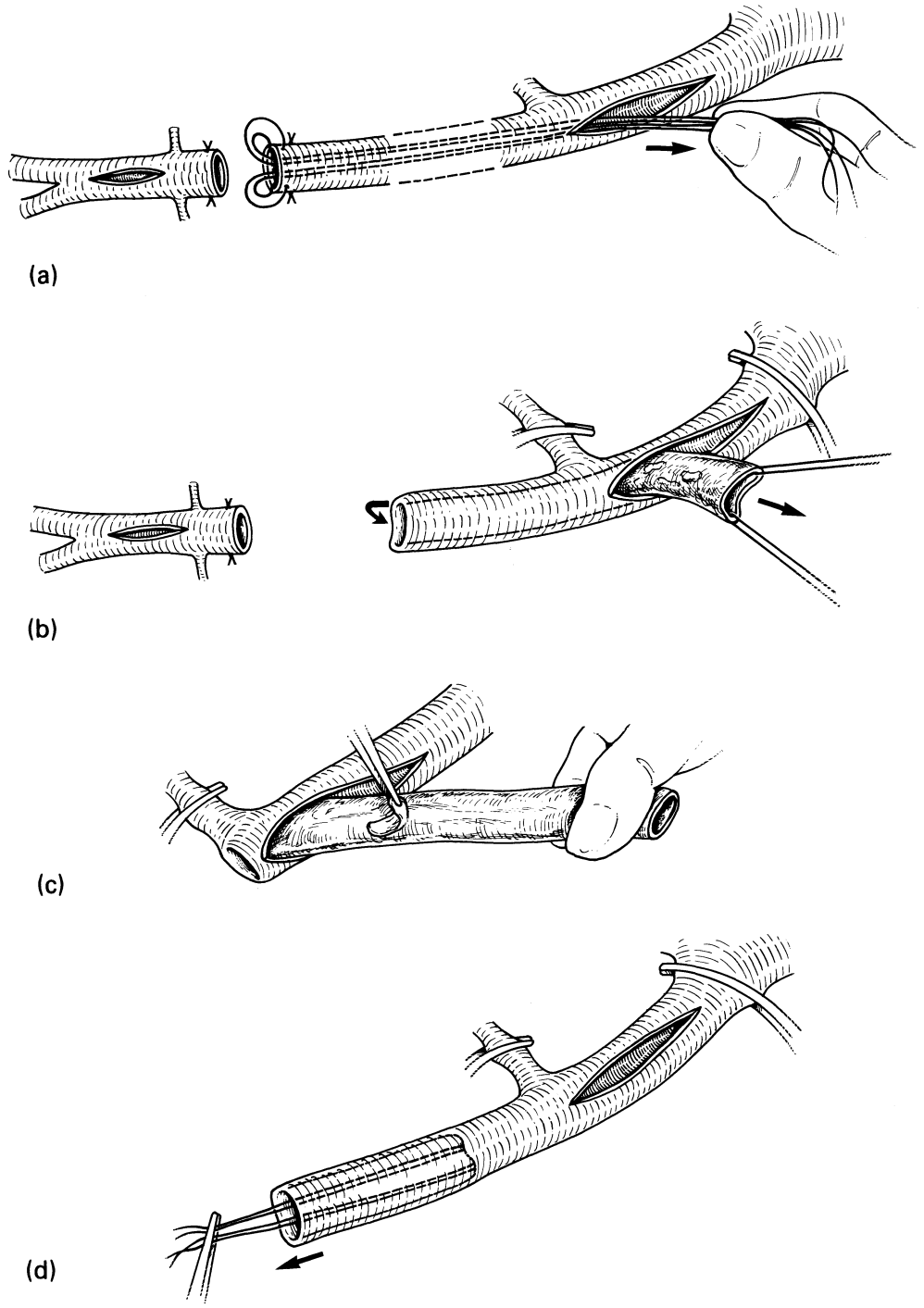


Figure 3.34 (a, b) the external iliac artery is turned inside-out via the common iliac arteriotomy, using traction sutures placed through the cut edge. (c) residual shreds of media removed. (d) artery restored to normal alignment

iliac artery. Ostial plaque may be separated with a spatula as the internal iliac artery is progressively everted into the common iliac lumen (Figure **3.35b**). More extensive plaque may be amenable to a similar procedure providing the bifurcation of the internal iliac artery is not involved (Figure **3.36a**). Any intimal fragments remaining after endarterectomy should be removed by wiping out with a moist pledget mounted on a small artery forceps (Figure **3.36b**).

If the bifurcation is involved, iliofemoral (or indeed aortoiliac) endarterectomy should not be attempted, since acute occlusion of the internal iliac artery is likely and one of the main benefits of the endarterectomy process will be lost.

In other patients the internal iliac artery is either

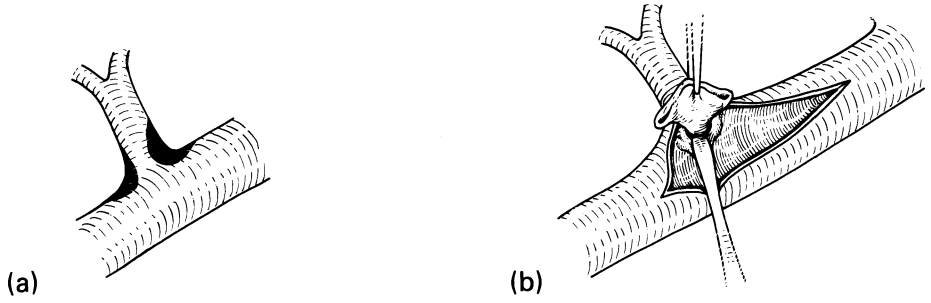


Figure 3.35(a, b) Localized plaque at the internal iliac orifice removed by eversion endarterectomy

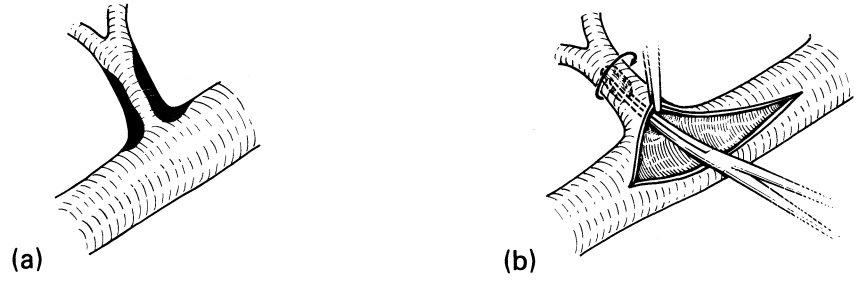


Figure 3.36(a, b) More extensive internal iliac atheroma. After eversion endarterectomy any remaining loose intimal fragments are removed by wiping out with a pledget

normal or minimally thickened (Figure **3.37a**). The best option in these cases is to arrest the common–external iliac endarterectomy at the internal iliac origin. Tack-down stitches are then used to control the intimal edge and prevent a dissection (Figure **3.37b**).

Once satisfactory clearance of the common and internal iliac arteries has been achieved, the common iliac arteriotomy is closed in two sections starting at each end. A soft clamp is then placed across the external iliac origin and flow restarted in the internal iliac territory (Figure **3.38**). Attention is now turned to re-anastomosing the distal external iliac artery and to completing the common femoral endarterectomy. It is usually preferable to extend the initial common femoral arteriotomy proximally to reach the external iliac transection site so that all shreds of media can be removed from the intervening segment (Figures **3.38, 39**). Distally the endarterectomy is usually

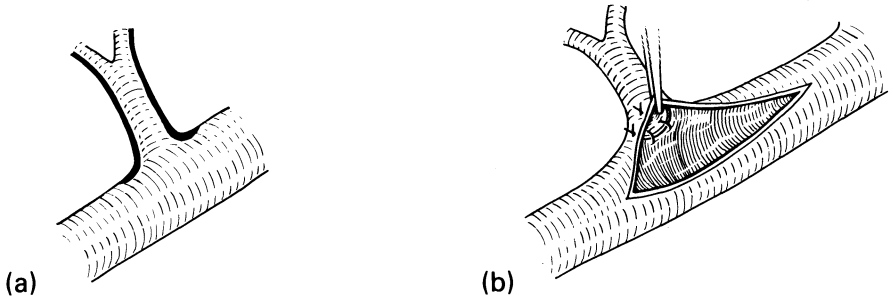


Figure 3.37(a, b) Minimal intimal thickening of the internal iliac artery. Endarterectomy should be limited to the common–external iliac axis with tack-down sutures to control the intima at the internal iliac origin

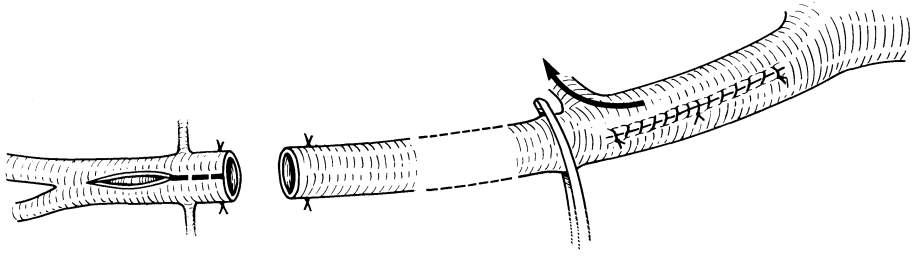


Figure 3.38 The common iliac arteriotomy is closed and flow restarted in the internal iliac territory. The common femoral arteriotomy is then extended proximally to allow complete clearance

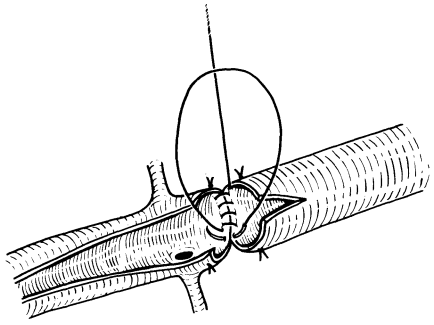


Figure 3.39 The common femoral arteriotomy is extended to meet the external iliac transection site and the remaining fragments of media are removed. Re-anastomosis of external iliac artery commenced

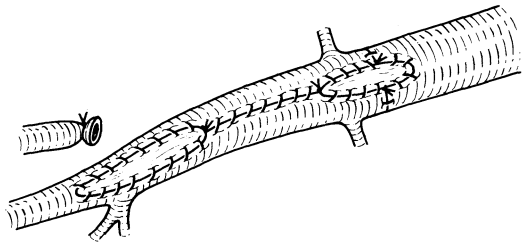


Figure 3.40 Completed distal end of the reconstruction. In this case the occluded superficial femoral artery has been detached and a profunda endarterectomy carried out with vein patch closure. An additional patch has been placed at the iliofemoral re-anastomosis site to avoid late stenosis

taken into the profunda femoris, though additional manoeuvres may be necessary if the superficial femoral artery is still patent (see Chapter 4). A vein patch is then used to close the profunda. The common femoral artery is closed by direct suture since extending the patch into this segment may invite late aneurysmal dilatation. Finally, the external iliac artery is re-anastomosed in end-to-end fashion with a small anterior vein patch to avoid late stenosis (Figure **3.40**).

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4

Surgery of the Common Femoral Bifurcation

The profunda femoris and its branches constitute a system which runs in parallel with the superficial femoral artery and which communicates with the popliteal and infrapopliteal vessels via a rich anastomotic network around the knee (Figure **4.1**). When the superficial femoral artery is occluded, the profunda femoris becomes the essential route for revascularizing the lower limb either in conjunction with an aortoiliac reconstruction[1,2] or as an isolated event[3]. It is the latter usage that is considered in the present chapter.

In order for isolated profunda surgery to succeed, certain criteria must be fulfilled. Thus inflow into the groin should be unimpeded, there should be a significant (>50%) stenosis or occlusion of the proximal profunda, the distal profunda and branches should be disease-free with well-developed collateral connections to the popliteal-tibial segment, and the distal run-off should be good[4,5]. In practice these criteria are not always met and this may detract from the clinical response.

Proximal profunda stenosis may be best identified by arteriography using oblique views. It is important not to overlook an additional aortoiliac lesion and adequate views of this segment together with the distal run-off should be obtained as a routine. As at other sites, arteriography may sometimes underestimate the extent of the disease process and a more accurate assessment may be provided by operative examination. This may require exposing the profunda and its branches

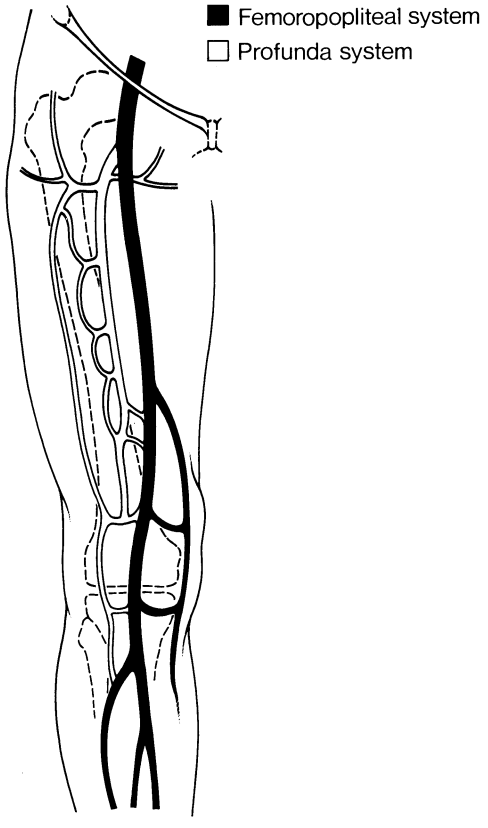


Figure 4.1 Anastomotic connections of the profunda femoris

down to the second or even the third perforating artery.

The degree of collateralization between the profunda and the popliteal–tibial vessels may be assessed by arteriography[6] or vascular laboratory criteria[7]. However it must be admitted that these methods are only approximate and the functional value of the collaterals may remain uncertain.

The quality of the distal run-off also has a decisive influence on the clinical response and is usually easier to document[8,9]. Unfortunately a poor run-off is a frequent finding in limb salvage cases and as a result the overall success of isolated profunda surgery in this group is no more than 50%[10]. Long-term results may be even less rewarding[11]. Among claudicants the proportion responding may be higher, though complete relief is much less frequent than after femoro–distal bypass because of the inherently greater resistance to flow offered by the thigh collaterals[10].

These considerations, together with the recent technical advances in femoro–distal grafting, have led to a marked reduction in the frequency with which isolated profunda reconstruction is now undertaken either for limb salvage or claudication. Moreover, many patients with proximal profunda stenosis can be managed by transluminal angioplasty[12] and this has obvious appeal in a condition where the surgical results remain difficult to predict.

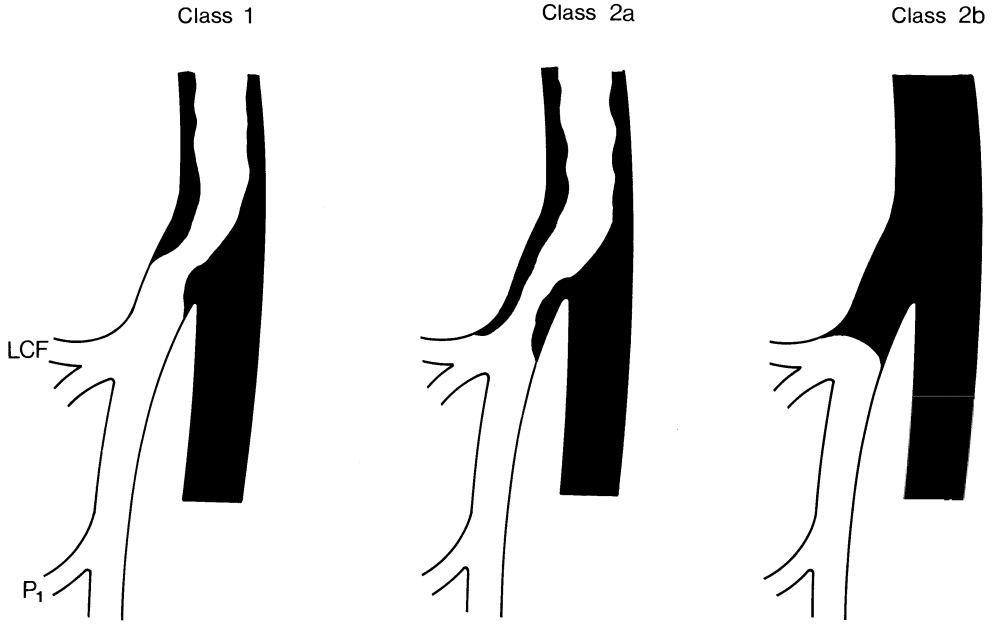


Figure 4.2 Pathology of the profunda femoris: class 1 – ostial stenosis; class 2 – main trunk involved with (a) stenosis, (b) occlusion

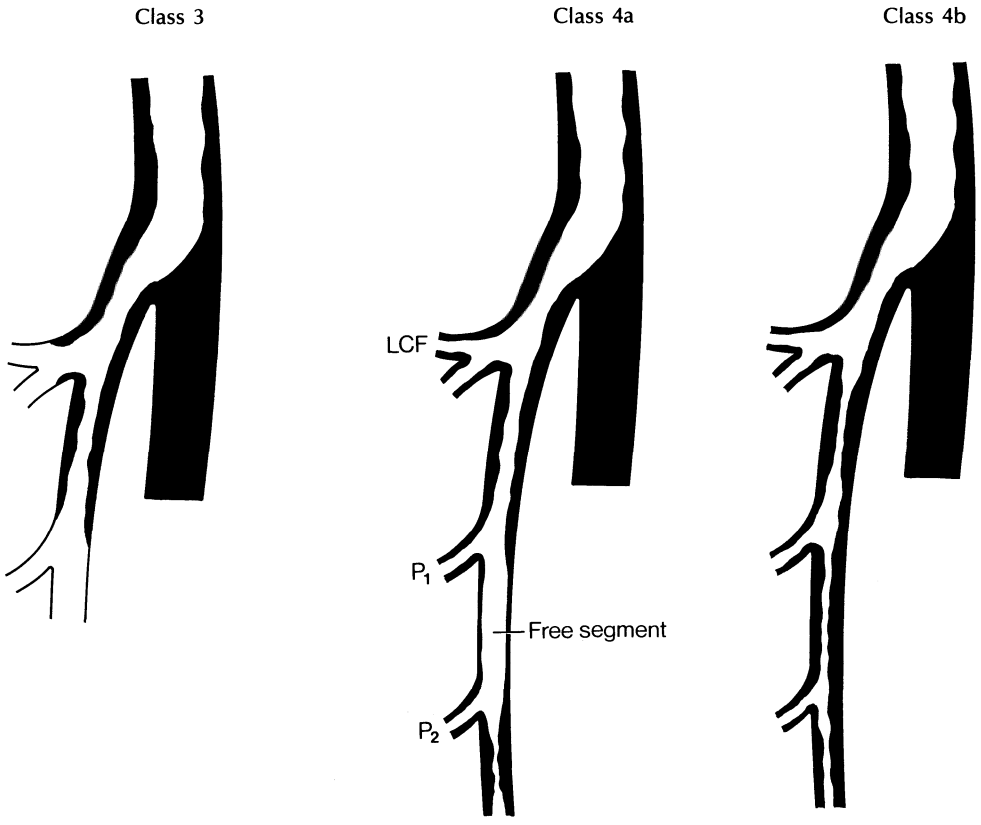


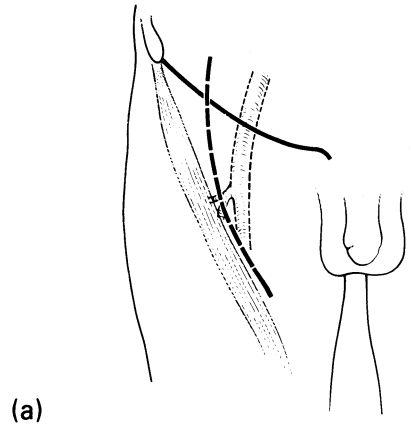
Figure 4.2 continued class 3 – atheroma extending beyond the lateral circumflex femoral artery (LCF) into the perforator trunk; class 4 – perforator trunk and branches (P_1, P_2, etc.) involved with (a) free segment, (b) total involvement

PATHOLOGY OF THE PROFUNDA FEMORIS

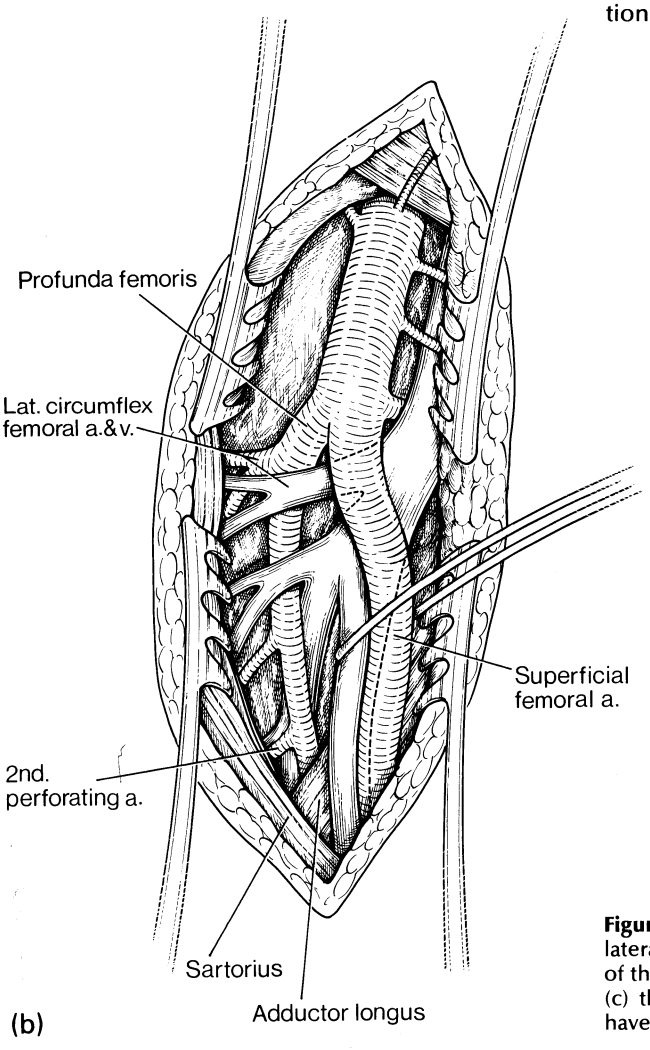
The operative indications and the method of profunda reconstruction are influenced by the extent of atheromatous involvement. Four pathological classes may be recognized (Figure **4.2**). In classes 1 and 2, which make up almost 90% of cases, surgery is easy, there is little risk of an immediate postoperative occlusion and a good functional result may be anticipated when the occlusive process is severe (e.g. class 2b). In class 3, acute postoperative thrombosis is again unlikely, but long-term patency is reduced, with 50% occlusion at 5 years in our own cases. Surgery should therefore be undertaken only for severe ischaemia. In class 4a, reconstruction may still be possible by means of a bypass graft implanted onto the free segment, but the presence of disease in the profunda branches means that the functional benefit is likely to be small. Reconstruction should therefore only be considered when there are no other therapeutic options available and the patient is faced with a major amputation. In class 4b, reconstruction of any kind is contraindicated because of the major risk of acute thrombosis leading to an unnecessarily high amputation.

EXPOSURE OF THE COMMON FEMORAL BIFURCATION AND PROFUNDA FEMORIS

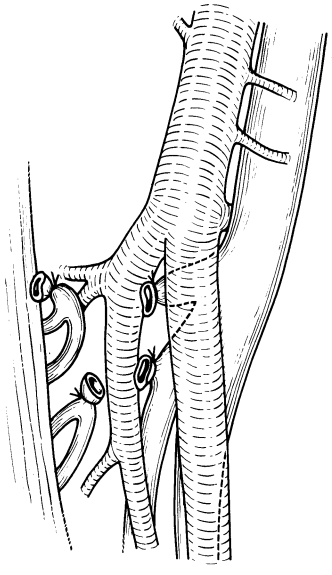
The patient is placed supine with the thigh externally rotated and slightly flexed over a sandbag. The arterial axis should be approached from the lateral side to avoid cutting into the lympho-adipose tissue which lies directly in front of the femoral sheath. Accordingly a skin incision is made commencing two finger-breadths above the inguinal ligament and extending distally with a slight curve along the medial border of sartorius for 15–20 cm (Figure **4.3a**). This incision is then deepened to the medial border of sartorius and the lymphatic fat pad reflected in a medial direction. The deep fascia is opened at the edge of



(a)



(b)



(c)

Figure 4.3(a–c) Exposure of the profunda femoris: (a) lateral incision avoiding lymphatic pad. (b) dissection of the profunda down to the second perforating artery. (c) the lateral circumflex femoral ± perforating veins have been divided to fully expose the profunda

sartorius and this incision is then extended up to the inguinal ligament and distally to the apex of the femoral triangle. This displays the femoral sheath which is now opened along its entire length. Distally, the saphenous nerve, which perforates the femoral sheath to descend alongside the superficial femoral artery, should be preserved to avoid troublesome postoperative neuralgia.

The common femoral artery is then mobilized taking care to avoid avulsing the superficial epigastric and/or circumflex iliac branches (Figure **4.4a**). The superficial femoral artery is freed for several centimetres and encircled with a Silastic loop. A similar loop is passed around the common femoral artery and gentle traction applied to bring the profunda origin into view. This is usually located 3–5 cm distal to the inguinal ligament (Figure **4.4a**). However, a more proximal or distal origin is not uncommon (Figure **4.4b, c**) and for this reason it is advisable as a routine to commence the arterial dissection at the level of the inguinal ligament and work distally. In the majority of cases the main trunk of the profunda descends for 2–3 cm before giving rise to the lateral circumflex femoral artery. Occasionally, however, the profunda trunk divides into two or three branches of approximately equal size immediately after its origin (Figure **4.4d**). This variation is the least favourable for endarterectomy.

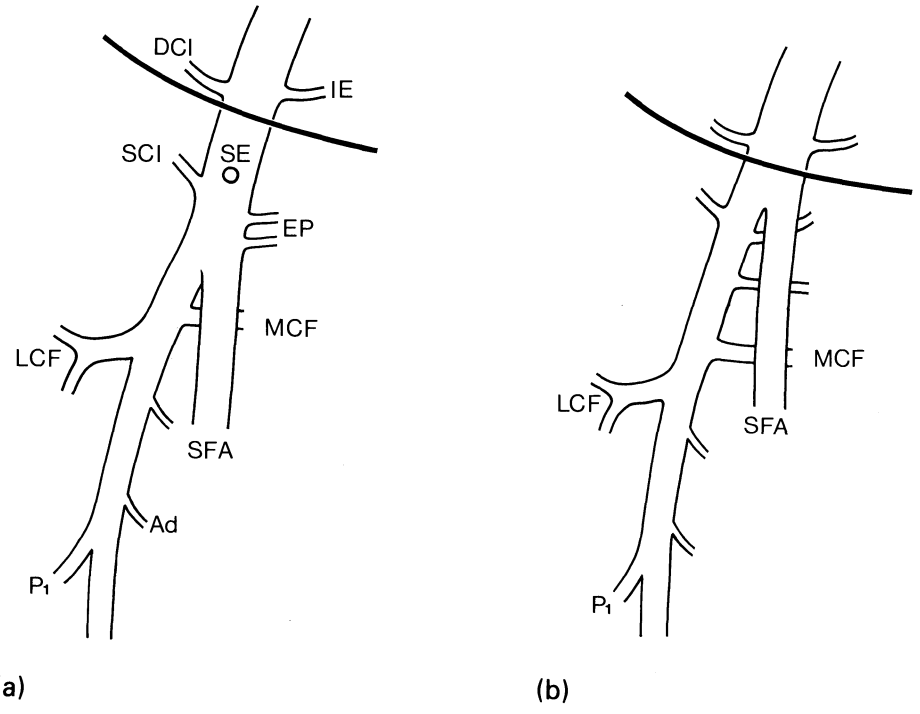


Figure 4.4(a–d) Some of the anatomical variations in the profunda femoris. *Key*: Arteries: DCI = deep circumflex iliac; IE = inferior epigastric; SCI = superficial circumflex iliac; SE = superficial epigastric; EP = external pudendal (superficial and deep); MCF = medial circumflex femoral; LCF = lateral circumflex femoral; P_1, P_2, etc., = perforators; Ad = adductor branches; SFA = superficial femoral.
(a) most frequent disposition. (b) high bifurcation of the common femoral artery with all branches arising from the profunda

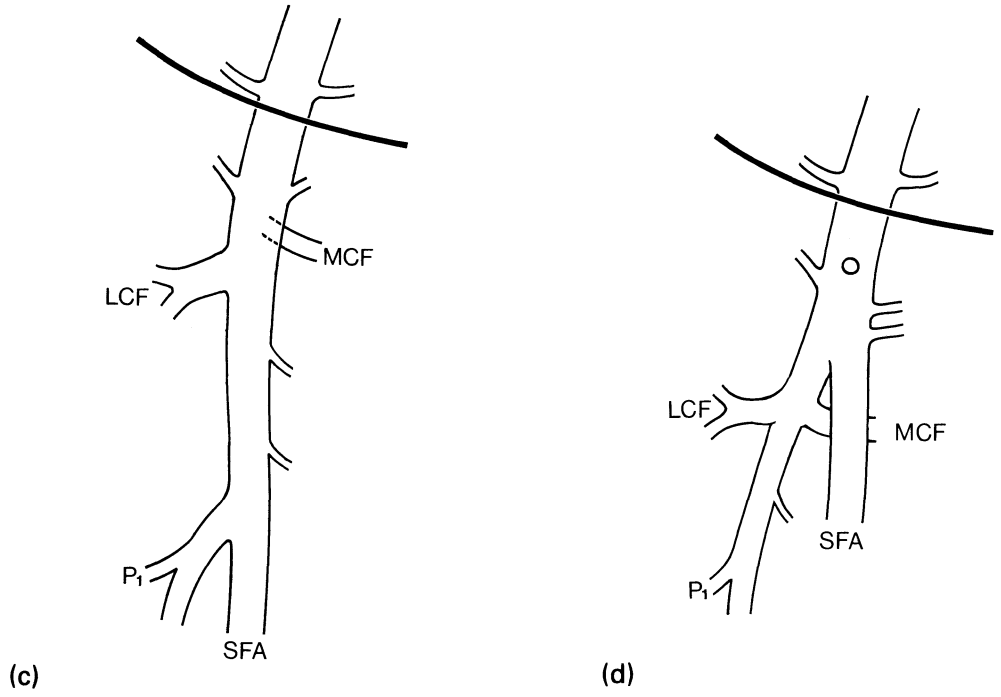


Figure 4.4 continued (c) 'double profunda' due to large lateral circumflex femoral branch and low bifurcation into superficial femoral artery and perforator trunk. (d) early branching of the profunda into three trunks of equal size

The key to exposing the proximal profunda is the lateral circumflex femoral vein which crosses in front of the artery to join the main venous channel (Figure **4.3b**). This should be divided and the distal end ligated. The proximal stump may be oversewn rather than ligated since a ligature may slip off during subsequent vascular mobilization. The division of the profunda into the lateral circumflex femoral artery and the perforator trunk is thereby exposed (Figure **4.3c**). Mobilization of the common femoral bifurcation is completed by identifying the medial circumflex femoral artery, which may arise from the posteromedial aspect of either the common femoral artery or the profunda femoris.

The dissection is then pursued distally to expose the perforator trunk. This requires adjustment in the position of the self-retaining retractor so that sartorius and rectus femoris are retracted laterally and adductor longus medially. The superficial femoral artery is reflected medially or sectioned if it is occluded. During this stage of the dissection care should be taken to avoid damaging the nerve to vastus medialis which descends on the muscle lateral to the perforator trunk. One or more perforating veins will usually require division before the perforator trunk is fully exposed. The venous anatomy of this region is subject to some variation and the profunda veins may empty into the main femoral vein separately or in combination (Figure **4.5a–c**).

The first and second perforating arteries which arise from the posterolateral aspect of the profunda are then identified. Usually the second perforator is given off as the main artery passes behind adductor longus. More distal exposure is seldom necessary but an extra centimetre or so of the artery can be exposed by incising the upper fibres of the latter muscle.

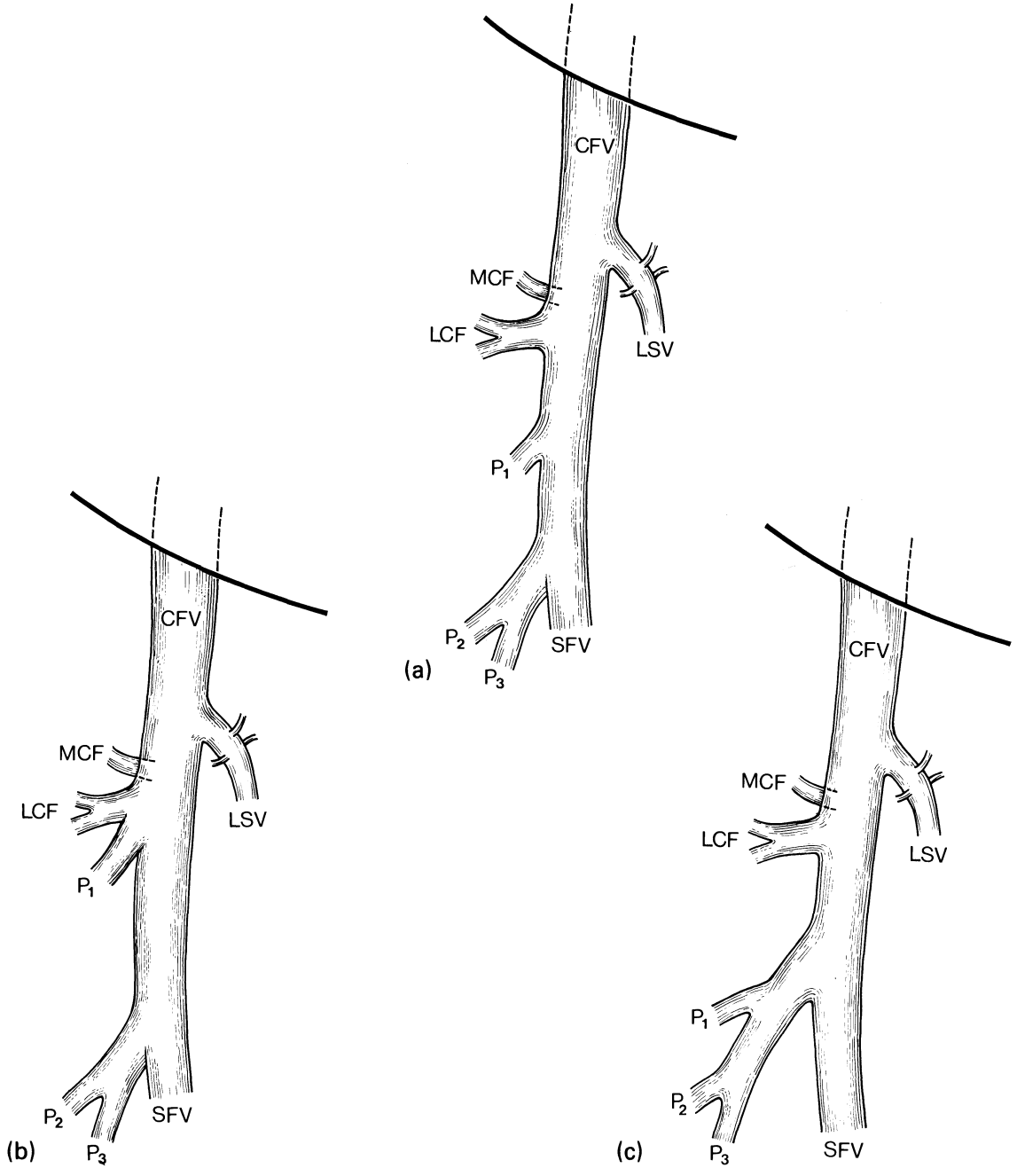


Figure 4.5 Venous variations. *Key*: Veins: CFV = common femoral; LSV = long saphenous; MCF = medial circumflex femoral; LCF = lateral circumflex femoral; P_1 = first perforator; P_2 = second perforator; P_3 = third perforator; SFV = superficial femoral.
(a) most frequent arrangement. (b) lateral circumflex femoral vein and first perforating vein open via a common channel. (c) confluence of first, second and third perforating veins to form a single trunk

Exposure of the mid-profunda femoris

The profunda femoris may be alternatively exposed in the midthigh without initial dissection of the proximal segment. This type of approach may be helpful in re-do aortoiliac reconstructions where access to the common femoral bifurcation is limited by postoperative scarring. It may also be indicated in cases of graft sepsis in the groin (see Chapter 14).

A midthigh incision is made in a line from the anterior superior iliac spine to the medial border of the patella (Figure **4.6a**). After incising the deep fascia, sartorius is retracted medially and rectus femoris laterally. The superficial femoral artery is located and displaced medially along with sartorius. This may necessitate division of a pedicle to vastus medialis. The profunda femoris is then identified some 2 cm or so deep to the superficial femoral artery as it descends in the interval between vastus medialis and adductor longus (Figure **4.6b**).

A more medial approach to the mid-profunda has also been advocated[13]. In this method the thigh is placed in flexion and full external rotation and the vessel exposed by retracting sartorius and rectus femoris laterally.

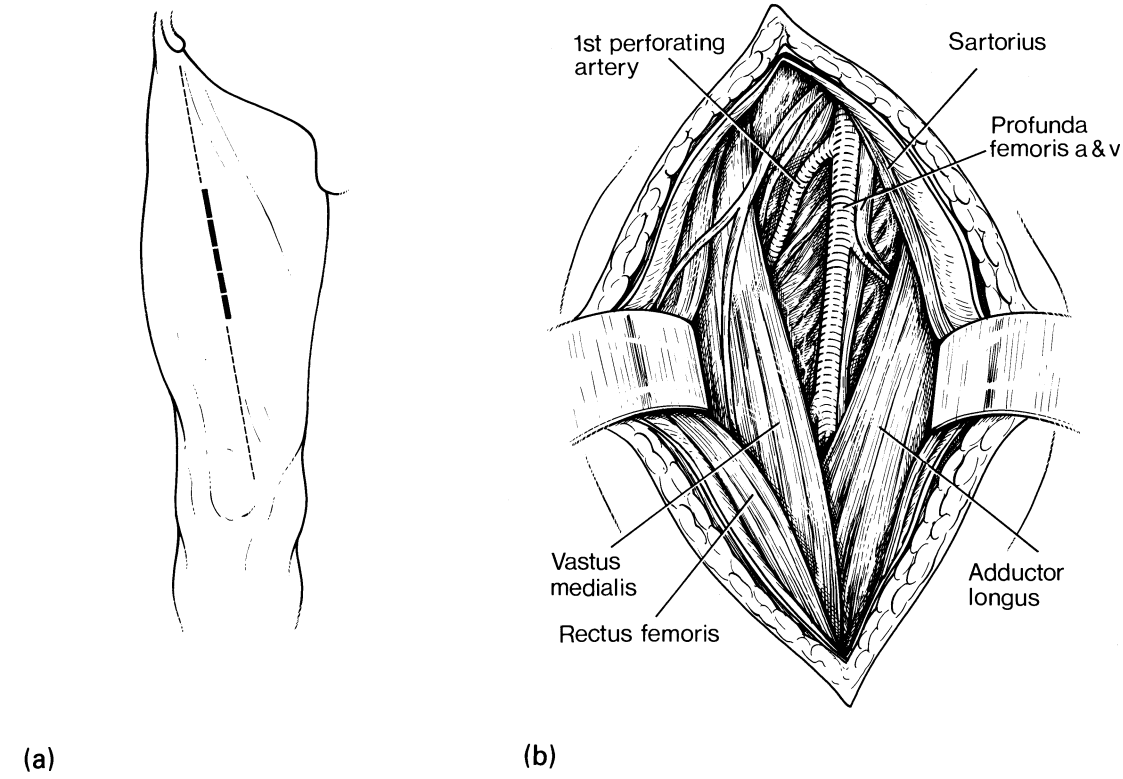


Figure 4.6(a, b) Midthigh exposure of profunda femoris: (a) skin incision. (b) profunda exposed by lateral retraction of rectus femoris and medial retraction of sartorius

TECHNIQUES OF RECONSTRUCTION

Profunda reconstruction figures prominently in aortic bypass or endarterectomy techniques which terminate in the groin and may also be occasionally undertaken at the time of a femoro–distal bypass. Its use in these situations is described elsewhere (Chapters 3 and 5). The following sections are concerned with reconstructive procedures confined to the common femoral bifurcation.

Endarterectomy

This is used principally for ulcerative or plaque lesions involving the orifice or proximal segment of the profunda. More extensive lesions are better treated by a bypass technique.

After displaying the profunda down to the distal limit of disease the patient is heparinized and a soft-jaw clamp placed across the proximal common femoral artery. The distal profunda and branch vessels are controlled with microvascular

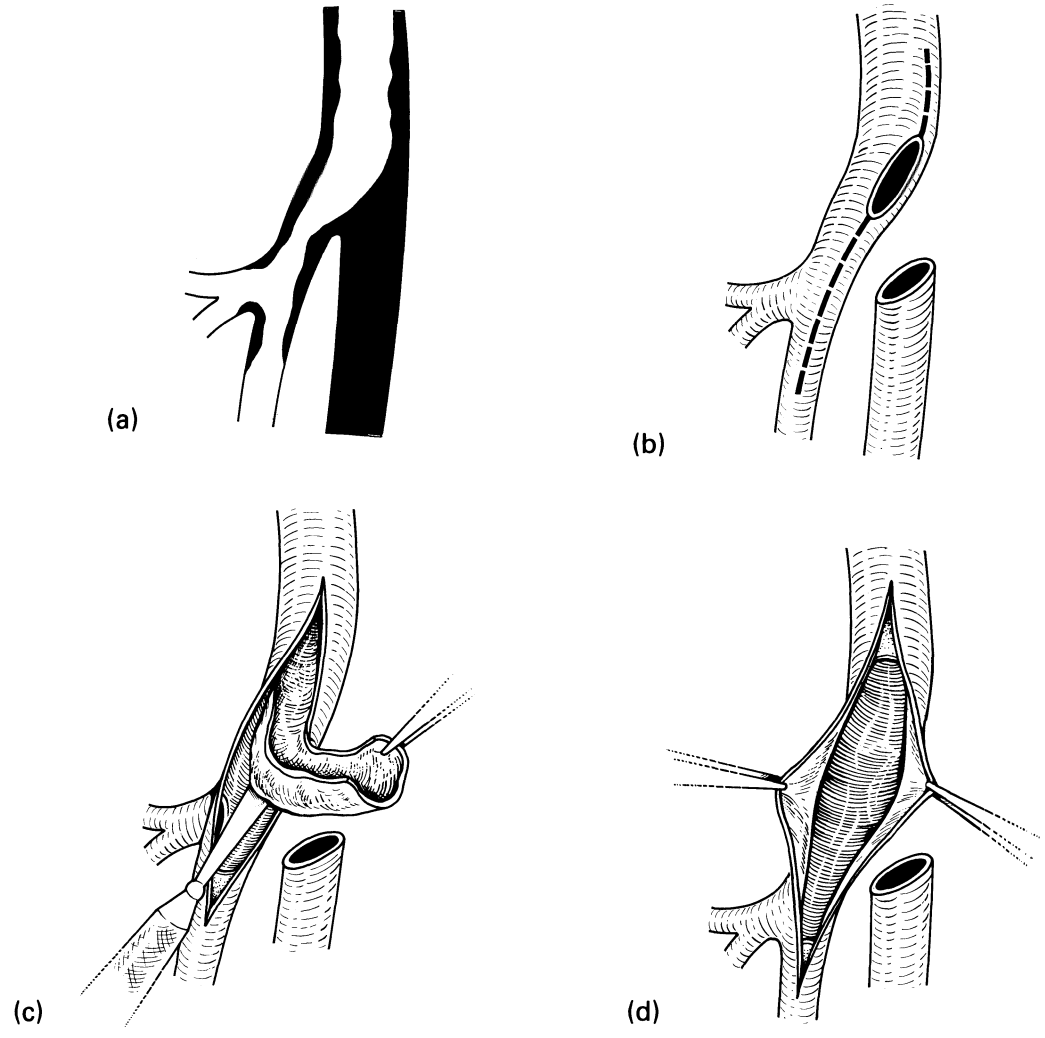
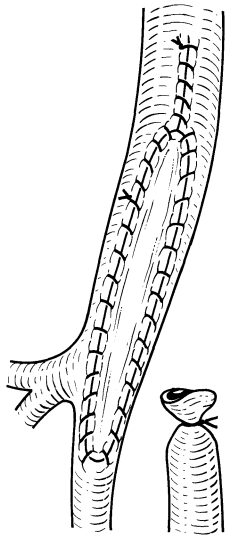


Figure 4.7(a–e) Endarterectomy of the common–deep femoral segment: (a) distribution of atheroma. (b) occluded superficial femoral artery amputated. (c, d) open endarterectomy using a Cannon spatula



(e)

Figure 4.7 continued (e) closure with a vein patch

clips or double-looped Silastic slings. If the superficial femoral artery is occluded at the origin it may be amputated to allow subsequent remodelling of the common–deep femoral axis in a smoothly coned manner (Figure **4.7a–e**). The opening at this detachment site is extended proximally and then distally down the profunda to beyond the macroscopic limit of the disease. An open endarterectomy of the profunda and the common femoral artery is then carried out commencing at the distal limit to ensure maximum control of this critical edge. In nearly all cases it should be possible to leave a clean, securely adherent distal intimal edge of minimal thickness, obviating the need for tack-down sutures. In contrast to the aortoiliac region, the best endarterectomy plane is the trans-medial one (see Chapter 1). If a deeper plane is sought with removal of the full thickness of the media, the remaining wall is likely to be too thin with a roughened surface resulting in either leakage or thrombosis. In addition, an intimal edge is more likely to be left at each branch vessel giving rise to branch occlusion on restoring flow. Because of the need to avoid a deep endarterectomy plane, calcific deposits in the wall of the profunda should be regarded as a contraindication to endarterectomy since these require full thickness media removal.

Particular care should be taken to remove any obstructing plaque from the orifice of the lateral circumflex femoral artery. This may be facilitated by everting the orifice into the profunda lumen. If the atheroma does not separate cleanly, a supplementary arteriotomy with patch angioplasty may be required to preserve patency in this important branch vessel (Figure **4.8**). Where there is palpable thickening in the wall of the lateral circumflex femoral artery as far as its bifurcation, it may be wiser to stop the endarterectomy at the origin and secure the intima with a few tack-down sutures (Figure **4.9a, b**). However, this is not ideal, and in such cases the profunda should be recon-

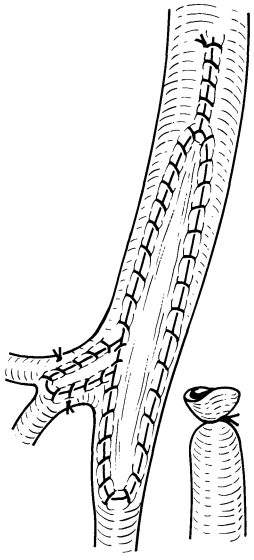


Figure 4.8 Supplementary arteriotomy to secure the intima in the lateral circumflex femoral artery. This is then closed with a small patch

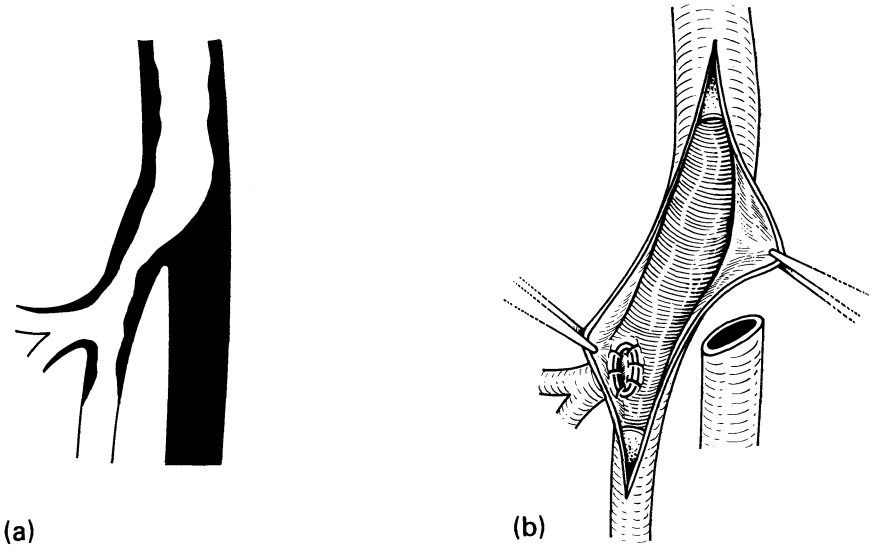


Figure 4.9(a, b) Diffuse thickening of the lateral circumflex femoral artery. The endarterectomy is terminated at the ostium and the intima secured with tack-down stitches

structed by a bypass technique rather than by endarterectomy (see Figure **5.14**).

Once the lateral circumflex femoral artery has been cleared, separation of the atheromatous core is continued retrogradely into the common femoral artery and the specimen sectioned near the proximal limit of the arteriotomy. Any remaining loose shreds are removed from the endarterectomy zone, following which the clamps are flushed and the lumen irrigated with heparin–saline.

The arteriotomy is then closed with a patch using 6/0 or 7/0 prolene. Only the profunda segment should be patched, the common femoral portion of the arteriotomy being closed by direct suture to avoid the risk of late aneurysmal dilatation. Autogenous saphenous vein is the preferred patch material because it can be sewn in easily to the endarterectomized profunda. It may be harvested from the opposite distal leg, preserving the more proximal segment in case femoro–distal or other bypass grafting is necessary at a later date. Usually half the vein width only is utilized to avoid creating an overlarge lumen. Dacron has also been used for profunda patching but tends to develop a rather thick pseudointima. In addition heavier sutures are required which may predispose to re-stenosis. PTFE is an improvement in both respects but its long-term behaviour is not yet known. Endarterectomized superficial femoral artery is immediately to hand but in our experience this material has been prone to premature degeneration and is no longer preferred.

The endarterectomy technique may require to be modified when the profunda is stenosed but the proximal superficial femoral artery is still patent (Figure **4.10a**). An open endarterectomy of the common–deep femoral segment may risk leaving an intimal edge at the superficial femoral origin with a subsequent intimal dissection. Tack-down stitches may reduce this risk but the superficial femoral artery is nearly always atheromatous and stitches here are not ideal. Equally an open endarterectomy of each branch of the common

femoral artery using a Y-shaped incision is unsatisfactory because closure inevitably leads to an overlarge lumen at the confluence of the patches. The most satisfactory alternative is to perform an open common–deep femoral endarterectomy as previously described and then to transect the superficial femoral artery at a point where the disease seems minimal (Figure **4.10a**). The proximal stump is then cleared by an eversion procedure (Figure **4.10b**). Re-anastomosis of the transected vessel avoids the risk of a dissection by including the distal intima in the suture line (Figure **4.10c, d**).

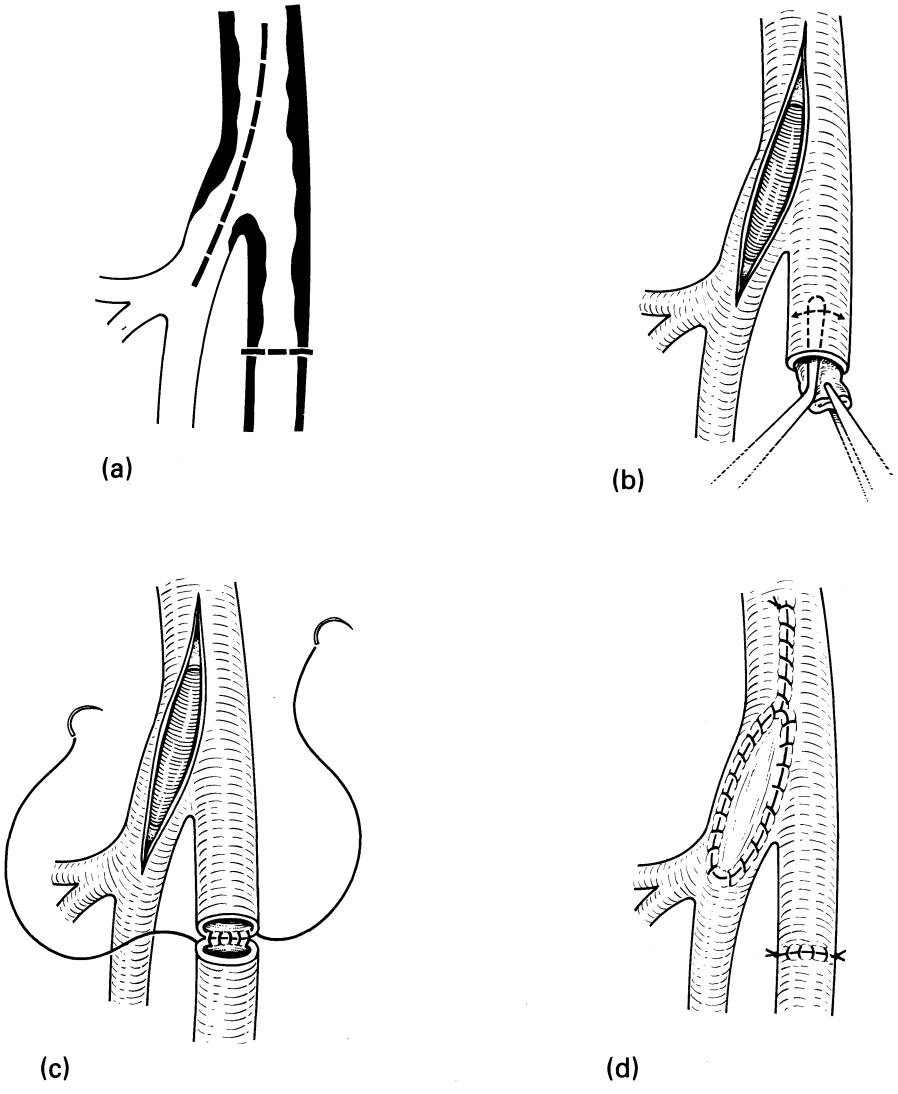


Figure 4.10(a–d) Endarterectomy of the common femoral bifurcation when the superficial femoral artery is still patent: (a) distribution of atheroma. (b) open endarterectomy of the common–deep femoral segment with section–eversion of the proximal superficial femoral artery. (c) superficial femoral artery re-anastomosed. (d) patch closure of the profunda

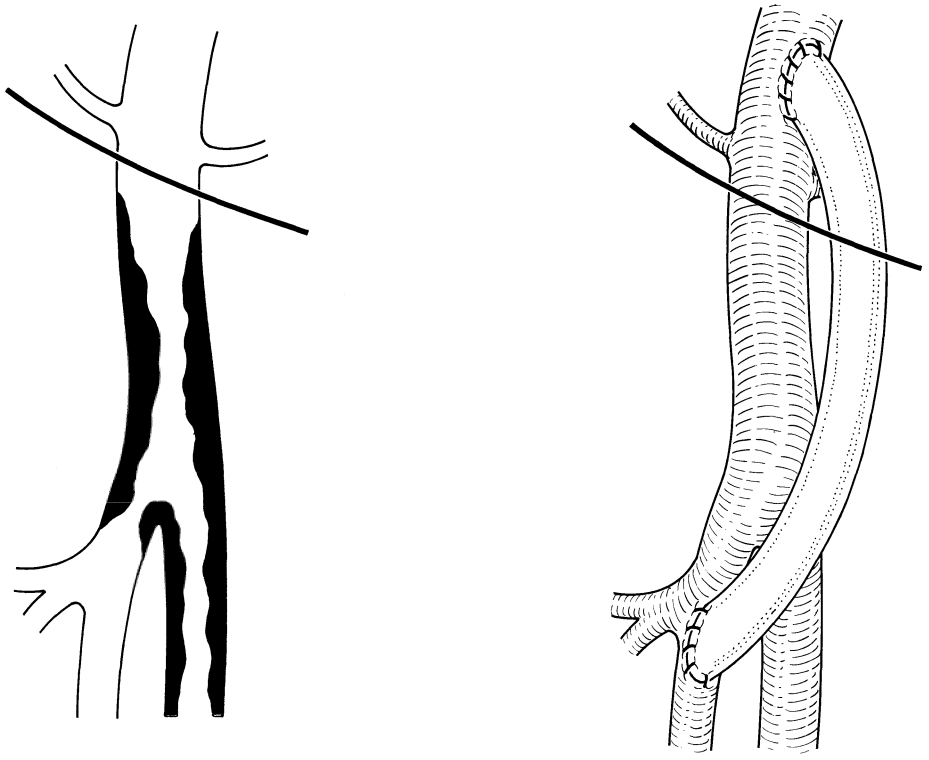


Figure 4.11(a, b) Profunda reconstruction by means of an external iliac–profunda PTFE graft. This avoids the risk of losing the superficial femoral artery during attempted common-deep femoral endarterectomy

Where the superficial femoral artery is more grossly diseased but still patent, this method is less suitable because of the overthick distal edge at the site of re-anastomosis. Endarterectomy is best avoided altogether in these circumstances, profunda reconstruction being more satisfactorily achieved using a bypass technique (Figure **4.11a, b**).

Bypass

Bypass grafting between the external iliac or proximal common femoral artery and the profunda femoris is used in preference to endarterectomy when there is occlusive disease of the profunda extending beyond the first perforating branch

(Figure **4.12**). Although a long endarterectomy is possible, the risk of losing at least one of the branch vessels is increased, particularly as the branches themselves are more likely to be involved when there is extensive main trunk disease. Failure to reopen the profunda branches reduces the possibility of popliteal re-entry and the beneficial effect of the endarterectomy is likely to be lost. In addition, it is difficult to insert a patch of 15–20 cm length without some variation in the calibre of the reconstituted lumen, and this may result in turbulence and mural thrombus deposition.

The second indication for bypass grafting is the presence of recognizable thickening in the lateral circumflex femoral artery (Fig. **4.9a**), or a grossly diseased but still patent superficial femoral artery (Figure **4.11a**). As already described, attempted profunda endarterectomy may lead to the loss of either vessel.

Patients with complete occlusion of the common femoral artery may also be better treated by an end-to-end external iliac–profunda graft (Figure **4.13**). Although endarterectomy is feasible here, direct graft replacement is simple and may give superior long-term results.

The technique of profunda bypass is relatively straightforward. Autogenous saphenous vein is the best graft material but, if this is unavailable, PTFE or a composite graft may be considered. In patients with common–deep femoral occlusion the graft may be interposed in end-to-end fashion, but in most cases an end-to-side bypass is preferred. The proximal attachment site for the graft may be either to the common femoral artery or the external iliac artery. The latter may be exposed by extending the groin incision proximally with division of the inguinal ligament. Alternatively, a separate supra-inguinal exposure may be utilized. Distal attachment depends on the extent of the pathological process, though as with bypasses elsewhere, graft length should be kept as short as possible to provide antegrade perfusion in the maximum number of branches.

Occasionally a profunda bypass may be combined with femoro–distal reconstruction. This may be indicated when profunda revascularization alone may not be sufficient to salvage the limb or when the common femoral artery is markedly diseased. In the latter circumstance preliminary profunda bypass will provide a more satisfactory attachment site for the femoro–distal reconstruction while at the same time maximizing profunda flow (see Figure **5.14**).

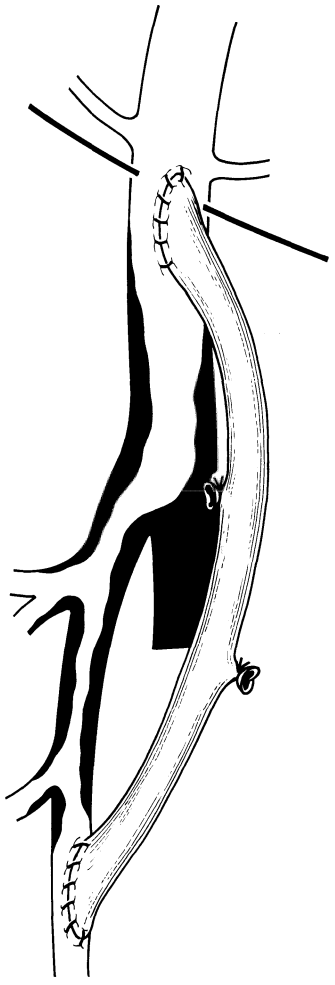


Figure 4.12 Atheroma extending beyond the first perforator: reconstruction by vein bypass graft

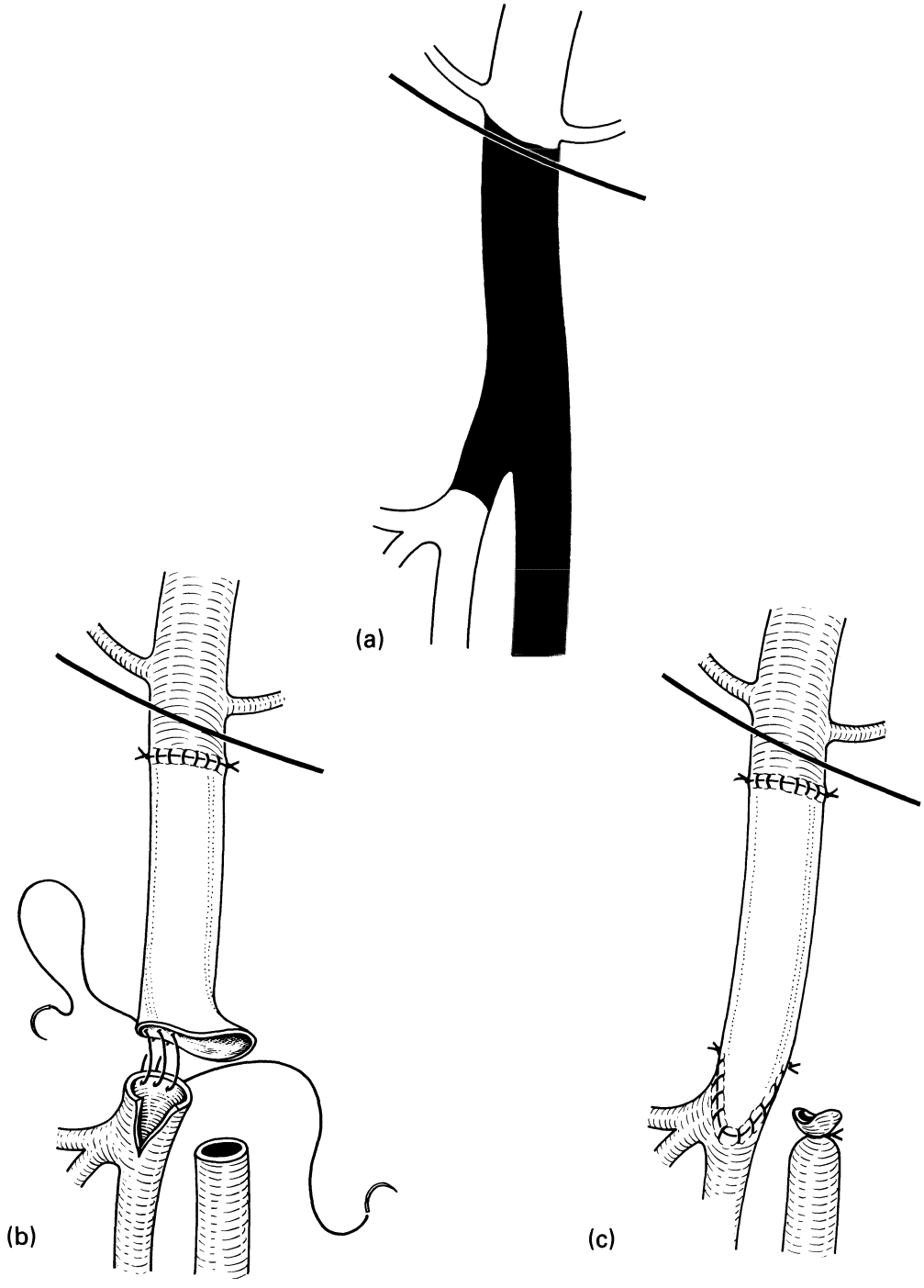


Figure 4.13(a–c) Common femoral artery occlusion treated by an interposition PTFE graft

COMMON FEMORAL ANEURYSMS

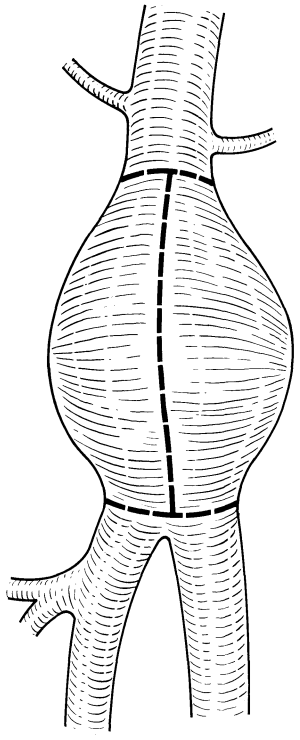
Femoral aneurysms may be post-reconstructive, traumatic, iatrogenic or atherosclerotic[14]. Only the latter are considered here. Most atherosclerotic aneurysms are asymptomatic and are discovered incidentally during examination for other problems. A minority present with a distal embolic episode, acute or chronic thrombosis, local pressure effects or rarely, rupture. In view of these possible complications surgery is advisable in virtually all cases. However, small aneurysms amounting to no more than a widening of the femoral pulse may be kept under observation.

As many as 70% of patients may have aneurysmal disease elsewhere[15,16], particularly in the aorta or in the popliteal and contralateral femoral arteries and more than one reconstruction may be ultimately required. The usual order of proceeding in these cases is to deal with the aortic or popliteal aneurysm first since these pose a relatively greater risk to life or limb respectively than their femoral counterparts.

The approach is via a longitudinal groin incision beginning above the inguinal ligament. As with aneurysms elsewhere, it is neither necessary nor advisable to completely excise the sac. Often these lesions are adherent to the femoral vein or nerve and if most of the sac is left intact a potentially hazardous dissection may be avoided. The external iliac artery is first exposed by dividing the inguinal ligament. The superficial and deep femoral vessels are then sought beyond the aneurysmal zone. If there is difficulty in dissecting the profunda due to the size of the aneurysm or because of periarterial fibrosis, it may be simpler to clamp the external iliac and superficial femoral arteries and control the profunda intraluminally once the sac is opened (see Chapter 14).

After heparinization, clamps are applied proximally and distally and the sac opened. Any small branches arising from the aneurysm are then oversewn from within. Several methods of reconstruction may then be available depending on the extent of the aneurysm and the presence or absence of occlusive disease in the superficial and deep femoral arteries.

If the femoral bifurcation is preserved and both branch vessels are free from ostial stenosis, a Dacron or PTFE prosthesis may be interposed or inlaid in end-to-end fashion. Where the bifurcation is rather splayed, a preliminary tailoring stitch may be useful prior to distal attachment (Figure **4.14a–f**).



(a)

Figure 4.14(a–f) Common femoral aneurysm not involving the bifurcation: (a) aneurysm incised

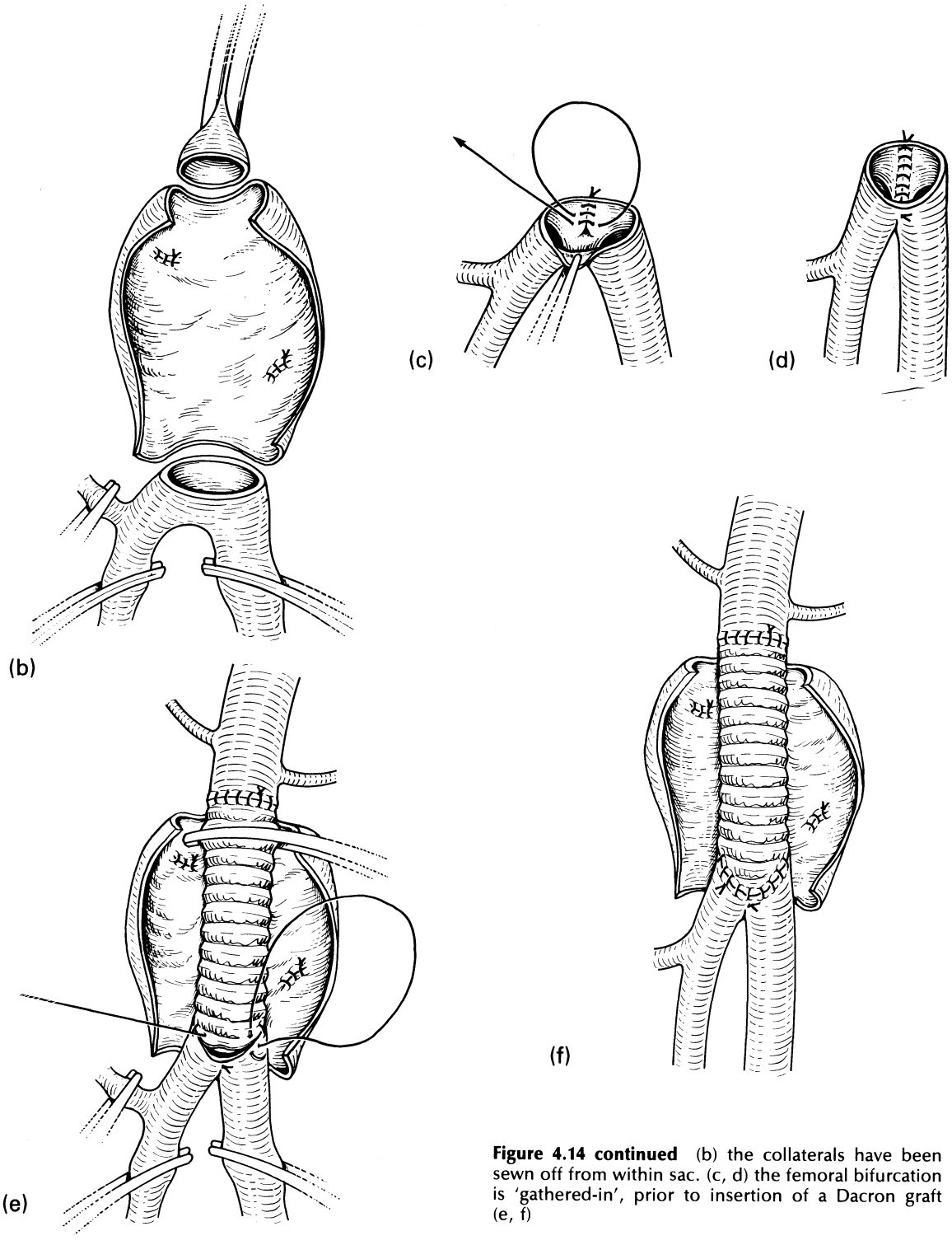


Figure 4.14 continued (b) the collaterals have been sewn off from within sac. (c, d) the femoral bifurcation is 'gathered-in', prior to insertion of a Dacron graft (e, f)

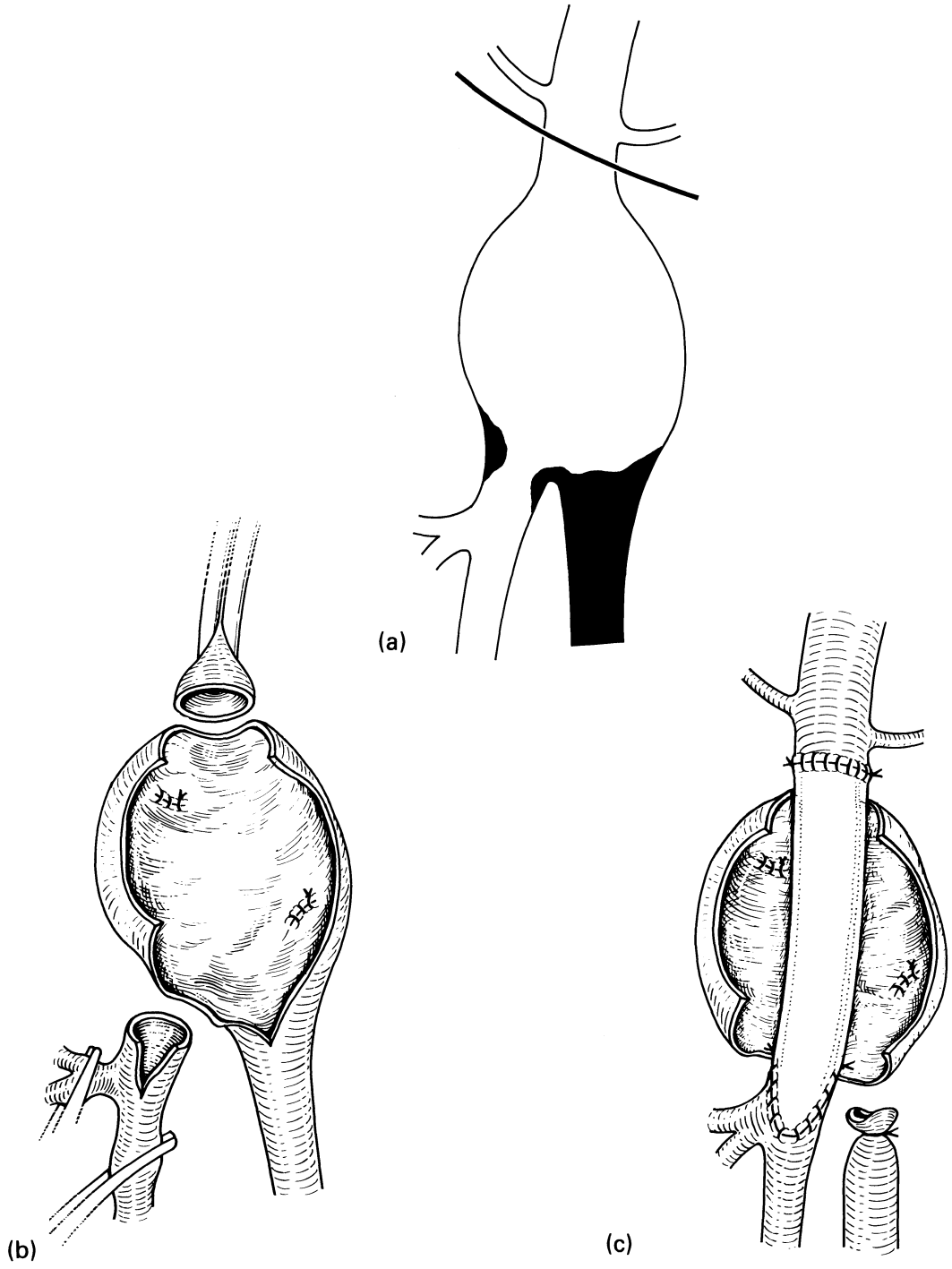


Figure 4.15(a–c) Common femoral aneurysm with a superficial femoral occlusion and profunda ostial stenosis: (a) pathology. (b) the profunda is detached and spatulated. (c) a PTFE graft is then interposed.

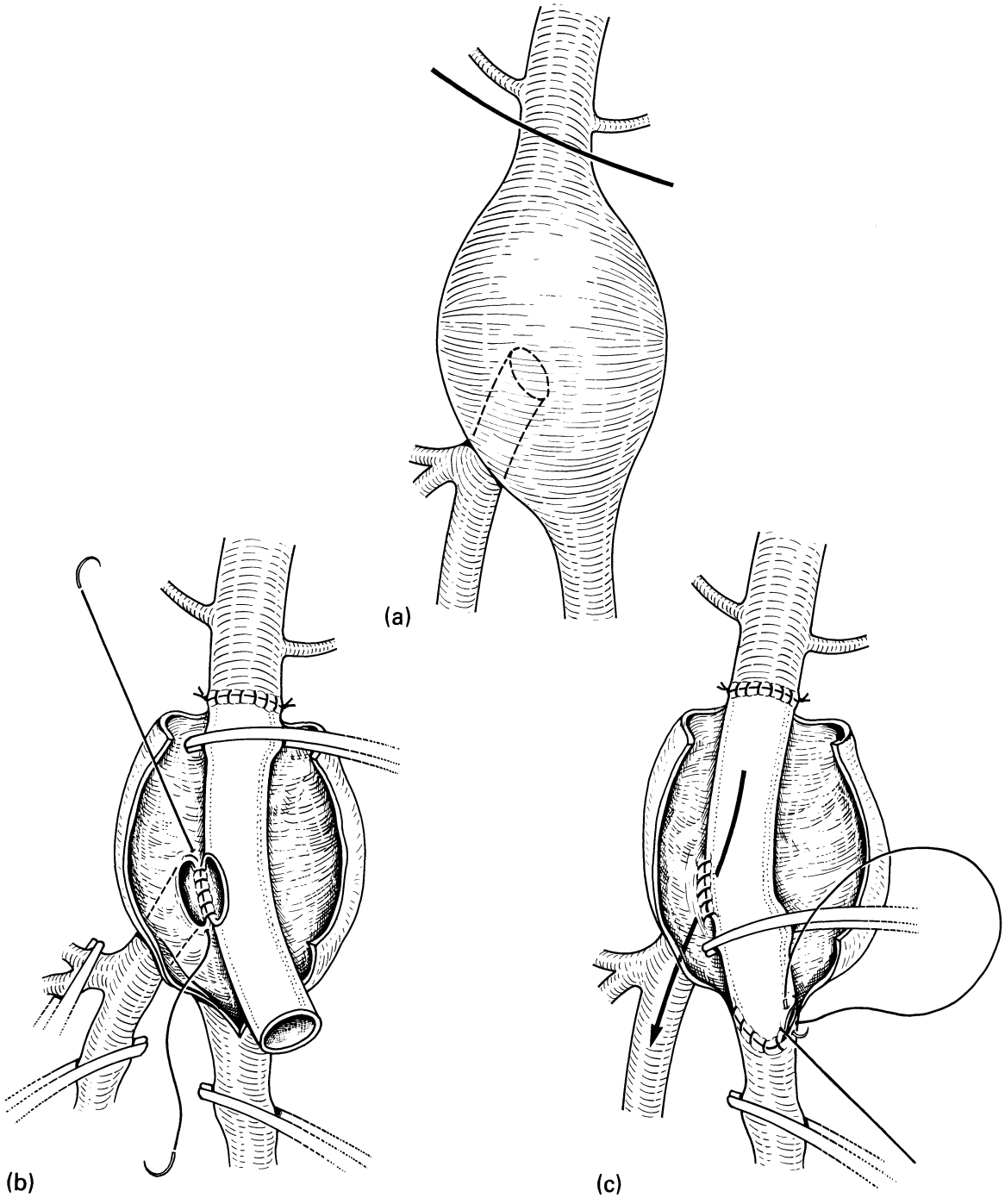


Figure 4.16(a–c) Aneurysm involving the common femoral bifurcation: (a) extent of lesion. (b) the sac has been opened and a PTFE graft anastomosed to the external iliac artery. The profunda femoris is attached by an inclusion technique. (c) profunda flow restored while the superficial femoral anastomosis is being completed

In many patients the superficial femoral artery is occluded and there is a variable amount of plaque at the profunda origin (Figure **4.15a**). One option in these cases is to detach the profunda and insert an external iliac–profunda graft after suitable spatulation of the distal stump (Figure **4.15b, c**). Alternatively, the profunda orifice may be oversewn from within the sac and an end-to-side bypass inserted bridging the pathological zone (as in Figure **4.18**).

If there is aneurysmal extension into the bifurcation and both femoral branches are patent, a double reconstruction will be necessary. One method is to place a graft end-to-end between the external iliac and superficial femoral arteries, the profunda being reattached by means of a Carrel button or by an inclusion technique (Figure **4.16a–c**). Alternatively, a branching graft arrangement may be utilized (Figure **4.17**). However, the easiest and most satisfactory method is to sew off the afferent and efferent orifices from within the aneurysm. An end-to-side bypass is then inserted between the external iliac and profunda arteries with an additional side-limb to the superficial femoral artery (Figure **4.18**). This technique avoids the problem of mismatch in size between graft and artery and enables the anastomoses to be undertaken at sites where the vessel wall is non-pathological. In addition, arterial mobilization is restricted to the anastomotic zones only, without the need to dissect the extremities of the aneurysm, so that venous or other injury should be completely avoided.

Occasionally the superficial femoral artery is

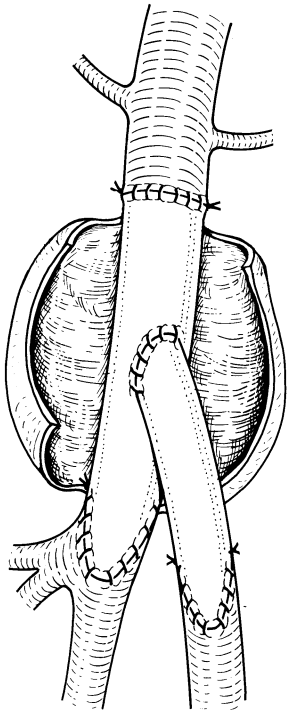


Figure 4.17 Aneurysm involving the common femoral bifurcation treated by interposition grafting of both branches (PTFE)

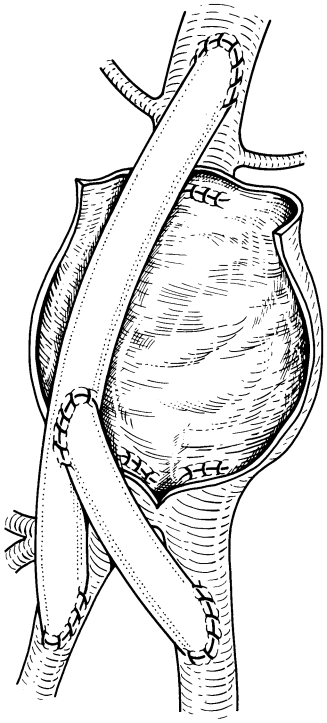


Figure 4.18 Preferred method of managing an extensive common femoral aneurysm. The aneurysm has been excluded and a double end-to-side bypass inserted

segmentally dilated throughout its length with or without a second overt aneurysm in the popliteal fossa (Figure **4.19a**). Mural thrombus may form in the ectatic segments and may give rise either to acute superficial femoral artery occlusion or distal embolization. These cases may be managed by excluding the common femoral aneurysm and the superficial femoral artery. An ilioprofunda bypass is then inserted with a femoro–distal extension limb to the popliteal or infrapopliteal vessels (Figure **4.19b**).

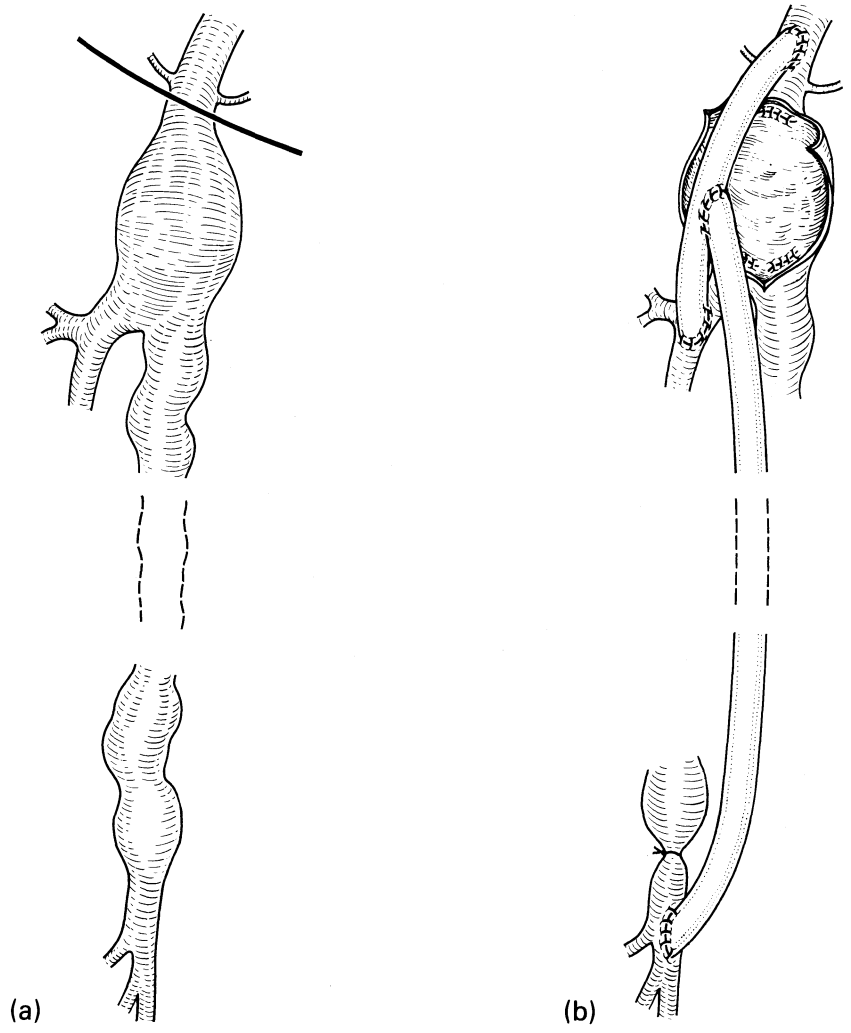


Figure 4.19(a, b) Common femoral aneurysm associated with diffuse ectasia (arteriomegaly) of the superficial femoral artery: (a) pathology. (b) double bypass with exclusion of the aneurysm and the ectatic segment

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5

Femoropopliteal Surgery

GENERAL CONSIDERATIONS

The most frequent site for atherosclerosis is in the vessels below the inguinal ligament and reconstructive surgery should therefore have the widest application at this level. In fact the frequency of additional disease distal or proximal to the superficial femoral segment has a limiting effect on the extent to which such surgery is undertaken.

The main indication for femoropopliteal reconstruction is limb-threatening ischaemia manifest by rest pain, ischaemic ulceration or gangrene. Intermittent claudication is also an indication for operative intervention if it causes serious disability and there are no precluding risk factors. Patients with less severe degrees of claudication may be managed conservatively in view of the relatively favourable natural history of the untreated condition[1].

The most widely accepted method of reconstruction is femoro–distal bypass. Endarterectomy of the superficial femoral artery is still utilized in some centres and has the advantage that the long saphenous vein is preserved for subsequent usage[2,3]. However, in most hands endarterectomy has had limited durability and is no longer widely used outside the common femoral bifurcation. Localized stenoses or short segmental occlusions which were formerly considered suitable for endarterectomy may now be treated by transluminal angioplasty (see Chapter 15).

ESSENTIALS IN FEMORO–DISTAL BYPASS

Adequate inflow

Unimpeded inflow into the groin is essential and this may be assessed by clinical examination, vascular laboratory criteria, and by biplanar or multiplanar arteriography. Minor arteriographic stenoses in the iliac segment may be accepted in elderly subjects with large vessels. However, more marked iliac lesions which reduce the luminal diameter by 50% may require a two-level reconstruction for full relief. One approach is to correct the iliac stenosis by percutaneous transluminal angioplasty followed by femoro–distal bypass. In patients with an iliac stenosis of 75% or more, reconstruction may be limited to the proximal segment alone, since restoration of perfusion to the profunda will relieve ischaemic symptoms in most cases (see Chapter 3).

Ulcerative or ectatic lesions in the aortoiliac segment should also be fully identified preoperatively. Such lesions may harbour mural thrombus and if overlooked may be the cause of intra- or postoperative embolization of the graft bed.

Adequate anastomosis site

The graft should be implanted on a segment of artery free from plaque or undue thickening. If

the site selected is too pathological, overdeep suture bites may be required or the intima may split during suture insertion.

The customary site for proximal graft anastomosis is the common femoral artery. If the artery wall is markedly thickened here, an intermediate vein patch may improve graft attachment. Alternatively if there is more marked atheroma with profunda stenosis, a preliminary endarterectomy should be undertaken or an external iliac–profunda bridging graft inserted in order to provide an adequate platform for the graft.

At the distal end, the graft may be attached either to the proximal or distal popliteal artery. Despite the haemodynamic advantages in keeping the bypass short[4,5], the distal popliteal segment is the preferred site of attachment since this is usually of better quality and is less subject to atherosclerotic progression[6]. Where the entire popliteal artery is thick-walled with reinforcement at the anterior tibial origin, a more distal anastomosis should be sought on the tibioperoneal trunk or one of the tibial vessels. Alternatively, operation may be withheld in patients with claudication, in view of the less favourable results of infrapopliteal bypass[7,8].

Adequate distal run-off

The quality of the run-off has an important bearing on graft patency and two patent infrapopliteal vessels should guarantee a successful outcome[9,10]. However, a more limited run-off may have to be accepted in limb salvage cases. This may include using an isolated popliteal segment[11] or attaching the bypass to a vessel in the distal leg or foot[12]. Some judgement is required, since if the run-off is too compromised and the graft fails acutely, an unduly high amputation may result.[13]. Satisfactory opacification of the distal run-off may be difficult to achieve in some cases and reactive hyperaemia, pharmacological vasodilation or intra-arterial digital subtraction techniques may be required[14,15]. Alternatively, if these are unavailable, an intraoperative arteriogram may be obtained prior to bypass placement[16,17].

GRAFT MATERIAL

Autogenous saphenous vein

This remains the material of choice, particularly for the more distal type of bypass. At proximal popliteal level the performance of prosthetic grafts is comparable[18], but from then on distally the advantages of vein become progressively more marked[19,20]. Autogenous saphenous vein is pliable, easy to tailor and amenable to fine suture technique. In addition, it remains supple when taken across the knee and is the best type of graft material for resisting infection. However, autogenous vein grafts are not entirely without problems, since a small but significant number may develop intimal hyperplasia or other structural changes predisposing to graft closure[21,22].

The saphenous vein may be used in reversed, nonreversed or *in situ* fashion. Until recently the reversed vein bypass was the standard method of femoropopliteal reconstruction but with the advent of improved methods of valve suppression, the *in situ* technique has become a practical alternative. Indeed the latter may hold a number of advantages. Thus the structural integrity and function of the endothelium may be better preserved in the *in situ* technique with an attendant improvement in early and perhaps late graft patency[23,24]. This beneficial effect on *in situ* graft structure and function has been attributed to preservation of the vasa vasorum but may in fact be more closely related to careful vein handling techniques[25]. A second major advantage of the *in situ* method is that it enables veins too small for reversed bypass to be accommodated[26]. This increased vein utilization and better graft patency have led to improved limb salvage particularly in patients undergoing femoro–infrapopliteal reconstruction[27]. A further benefit of *in situ* bypass is that by having the largest diameter of the vein proximally and the small end distally, anastomoses are better matched and graft haemodynamics enhanced. Again these advantages become more accentuated the more distal the bypass.

In a number of patients the saphenous vein has already been removed or is unsuitable as a result of sclerosis or varicose change. A poor quality

vein performs poorly[21] and an alternative method of reconstruction should be adopted. This may include using the contralateral saphenous vein or one of the veins from the arm. The best parts of both long saphenous veins may be joined together to form a satisfactory conduit but sacrifice of both veins represents a major loss of graft material. Cephalic or basilic veins are more difficult to handle and are not generally recommended in view of their tendency to structural degeneration[28]. If part of the saphenous vein is available this may be used in conjunction with PTFE as a composite graft or as a sequential bypass[29]. Marginally short veins may be accommodated by attachment to the profunda instead of the common femoral artery, if necessary with a proximal extension in the form of a long patch. Alternatively the superficial femoral artery may very occasionally serve as graft source if well preserved[30]. Preliminary endarterectomy of the proximal superficial femoral artery with a short distal vein bypass has also been utilized but long-term results have been rather poor due to degeneration in the endarterectomy zone[31].

PTFE

Expanded polytetrafluoroethylene (PTFE) is one of the alternatives which may be considered when there is insufficient good quality saphenous vein. Non-tapered 6 mm grafts are preferred. Unlike Dacron grafts, PTFE can be taken across the knee joint without risk of kinking. A particular advantage of PTFE is the relative ease of graft thrombectomy[32] which makes management of graft occlusion much easier than in the case of autogenous vein or other bypasses. Although effective at popliteal level, PTFE may be less satisfactory for more distal bypass[8,18,19]. A major factor in early closure of these distal bypasses appears to be intimal hyperplasia in the vicinity of the distal anastomosis. An intermediate vein patch may be one method of avoiding this problem[33]. However, patching may lead to an overlarge lumen with stasis and a composite PTFE–vein graft or a sequential bypass may be a better option.

Human umbilical vein

Glutaraldehyde-tanned umbilical vein has been used in some centres, particularly for bypass to the infrapopliteal arteries[34]. However, this material is expensive and its intraoperative preparation cumbersome. In addition, long-term results in most hands have been disappointing[20,35].

Dacron

Dacron may also be used for femoropopliteal reconstruction and has functioned reasonably well above the knee[36,37]. However, results of below-knee popliteal bypass have been less satisfactory due partly to kinking and fracture of the pseudointima at the knee[6]. In addition there are technical difficulties in anastomosing a stiff, relatively thick-walled prosthesis to a small distal vessel and for these reasons Dacron is no longer utilized in this situation.

Composite grafts

A composite graft consisting of a proximal prosthetic and distal venous segment may be considered when there is only a limited length of vein available. These grafts retain the advantage of a distal vein–artery anastomosis which is particularly important at infrapopliteal level.

Dacron–vein composites have been used in the past, but are liable to stenosis or false aneurysm at the intermediate anastomosis[29]. PTFE–vein grafts appear to be less prone to this problem, provided that a long spatulated intermediate anastomosis is utilized, and results have been superior to PTFE alone for infrapopliteal bypass in some series[38].

BYPASS TO THE POPLITEAL ARTERY

IN SITU VEIN BYPASS TO THE DISTAL POPLITEAL ARTERY

The operative field should include the entire limb and lower abdomen. Where there is gangrene or ischaemic ulceration, particular care must be taken to isolate the foot from the operative zone. In addition the drapes should be placed so that the knee can be freely flexed and extended without causing a breach in asepsis. The operator faces the affected limb which is placed with the knee flexed to 60° and the thigh abducted and externally rotated (Figure **5.1**).

The operation starts with exposure of the distal part of the popliteal artery in order to check local operability. A skin incision is made from the posterior border of the medial femoral condyle extending distally in a curve just behind the posteromedial border of the tibia to end 6–8 cm below the tibial tuberosity (Figure **5.1**). The long saphenous vein is identified in the posterior flap. If the vein is of suitable size and quality it is dissected out and its branches ligated and divided. Ligatures should be placed 0·5 cm clear of the main vein to avoid stenosis when the vein is subsequently arterialized.

The deep fascia is then incised in the line of the skin incision to open the retrotibial space. Proximally the fascial incision is extended along the inferior border of the semitendinosus tendon. Each of the pes anserinus tendons is then fully mobilized from the muscle belly to the tibial insertion. Once this is done, these tendons can be easily retracted without the necessity for division.

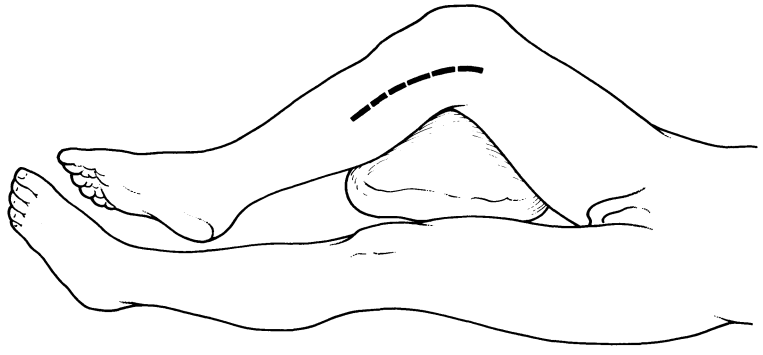


Figure 5.1 Position of the leg for femoropopliteal bypass. The incision for exposure of the distal popliteal artery is outlined

The medial head of the gastrocnemius is then retracted posteriorly to display the distal popliteal vessels (Figure **5.2a**). Usually the artery is surrounded by a venous plexus formed by cross-channels between the two popliteal veins. These cross-channels should be divided to allow a sufficient length of artery to be cleared.

The dissection then proceeds distally towards the soleal arcade. This may be incised using a right-angled clamp to protect the underlying vascular bundle. Ligation and division of the anterior tibial vein will then expose the distal popliteal artery and its division into the anterior tibial artery and tibioperoneal trunk (Figure **5.2b**). According to the state of the artery wall at this level and the clinical symptoms, a decision is made to proceed, abstain or go more distally.

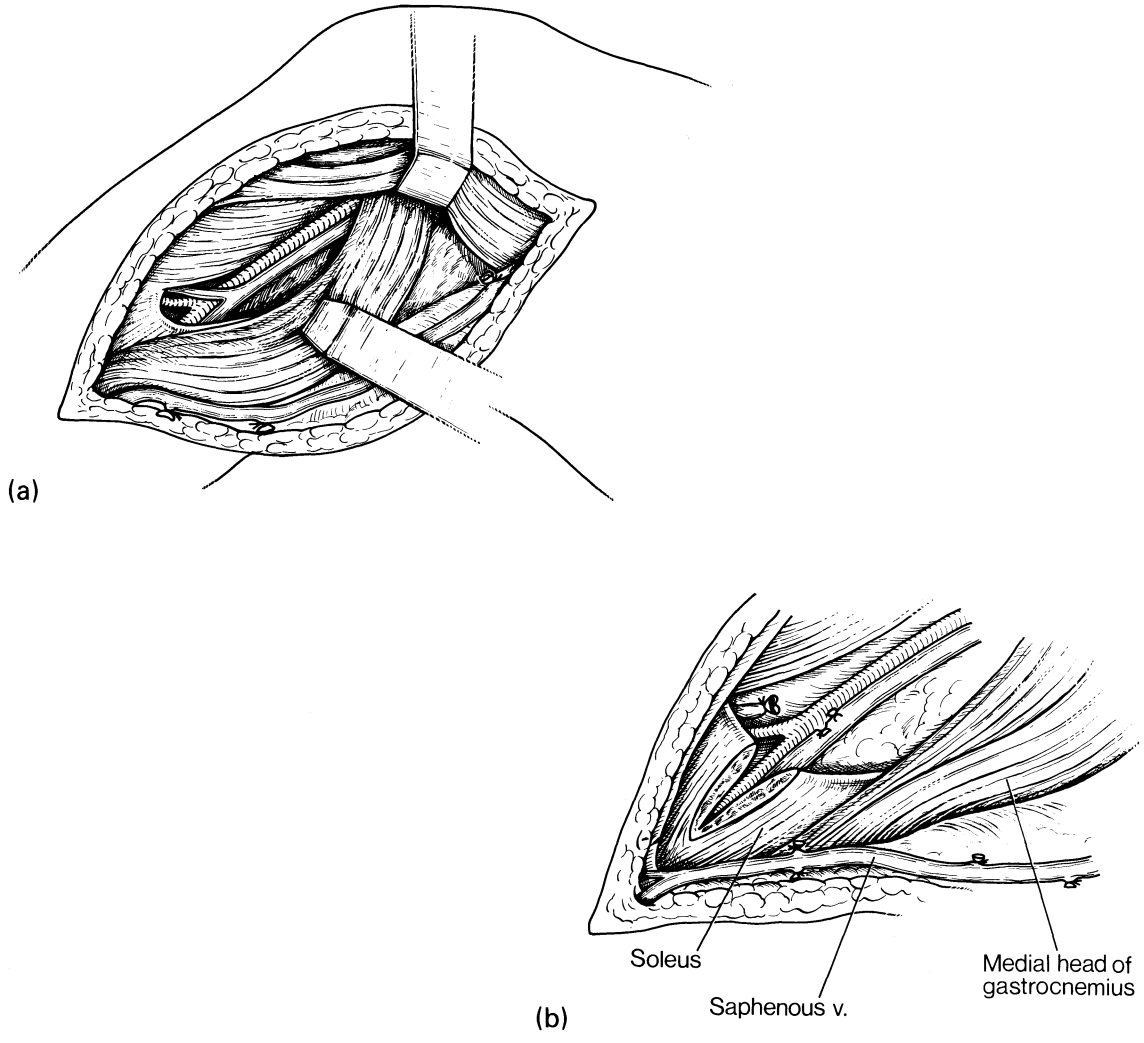


Figure 5.2(a, b) Exposure of the distal popliteal artery: (a) the tendons of sartorius, gracilis and semitendinosus are retracted superiorly and the medial head of gastrocnemius posteriorly to expose the distal popliteal vessels. (b) the anterior tibial artery and tibioperoneal trunk are exposed by incising the soleal arcade. The anterior tibial vein has been divided

The operative zone now moves to the groin. A longitudinal incision is made over the saphenofemoral junction. If the terminal portion of the saphenous vein is found to be ectatic or shows phlebitic thickening, the operative plan should be changed. Outside this event, the tributaries of the saphenous vein are ligated, preserving the largest for subsequent intraoperative arteriography. The front and sides of the common femoral vein are then cleared and after applying a partial occlusion clamp, the saphenous vein is detached together with a small cuff of common femoral vein (Figure **5.3a**). This cuff is a useful means of enlarging the arterial anastomosis and will ensure that the vein reaches the common femoral artery without tension. The common femoral vein is then carefully closed with 6/0 prolene, taking the smallest bites possible (Figure **5.3b**).

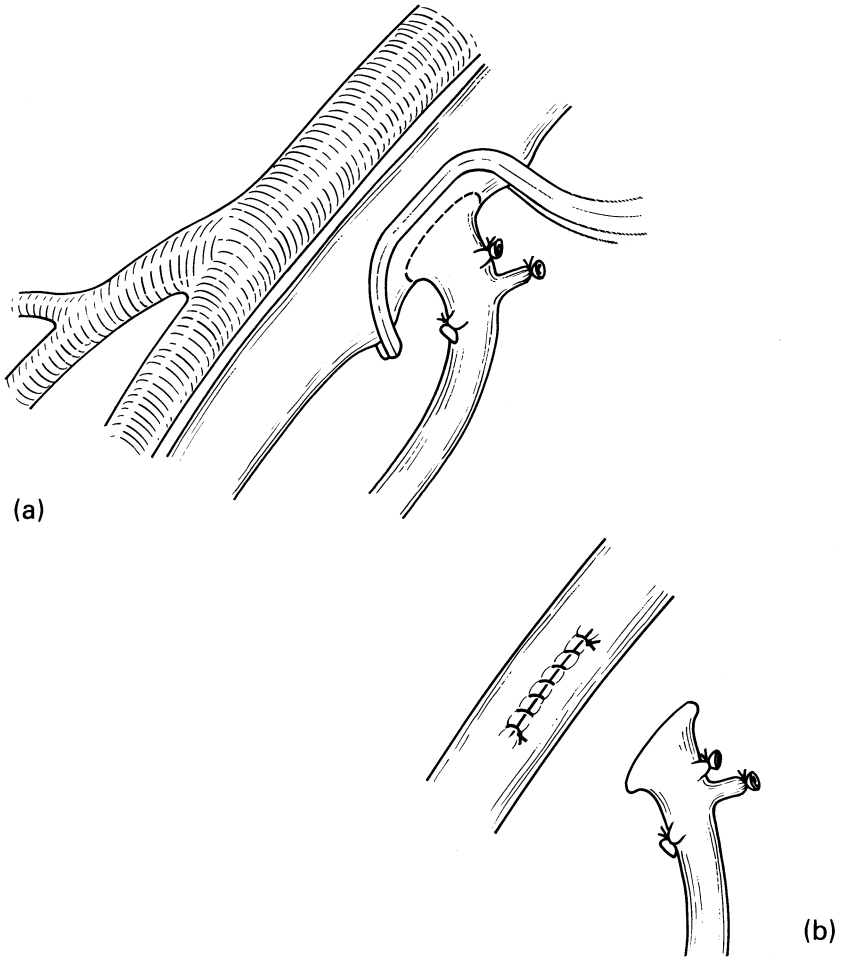


Figure 5.3(a, b) (a) A partial occlusion clamp is applied to the saphenofemoral junction. (b) the saphenous vein is detached, together with a cuff of common femoral vein. The latter is then closed with a fine suture

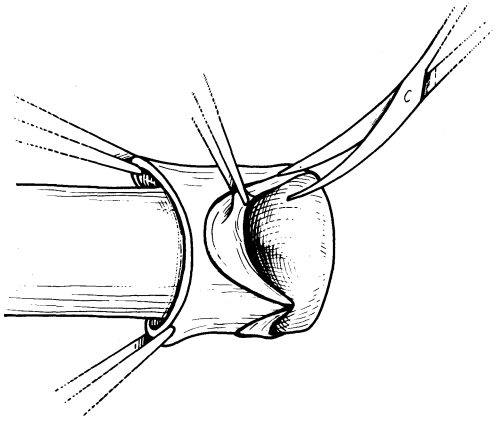


Figure 5.4 Eversion of the proximal end of the saphenous vein to allow excision of the ostial and preostial valves

The patient is heparinized (1 mg/kg body weight) and the saphenous vein turned back on itself for 6–8 cm as far as the preostial valve. The ostial and preostial valves are then excised taking care to avoid damage to the adjacent vein wall (Figure **5.4**). If a lateral thigh tributary is present this will need prior division to allow access to the preostial valve situated immediately below. Similarly if a medial accessory saphenous vein is present this should be ligated to avoid a subsequent arteriovenous fistula.

The common femoral artery is then exposed via a more laterally placed fascial incision, to avoid cutting into the lymphoadipose tissue anterior to the femoral sheath. If there are prominent lymphatic glands, the detached vein may be tunnelled under this tissue to reach the artery; otherwise it is transferred directly across. Clamps are applied and an arteriotomy made on the anteromedial aspect of the common femoral artery. An end-to-side anastomosis is then undertaken using 6/0 prolene. On completion, the arterial and venous axes are declamped (Figure **5.6**).

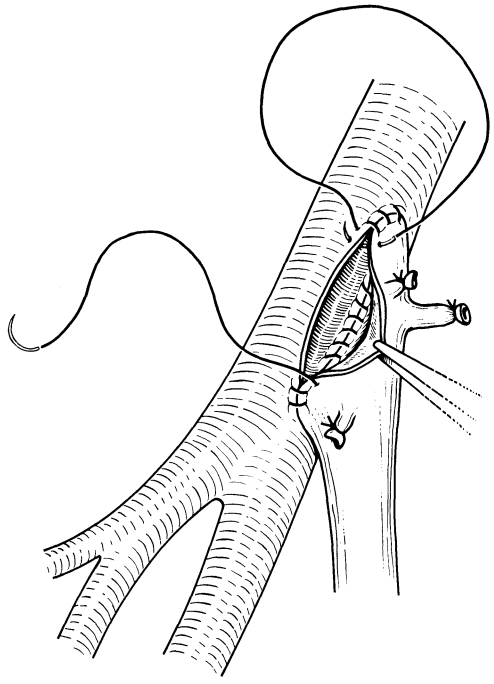


Figure 5.5 Saphenous vein anastomosed to the common femoral artery

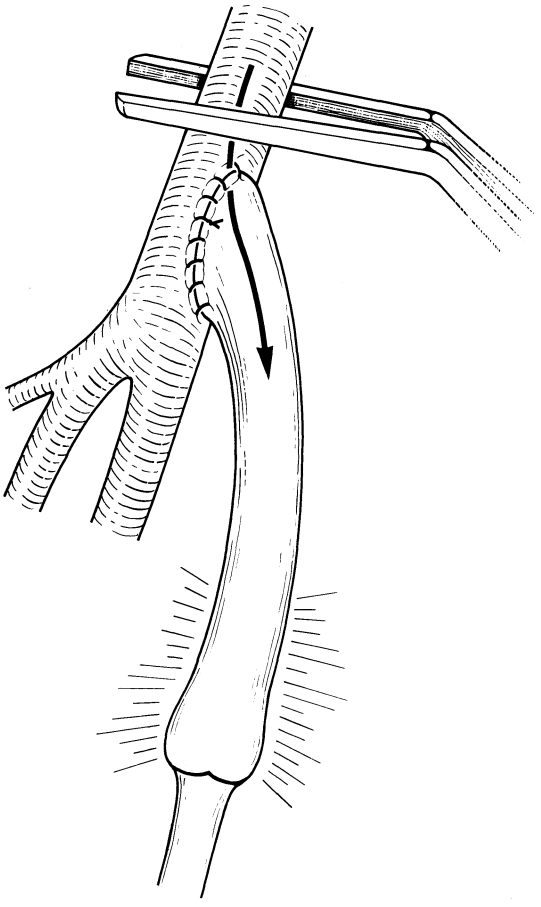


Figure 5.6 The arterial axis is declamped allowing entry of blood into the graft as far as the first competent valve. (branches of saphenous vein omitted for clarity)

The next stage concerns valve suppression which may be quickly and efficiently achieved using a Cartier stripper. This has a smooth, conical head and a scalloped base (Figure **5.7**). The saphenous vein is sectioned at midcalf level, i.e., well below the prospective anastomosis site. A 3 mm Cartier stripper is then passed up the vein from below with the knee straightened. The stripper should be passed to just below the saphenofemoral anastomosis and then gently withdrawn with a spiral movement. The valve cusps, which have been rendered tense by the entry of arterialized blood from above, are then engaged by the stripper and disrupted. This process is repeated in succession down the length of the vein (Figure **5.8a**). A spiral movement is essential during stripper withdrawal to ensure that both cusps are engaged at each valve ring. The manoeuvre is then repeated three or four times, controlling the escape of blood from the distal end of the vein between finger and thumb (Figure **5.8b**).

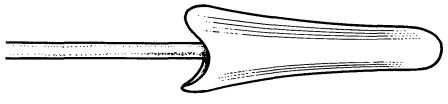


Figure 5.7 Cartier stripper. Note the scalloped edge at the base

Slightly larger strippers may then be used to ensure full valve ablation. However, care must be taken not to use an over-large stripper, because withdrawal may risk an intimal split in the narrow distal segment of the vein. If the stripper should get caught at the entry site of a venous tributary, it may be disengaged by gentle re-passage proximally followed by repeat withdrawal in a circular manner. Once all the valves have been disrupted there should be a thrusting arterial pulse at the extremity of the vein.

Before anastomosing the arterialized vein to the distal popliteal artery, precautions should be taken to ensure that the length and lie of the graft are correct. This is one of the most critical parts of the *in situ* technique, since the graft must pass in a smooth curve from a subcutaneous position to join the vascular axis deep within the popliteal fossa. The optimum anastomosis site for popliteal bypass is close to the anterior tibial origin. A more proximal graft attachment risks angulation over the medial hamstrings, while if the graft is too short it may be trapped between the medial head of gastrocnemius and the back of the tibia.

The arterialized vein is ligated at the distal end and the medial surface marked with a stitch to avoid subsequent torsion. The knee is then extended to assess the graft length and after slight flexion the arterialized graft and popliteal artery

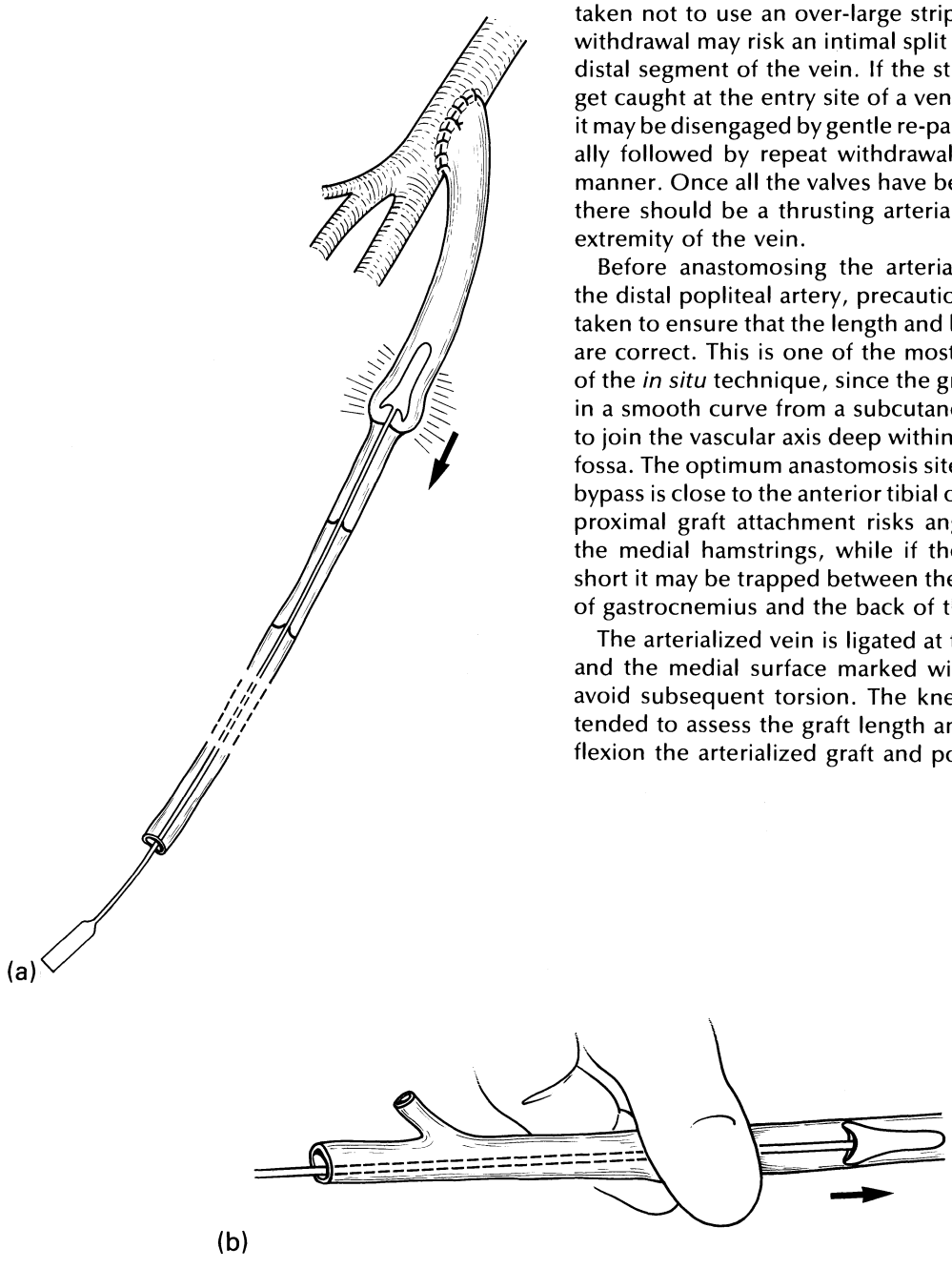


Figure 5.8(a, b) Suppression of saphenous vein valves: (a) the valve cusps are made tense by entry of arterialized blood from above and are then engaged by the Cartier stripper. (b) stripper re-passed using digital control to prevent escape of blood from the distal end of the vein

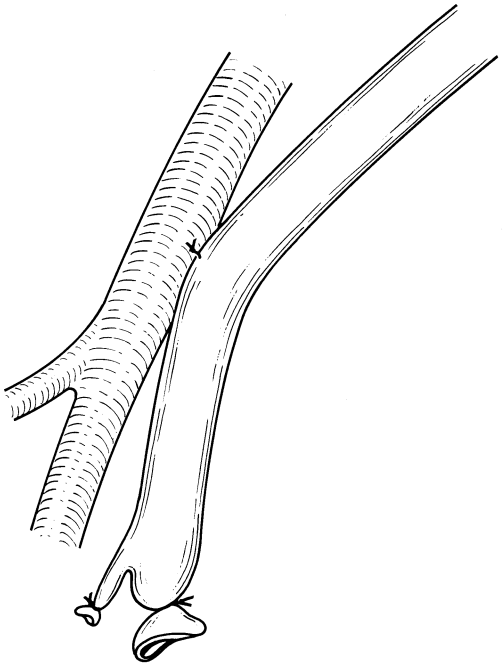


Figure 5.9 Graft length gauged by approximating the arterialized graft and the distal popliteal artery with an adventitial stitch

are approximated with an adventitial stitch (Figure **5.9**). The knee is then re-extended to verify that the length is correct. If necessary the position of the approximating stitch is changed and the procedure repeated until the length and lie are correct.

The knee is flexed to 90° to allow maximum access for anastomosis and the vein and artery are controlled with a soft clamp and a double Silastic loop respectively. Alternatively, vascular control may be achieved using a pneumatic tourniquet at mid-thigh. A longitudinal arteriotomy is then made on the anteromedial aspect of the artery. This should be more than twice the diameter of the vein in length. The vein is then sectioned, the corners trimmed and a matching cutback made. An end-to-side anastomosis is then constructed with 7/0 prolene, commencing at the heel and closing the lateral (deep) side first (Figure **5.10a, b**). Temporary insertion of a stent may be useful

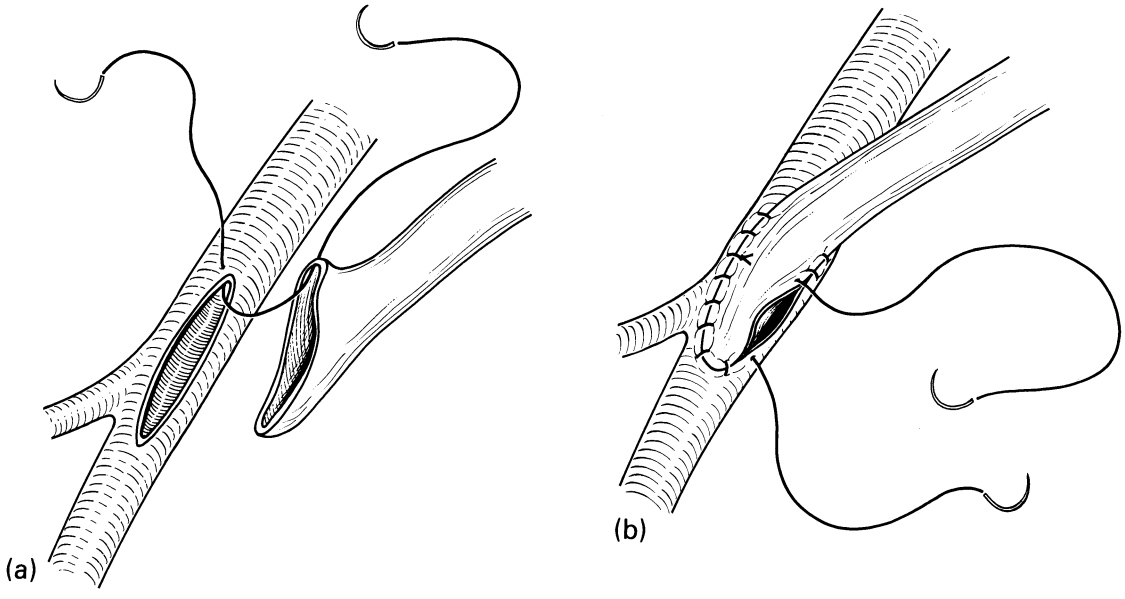


Figure 5.10(a, b) Vein graft anastomosed to the distal popliteal artery adjacent to the anterior tibial origin

when sewing the apex. Before completing the suture line, the artery and vein graft are flushed in turn and the lumen irrigated with heparin-saline. The last few sutures are then placed and the two suture arms tied together with the anastomosis exposed to arterial tension. This avoids any risk of a purse-string effect. The knee is then re-extended to confirm the absence of torsion, kinking or entrapment (Figure **5.11**).

Any branches of the vein which have been left too long may now be re-ligated closer to the graft. In addition, the remaining branches between the knee and the adductor canal are now identified and ligated to avoid a postoperative arteriovenous fistula. Ligation of these branches is deferred until this stage in order to provide a run-off for the graft while the distal anastomosis is being completed.

Intraoperative arteriography is then performed via a cannula introduced into the previously preserved branch at the proximal extremity of the vein. The proximal graft and anastomosis may be visualized by means of a film cassette placed under the thigh and buttock in a holder built into the operating table. The distal end of the graft and run-off are best shown by wrapping a cassette in a sterile drape and placing this diagonally under the area of interest. 20–30 ml of Conray 60 are hand injected at 5 ml/sec and the film exposed as the injection is being completed. Apart from providing a check on the course of the graft, the status of the valve sites and the adequacy of the distal anastomosis, arteriography will demonstrate residual arteriovenous communications in the segment between the groin and popliteal exposures. Any large communications should be explored locally and ligated, though small fistulae may be left to thrombose spontaneously. If appearances are satisfactory, the heparin may be partly reversed and the wounds closed with suction drainage.

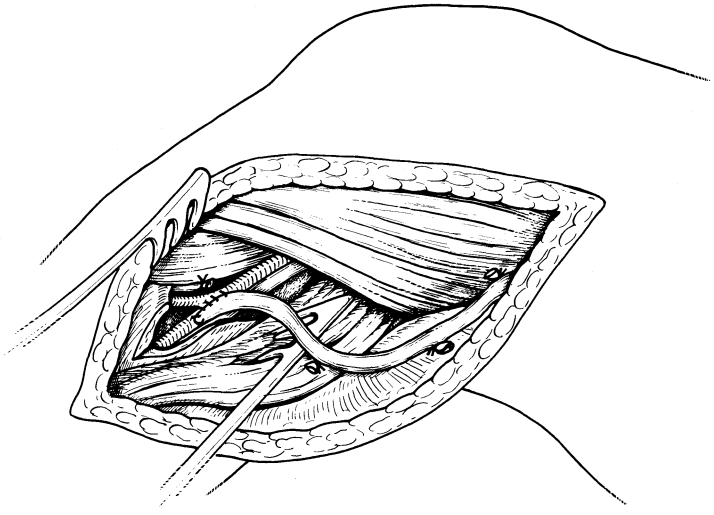


Figure 5.11 Completed *in situ* vein bypass

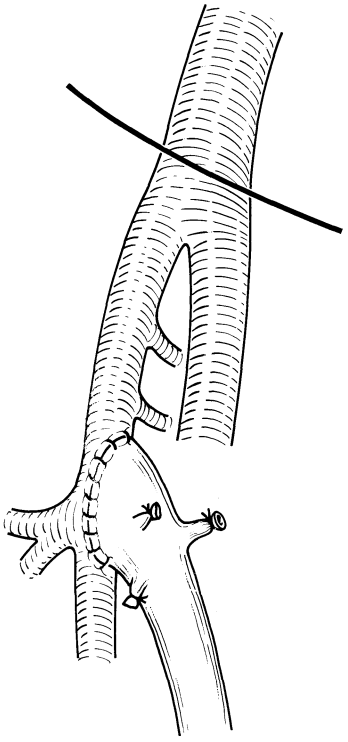


Figure 5.12 *In situ* graft attached to the profunda femoris when the common femoral bifurcation is high

VARIATIONS

Proximal anastomosis

At the proximal end the vein graft may be anastomosed to the profunda femoris if the common femoral bifurcation is high (Figure **5.12**). In other cases atheromatous involvement of the common femoral artery may necessitate additional endarterectomy and profundaplasty (Figure **5.13a, b**). Where there is a risk of losing branches such as the lateral circumflex femoral artery or the proximal superficial femoral artery during attempted common femoral endarterectomy, a more satisfactory solution may be to place a PTFE bypass between the external iliac and profunda femoris arteries and then to use this as the source for the vein graft (Figure **5.14**).

Distal anastomosis

The manner in which the popliteal artery divides is subject to some variation and this may lead to a change in the surgical plan. Thus, either the

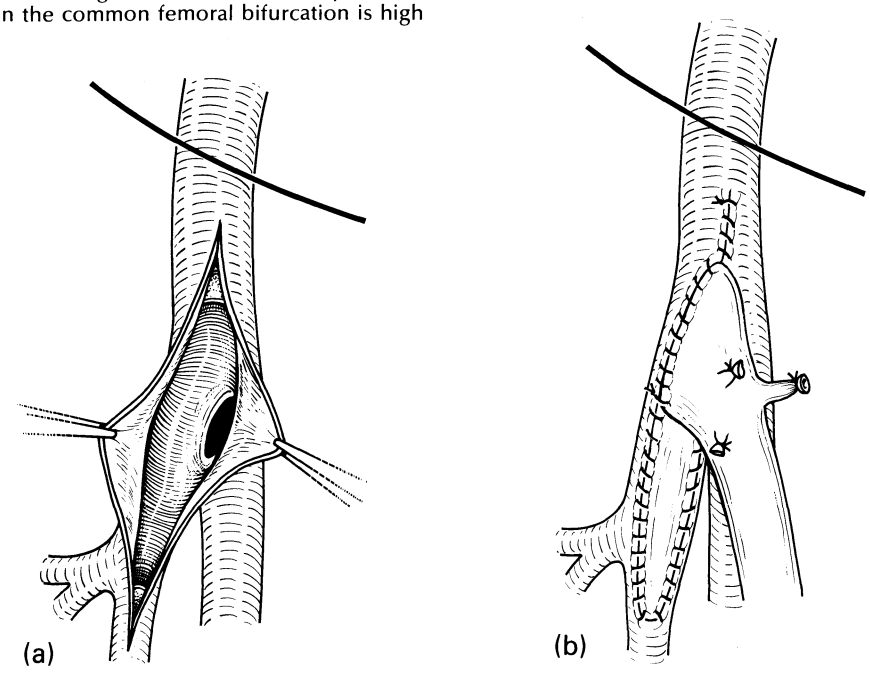


Figure 5.13(a, b) *In situ* bypass combined with common–deep femoral endarterectomy

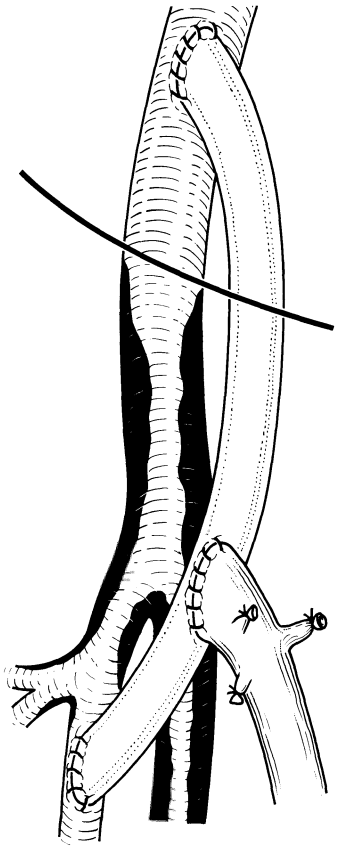


Figure 5.14 Associated common femoral atheroma. In this case a bifurcation endarterectomy might lead to loss of the lateral circumflex femoral artery or the proximal superficial femoral artery. An external iliac–profunda PTFE bypass provides a safe implantation site for the femoro-distal graft with additional profunda revascularization

anterior or the posterior tibial artery may occasionally arise more proximally than usual at knee joint level. Attempts to secure a popliteal attachment may then lead to awkward angulation of the graft over the medial hamstrings or entrapment between the medial head of gastrocnemius and the back of the knee. In order to avoid this problem the *in situ* bypass should be extended to one of the infrapopliteal vessels. Alternatively, if a popliteal attachment is preferred the saphenous vein should be removed from its bed and routed anatomically either in reversed or nonreversed fashion[25].

Valve suppression

This may be achieved by a variety of methods other than that described above. One of the earliest techniques involved valve excision via a series of transverse venotomies[39]. Although theoretically attractive, this is rather tedious and venotomy closure may risk subsequent stenosis. Valve incision using specially adapted scissors inserted from the end of the vein or via a side branch is a more recent approach which has been utilized in some centres[26]. Other techniques involve use of the Mills[40] or Hall valvulotomes[41], the latter being provided with a smooth cylinder ahead of the cutting edge to hold the vein open. Antegrade valve ablation has also been described using a smooth-tipped vein stripper introduced into the vein from above[42]. However, in order to produce effective valve suppression in the proximal segment a relatively large stripper head should be used and this may increase the risk of distal intimal injury or impaction.

Retrograde valve cutters give rise to very few complications, providing they are used with care. As previously described, a spiral movement is necessary on withdrawal to suppress both leaflets of the valve. In addition, care must be taken to avoid engaging the proximal graft anastomosis. If the stripper becomes impacted at the entry of a branch vein and cannot be easily disengaged, direct exposure is advisable, rather than continued traction. The vein should be opened and the stripper freed following which the venotomy may be closed using the branch vein as a patch. If the vein is more extensively traumatized, the safest solution is to resect the damaged segment and replace this with a short PTFE graft. Stripper impaction is more likely to occur when the saphenous vein is reduplicated in the thigh. In addition haemodynamics are then less favourable than in a single channel graft and in consequence whenever this anomaly is encountered it is advis-

able to interpose a prosthetic segment in the thigh at the outset.

A further potential complication of valvulotome usage is the occurrence of a split at the distal end of the vein due to using an overlarge stripper. This may be managed by harvesting the undamaged proximal vein and using this in reversed form as the distal portion of a composite graft.

REVERSED VEIN BYPASS

The position and preparation of the patient are the same as for *in situ* bypass. The first stage again involves a retrotibial approach with successive verification of the state of the saphenous vein and the distal popliteal artery. Spasm of the vein induced by dissection may give a false impression of size and if necessary an isolated segment of vein should be gently distended by injecting heparinized blood via a side branch before a final decision is made.

The groin is then opened via a longitudinal incision with medial prolongation in the direction of the saphenous vein. As before, separate lateral and medial fascial incisions are utilized for exposing the artery and saphenofemoral junction respectively, in order to avoid cutting into lymphatic tissue.

A series of interrupted skin incisions are then made down the leg for the purpose of harvesting the vein (Figure **5.15**). These incisions must be placed directly over the vein, since undermining of the skin edges may invite postoperative necrosis. A long continuous incision gives slightly better exposure, but may be attended by impaired wound healing. Particular care must be taken to avoid avulsing any small branches during dissection of the vein since subsequent repair may narrow the graft. All branches should be divided and ligated at a distance from the main vein with religation in closer proximity, if necessary, once the vein has been exposed to arterial tension. Marker stitches or methylene blue may be used to mark the anterior surface of the vein though torsion may be more easily avoided by the technique described below, in which the vein is arterialized prior to tunnelling. Once sufficient length has been exposed the vein is sectioned and removed.

Before closing the saphenofemoral junction, blood is withdrawn into a heparinized syringe and used to irrigate the graft. Any leaks are closed with 6/0 or 7/0 prolene sutures (Figure **5.16b**). Overdistension should be avoided as should irrigation with saline, both of which may favour the development of intimal hyperplasia[43,44]. The vein is then set aside in heparinized blood ready for use.

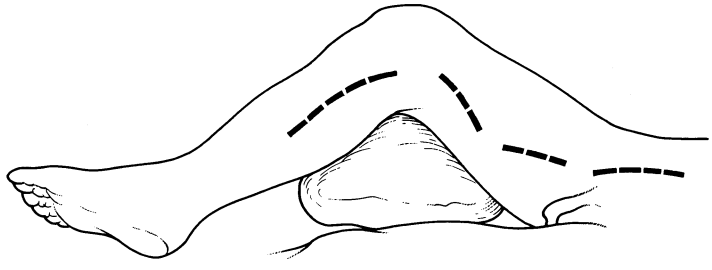


Figure 5.15 Reversed vein bypass. Incisions for vein harvest and arterial exposure

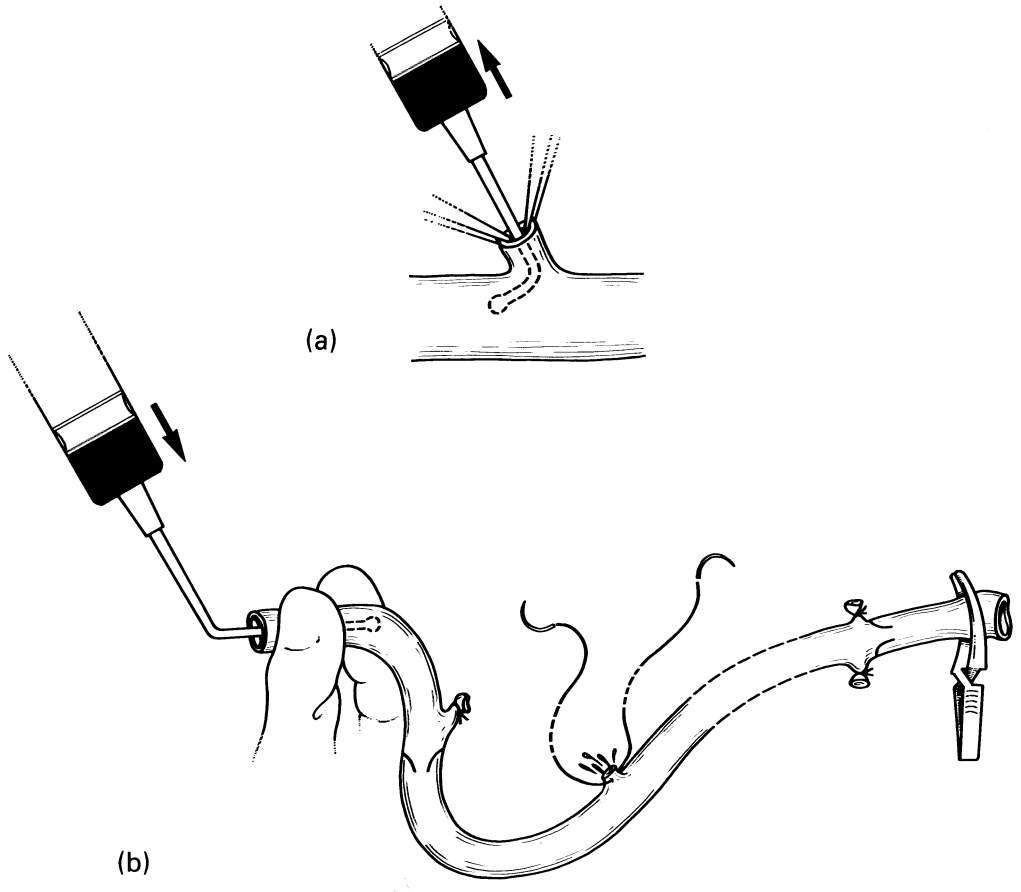


Figure 5.16(a, b) Heparinized blood is aspirated from the common femoral vein and used to irrigate the graft

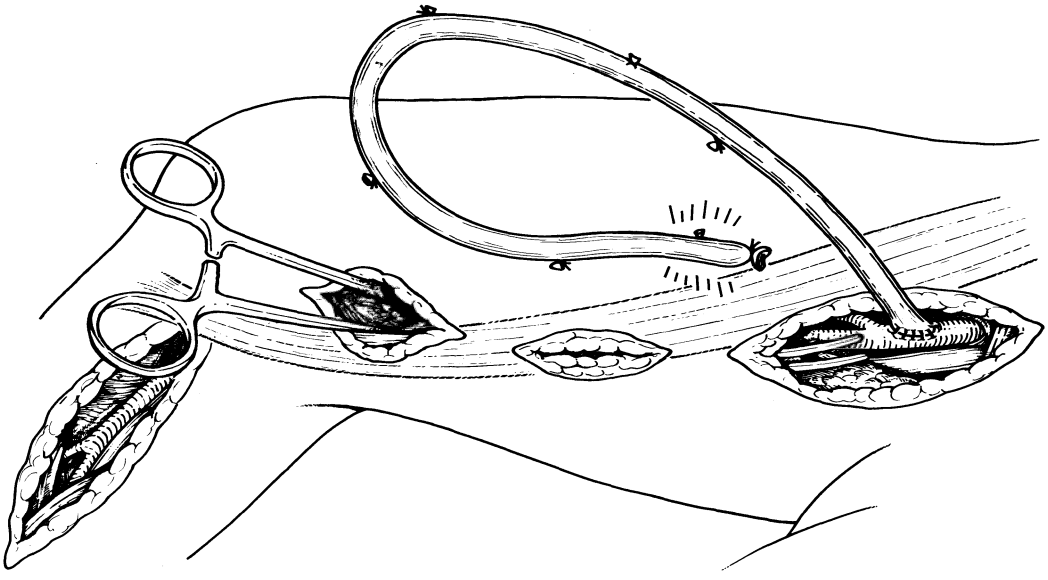
A tunnel is now developed deep to sartorius. The above-knee incision used for vein harvest is deepened to expose the upper popliteal fossa and then a tunnel is developed by blunt dissection between here and the groin exposure. Although tunnelling instruments are popular, there may be some risk of avulsing small venous or arterial branches during instrument passage and digital dissection is safer. Distally the tunnel is continued deep to the medial head of gastrocnemius to reach the retrotibial exposure. When the tunnel is completed a tape may be left in place as a guide for subsequent graft passage.

Systemic heparin is then administered in a dose of 1 mg/kg body weight. Two options for graft implantation are available. If the distal end is completed first, the least accessible anastomosis is made easier. However, graft length is more difficult to judge as the vein is not arterialized and there may be an increased risk of torsion. We

therefore prefer to perform the femoral anastomosis first, which has the additional advantage that the site of vein transection can often be chosen so that a venous branch is incorporated into the future anastomotic heel.

A longitudinal arteriotomy is made in the common femoral artery and after appropriate trimming and spatulation the vein graft is attached end-to-side. If the common femoral artery is severely thickened, an intermediate vein patch (Linton) may be used to avoid narrowing the graft inflow. This provision may be particularly important in the longer types of reversed vein bypass. Alternatively, if there is additional profunda stenosis a common–deep femoral endarterectomy or ilioprofunda bypass may be inserted before implanting the vein graft, as previously described.

Once the proximal anastomosis is complete the clamps are removed and the vein graft allowed to pulsate freely after ligating the distal end. The distended graft is then drawn down the preformed tunnel to the retrotibial exposure (Figure **5.17**).



5.17 The proximal anastomosis has been completed. The arterialized graft is then drawn down the subsartorial tunnel with the aid of an aortic clamp

The preferred anastomosis site is at or immediately proximal to the origin of the anterior tibial artery. As with *in situ* bypass, a temporary approximating stitch may be placed between the vein graft and the artery to help in judging graft length. The knee is then extended to confirm that this approximation is correct. An arteriotomy is then made in the distal popliteal artery and the spatulated end of the vein graft anastomosed in end-to-side fashion with 6/0 or 7/0 prolene. After removing the clamps a final check should be made to ensure that the graft is lying freely in the tunnel without kinking or compression from extraneous bands (Figure **5.18**). Intraoperative arteriography is then performed either via a 19-gauge needle inserted into the common femoral artery above the proximal anastomosis or by means of cannula introduced into a convenient vein branch. If appearances are satisfactory, the heparin may be partly reversed and the incisions closed with suction drainage.

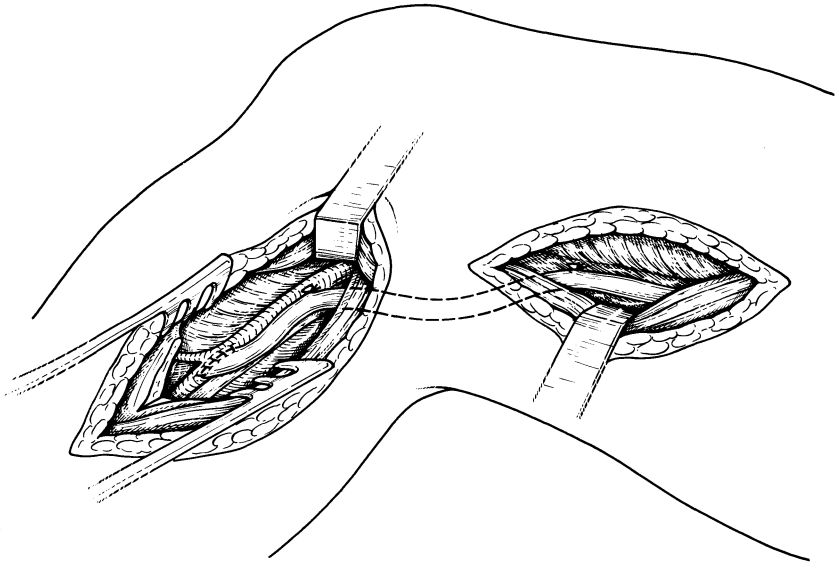


Figure 5.18 Distal attachment of the reversed vein bypass has been completed

BYPASS TO THE PROXIMAL POPLITEAL ARTERY

In a minority of cases the proximal popliteal artery is of good quality and suitable for bypass attachment. Femoro–proximal popliteal bypass is particularly useful when autogenous vein is unavailable, since prosthetics may then be used with a reasonable chance of success[18,36].

Access is achieved via an incision commencing five to six finger-breadths above the femoral condyle and descending to the knee-joint level (Figure **5.19a**). Sartorius is retracted posteriorly and the fascial roof of the adductor canal opened, taking care to preserve the descending genicular artery and accompanying saphenous nerve (Figure **5.19b**). The adductor magnus tendon is divided to fully expose the femoropopliteal junction (Figure **5.19c**). Significant plaque in the mid-popliteal segment may be overlooked on arteriography and it is important to palpate this segment before proceeding. If a lesion is detected here the medial head of gastrocnemius should be divided and a more distal attachment sought.

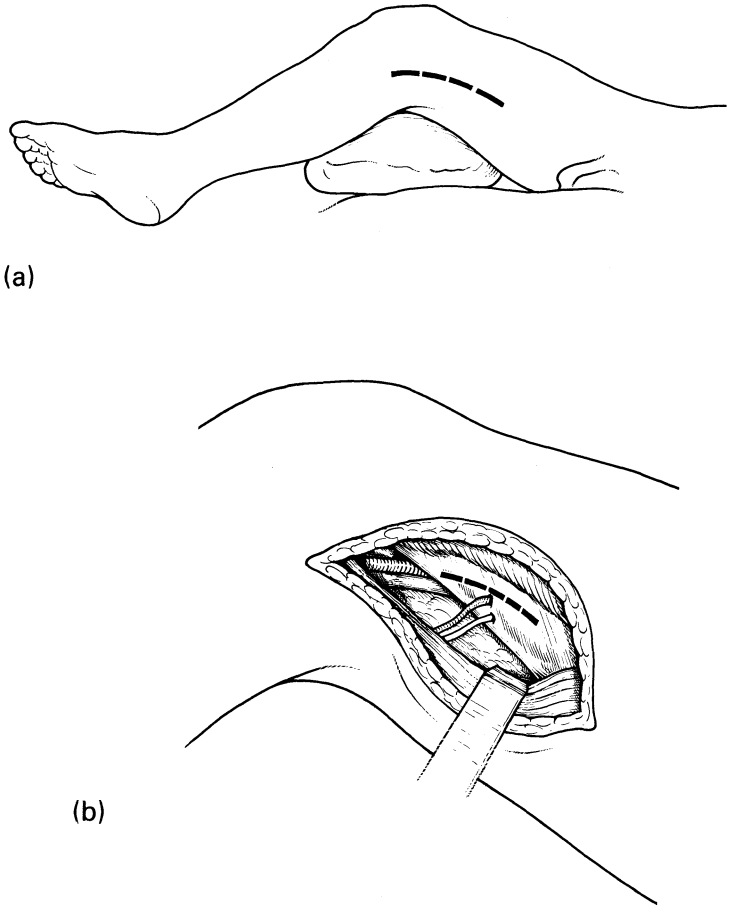


Figure 5.19(a–c) Exposure of the proximal popliteal artery: (a) skin incision. (b) sartorius is retracted posteriorly. The dotted line indicates the incision in the fascial roof of the adductor canal

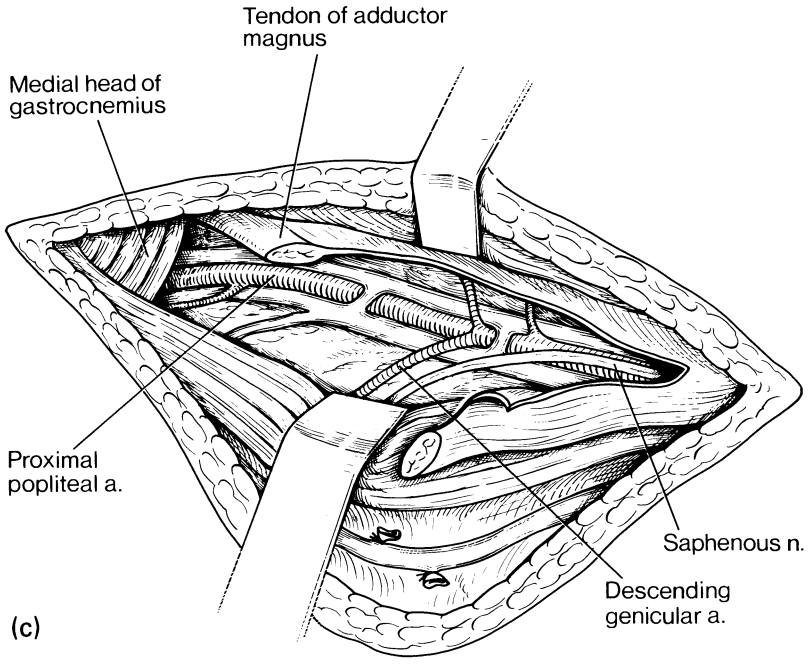


Figure 5.19 continued (c) femoropopliteal junction fully displayed by section of the adductor magnus tendon

BYPASS TO THE ISOLATED POPLITEAL SEGMENT

Arteriography may sometimes reveal a patent popliteal segment in relation to the sural arteries with proximal and distal occlusion. Bypass grafting to such a segment is one of the options which may be considered when attempting limb salvage.

Access may be achieved by a midpopliteal incision. The tendons of sartorius, gracilis and semitendinosus are retracted superiorly and the medial head of gastrocnemius divided. If the patent segment extends below the knee joint level, an *in situ* bypass may be possible. In other cases a reversed vein or PTFE graft is utilized.

Results intermediate between femoropopliteal and femorotibial bypass have been reported with this technique[11,45]. However, where there is extensive foot necrosis or infection, isolated popliteal bypass is unlikely to increase perfusion sufficiently to achieve healing and it should then be combined with a distal extension (Figure **5.20**).

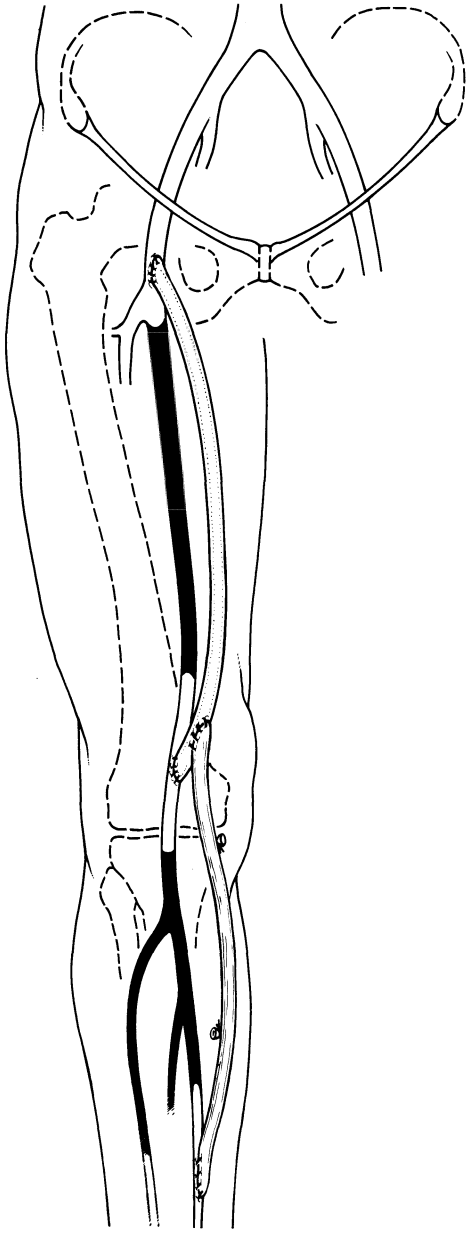


Figure 5.20 Sequential bypass. A PTFE graft has been placed to an isolated popliteal segment with a venous extension limb to the distal posterior tibial artery

FEMOROTIBIAL AND FEMOROPERONEAL BYPASS

In many patients atherosclerotic involvement of the popliteal artery and trifurcation precludes femoropopliteal bypass. Revascularization may then require bypass extension to the more distal leg or foot. These small vessel bypasses are more difficult to perform and 20% or more may close within the first few months of insertion[8,12,19]. In view of this less certain outcome, femoro–infrapopliteal bypass is best restricted to limb salvage cases only.

Although grafts to infrapopliteal arteries that are obstructed distally may occasionally remain patent, most do not, and a minimum requirement should be distal communication with a pedal arch either directly or indirectly via large bridging collaterals[46]. However, in some cases the distal run-off may only be adequately demonstrated once the bypass is functioning and lack of filling of the pedal arch preoperatively should not necessarily contraindicate a surgical attempt[12].

Where more than one infrapopliteal vessel is open, the choice of anastomosis site depends on relative vessel size, freedom from disease and on cross-communications. As a general rule the order of preference is posterior tibial, anterior tibial and then the peroneal artery. In all cases the anastomosis should be placed as proximally as possible to shorten the bypass and to provide antegrade perfusion in the maximum number of collaterals. Sequential bypass to more than one vessel may also be considered when the run-off is severely restricted[47,48]. A proximal side-to-side relay anastomosis may be undertaken to an open popliteal or proximal tibial segment with a second end-to-side anastomosis lower down the leg. Alternatively a Y-extension arrangement may be utilized[49]. An additional method of increasing the graft run-off is to create a common ostium arteriovenous fistula at the distal implantation site[50]. Although good results have been reported in some series others have found this approach less helpful[51].

The technique of graft insertion is similar to that described for femoropopliteal bypass. However certain aspects of distal graft attachment require

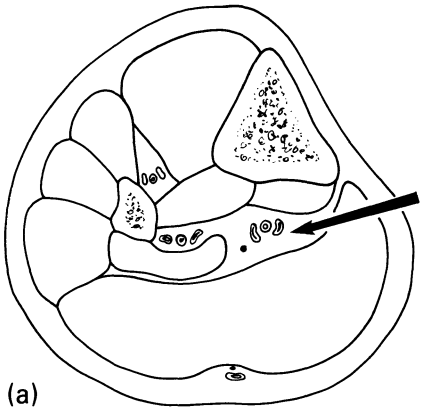
emphasis. Vascular control should be achieved with Silastic loops or intraluminal occluders rather than conventional clamps in view of the particularly delicate nature of the infrapopliteal vessels. A pneumatic tourniquet around the distal thigh is an alternative option. In the case of a reversed vein or non-vein bypass there may be a marked disparity in size between graft and artery and a relatively long arteriotomy and graft cutback may be necessary. Anastomosis to the dorsalis pedis artery, for example, may need to be up to 3·5 cm in length. This problem is less evident with *in situ* vein bypass and, as previously indicated, this type of reconstruction has other advantages when revascularizing the more distal arteries.

Optical magnification, ophthalmic instruments and fine prolene sutures (up to 8/0 or 9/0) may be needed for anastomoses in the distal leg or foot. Loosely looped sutures are placed at the anastomotic heel and the graft gently eased down onto the artery. The distal anastomotic angle is then closed with interrupted sutures over a coronary artery dilator or intraluminal occluder. Careful irrigation of the suture line and graft lumen prior to completing the anastomosis is particularly important since a little fibrin or platelet deposition can lead to graft occlusion. Anastomoses at this level are more prone to technical error, and intraoperative arteriographic control should be regarded as mandatory.

BYPASS TO THE POSTERIOR TIBIAL ARTERY

The posterior tibial artery is the most easily accessible of the infrapopliteal vessels. The proximal segment may be exposed via the popliteal fossa. The medial head of the gastrocnemius is retracted posteriorly and the soleal arcade incised taking care to protect the underlying vessels. Progressive distal mobilization of the tibioperoneal trunk will then display the proximal posterior tibial artery.

The more usual site for posterior tibial bypass is at midcalf level. This segment of the artery may be exposed by an incision approximately 10 cm in length just behind and parallel with the medial border of the tibia (Figure **5.21a, b**). After incising the deep fascia of the leg, gastrocnemius is retracted posteriorly and the soleus detached from the tibia (Figure **5.21c**). Posterior retraction of soleus will then reveal the neurovascular bundle lying in the groove between flexor digitorum longus medially and tibialis posterior laterally (Figure **5.21d**). Care must be exercised when retracting soleus and gastrocnemius to avoid avulsion of small arterial and venous branches passing out at right angles from the main vascular bundle. The artery is usually accompanied by two veins with several crossbridges. The latter should be divided and a 2–3 cm segment cleared ready for implantation.



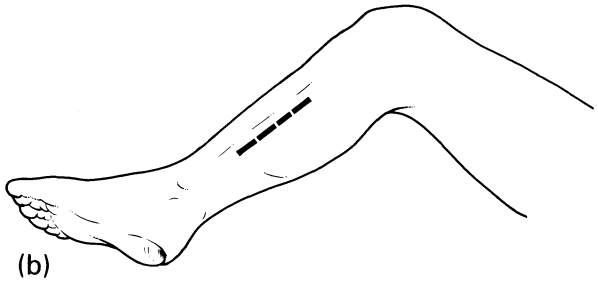


Figure 5.21(a–d) Exposure of the posterior tibial artery in the middle third of the leg: (a) transverse section of leg showing medial approach. (b) skin incision

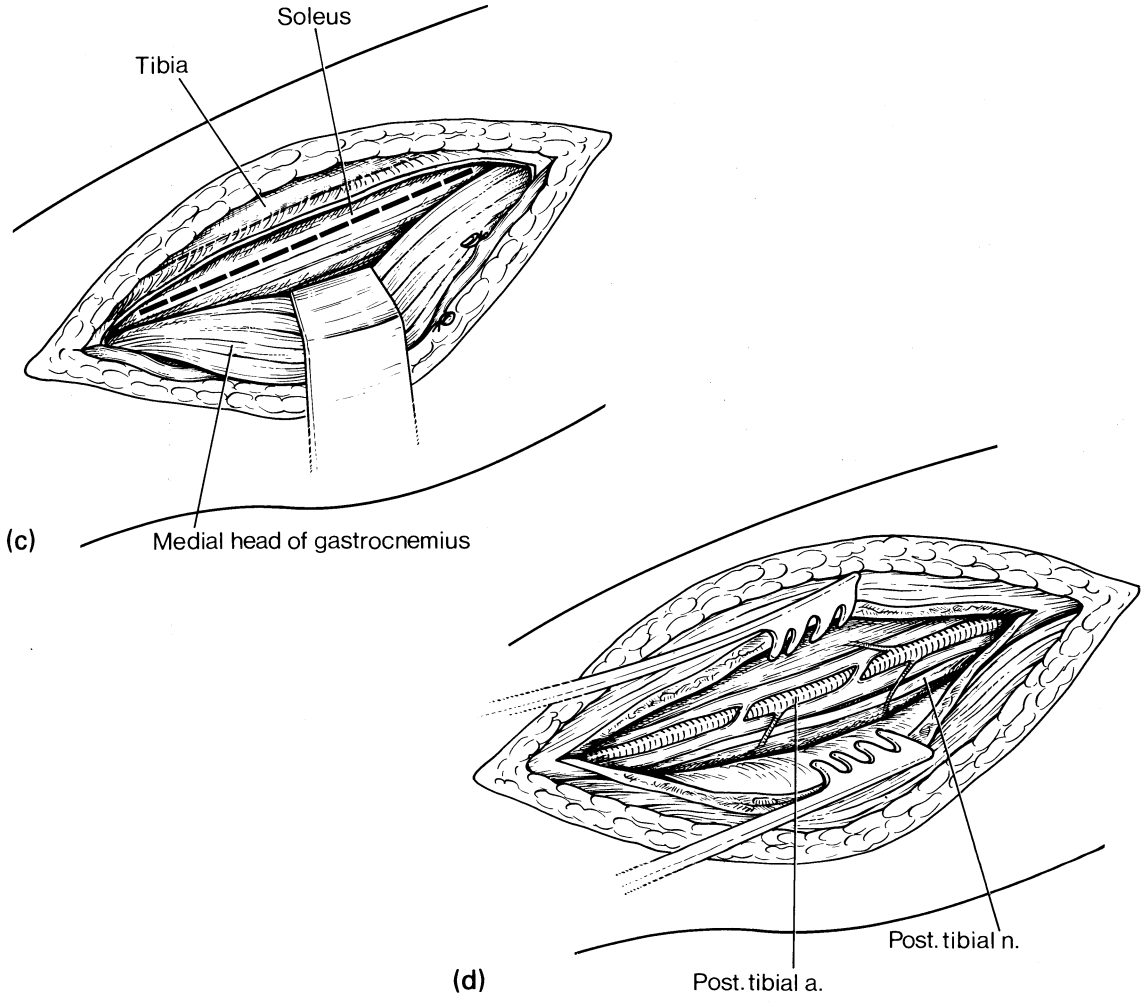


Figure 5.21 continued (c, d) trans-soleal exposure of the posterior tibial vessels.

In the case of an *in situ* vein graft, the saphenofemoral anastomosis is completed and the vein divided at the ankle. The valves are then disrupted using Cartier strippers, as previously described. Routeing the graft to the distal anastomosis site is particularly easy at this level because of the short horizontal distance between the normal vein course and the arterial axis. Reversed vein or other grafts may be tunnelled subsartorially to the popliteal fossa and then deep to the medial head of gastrocnemius to enter the midcalf exposure.

The posterior tibial artery may also be exposed in the distal third of the leg via a longitudinal incision midway between the Achilles tendon and the posteromedial border of the tibia (Figure **5.22a**). The thick investing fascia anterior to the Achilles tendon is incised and the neurovascular bundle located between flexors digitorum and hallucis longus (Figure **5.22b**). *In situ* vein bypass grafts may be routed without problem at this site. Reversed vein or other grafts are tunnelled from the groin to the popliteal fossa usually deep to sartorius and then led down to the ankle either just beneath the deep fascia or in the subcutaneous position.

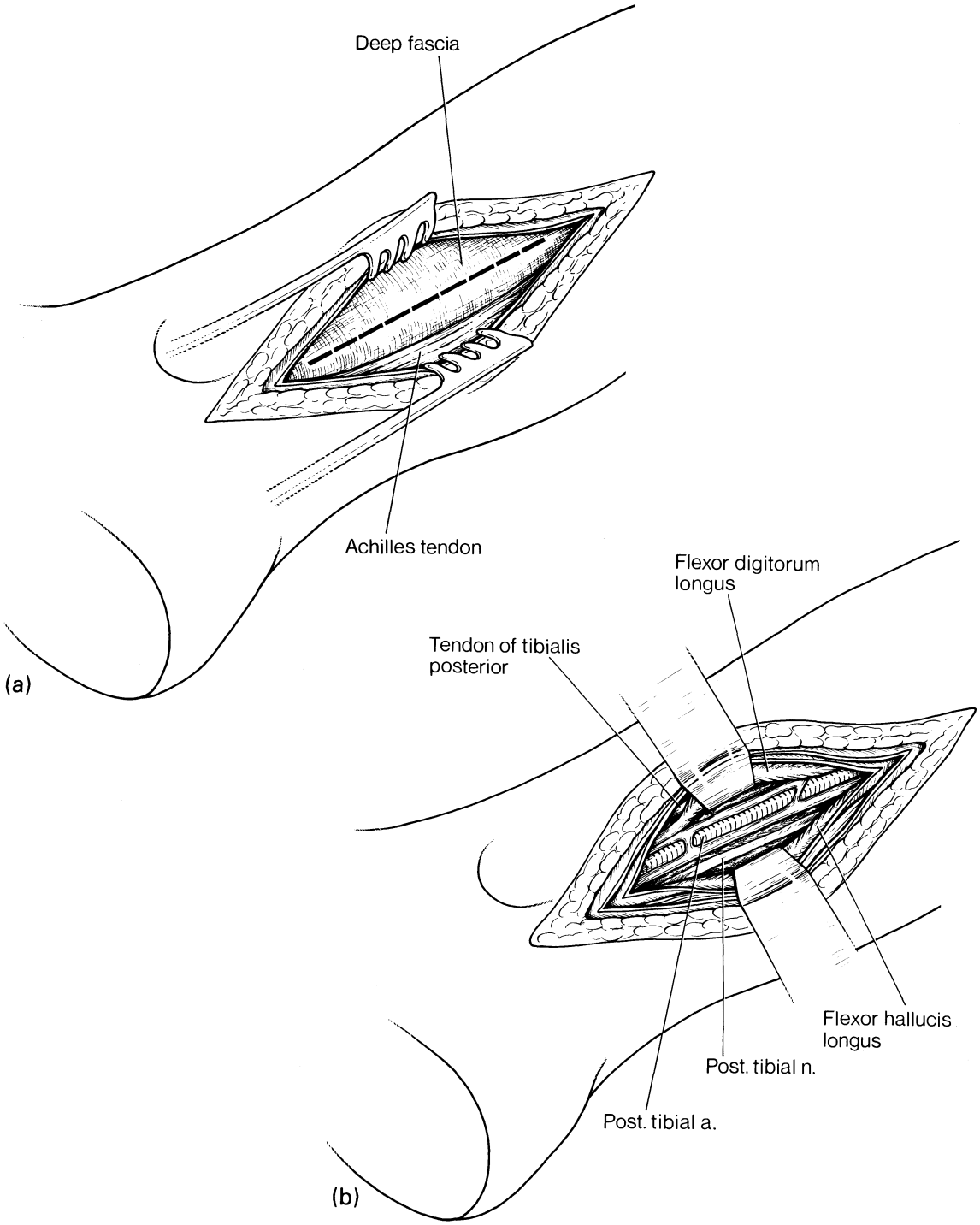
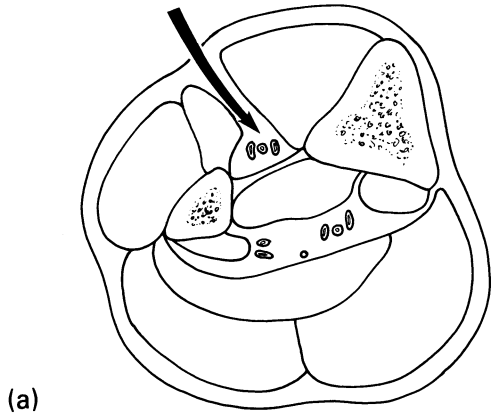


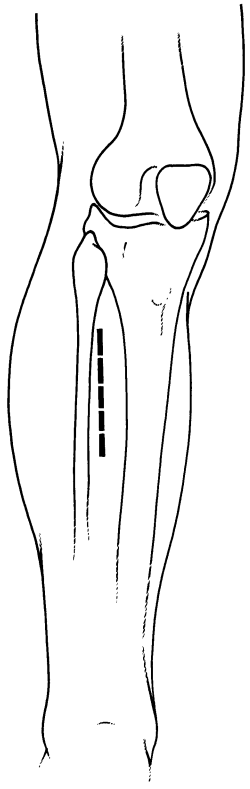
Figure 5.22(a, b) Exposure of the posterior tibial artery in the distal third of the leg: (a) the deep fascia is incised anterior to the Achilles tendon. (b) the neurovascular bundle is exposed by retraction of the flexor tendons

BYPASS TO THE ANTERIOR TIBIAL ARTERY

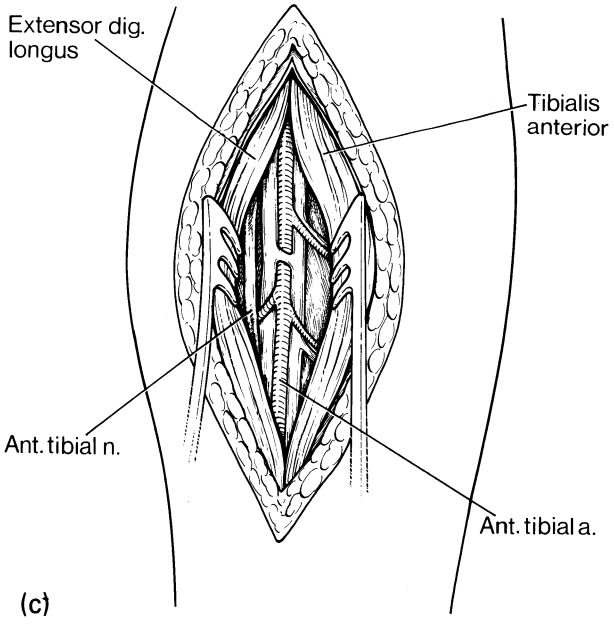
Access to the anterior tibial artery in the upper and middle thirds of the leg may be best accomplished by an anterior approach (Figure **5.23a–c**). The limb is positioned with the knee flexed 60–90° and the foot dorsiflexed to relax the anterior compartment muscles. A longitudinal incision 10 cm or so in length is then made over the appropriate segment of patent artery. This incision should lie just lateral to the crest of the tibia over the palpable groove between the tibialis anterior and extensor digitorum longus. After incising the deep fascia, the two muscles are separated to expose the neurovascular bundle lying deeply on the interosseous membrane (Figure **5.23c**).



(a)



(b)



(c)

Figure 5.23(a–c) Exposure of the anterior tibial artery in the proximal-middle third of leg: (a) transverse section of the leg showing anterior approach. (b) skin incision. (c) anterior compartment muscles separated to reveal the neurovascular bundle on the interosseous membrane

As before, the artery should be carefully freed from the accompanying veins with division of venous cross-bridges as necessary. Several branch arteries may be encountered and these should be preserved as they will form part of the graft outflow tract. The anterior tibial artery is then held aside with Silastic loops and the interosseous membrane widely incised. This step is important to ensure that the graft will be free from compression.

In situ bypass grafts are slightly more awkward to tunnel here than to the posterior tibial artery. Soleus should be widely detached from the medial border of the tibia to allow the graft to pass laterally in a smooth curve between the superficial and deep posterior compartments of the leg. The graft is then led out through the opening in the interosseous membrane to the anterior exposure. Sufficient length should be allowed so that the graft will fall comfortably into position parallelling the distal artery. Reversed vein and prosthetic grafts may be tunnelled in an approximately anatomical route with the aid of a relay incision in the distal popliteal fossa. Simultaneous digital dissection from the anterior exposure and from the popliteal fossa will then complete the tunnel (Figure **5.24**).

Bypass grafts may also be placed to the anterior tibial artery in the distal third of the leg. This segment may be approached via an anterior incision just above the ankle (Figure **5.25a**). The tibialis anterior tendon is first identified adjacent to the anterior border of the tibia. Outside this is the tendon of extensor hallucis longus which overlies the neurovascular bundle (Figure **5.25b**). After dorsiflexing the foot to relax the anterior compartment muscles, tibialis anterior is reflected medially and extensor hallucis longus laterally to expose the distal artery.

In situ vein bypass to the distal anterior tibial artery may be accomplished by sectioning the saphenous vein beyond the ankle and swinging it laterally in front of the tibia. Reversed vein grafts may be tunnelled subfascially or subcutaneously down the medial side of the leg before coursing around the tibia. Occasionally it may be necessary to divide some of the extensor tendons to improve the lie of these distal grafts.

The dorsalis pedis may occasionally be the only suitable artery for distal graft attachment. The artery runs from the midpoint of the ankle towards the first interosseous space and may be exposed by raising an appropriate flap on the dorsum of the foot (Figure **5.26a**). The vessel is best identified proximally between the tendons of extensor hallucis and digitorum longus and then traced distally where it lies between extensor hallucis brevis and longus (Figure **5.26b**). *In situ* vein bypass is particularly appropriate at this level because of the proximity of the saphenous vein and the length of graft required.

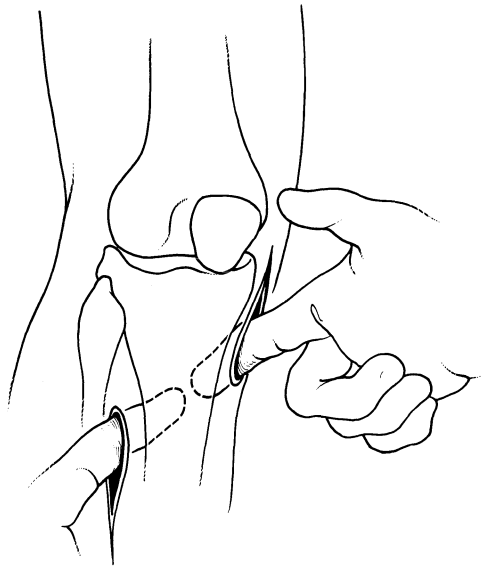


Figure 5.24 Bypass tunnel developed by simultaneous digital dissection from the anterior tibial exposure and from the popliteal fossa

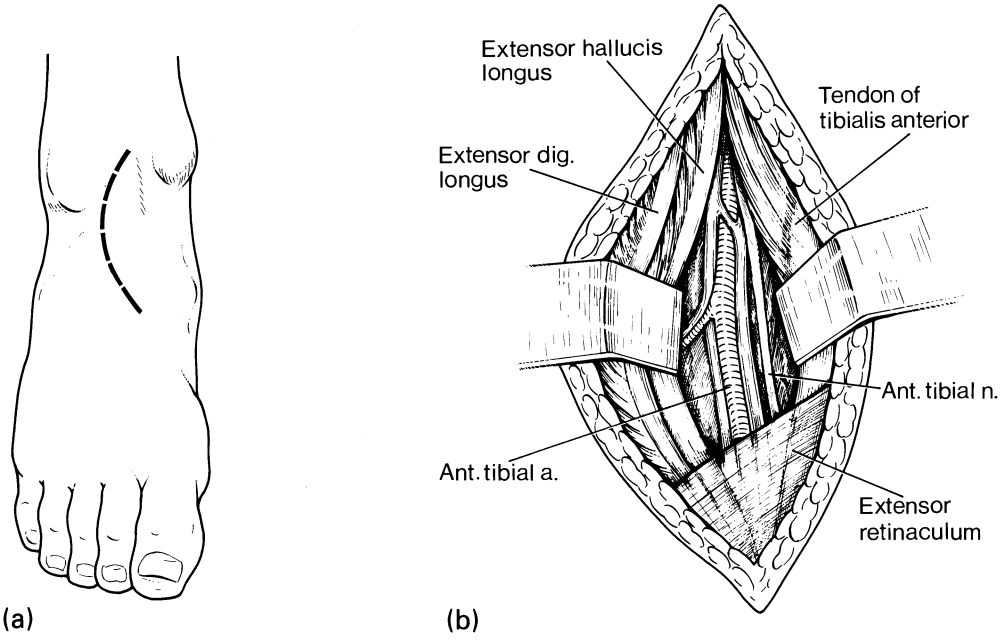


Figure 5.25(a, b) Exposure of the anterior tibial artery in the distal third of the leg: (a) skin incision. (b) the extensor tendons are retracted to expose the neurovascular bundle

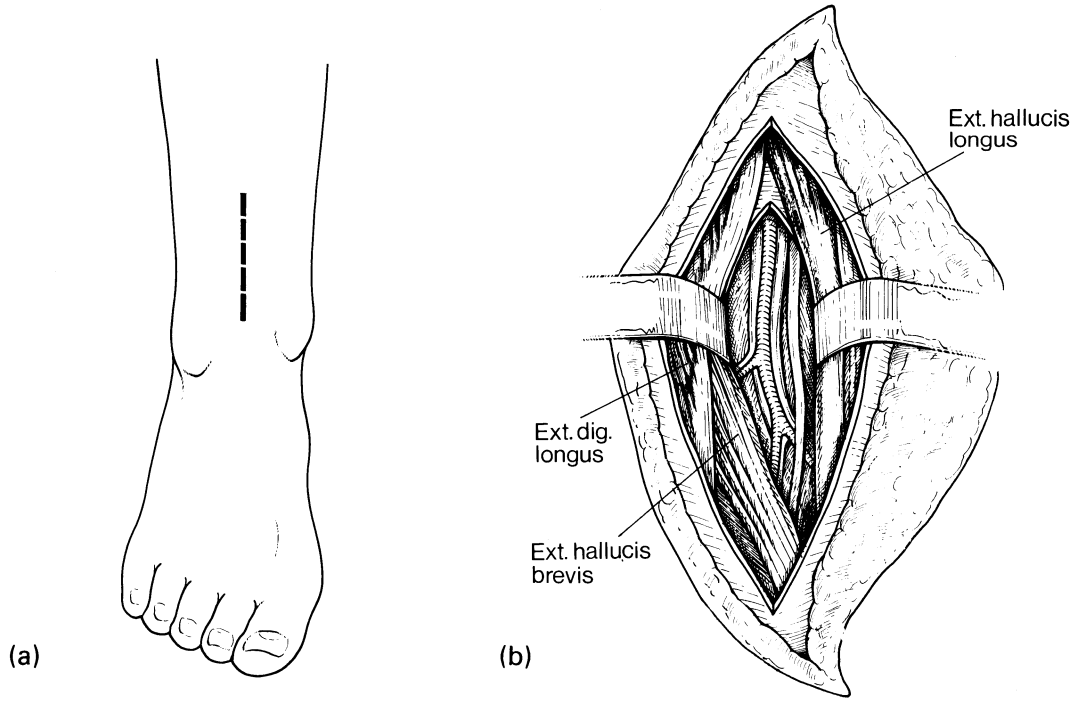


Figure 5.26(a, b) Exposure of the dorsalis pedis artery: (a) skin raised as a flap over the course of the artery. (b) dorsalis pedis artery exposed distally between extensor hallucis longus and extensor hallucis brevis

BYPASS TO THE PERONEAL ARTERY

Although the peroneal artery may be exposed from the medial side of the leg, a lateral approach with segmental fibulectomy is easier. The limb is placed with the knee flexed almost to 90° and the foot internally rotated. An incision 12–15 cm in length is made over the lateral aspect of the middle third of the leg (Figure **5.27a**). This incision should lie quite posteriorly over the intermuscular septum between triceps surae and the peroneal muscles. The plane of separation between these two compartments is then deepened to expose the posterolateral border of the fibula (Figure **5.27b**). A 10 cm segment of bone is cleared of soft tissue with a rougine and resected using a Gigli saw, taking care to avoid damaging the peroneal vessels which are immediately beneath (Figure **5.27c**). The artery is then prepared for anastomosis by separating it from the investing veins as previously described.

In situ vein grafts are passed laterally between the superficial and deep posterior compartments of the leg after detaching soleus from the tibia. Reversed vein or other grafts may be tunnelled directly from the popliteal fossa.

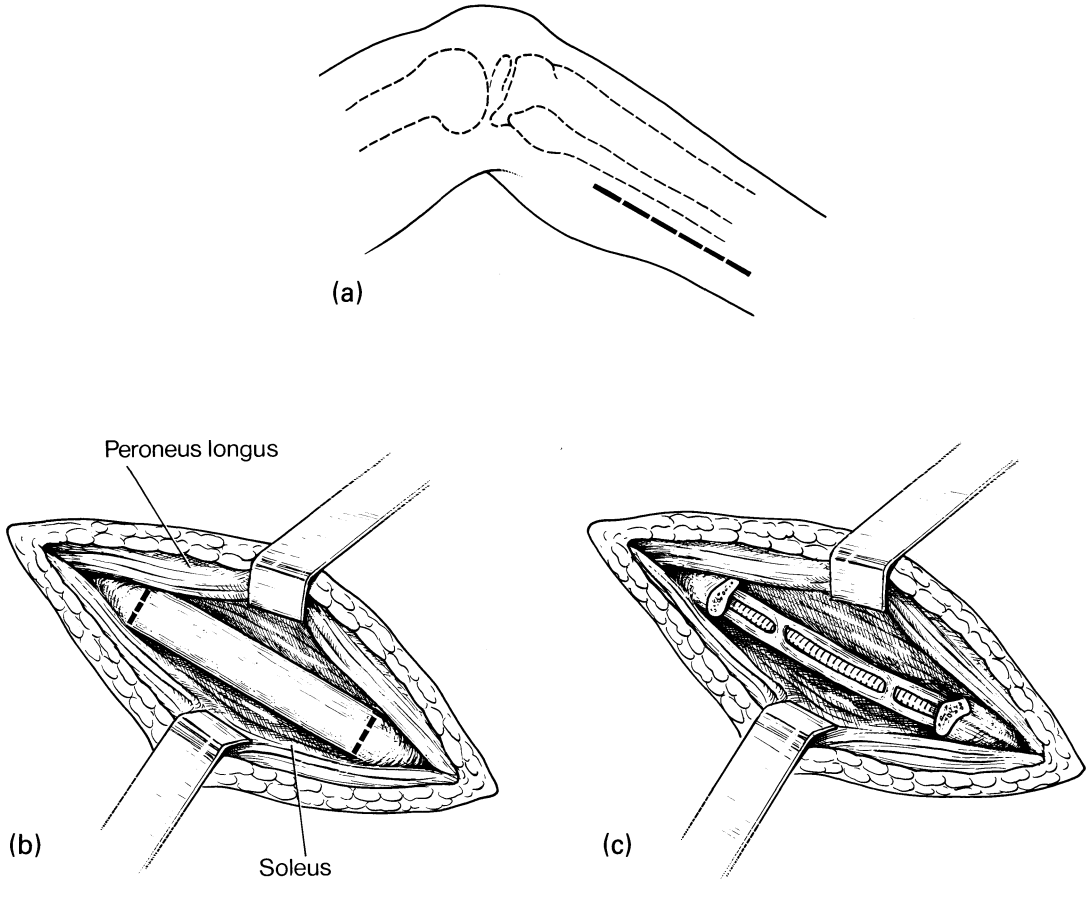


Figure 5.27(a–c) Exposure of the peroneal artery: (a) skin incision. (b) fibula exposed. (c) fibula resected to reveal the peroneal vessels

POPLITEAL ANEURYSMS

Atherosclerotic popliteal aneurysms are among the most common forms of peripheral aneurysm. Most patients are elderly and there is a marked male predominance. Aneurysms at other sites, particularly the aorta, may be additionally present in up to 50% of cases, this figure rising to nearly 80% when both popliteal arteries are aneurysmal[52].

These aneurysms are particularly prone to complications, possibly as a result of repeated compression with knee movement. Thus severe ischaemia from acute thrombosis or embolization may be the presenting feature in up to one third of all cases[53]. Others may present with claudication due to more chronic occlusion or with punctate distal ischaemic lesions from atheromatous microembolization. Local symptoms may also occur either from venous or nerve compression or from rupture. In view of the frequency and seriousness of these complications, surgical treatment is indicated in virtually all cases. Once complications have occurred, the outcome is much less certain[54] and for this reason surgery is best undertaken as soon as the diagnosis is made.

In doubtful cases an ultrasound scan may be helpful in determining popliteal artery size. This may also strengthen the case for surgery by demonstrating intraluminal clot. A full arteriographic assessment should then be made in order to plan the reconstruction and to delineate aneurysmal disease elsewhere. If coexistent aortoiliac aneurysmal disease is identified this should be dealt with first in view of the potentially greater threat to life. Femoral aneurysmal disease may be dealt with either at the same time as the popliteal aneurysm or at a later stage.

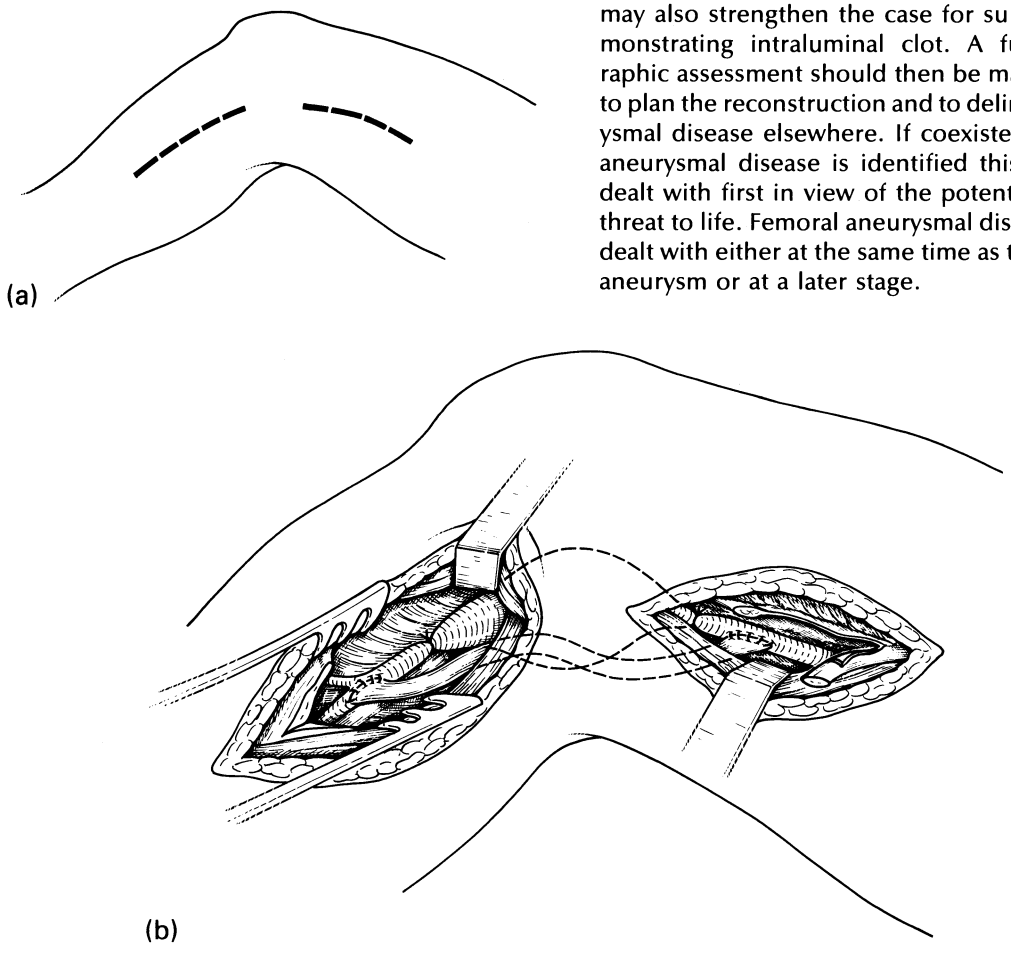


Figure 5.28(a, b) Method of managing a small popliteal aneurysm: (a) proximal and distal control via two separate incisions. (b) the aneurysm is excluded and a vein bypass graft inserted

SURGICAL MANAGEMENT

Access for reconstruction is best achieved via a medial approach. This allows flexibility should proximal or distal extension be required and will allow easy harvest or *in situ* use of the long saphenous vein, which is the preferred graft material[53].

One method of treating small popliteal aneurysms is by exclusion-bypass using separate proximal and distal incisions (Figure **5.28a, b**). Although this approach is simple and quick there is a risk of continuing aneurysmal expansion due to uninterrupted collateral inflow[55] and most cases are best treated by direct exposure and laying open of the sac. This is particularly advisable with large aneurysms in order to decompress the popliteal fossa and to allow anatomical routing of the graft.

The aneurysm may be exposed by dividing the tendons of sartorius, gracilis, semitendinosus and semimembranosus together with the medial head of gastrocnemius (Figure **5.29a**). Manipulation of the aneurysm may risk intraoperative embolization and it is therefore advisable to obtain proximal and distal control at a distance before approaching the lesion.

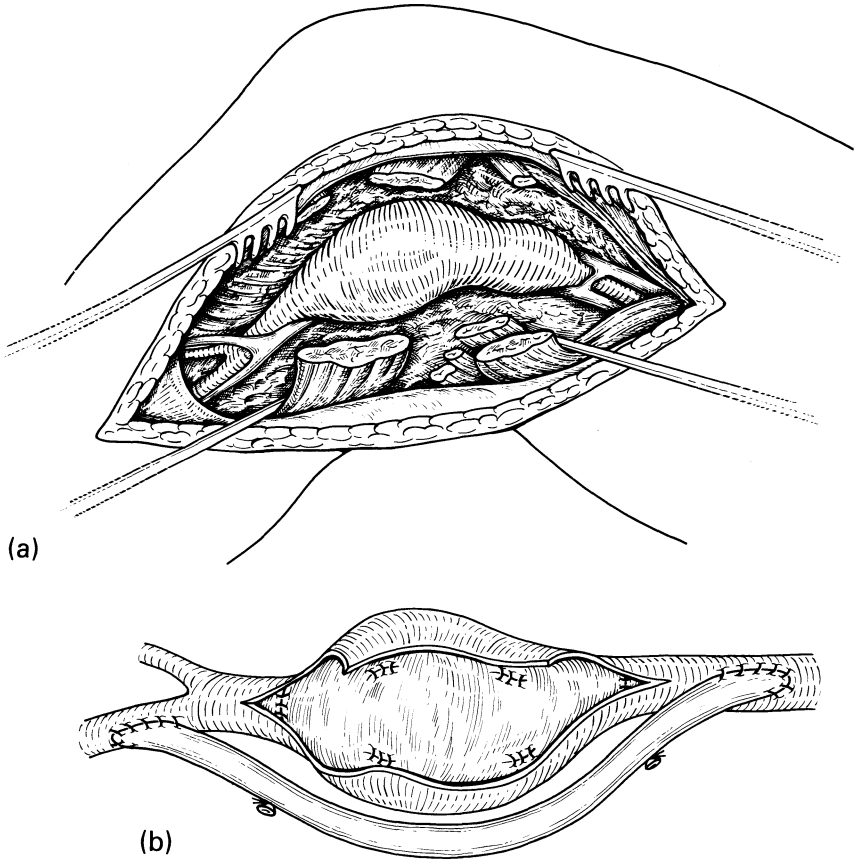


Figure 5.29(a, b) Preferred method of treating popliteal aneurysms: (a) medial approach with division of the pes anserinus tendons and the medial head of gastrocnemius. (b) the sac has been opened and the main channel and collaterals oversewn from within. An end-to-side vein bypass is then inserted

After heparinization, clamps are applied and the aneurysm opened. All branches including the main channel should then be sewn off from within the sac. Revascularization may be most satisfactorily achieved by means of a saphenous vein graft inserted end-to-side above and below the aneurysmal zone (Figure **5.29b**). This arrangement avoids any mismatch in size between graft and artery and allows graft attachment well beyond the diseased zone. In some patients the outflow tract may be occluded by previous embolization and the bypass graft may then require extension to midcalf level or beyond.

An alternative method of graft replacement is by an endosaccular technique (Figure **5.30a, b**). This may be particularly indicated when the distal vessels are diffusely thickened making infrapopliteal bypass less satisfactory. Prosthetics may be more suitable than autogenous vein for this kind of reconstruction. On completion the aneurysm sac should be left in place rather than attempting to wrap it around the prosthesis as at other sites. A sufficiently tight wrap may risk damaging the popliteal veins or nerves embedded in the wall, while if the edges of the sac are only loosely approximated a perigraft haematoma may result.

Management of acute thrombosis may be difficult and a significant number of these cases may require eventual amputation[54]. If the extremity is not in immediate jeopardy, a local intra-arterial infusion of streptokinase via a femoral catheter should be considered[56]. If this restores distal pat-

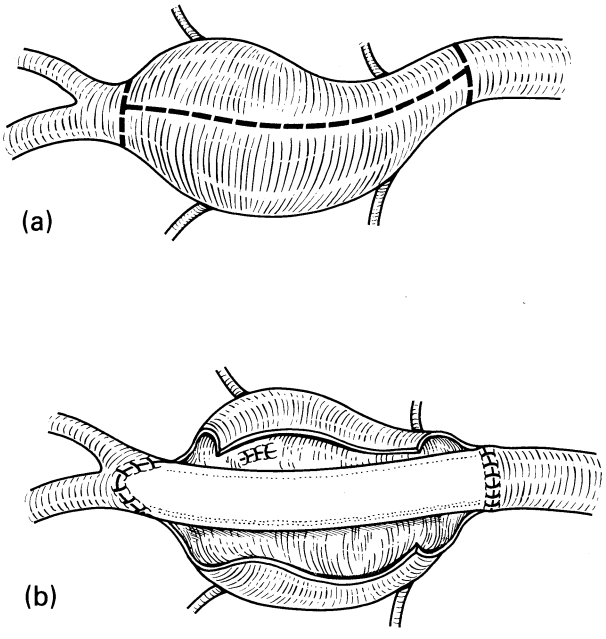


Figure 5.30(a, b) Direct replacement using a PTFE segment with inlay anastomoses

ency, the aneurysm can then be grafted using any of the above techniques. Where ischaemia is more severe, and the limb in danger, immediate operation should be undertaken and an attempt made to establish distal patency using Fogarty catheters from the popliteal fossa and if necessary from the ankle. Providing the occlusion is recent it should be possible to clear the distal tree despite seemingly unfavourable arteriographic appearances. The aneurysm is then replaced as outlined above.

In a small number of cases the aneurysmal process extends into the popliteal bifurcation (Figure **5.31a**). If the anterior tibial artery is still patent, a double bypass may be required. Detachment of the anterior tibial with a cuff of aneurysm may allow graft placement within the same operative zone (Figure **5.31b**). However, this may be difficult and it is usually necessary to oversew the anterior tibial origin and then carry an extension limb from the main graft down to the anterior tibial artery at midcalf level.

Popliteal aneurysms may be found in conjunction with diffuse ectasia (arteriomegaly) of the superficial femoral artery (Figure **5.32a**). Mural thrombus may form in the ectatic segments and give rise to embolization or extensive occlusion. In order to prevent these complications the ectatic segment should be excluded along with the popliteal aneurysm. The limb may then be revascularized by means of an *in situ* vein bypass (Figure **5.32b**).

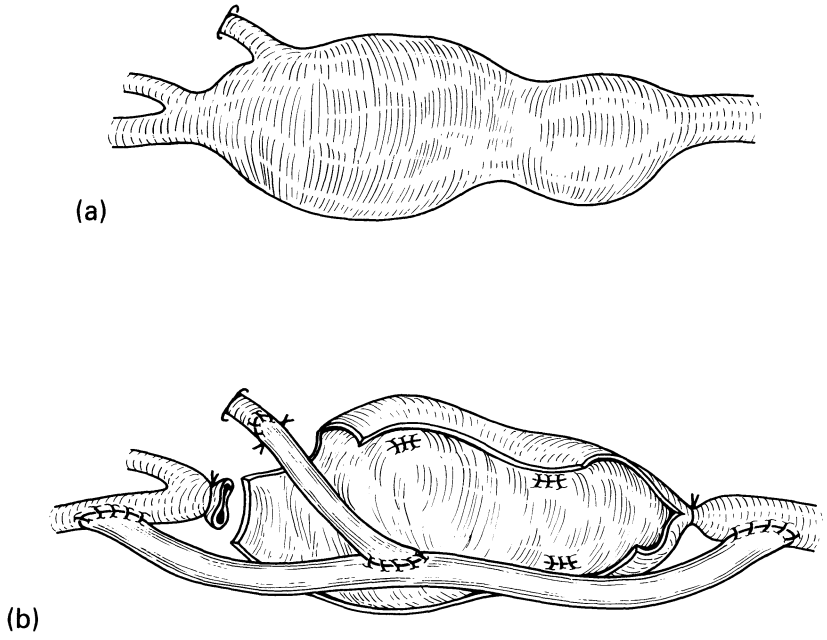


Figure 5.31(a, b) (a) Aneurysm extending into the tibioperoneal trunk. (b) Reconstruction using two segments of saphenous vein

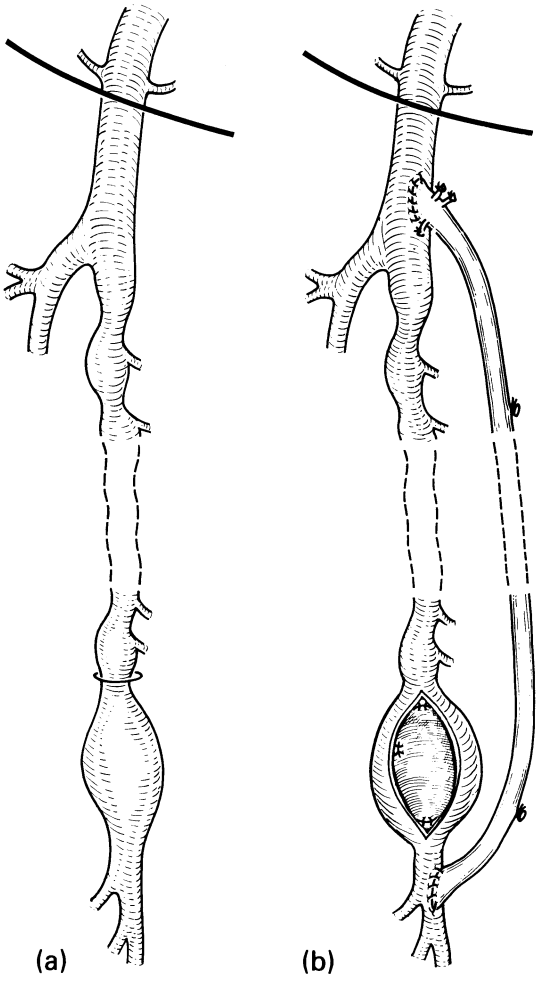


Figure 5.32(a, b) Popliteal aneurysm associated with diffuse ectasia of the superficial femoral artery: (a) pathology. (b) the aneurysm and proximal ectatic segment have been excluded and an *in situ* vein bypass inserted. Some flow may continue in the proximal branches of the superficial femoral artery

OTHER POPLITEAL CONDITIONS

POPLITEAL ENTRAPMENT

Rarely the popliteal artery may be compressed by soft tissue elements behind the knee[57]. The most common of these is an abnormally sited medial head of gastrocnemius (Figure **5.33b**). As a result of this entrapment the artery wall may become thickened and thrombosis may supervene. In other cases there may be post-stenotic dilatation or frank aneurysmal change.

This condition should be considered in any young subject who develops calf and foot claudication and who lacks evidence of an embologenic cardiac lesion or vascular disease elsewhere. If the patient is seen at the stage of intermittent compression, the popliteal and pedal pulses may be present at rest but abolished by passive dorsi-

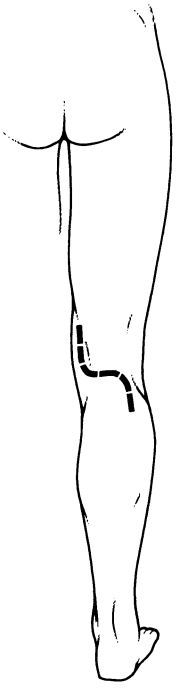


Figure 5.33(a, b) Popliteal entrapment: (a) posterior approach to the popliteal fossa

flexion or active plantar flexion. Later on, if thrombosis supervenes, the popliteal and usually the pedal pulses are absent.

Characteristic arteriographic and B scan findings include medial deviation of the proximal popliteal artery, midpopliteal stenosis with or without post-stenotic dilatation, or segmental occlusion. In 25% of cases the contralateral artery may show signs of similar compression and a bilateral operation is then advisable.

Access may be achieved via either a posterior or a medial approach. The former gives better exposure of the soft tissue anomaly (Figure **5.33b**) though the latter is preferable if arterial reconstruction seems likely. Once the compressive element has been identified it should be divided and the artery released. In most symptomatic patients intrinsic arterial damage will already be present and a reconstruction is then necessary. This may be best achieved by vein bypass graft with proximal and distal end-to-side attachment. If the condition is complicated by recent thrombosis or distal embolization, a catheter thrombectomy should be undertaken prior to reconstructing the popliteal artery.

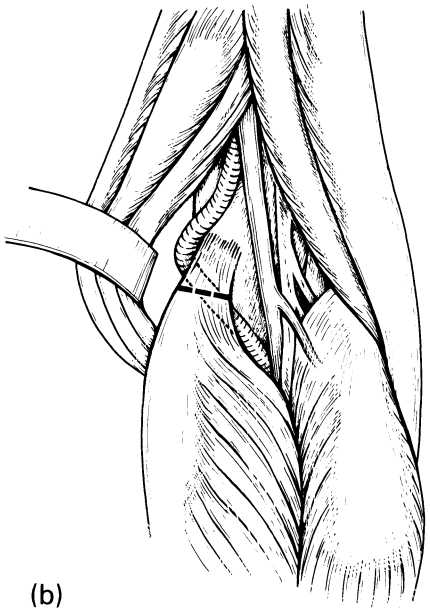


Figure 5.33 continued (b) entrapment of the artery beneath an abnormally sited medial head of gastrocnemius.

ADDUCTOR CANAL SYNDROME

A similar entrapment syndrome may occur at the adductor hiatus as a result of compression of the superficial femoral artery by a fibrofascial band[58]. The condition presents as claudication in young adults and, as in the case of popliteal entrapment, segmental arterial occlusion is often present by the time symptoms occur. Treatment consists of dividing the fascial band, usually with additional vein bypass grafting.

ADVENTITIAL CYSTIC DISEASE OF THE POPLITEAL ARTERY

Rarely the adventitia of the popliteal artery is the site of cystic degenerative change[59]. The exact aetiology of this condition is unknown, though one view is that the cysts develop from synovial rests sequestered in the artery wall during development[60]. These cysts may be unilocular or multilocular with contents similar to those found in ganglia.

The condition may present with a sudden onset of severe claudication in a young or middle-aged subject. This sudden onset of symptoms is thought to be due to a rapid change in the size of the cyst. If the intracystic pressure exceeds that in the artery lumen, there may be complete occlusion with or without thrombosis. The typical arteriographic or B scan finding is either a crescentic filling defect (scimitar sign) or an hour-glass stenosis. In later cases there may be a localized complete occlusion typically just proximal to the joint line.

If thrombosis has not yet occurred, the simplest form of treatment is to evacuate or enucleate the cyst via a partial-thickness incision of the artery wall. Where evacuation fails to restore arterial patency, or where there is established thrombotic occlusion, an arterial reconstruction will be necessary. This is most satisfactorily achieved by a vein bypass graft.

COMPLICATIONS OF FEMOROPOPLITEAL SURGERY

WOUND COMPLICATIONS

As with reconstructive procedures elsewhere, local wound complications may occasionally occur. These particularly concern the groin and may include haematoma, lymphatic leakage and skin necrosis. Further details are described in Chaper 14.

EARLY GRAFT OCCLUSION

Most early graft occlusions, i.e. those occurring within 30 days of operation, are due to technical or judgemental errors (Figures **5.34–39**). The former include anastomotic narrowing or intimal flaps, particularly at the distal end, unevacuated thrombus, intraoperative embolization and errors in graft length or trajectory. Judgemental errors include acceptance of a run-off bed which is inadequate to maintain graft flow, failure to identify an embologenic lesion in the aortoiliac segment and attempted graft implantation at a site where the vessel wall is too thick or degenerate.

Many intraoperative errors are obvious at the time of their committal. However, a significant number are only detected by intraoperative arteriography and this should be undertaken as a routine with this type of surgery. Any faults can then be corrected before the patient leaves the operating room.

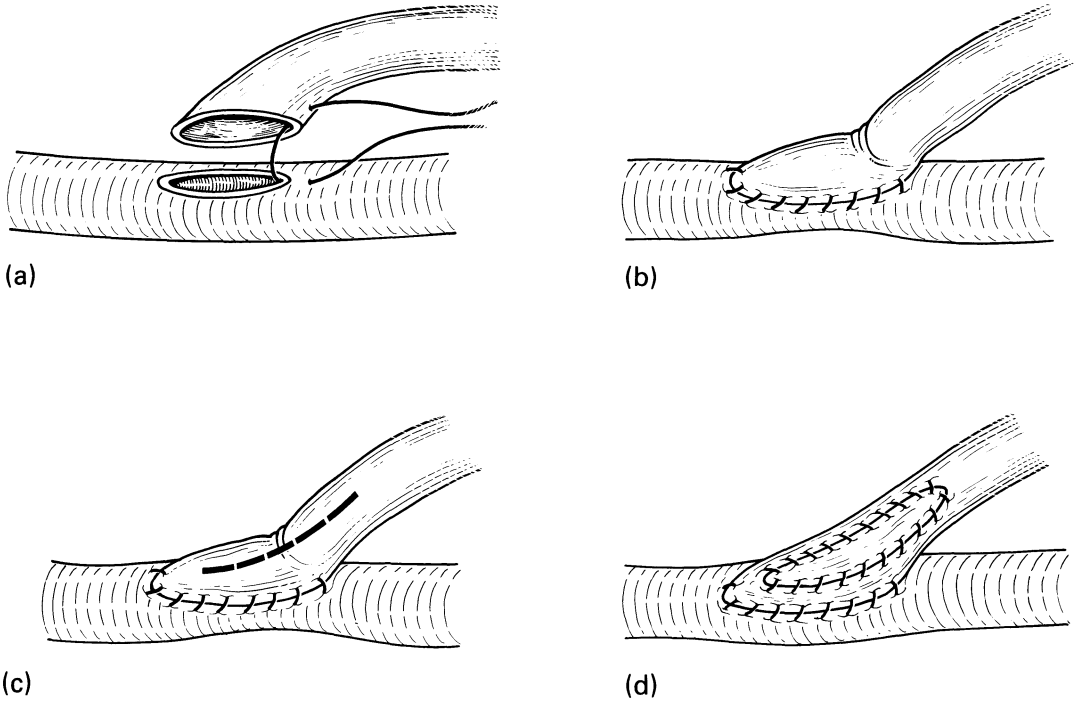


Figure 5.34(a–d) (a, b) Stenosis at the heel of a vein graft anastomosis due to over-deep suture placement. This may be corrected by inserting a patch in the dorsum of the graft (c, d)

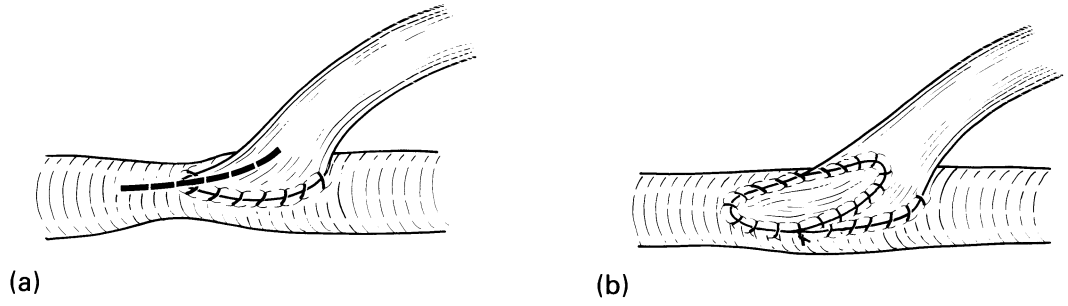


Figure 5.35(a, b) (a) Stenosis at the apex of a vein graft anastomosis. (b) Error corrected by patch insertion

Early postoperative graft occlusion usually places the limb in jeopardy and after heparinization the patient should be returned to the operating room. It is usually advisable to expose both ends of the bypass to control subsequent catheter manipulations. A longitudinal incision is then made in the hood of the distal anastomosis and clot removed from the artery proximally and distally using a small Fogarty catheter. Retrograde thrombectomy of the graft is then achieved. In the case of a reversed vein graft retrograde catheter passage may be impeded at the valve rings. However, by gentle manipulation, aided if necessary by transient balloon inflation to render the valves incompetent, the catheter can usually be slipped past.

If there has been a delay between vein graft occlusion and reoperation, graft replacement is preferable to thrombectomy because of likely thrombus adherence at the valve rings and secondary changes in the vein endothelium. Occluded PTFE grafts are much easier to thrombectomize and usually complete clearance can be obtained even when some time has elapsed between occlusion and reoperation[32].

After the graft has been cleared of clot, the distal anastomosis is inspected for narrowing or an intimal flap. The former may be managed by patching or relocation of the anastomosis at a more distal site (Figures **5.34–36**). An intimal flap is seen more frequently with prosthetic than venous grafts (Figure **5.37**). It can be managed by

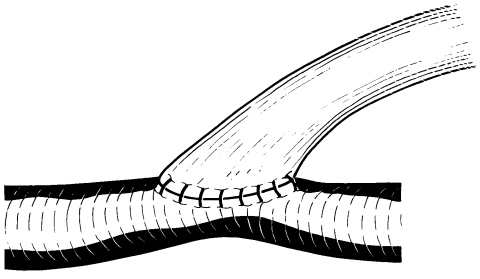


Figure 5.36 Anastomotic stenosis due to implanting the graft on a thick-walled artery. Correction requires relocation of the anastomosis or use of an intermediate vein patch

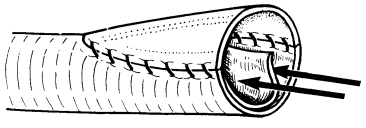


Figure 5.37 Intimal dissection at distal PTFE–artery anastomosis. This may be managed by opening the anastomosis, securing the intima with tack-down sutures and inserting a patch. Alternatively the graft may be extended to a more distal site

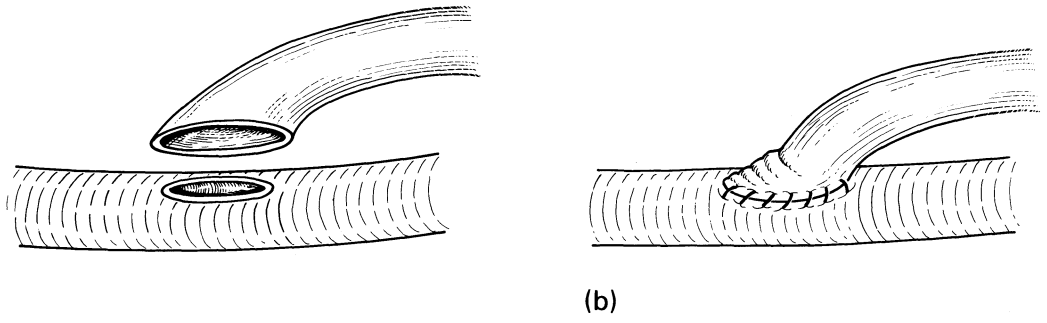


Figure 5.38(a, b) Wrinkling of vein graft anastomosis due to an overlong venous cutback. Correction requires redoing the anastomosis with trimming of the vein or arteriotomy extension

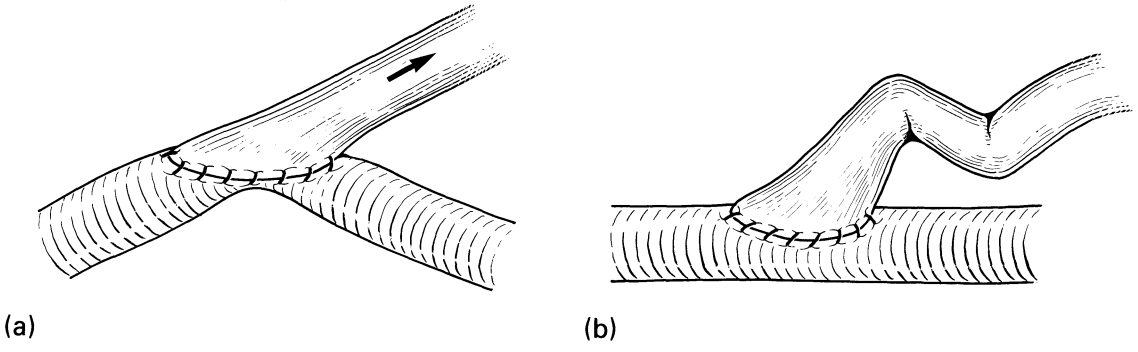


Figure 5.39(a, b) Errors in graft length: (a) the vein graft has been cut too short, resulting in angulation and stenosis at the distal anastomosis. A minor degree of shortening may be corrected by soft tissue release, e.g. division of the medial hamstrings or the medial head of gastrocnemius. If there is a marked deficiency, an additional graft segment should be interposed. (b) an overlong graft with consequent kinking. Some slack may be taken up by creating a more sinuous course for the graft. If still too long, a portion of the graft should be resected

extending the graft incision distally into the artery. The loose intima is then excised and the distal edge secured with tack-down stitches. The graft–artery incision is then closed with a patch. An alternative approach is to extend the bypass to a more distal level. Irrespective of the revisionary measures necessary, a completion arteriogram should be routinely obtained to confirm the adequacy of the reconstruction and of the distal runoff.

If a local anastomotic problem is not apparent, the graft incision should be closed with 6/0 or 7/0 prolene and an arteriogram obtained. This may show previously unrecognised distal disease. Management then requires a venous or less preferably a PTFE extension from the thrombectomized graft to a distal patent vessel (see Figure **5.40**).

In a number of patients the cause of the graft failure may not be apparent and the operative procedure is limited to thrombectomy. Apart from initial unevacuated thrombus in the graft, embolization from the aortoiliac segment is a possible explanation that must be kept in mind in these cases. Re-examination of the original arteriogram may help plan further action.

DEGENERATIVE CHANGES IN VEIN GRAFTS

Femoropopliteal vein bypass grafts are liable to degenerative changes which contribute significantly to overall graft failure[21,22]. Intimal hyperplasia is a particular complication of reversed vein grafts and may be due at least in part to faulty harvesting techniques such as the use of saline for graft distension, excess dilatation and hypothermia[43,44,61]. Turbulence and stasis at valve sites may also predispose to intimal thickening[62] and this has been advanced as an argument for utilizing avalvular or valve-ablated segments of vein as a routine in cardiovascular surgery[63]. Other causes of vein graft stenosis include clamp trauma and branch ligation too close to the wall of the graft.

Graft stenosis may be marked by reappearance of claudication with diminution in distal pulses and in Doppler ankle pressure. Similar features may also be produced by anastomotic stenoses or atherosclerotic progression proximal or distal to the graft, and arteriography is required to determine the precise underlying cause. This should be obtained with some urgency since delay may invite progression from stenosis to graft occlusion which is much more difficult to correct. Not all stenotic changes are accompanied by recurrence of clinical symptoms and a policy of routine postoperative surveillance using vascular laboratory testing or digital subtraction angiography is very helpful in detecting graft failure at an early and correctable stage.

Some vein graft stenoses may be successfully treated by percutaneous angioplasty (see Chapter 15). A similar approach may also be used for dealing with anastomotic intimal hyperplasia or with limited atherosclerotic progression proximal or distal to the graft. Where angioplasty is not feasible a number of surgical options are available. Inflow disease may require aorto-, ilio- or femoro-femoral bypass with distal end-to-side attachment to the femoro-popliteal graft. Stenosis at the proximal anastomosis may be treated by patching or preferably by a bridging graft between the external iliac artery and the proximal end of the femoropopliteal bypass. Short stenoses within the graft may be treated by patching, lengthier lesions by segmental replacement or by end-to-side bypass extension. Distal anastomotic narrowing or atherosclerotic progression in the run-off bed may also be managed by extending a new section of vein from the original graft to distal uninvolved territory (Figure **5.40**).

Aneurysmal dilatation of vein grafts may also be encountered, though less frequently than stenotic disease. Reversed vein grafts are again more prone to this complication than *in situ* bypass. Local dilatation may occur adjacent to one or other anastomosis or there may be multiple dilated zones throughout the graft, in some cases as a result of secondary atherosclerotic degeneration.

Patients with marked local or diffuse dilatation are at risk from embolization of the run-off bed or acute graft thrombosis, and for these reasons reoperation is advisable. A local juxta-anastomotic dilatation may be corrected by disconnecting the graft and replacing the dilated segment with a venous or PTFE extension to a new anastomotic site. Diffuse dilatation requires a completely new graft.

LATE GRAFT OCCLUSION

Late femoropopliteal graft occlusion is commonly due to distal (or proximal) atherosclerotic progression, intimal hyperplasia at or just beyond the distal anastomosis, or to intrinsic degenerative changes within the graft itself (see above). Other factors may include an uncorrected anastomotic fault, clamp injury to the artery adjacent to either anastomosis, false aneurysm formation or sepsis. A sudden reduction in cardiac output may also on its own produce graft occlusion, though usually there is an additional morphological abnormality in the graft or run-off bed.

If the limb is not threatened, further treatment may not be justified. The remaining cases should be re-evaluated by arteriography. This should include films of the aortoiliac segment and if possible of the run-off into the foot. The first line of treatment in recent graft thrombosis may be a low-dose streptokinase infusion via a contralateral catheter[64]. Once lysis has been achieved any underlying stenosis may be dilated by balloon catheter or surgically corrected (see Chapter 15). The best results are obtained in recent occlusions

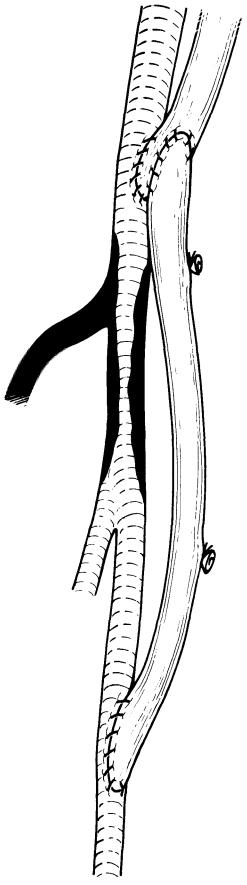


Figure 5.40 Graft failure due to atherosclerosis in the run-off bed. An end-to-side bypass has been extended to a distal patent vessel, in this case the posterior tibial artery.

though occasionally patency may be restored in quite late cases.

Lysis by low-dose streptokinase may take several hours and where limb ischaemia is critical an immediate surgical approach is necessary. Surgery may also be required if streptokinase therapy is unsuccessful or has to be discontinued because of complications (see Chapters 6 and 14). Although successful thrombectomy and graft revision has been reported in patients with late occluded vein bypass grafts[22], most patients are better treated by complete graft replacement[65,66]. The new bypass is usually inserted proximal and distal to the occluded zone, though occasionally a cuff of the old graft can be preserved at one or other end[67]. If possible, autogenous vein should be used for these secondary reconstructions[66]. Often both saphenous veins will have been removed in these patients and a PTFE or composite graft may then be inserted.

In the case of an *in situ* vein bypass, part of the graft may remain patent due to persistent arteriovenous communications. If the distal graft segment is patent, a PTFE graft may be placed in the thigh to re-establish continuity. Where the proximal segment of the *in situ* bypass has remained patent, a new venous section may be used to extend the bypass to a patent infrapopliteal vessel. Alternatively the residual proximal segment may be harvested and used as the distal end of a PTFE–vein composite.

In contrast to autogenous vein grafts, a thrombosed PTFE graft can usually be reopened by catheter thrombectomy from a distal graftotomy[32]. This should then be supplemented by appropriate graft revision. If the cause of the occlusion is intimal hyperplasia at the distal anastomosis (Figure **5.41a**) one approach is to extend the graft incision distally into the host vessel followed by patch graft angioplasty (Figure **5.41b–d**). Alternatively a distal extension limb may be used to bypass the stenotic zone (see Figure **5.40**). If the graft occlusion does not appear to be due to an anastomotic stenosis the exploratory graft opening should be closed and an intraoperative arteriogram obtained. If run-off disease is then demonstrated, an extension limb is added from the thrombectomized graft to a distal patent vessel as previously described.

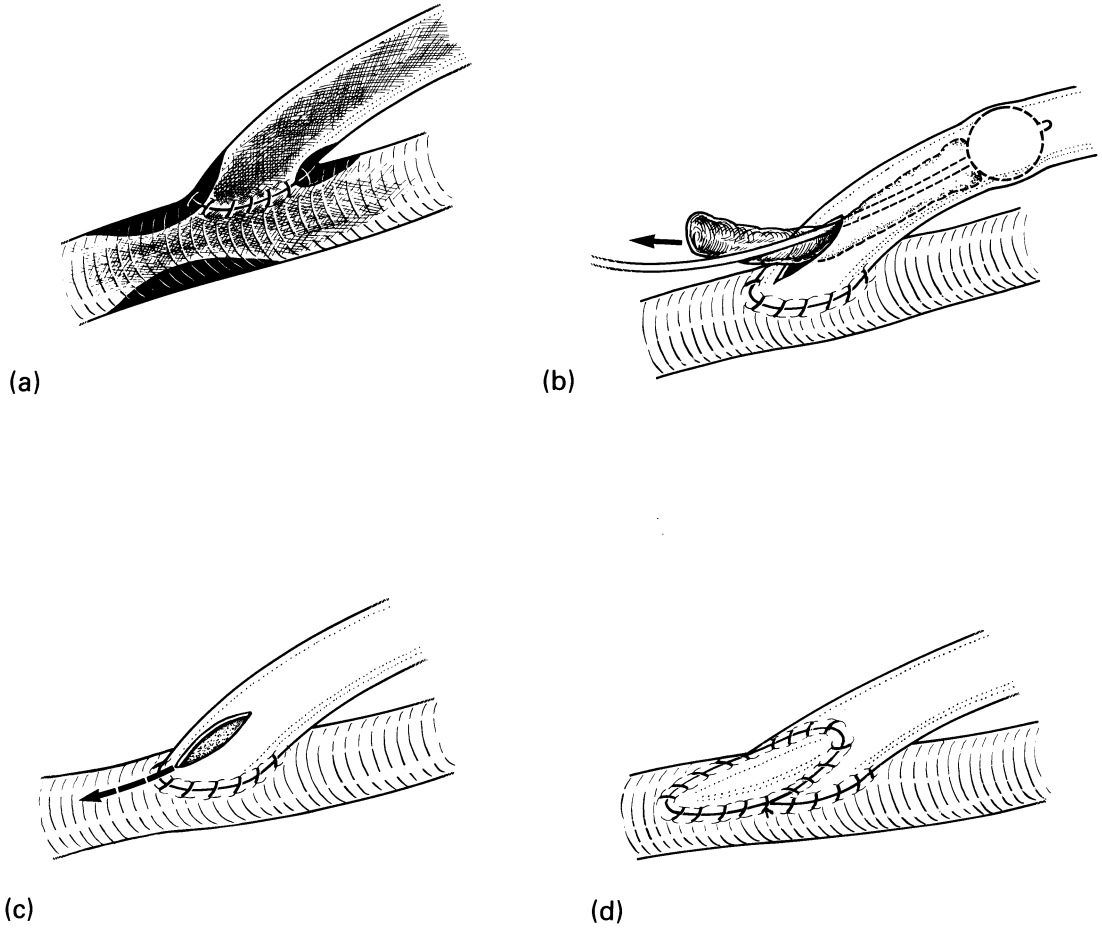


Figure 5.41(a–d) PTFE graft occlusion due to anastomotic intimal hyperplasia: (a) pathology. (b) thrombectomy via a distal graft incision. (c) the graft incision is extended into the distal artery to lay open the stenosis. (d) a PTFE patch is then inserted

FALSE ANEURYSM

This is usually, but not exclusively, a complication of prosthetic grafts (see Chapter 14). Surgical correction is advisable because of the risks of distal embolization, graft occlusion or rupture. Periarterial fibrosis may make dissection difficult and the easiest approach is to clamp the external iliac artery and the graft at a distance from the aneurysm. The latter is then opened and back-bleeding from the profunda controlled with a balloon catheter. Reconstruction may then be achieved by an ilioprofunda bypass with a side limb to the detached femoropopliteal graft. If there is an additional false aneurysm or stenosis at the popliteal anastomosis, the entire femoropopliteal graft should be replaced (Figure **5.42a, b**).

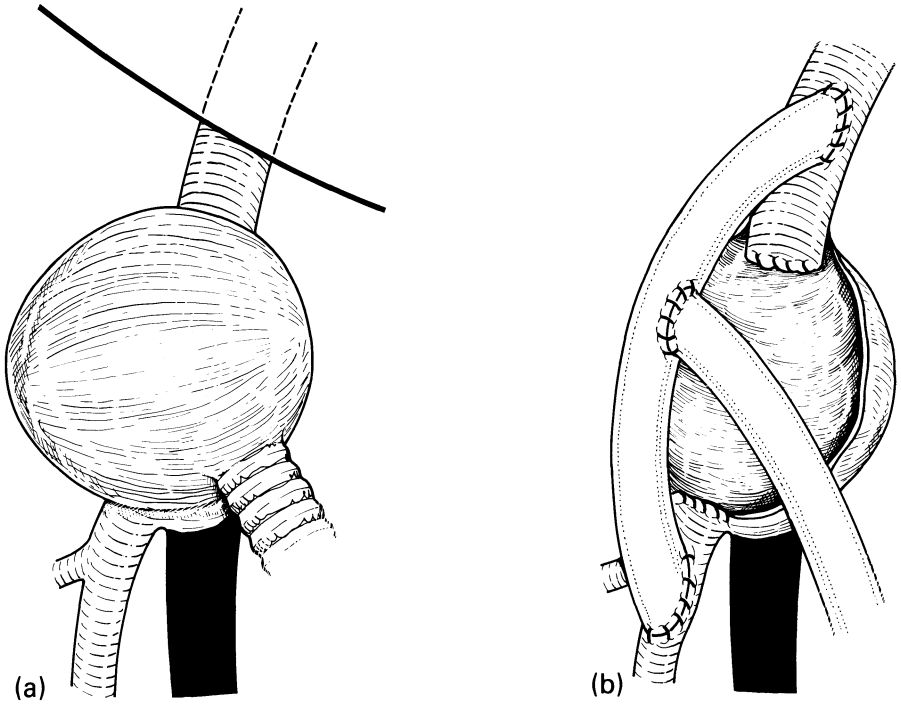


Figure 5.42(a, b) (a) False aneurysm at the proximal anastomosis of a Dacron femoropopliteal bypass graft. (b) An ilioprofunda PTFE graft has been inserted with a new femoro–distal extension. The latter may be either PTFE or a composite PTFE–vein graft if there is insufficient saphenous vein

INFECTION

This is also more frequently seen with prosthetic than venous bypass grafts. Treatment is difficult with a relatively high amputation rate[68].

If the suture line is not involved, local irrigation with povidone–iodine and systemic antibiotics may allow healing (see Chapter 14). Once the suture line or graft lining is involved, as evidenced by bleeding, false aneurysm, graft occlusion or septic embolization, the graft should be removed and the arterial axis interrupted at either end. Revascularization may then be achieved by an *in situ* autogenous ilioprofunda bypass or, more safely, by an extra-anatomic bypass to the mid-profunda passing laterally around the infected

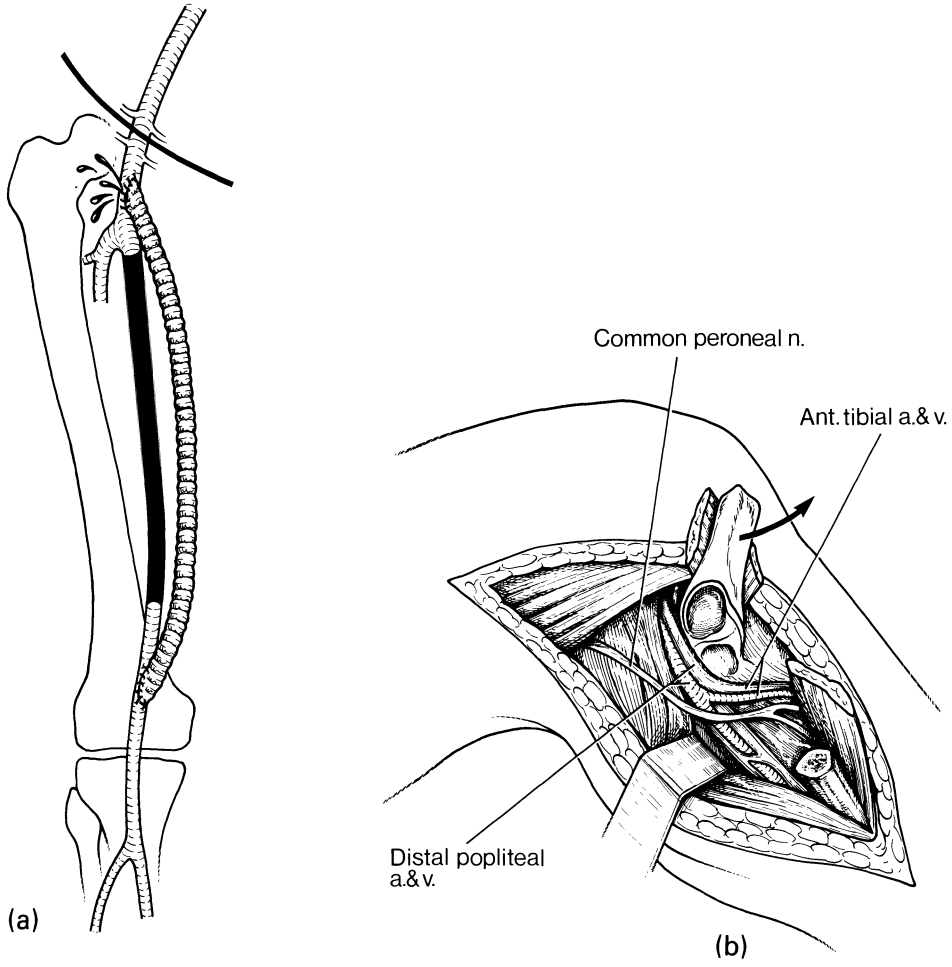


Figure 5.43(a–c) Management of an infected femoropopliteal prosthesis: (a) haemorrhage due to infection in the groin. (b) lateral approach to the distal popliteal artery and tibioperoneal trunk. The fibula has been divided and the proximal segment hinged superiorly prior to removal

Figure 5.43 continued (c) the infected Dacron prosthesis has been removed and the femoral vessels ligated. A new PTFE bypass has been inserted using a lateral extra-anatomic route

groin (see Figure **14.22**). However, if the profunda is unsuitable or if the popliteal re-entry is limited, a bypass to infrapopliteal level will be required. The new bypass may be attached to the external iliac artery via a separate clean field and then routed laterally down the leg to join the tibioperoneal trunk or one of its branches. Access for distal graft attachment may be conveniently achieved by resecting the proximal fibula (Figure **5.43a–c**).

Where infection complicates a femoropopliteal vein graft, the same principles of exclusion of the septic zone followed by revascularization at a distance apply. However, unlike infection in prostheses, the non-infected part of the vein graft can often be preserved, simplifying revascularization.

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6

Arterial Embolization

The great majority of major peripheral emboli are cardiac in origin. Atherosclerosis has replaced rheumatic valvular disease as the commonest cardiac pathology, myocardial infarction or atrial fibrillation being the underlying event in most cases[1,2]. Other possible cardiac sources include prosthetic valves, endocarditis or atrial myxoma.

In approximately 10% of patients major emboli arise from an extracardiac source such as an aortic or peripheral aneurysm or an ulcerative plaque[3,4]. In some of these cases much smaller emboli consisting of atheromatous debris or thrombus are released into the distal circulation, producing digital ischaemia with retained distal pulses (blue toe syndrome)[5]. A previous aortic bypass graft may also occasionally act as the source of an embolus if it contains loose mural thrombus or if a false aneurysm or sepsis are present (see Chapter 14).

In a significant number of cases, perhaps as many as 20%[6,7], an exact source for the embolus is never found.

Major emboli lodge at bifurcations or the origin of large branches, at sites of anatomical narrowing such as the adductor or scalene canal, and at sites of pathological narrowing such as an atherosclerotic superficial femoral artery. Approximately 80% of peripheral emboli affect the lower limb with the common femoral bifurcation accounting for almost half of all cases[8,9]. A single embolus is most common but sometimes multiple emboli occur either in the same extremity or at different sites including the cerebral or visceral arteries.

The usual clinical features of peripheral embolization are pain, pallor or cyanosis, a cool extremity, loss of pulses, altered sensation and paresis. In a minority of cases emboli produce subacute or chronic ischaemia[10], while occasionally they may be entirely silent[11].

THE MANAGEMENT OF MAJOR LIMB EMBOLIZATION

The general principles of treatment are similar in all arteries and may be described by taking as example an embolus at the common femoral bifurcation.

Common femoral embolization presents with acute ischaemic signs from the distal thigh or knee downwards. A pulse may be palpable at or just above the inguinal ligament, but distal pulses are absent. As soon as the diagnosis is made, systemic heparin should be given (1 mg/kg body weight) to minimize secondary propagation of thrombus in the distal arterial tree. Many of these patients have congestive failure or an arrythmia which may merit intensive medical therapy. As far as possible such treatment should proceed

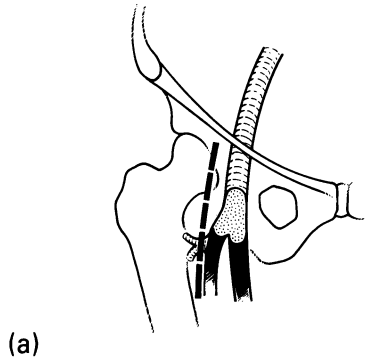
concomitantly with arrangements for surgery so that the latter can be undertaken with minimum delay. As with most other arterial procedures, antibiotic cover should be commenced prior to surgery.

Although embolectomy can often be accomplished under local anaesthesia with particular advantage in the critically ill patient, recent advances in general anaesthesia have led to use of the latter on an increasing scale. Certainly general anaesthesia is more comfortable for the patient and surgeon and is particularly useful when the patient is obese or unable to lie flat, making access to the groin difficult.

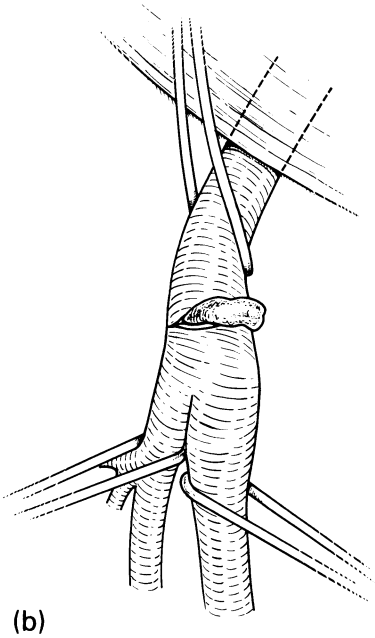
Preoperative arteriography is not absolutely essential with a typical common femoral embolus. However, where there is a history of peripheral vascular disease or the diagnosis is in doubt, arteriography should always be obtained.

The entire extremity should be prepped and draped to allow for possible popliteal or ankle exploration or for femoro–distal bypass insertion. The common femoral bifurcation is then exposed via the usual longitudinal incision just lateral to the vascular axis (Figure **6.1a**). Typically, the common femoral artery is found to be nonpulsatile, distended, and with a bluish discoloration.

If necessary, a further systemic dose of heparin may now be given and Silastic loops then placed around the common, superficial and deep femoral vessels. Clamps should be avoided because of the risk of fragmenting intraluminal clot. An arteriotomy is then made in the distal common femoral artery. If the vessel is particularly athero-



(a)



(b)

Figure 6.1(a–e) Common femoral embolectomy: (a) embolus with secondary thrombus. (b) transverse arteriotomy with embolus extruding

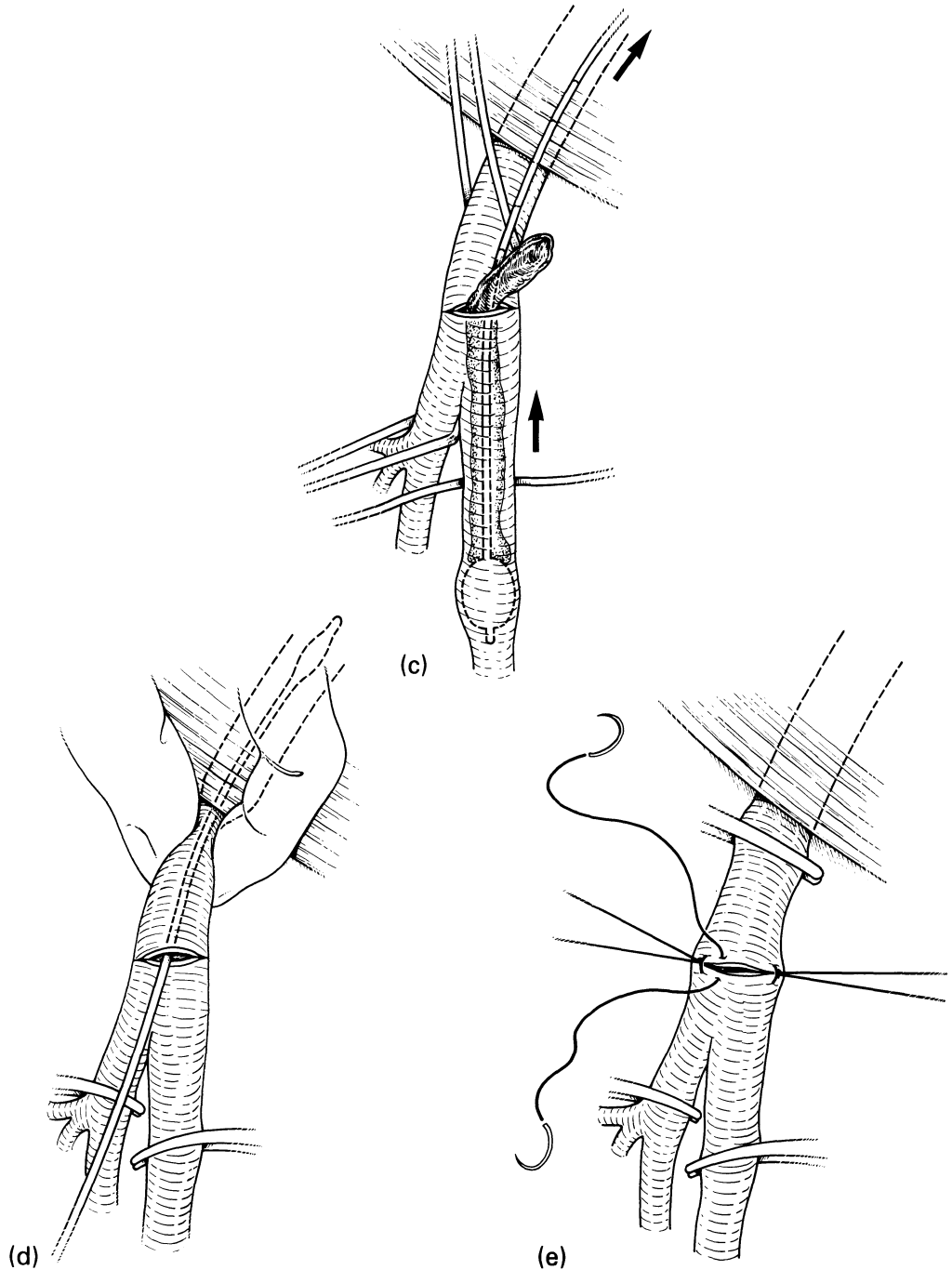


Figure 6.1 continued (c) Fogarty catheter retrieving thrombus from the superficial femoral artery. (d) retrograde catheterization of the iliac axis. (e) arteriotomy closure using double-armed sutures

sclerotic or small, a longitudinal incision is used to minimize the risk of wall damage during instrumentation. Once embolectomy has been achieved the arteriotomy may be closed with a small patch. A longitudinal arteriotomy may also be indicated if there is significant profunda ostial stenosis. In these cases the incision is extended into the proximal profunda to allow a common-deep femoral endarterectomy. With these exceptions a transverse arteriotomy is routinely employed as this will provide adequate access and can be closed directly without risk of narrowing the lumen. However it is important to ensure that the incision extends over the anterior half of the vessel circumference. If a more limited opening is made there is a risk of splitting at the angles on balloon retrieval or of intimal separation on reinserting the catheter.

As soon as the arteriotomy is made the embolus may extrude spontaneously or may be eased out by digital pressure with an ensuing gush of blood (Figure **6.1b**). The embolus should be inspected for fragmentation and sent for culture and histological examination.

The next stage is to obtain distal clearance. A Fogarty catheter, usually 4F size, is passed down the superficial femoral artery as far as the foot. The balloon is inflated with one hand until the elastic resistance of the vessel wall can be felt, while the other hand gently withdraws the catheter. It is essential for the surgeon himself to carry out both manoeuvres since only by the continuous sensation of contact with the artery wall can complete clearance be achieved on the one hand, and intimal injury avoided on the other. As the balloon travels up the leg, the degree of inflation should be progressively increased to maintain wall contact. Finally, the balloon is withdrawn through the arteriotomy, bringing with it additional embolic material and consecutive clot (Figure **6.1c**). If the latter has a tapering tail and removal is followed by a vigorous back-bleed, this is encouraging. If the clot is fragmented or back-bleeding poor, the catheter should be re-passed and the procedure repeated.

If the back-bleeding remains poor, or the catheter cannot be passed distally, a soft catheter or a cannula should be inserted into the superficial femoral artery and an arteriogram obtained. Catheter hold-up is more likely to be due to co-existent atherosclerosis than fresh clot and a femoro–distal bypass may be subsequently required. In other cases arteriography may show retained clot in the popliteal–tibial arteries. For anatomical reasons, a catheter passed from the groin is very likely to enter the same infrapopliteal vessel each time, particularly the peroneal artery[12]. One method of cannulating additional infrapopliteal vessels is to pass a 3F embolectomy catheter as far distally as possible from the groin, followed by a second similar catheter inserted with the knee slightly flexed. Deflection by the first catheter may then allow entry into a previously uncannulated infrapopliteal vessel. If available, fluoroscopy will facilitate this type of manipulation[13].

If distal clearance seems complete, an irrigating catheter is passed to the foot and heparin–saline infused while the catheter is withdrawn. The superficial femoral artery is then clamped at its origin and attention is turned to clearing the profunda femoris. Embolic material may be recovered from this vessel with surprising frequency. Once the profunda is clear, heparin–saline is instilled and the vessel similarly clamped.

Even if the inflow appears unimpeded following the initial extraction of the embolus, it is advisable to routinely pass a Fogarty catheter into the aorta using digital control of the common femoral artery (Figure **6.1d**). The balloon is inflated and the catheter progressively withdrawn taking care to decrease balloon inflation at the orifices of the common and external iliac arteries. The down-bleed is then re-tested and the iliac axis heparinized and clamped.

Arteriotomy closure is then commenced by placing a double-armed suture from within–out at each angle. Traction on these stitches will then appose the two edges while the remainder of the arteriotomy is closed with interrupted sutures (Figure **6.1e**). The intima often has a tendency to retract beneath the cut edge and double-armed sutures passed from within–out on each side will ensure inclusion of all layers in each bite. Before completing the suture line, the clamps should again be flushed and the lumen well rinsed with heparin–saline.

A completion arteriogram should then be obtained. Adequacy of distal clot retrieval is traditionally based on the presence of good back-

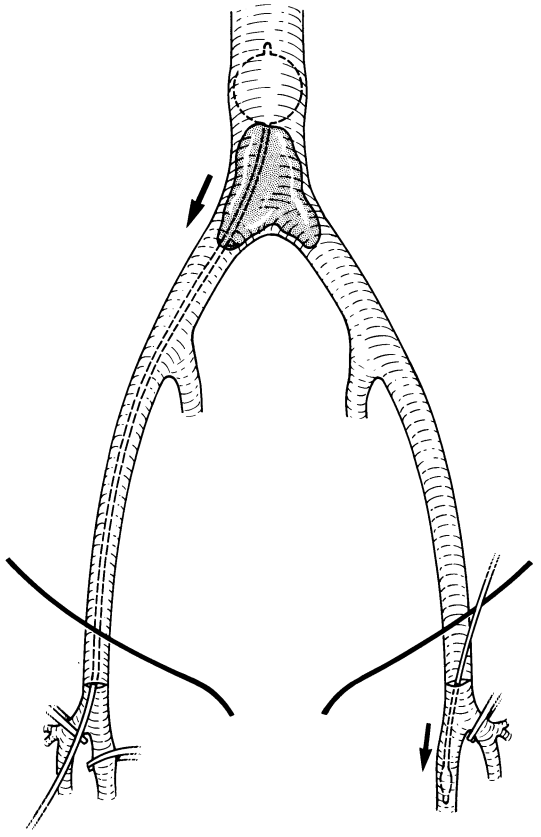
bleeding, the ability to completely pass the Fogarty catheter and failure to retrieve further thrombus on recatheterization. However, such criteria are not completely reliable and the only satisfactory method of assessing distal patency is by arteriography. In addition arteriography may provide early warning of possible catheter complications (see below). If a retained fragment of clot is demonstrated, for example in the anterior tibial territory, this may be removed via a popliteal arteriotomy or by retrograde embolectomy from the ankle[14,15]. Once distal patency is assured the groin is closed in layers with suction drainage.

Postoperative anticoagulation is advisable in view of the risk of recurrent embolization[16,17]. However, it should be recognized that even with full anticoagulant cover further embolic episodes may still occur[2]. A continuous intravenous heparin infusion is most commonly utilized and the dose rate adjusted to maintain the activated clotting time at approximately one and a half times the control value. Full anticoagulation with oral agents may then be substituted after 3 or 4 days.

Postoperative cardiological supervision is essential, since the cardiac status governs the ultimate prognosis. Although results in terms of limb salvage have improved and should now approach 90%, operative mortality for embolectomy remains of the order of 15–30%[1,6,17]. Predictably, most of these deaths are due to cardiac causes, with the highest mortality occurring when embolization complicates myocardial infarction[18]. In all cases, a careful search should be made for the source of the embolus by means of echocardiography and other cardiological studies, or by aortography if an extracardiac source is suspected. If a remediable source is identified appropriate measures may then be instituted, e.g. cardiac valve replacement or resection of an aortic or peripheral aneurysm. In the latter instances, complete removal of the embolic source will obviate the need for long-term anticoagulation.

Aortic bifurcation embolectomy

Aortic saddle embolus usually occurs in patients with severe cardiac disease and historically this is the most lethal form of peripheral embolization[3,19]. The condition is marked by the sudden onset of bilateral lower extremity ischaemia. In contrast to more peripheral emboli, symptoms may take several hours to develop. This more attenuated clinical course may reflect incomplete obstruction of the aortic bifurcation. Typically



(a)

Figure 6.2(a–c) Saddle embolectomy: (a, b) proximal and distal clearance using catheters from each groin

numbness and pain are marked with ischaemic signs from the midthigh or groin region distally. Femoral pulses are absent or diminished.

Retrograde transfemoral embolectomy offers particular advantages in this group of patients, many of whom would not withstand a direct approach to the aorta. A relatively minor disadvantage of retrograde balloon embolectomy is that embolic material or thrombus in the internal iliac arteries cannot be removed.

Prepping and draping should include provision for possible axillofemoral bypass in case embolectomy proves unsuccessful. Both groins are opened and the common, superficial and deep femoral arteries are controlled with Silastic loops. A common femoral arteriotomy is made and the distal arterial tree is cleared of thrombus with either a 3F or 4F Fogarty catheter. In most cases there is little embolic material in the femoral vessels. Heparin–saline is then infused distally and the vessels clamped (Figure **6.2a, b**).

A 5F or 6F catheter is passed retrogradely into the aorta from either side and the balloon inflated. The latter is then withdrawn with appropriate slight deflation at the origins of the common and external iliac arteries. This manoeuvre is repeated

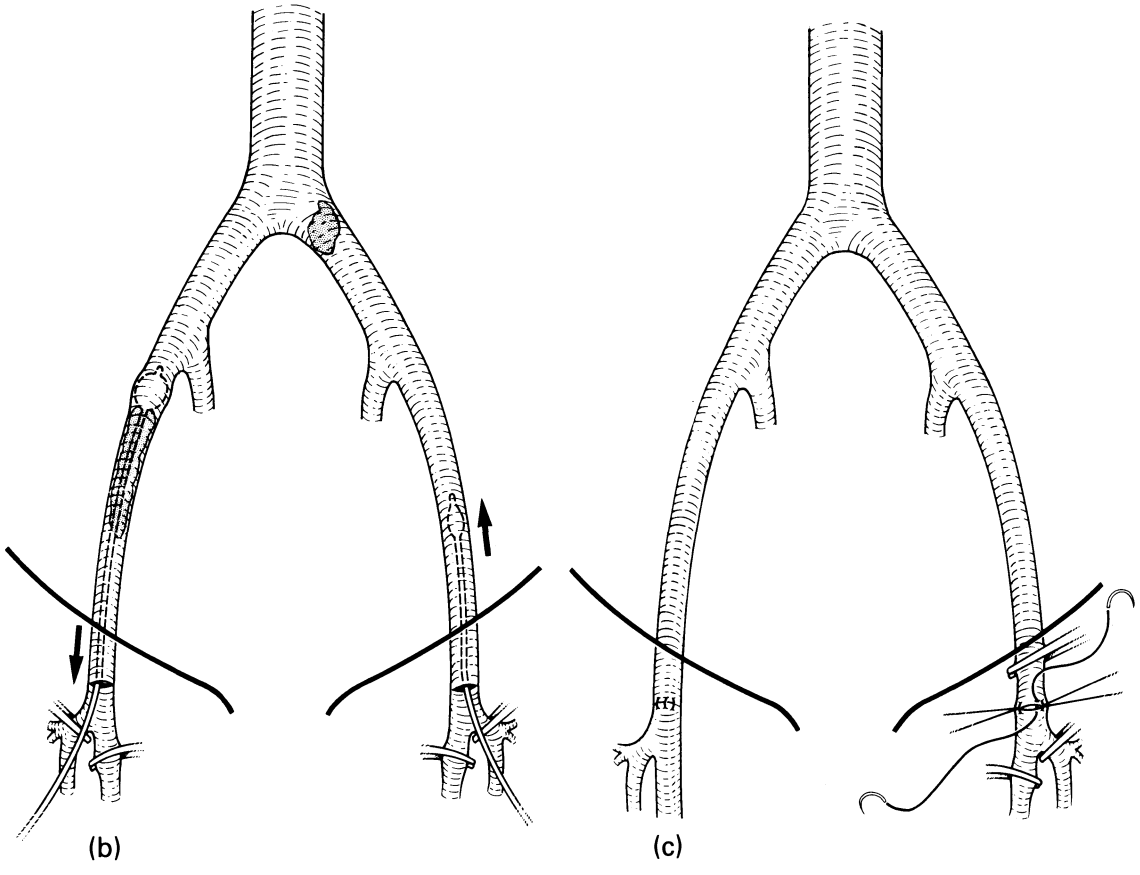


Figure 6.2 continued (c) after patency has been restored the arteriotomies are closed

until the aortic bifurcation is completely free and a vigorous downbleed is achieved on both sides. The iliac axes are then heparinized and clamped and the femoral arteriotomies closed as previously described (Figure **6.2c**).

Two catheters working in concert from each side should achieve clearance in most cases. However if a good down-bleed is not restored, retrograde arteriography should be undertaken to visualize the aortic bifurcation. An axillofemoral bypass or, in the better risk case, an approach to the aorta may then be considered.

Iliac embolectomy

An embolus lodging at the common iliac bifurcation may produce a unilateral pulse deficit and signs of ischaemia from midthigh or groin distally. Bilateral iliac embolization is indistinguishable from the clinical picture of an aortic saddle embolus.

The operative field should include both groins because of the risk of contralateral thrombus migration during retrograde catheterization. In addition it will cover the possible need for a femorofemoral bypass should embolectomy prove unsuccessful.

A common femoral arteriotomy is made on the ischaemic side and the distal tree is cleared of thrombus. Proximal embolectomy is then performed, using a 5F balloon catheter. During catheter insertion and withdrawal the opposite femoral artery should be temporarily compressed to avoid a new embolic episode. Once a brisk down-bleed has been restored, the femoral arteriotomy is closed. If proximal clearance is incomplete or catheter passage difficult due to iliac tortuosity, a femorofemoral crossover graft offers a simple and quick solution (see Chapter 3).

Popliteal embolectomy

Popliteal emboli usually produce pain or numbness in the foot with pallor, cyanosis and coldness from the midcalf downwards. A proximal popliteal pulse may be palpable, but pedal pulses are absent. Motor function may be deceptively good initially.

Although there has been a tendency to treat some of these cases conservatively in the past, this cannot be recommended. Popliteal embolectomy is highly effective and should be considered in all patients with severe ischaemia. In the better risk case this may be achieved by a direct popliteal approach under general anaesthesia. Alternatively, in poor risk patients, transfemoral embolectomy, supplemented if necessary by retrograde clearance from the ankle may be equally successful and can be performed under local anaesthesia.

Where the limb is not in immediate jeopardy, intra-arterial streptokinase via a femoral catheter may be utilized. This will lyse non-atheromatous emboli and may be particularly appropriate in patients with operative risk factors[20,21]. However, it should be emphasized that streptokinase therapy may occasionally lead to haemorrhagic complications or to further embolization as a result of loosening of thrombus from the primary embolic site (left ventricle, aortic aneurysm etc.)[22,23]. Caution is therefore required when adopting this approach and blood parameters should be carefully monitored to avoid a systemic lytic effect (further details see Chapter 15).

Clot lysis may take an unpredictable length of time and patients with limb-threatening ischaemia should be managed by surgical means. Preoperative arteriography is helpful in these cases in order to confirm proximal patency and to determine the exact site and nature of the occlusion. This may be performed by hand injection in the operating room to avoid delay.

A direct operative approach through the distal popliteal fossa is preferred where possible, as this will allow catheterization of all three infrapopliteal vessels[24]. This facility is particularly important in the late case where adherent clot may be difficult to clear via a transfemoral or retrograde approach. Although popliteal exploration is possible under a local anaesthetic, this is not ideal and general anaesthesia is preferred.

A standard medial approach is made to the

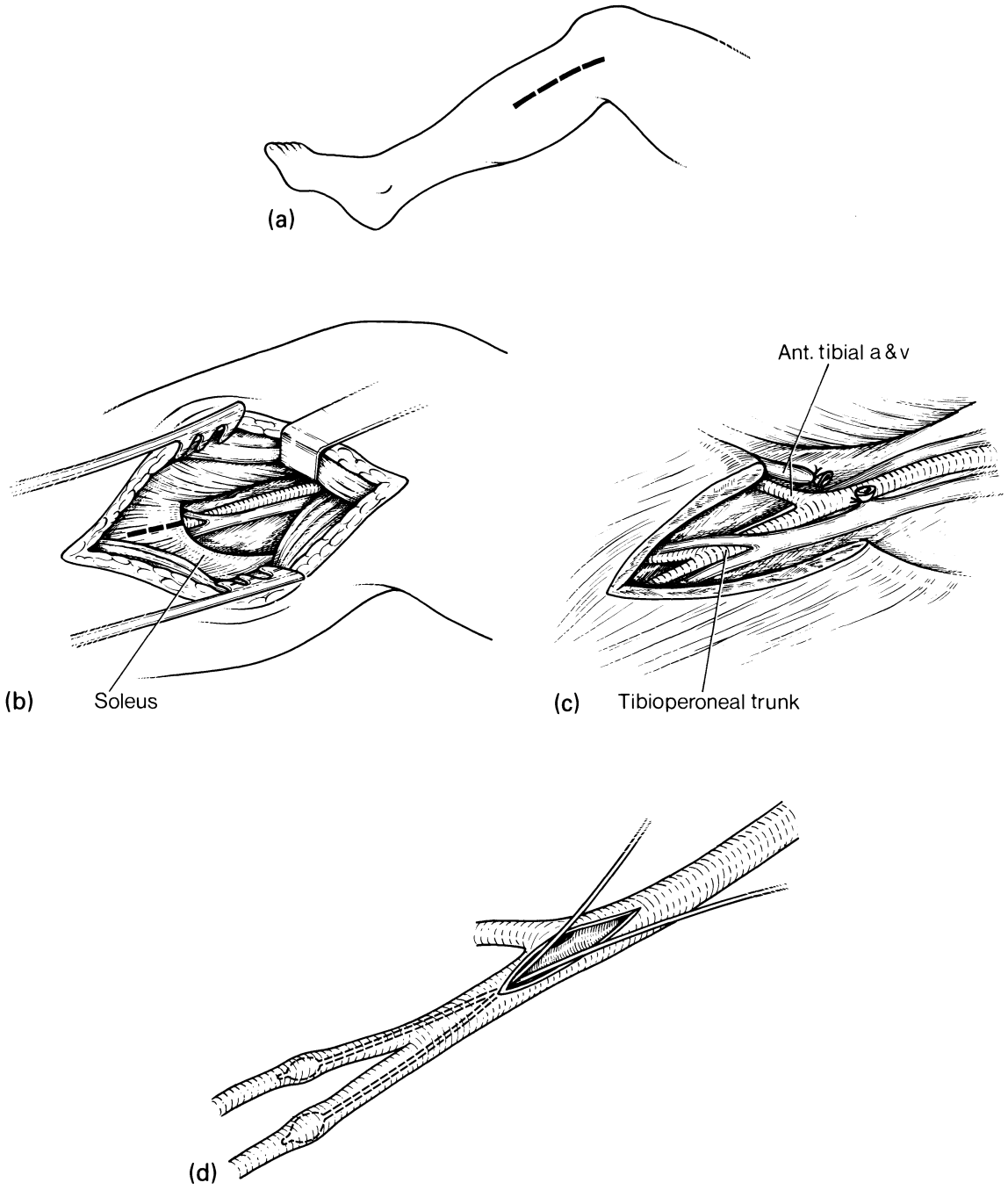


Figure 6.3a–f Popliteal embolectomy: (a–c) exposure of the distal popliteal artery and tibioperoneal trunk. (d) distal clearance of all three popliteal branches via a longitudinal arteriotomy opposite the anterior tibial origin

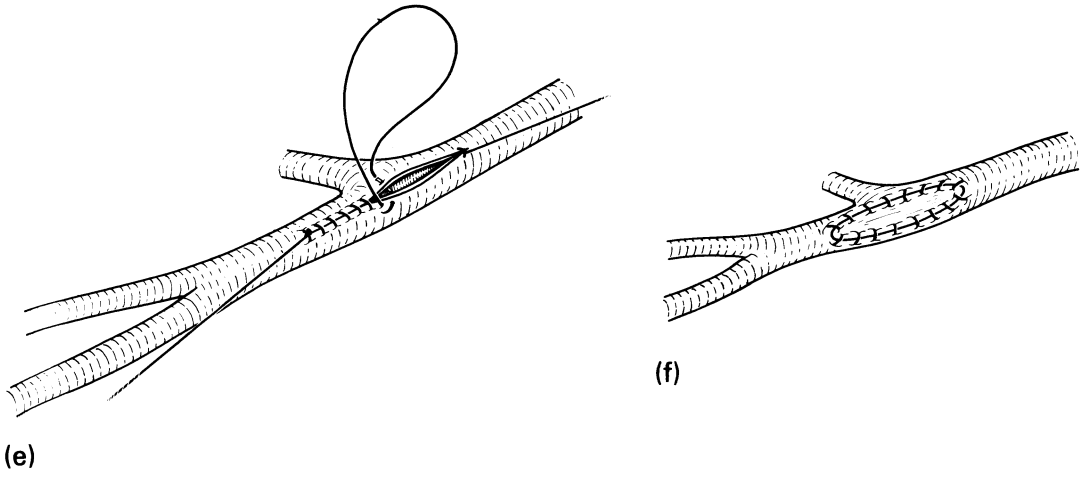


Figure 6.3 continued (e) direct closure of the arteriotomy. (f) alternative closure with a vein patch

distal popliteal artery (Figure **6.3a**). The medial head of gastrocnemius is retracted posteriorly and the soleal arcade divided to expose the anterior tibial artery and the tibioperoneal trunk (Figure **6.3b, c**). All the vessels are controlled with Silastic loops and a longitudinal arteriotomy is made in the distal popliteal artery opposite the anterior tibial origin. This opening is then extended into the tibioperoneal trunk (Figure **6.3d**). A distally sited transverse arteriotomy is an alternative when the popliteal artery is of large calibre but access is then more limited.

A 3F or 4F balloon catheter is first passed proximally to confirm that the superficial femoral segment is patent. Distal clearance is then obtained by passing a 2F or 3F catheter into each of the infrapopliteal vessels in turn. Simultaneous introduction of two catheters may ensure catheterization of the posterior tibial as well as the peroneal artery, as previously described (Figure **6.3d**). If there is difficulty, the operative exposure may be extended more distally to allow both vessels to be manipulated directly.

If a 2F catheter cannot be passed beyond the ankle or back-bleeding is poor, an angiogram should be obtained. Distal clot may then necessitate exploration of the tibial arteries at the ankle (see below). In other cases the distal tree may seem clear and after irrigation with heparin–saline, the popliteal arteriotomy is closed. A direct closure with a running 7/0 prolene stitch may be possible, providing very small bites are taken. If the artery wall is at all diseased or the artery of small calibre, vein patch closure should be utilized (Figure **6.3f**). Finally, a completion arteriogram is obtained to confirm distal patency.

Infrapopliteal vessels

Embolization of the infrapopliteal vessels may be encountered more frequently nowadays since many emboli are of a smaller size consequent upon the change in the underlying cardiac pathology[1]. In other cases the infrapopliteal vessels become occluded as a result of fragmentation of a larger proximal embolus.

Patients who present with primary infrapopliteal embolization may be initially treated with a low-dose intra–arterial streptokinase infusion in view of the slightly greater technical difficulties of embolectomy at this level[20]. Atheromatous emboli from the aorta or proximal arteries are not lysable and these cases together with those who have residual infrapopliteal clot following transfemoral or transpopliteal embolectomy may be managed by retrograde catheter extraction from the ankle[14,15].

The posterior tibial artery is exposed by a curvilinear incision midway between the medial malleolus and the Achilles tendon. The flexor retinaculum is then divided and the artery located between the tendons of tibialis posterior and flexor digitorum longus anteromedially and flexor hallucis longus posterolaterally. The dorsalis pedis artery is exposed by a dorsal incision between the first and second metatarsals (see Chapter 5).

After gaining loop control, a transverse arteriotomy is made in the relevant artery. A 3F catheter is then passed retrogradely into the popliteal artery and proximal embolectomy achieved. A 2F catheter is passed as far distally as possible and similar clearance achieved. The arteriotomy is then closed with interrupted 8/0 or 9/0 prolene sutures. Optical magnification is helpful in making and closing arteriotomies at this level.

LATE EMBOLECTOMY

A number of patients do not present until several hours or days after the onset of ischaemic symptoms. Although early operation, i.e. within 12 hours, is associated with increased limb salvage[1,3,25], the critical determinant is the severity of the ischaemia rather than the absolute time interval involved[9,26]. If ischaemia is total, irreversible changes will start to occur in the muscles by 4–6 hours[27]. However, if there is still some collateral flow, limb viability may be maintained and a successful embolectomy may be achieved many hours or even days later. The best guide to limb viability is the state of the calf muscles. If these are still soft then the limb can still be salvaged no matter how long the delay[28]. Skin mottling and even sensory or motor loss may be largely or completely recoverable and are therefore unreliable indices[10,29]. Rigor of the muscles or frank gangrene are the only contraindications to attempted revascularization.

Other patients may present subacutely with claudication or mild rest pain in the foot rather than the classical features of acute ischaemia[10]. The presence of a viable limb in these patients is an assurance that the distal vessels are free from thrombus and that revascularization should be feasible. Late embolectomy may be achieved on standard lines though thrombus adherence may necessitate an arteriotomy directly over the embolus or catheter insertion at more than one site. Intraoperative arteriographic control is particularly important in late cases to confirm the effectiveness of the clearance. If catheter thromboembolectomy is unsuccessful a bypass graft should be placed to a distal patent vessel.

ADVANCED ISCHAEMIA

Where ischaemia has been particularly severe preoperatively, fasciotomy is advisable as a routine since there may be quite massive swelling and oedema following revascularization. Mannitol may also be beneficial in limiting local limb oedema[29]. Restoration of the circulation to a severely ischaemic limb may result in a washout of acid metabolites and potassium with an attendant risk of cardiac arrythmias, potentially fatal myocardial depression, or renal impairment. Renal function is particularly likely to be impaired if myoglobin is deposited in the tubules (myonephropathic–metabolic syndrome[30]).

Several measures should be adopted to prevent the systemic effects of revascularization in patients with severe ischaemia. Intravenous fluid loading should be commenced prior to surgery

and be adjusted according to central venous or left atrial pressures. Additional frusemide may also be helpful in promoting urine flow. Bicarbonate is required to correct acidosis and is best given prior to clamp release. Adequate alkalinization is particularly important if myoglobinuria is present or anticipated, since myoglobin is more likely to be precipitated in the renal tubules in an acid urine. Hyperkalaemia may be controlled by glucose–insulin, ion exchange resins or, if necessary, by haemodialysis. Others have advocated venotomy and drainage of the venous effluent during the first pass of arterial blood in order to prevent the initial wash-out of acid metabolites and the release into the lungs of venous microthrombi from the ischaemic limb[31]. Where circulatory stasis has been particularly severe, an additional venous occlusion may be present and may require venous thrombectomy.

EMBOLECTOMY IN THE PRESENCE OF ATHEROSCLEROSIS

The arrival of an embolus in a markedly atherosclerotic vessel may disrupt the previous equilibrium and cause certain technical difficulties. Simple embolectomy may still succeed in restoring patency in some cases but this may be relatively short-lived. In other cases complete clearance may be impossible to achieve, or a sub-intimal dissection may occur during catheter manipulations (see below). Either of these circumstances will require definitive arterial reconstruction, usually by means of a bypass graft[32]. Persistent or recurrent aortoiliac occlusion may require extra-anatomic bypass while occlusion in the superficial femoral segment may be managed by a femoro–distal bypass. Embolization supervening on a diseased popliteal trifurcation may be particularly difficult to manage because the infrapopliteal vessels may also be diseased and there may be extensive distal clot propagation. If patency can be re-established in the distal calf or foot an *in situ* bypass may salvage the limb (see Chapter 5). Alternatively if the distal tree cannot be cleared, a local low-dose streptokinase infusion may be considered in an attempt to avert amputation.

EMBOLECTOMY IN THE UPPER LIMB

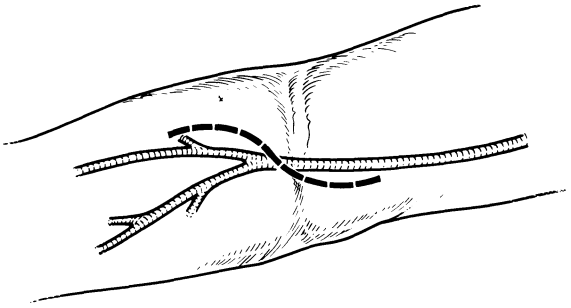
Approximately 20% of major peripheral emboli involve the upper limb. The principal sites of lodgement are the subclavian, axillary and brachial vessels with the brachial bifurcation being most frequently affected[33,34].

An underlying cardiac source is usually evident, e.g. a cardiac arrhythmia, previous myocardial infarction, valvular disorder or ventricular aneurysm. Non-cardiac sources include ulcerative lesions in the arch or subclavian–axillary vessels and thoracic outlet compression (see Chapter 11). As in the lower limb non-cardiac emboli tend to be smaller and produce occlusion at digital or palmar arch level with retained limb pulses[35,36].

Although there has been a tendency to regard upper limb emboli as innocuous, this is not always the case. Certainly collateralization is more readily established in the upper than in the lower limb, but emboli may still result in functional impairment of the hand and very occasionally in limb loss. Because of the simplicity and general effectiveness of embolectomy in the upper limb, a surgical approach is advisable in all cases.

Most major upper limb emboli can be extracted through a distal brachial arteriotomy[32]. This site has the advantage that both forearm vessels can be directly cannulated. An S-shaped incision is made in the antecubital fossa and the brachial bifurcation exposed by dividing the bicipital aponeurosis (Figure **6.4a, b**). Loops are placed around the proximal and distal vessels and a transverse arteriotomy made in the distal brachial artery. An embolus at this site may then be directly extracted, following which the radial and ulnar arteries should be catheterized in turn to remove propagated clot (Figure **6.4c, d**). It is important to clear both forearm vessels since single vessel patency may not result in full symptomatic relief. Heparin is then instilled distally, and after confirming proximal patency, the arteriotomy is closed with interrupted sutures.

If there is retained distal clot on the control angiogram, or wrist pulses are restored but the hand remains ischaemic, the radial or ulnar artery should be opened at the wrist and a 2F catheter passed distally into the hand[37].



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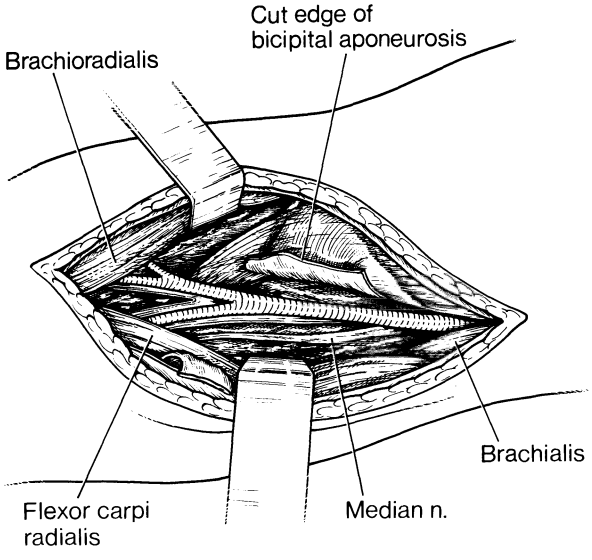


(b)

Figure 6.4(a–d) Brachial embolectomy: (a, b) exposure of the brachial artery bifurcation

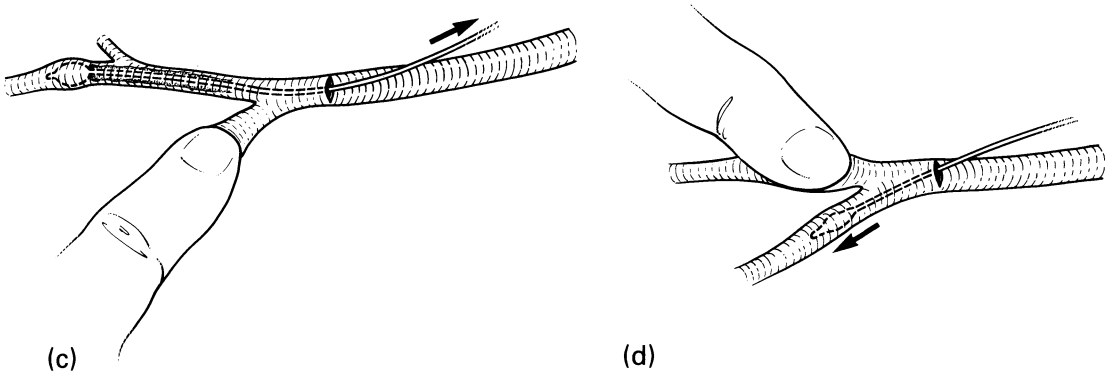


Figure 6.4 continued (c, d) a Fogarty catheter is directed into the radial and ulnar arteries in turn using alternate digital compression

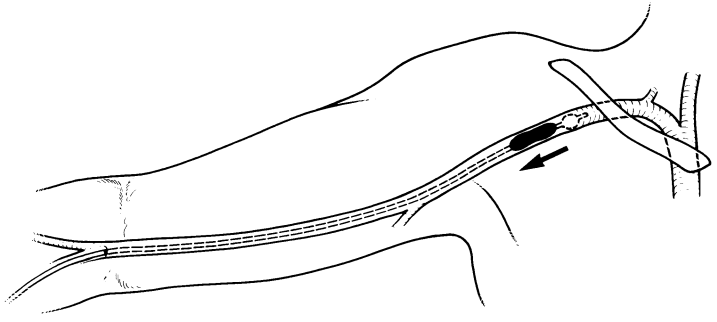


Figure 6.5 Subclavian–axillary embolectomy by means of retrograde catheterization from the antecubital fossa

Emboli in the axillary or subclavian arteries may also be removed by transbrachial retrograde catheterization (Figure **6.5**). In the case of a right subclavian embolus there is a theoretical risk that thrombotic material may be dislocated into the common carotid artery during retrograde catheter passage. Temporary compression of the proximal right common carotid artery may therefore be advisable while the catheter is being advanced and withdrawn.

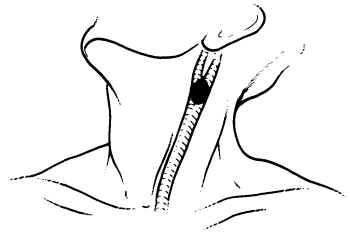
NON-LIMB EMBOLIZATION

Embolization of non-limb arteries occurs with greater frequency than is generally realized, many smaller emboli remaining clinically silent or producing symptoms which are attributed to other causes[30]. At the other extreme, more major emboli may be rapidly fatal[25]. From a surgical point of view the most important sites of non-limb embolization are the carotid bifurcation and the renal and superior mesenteric arteries.

Carotid artery embolectomy

Most cerebral emboli of cardiac origin are small and cause occlusion of intracranial arteries rather than the common carotid bifurcation. Nevertheless, an embolus at the latter site should be considered in any patient with an embologenic cardiac lesion who sustains a sudden neurological deficit (Figure **6.6a**). If this occurs while the patient is in hospital, e.g. following valve replacement, there should be sufficient time to undertake emergency carotid arteriography followed by surgical exploration.

The carotid bifurcation is exposed along standard lines (see Chapter 9). After heparinization a transverse arteriotomy is made in the distal common carotid artery and the embolus directly extracted. A Fogarty catheter is then passed up the internal carotid artery to the base of the skull and gently withdrawn (Figure **6.6b**). A similar manoeuvre is carried out in the external carotid artery. After all the thrombotic material has been removed the vessels are clamped and the lumen rinsed. The arteriotomy is then closed with interrupted sutures. Air is evacuated from the operative zone before releasing flow firstly into the external carotid artery and then into the internal carotid territory (see Chapter 9).



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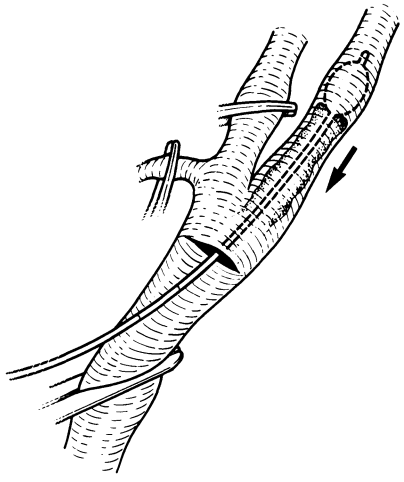

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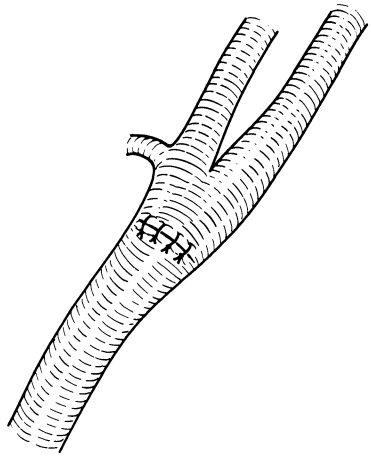
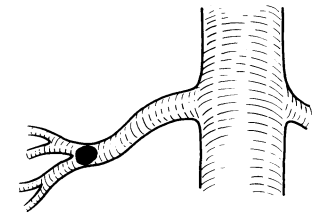


(c)

Figure 6.6(a–c) Carotid artery embolectomy: (a) site of embolus at the carotid bifurcation. (b) thrombus retrieval from the internal carotid artery. (c) arteriotomy closure



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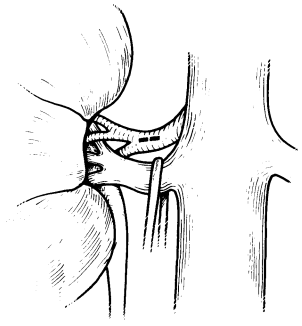

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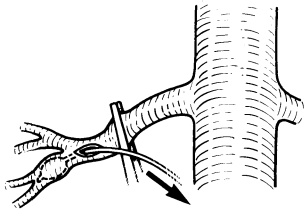

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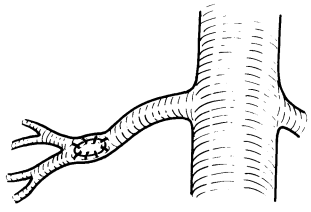

(d)

Figure 6.7(a–d) Renal artery embolectomy: (a) site of embolus in the right renal artery. (b) longitudinal arteriotomy. (c) the embolus is removed and the branch arteries cleared using a small Fogarty catheter. (d) the arteriotomy is closed with a vein patch

Renal artery embolectomy

Renal artery embolization may be suspected in any patient with cardiac disease who develops sudden flank pain. Vomiting, fever and a leukocytosis may be associated and there is likely to be albuminuria and microscopic haematuria. An intravenous pyelogram or scan may show a non-functioning kidney, though if the occlusion involves a branch artery appearances may be relatively normal. Arteriography should be immediately undertaken to confirm the diagnosis.

Although total circulatory interruption inevitably results in loss of the kidney, viability is often maintained by collateral flow and by residual flow around the embolus. Treatment may hence be feasible despite considerable delay.

Many patients who sustain renal emboli are poor operative risks and first line treatment nowadays may be low-dose intra-arterial streptokinase[38,39] or percutaneous recanalization[40]. Alternatively transfemoral catheter thromboembolectomy may be carried out, the embolus being either dislodged into a safer vessel[41] or retrieved via a femoral arteriotomy[42].

A surgical approach may be indicated in the better risk patients, particularly when there are bilateral main artery emboli or a major embolus to a solitary kidney[43]. Surgical exposure of the renal arteries is described in Chapter 13. Although emboli may be extracted via an aortotomy, a direct incision over the embolus may be preferable (Figure **6.7a, b**). After heparinization the renal artery is clamped proximally and a longitudinal arteriotomy made, stopping short of the bifurcation. The embolus is then removed and the distal tree cleared with a 2F catheter. After rinsing with heparin–saline, the arteriotomy is closed with a small patch (Figure **6.7d**). Where embolectomy is undertaken late there is a significant incidence of technical failure and an aortorenal bypass may be needed to revascularize the kidney.

Superior mesenteric embolectomy

Mesenteric ischaemia due to embolization may be eminently treatable if recognized promptly. The technique is similar to that in other arteries. Further details are described in Chapter 12.

COMPLICATIONS ASSOCIATED WITH USE OF THE FOGARTY CATHETER

Despite the great advantage of the Fogarty catheter, its use may be associated with certain risks which, though rare, should be recognized[44,45]. These hazards are more frequent in fragile vessels such as the infrapopliteal or profunda femoris arteries and in the presence of atherosclerotic disease.

Introduction of the catheter may cause a subintimal dissection (Figure **6.8a**) or rarely a complete perforation of the wall (Figure **6.8b**). As a result of the latter a false aneurysm may form, or if an adjacent vein is also injured there may be an arteriovenous fistula. Excessive balloon inflation may rupture the artery wall (Figure **6.8c**) or the balloon itself may burst with distal embolization of the fragments (Figure **6.8d**). Balloon retrieval is perhaps the stage which gives rise to most complications. If the balloon is slightly overinflated it may detach the intima and result in an extensive dissection particularly if the vessel is atherosclerotic (Figure **6.8e**). This may lead to immediate thrombosis or, if the injury is less severe, to stenosis and late occlusion.

Rather more subtle damage may also occur after use of balloon catheters. Thus repeated catheter passage may impair endothelial fibrinolytic activity[46]. In addition there have been isolated reports of diffuse arterial narrowing suggestive of intimal hyperplasia[47] or even premature atherosclerosis[48] after catheter thromboembolectomy.

Routine use of intraoperative arteriography will detect most catheter injuries. Evidence of damage may include contrast leakage, arteriovenous fistulae, discrete irregularities of the artery wall, segmental narrowing or complete occlusion. In the absence of arteriographic control, many postoperative occlusions may be wrongly attributed to a poor distal arterial tree or extensive consecutive thrombus.

Management of catheter injuries depends on the vessel involved, the type of injury and the status of the other vessels. Thus, perforation of a branch vessel or one of the infrapopliteal vessels with subsequent haemorrhage may be treated simply by ligation. Occlusion of an infrapopliteal artery may be ignored if the other calf vessels are patent. In contrast, injury to the superficial femoral, proximal profunda or popliteal arteries should always be repaired. Techniques are the same as for vascular injuries elsewhere and include simple suture, angioplasty or resection–anastomosis. Extensive intimal dissection in an atherosclerotic vessel may require bypass grafting.

In order to avoid these complications certain precautions should be adopted when using a Fogarty catheter. Thus, the stylet should be removed and the balloon tested before passage. Balloon inflation may be best achieved using a small lubricated glass syringe filled with a saline–air mixture[14,49]. Air alone may not provide enough traction to achieve complete clearance and control over balloon inflation may be poor[50]. On the other hand, a saline-filled balloon may be too rigid and may increase the risk of intimal injury.

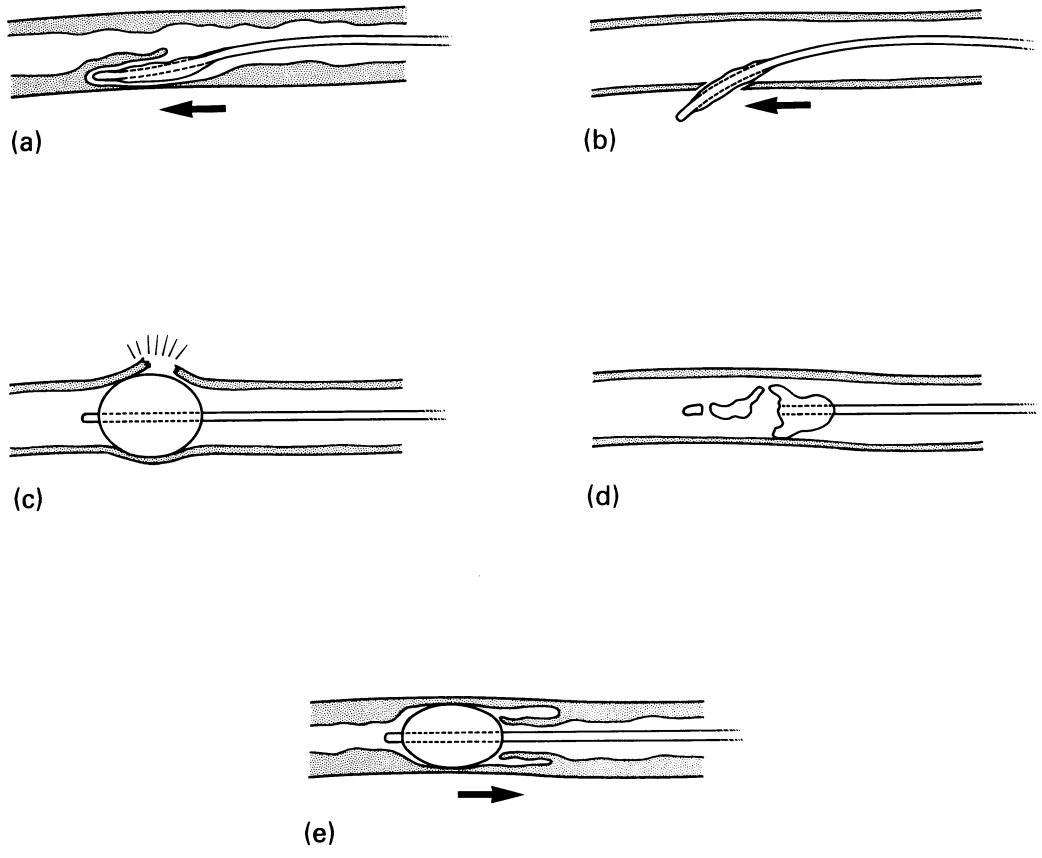

Figure 6.8(a–e) Complications associated with the use of balloon catheters: (a) subintimal dissection during catheter advancement. (b) perforation. (c) rupture of artery. (d) balloon rupture. (e) extensive dissection in an atherosclerotic vessel caused by overinflation of balloon during withdrawal

In addition the catheter should always be inserted gently and never forced into the artery. If an obstruction is encountered this is much more likely to be due to atherosclerosis than recent thrombus and an immediate arteriogram should be obtained. As previously emphasized, catheter withdrawal should be in a smooth gradual manner with the operator himself matching the degree of balloon inflation against the elastic resistance of the artery wall. Injuries both overt and subtle will be minimized by keeping the number of catheter passes to a minimum and avoiding balloon hyperinflation.

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7

Vascular Injuries

GENERAL CONSIDERATIONS

Vascular injuries are being seen with undiminishing frequency mainly as a result of road traffic accidents and acts of civil violence. Iatrogenic causes, particularly arterial catheterisation, and domestic or industrial accidents account for most of the remainder. Vascular trauma may be of two kinds: penetrating and blunt. Examples of penetrating injuries include gunshot and knife wounds and accidents involving glass or other sharp fragments. Blunt trauma may involve fractures or dislocations, and crushing, stretching or deceleration forces. Certain skeletal injuries, such as supracondylar fractures of the humerus or femur and posterior dislocation of the knee, are so likely to be complicated by vascular injury that the latter should be assumed present until proved otherwise. An important feature of blunt trauma is that the vascular damage tends to be more extensive than that associated with penetrating injury.

The main pathological types of arterial injury are laceration, transection, contusion, false aneurysm and arteriovenous fistula (Figures **7.1–3, 11, 12**). Most often laceration and transection result from penetrating injury and contusion from blunt trauma but exceptions abound, e.g. bony fragments from a fracture caused by blunt trauma may lacerate an artery while a high velocity missile may cause contusion in an adjacent vessel.

Although some lacerations such as those following a surgical accident have a clean edge, most are ragged with a variable degree of adjacent wall damage (Figure **7.1b**). Retraction of the media tends to hold the defect open so that haemorrhage may be profuse. Complete transections in contrast are usually associated with less bleeding since the ends retract and seal off (Figure **7.1c**). Rarely the adventitia and media only are divided and the intima remains intact. However the latter is almost invariably contused and unless the damaged zone is excised, thrombosis may result.

Contusions may consist of no more than a relatively minor haematoma in the adventitia. In other cases an intimal split or dissection may occur

Figure 7.1(a–c) Penetrating arterial injuries: (a) clean laceration. (b) ragged laceration. (c) complete transection with retraction of the extremities

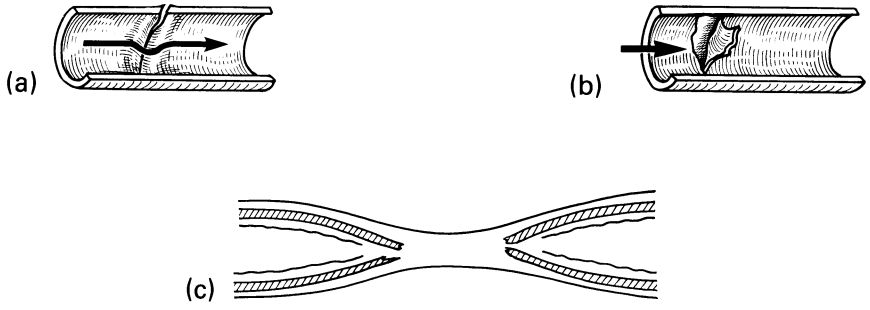


Figure 7.2(a–c) Non-penetrating arterial injuries: (a) intimal split with initial preservation of flow. (b) intimal dissection. (c) rupture of the intima and media

(Figure **7.2a, b**) or there may be disruption of both the intima and media (Figure **7.2c**) leading to occlusion or, much less commonly, to a true aneurysmal dilatation. More severe blunt trauma may result in complete separation of the vessel. Of these lesions, an intimal split may be singled out as being particularly treacherous, since flow may be initially preserved only to disappear several hours later when thrombosis supervenes. By this time the limb may be encased in a plaster cast or dressings and recognition may be delayed.

The clinical signs of arterial injury include absent distal pulses, a cool extremity, distal pallor, sensory-motor loss, external haemorrhage, an expanding or pulsatile haematoma and a bruit or thrill. It should be emphasized that pulse status may not be completely reliable[1]. The pulse wave can be propagated across soft clot or intimal flaps so that distal pulses may occasionally be palpable despite the presence of an arterial injury[2]. Distal pulses may also be deceptively retained in the case of partial transection injuries where the breach has been temporarily sealed by clot. Conversely pulses may be absent yet the artery is intact. Thus marked hypotension alone may cause loss of distal pulses, while in some cases of blunt trauma the artery may be trapped or angulated at a fracture site without any intrinsic injury. Absent

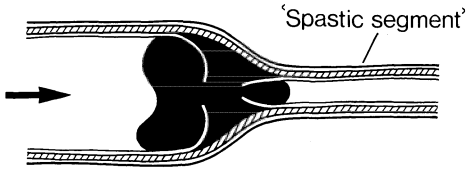


Figure 7.3 Intimal injury masquerading as 'spasm'. The pulse wave may travel across soft clot and the collapsed distal segment may be wrongly attributed to spasm

pulses may also be due to arterial spasm, particularly in children. However, this is rare and loss or diminution of distal pulses should always be attributed to intimal injury until proved otherwise either by arteriography or exploration. Even at operation appearances can be misleading since clot can pulsate and a narrowed distal segment may be wrongly attributed to 'spasm' unless the artery is opened or an arteriogram obtained (Figure **7.3**).

If there are clear signs of arterial injury in relation to a wound or fracture, arteriography may add little to management[3]. However, in some circumstances arteriography may be very helpful in surgical planning. This particularly applies to injuries at the base of the neck, multiple injury sites in a limb or where a missile trajectory parallels the artery for some distance. An even more important role for arteriography is in excluding arterial injury when clinical signs are equivocal or there is a penetrating wound in proximity to a major vessel[4,5]. Demonstration of a normal arterial tree in these cases (reinforced if necessary by ultrasound assessment) may avoid an otherwise unnecessary surgical exploration.

OPERATIVE MANAGEMENT

Some general principles of management may be described taking as example an injury to the superficial femoral artery associated with a fractured shaft of femur.

If there is haemorrhage from an open wound this should be controlled by local compression while fluid resuscitation is commenced and the patient transferred to the operating room. Tourniquets should be avoided in extremity injuries because they aggravate muscle ischaemia and on release may give rise to acidosis and possible renal impairment.

In the operating room, local compression is maintained while the limb is cleaned and prepped. The contralateral thigh should also be included in this process in case a saphenous vein graft is required for arterial or venous repair. At the last moment local compression is removed and the prepping and draping quickly completed. The wound is then extended as necessary in the line of the superficial femoral vessels (Figure **7.4a, b**) and proximal and distal control obtained at a distance from the traumatized zone. The latter is then explored and the clamp positions readjusted in closer proximity. The ends of the artery are then debrided and a catheter thrombectomy performed proximally and distally, followed by distal infusion of 20 mg heparin. Systemic heparinization is a more effective method of preventing distal thrombosis but is often contraindicated by injuries elsewhere.

Traditionally the femoral fracture is now reduced and the skeleton stabilized prior to vascular repair[1,6]. Stability can be rapidly achieved by cross-pins and an external fixation device of the Hoffman type[7]. Internal fixation has alternatively been advocated[8] but there is at least a theoretical risk of bone sepsis with penetrating injuries and internal fixation may delay arterial repair. However, it remains an appropriate option for dealing with closed fractures when the limb is not in immediate jeopardy.

The threat by the bone ends to the vascular repair has perhaps been overemphasized in the past and in most injuries vascular repair may precede skeletal fixation[9]. This is particularly important when the limb has already been severely

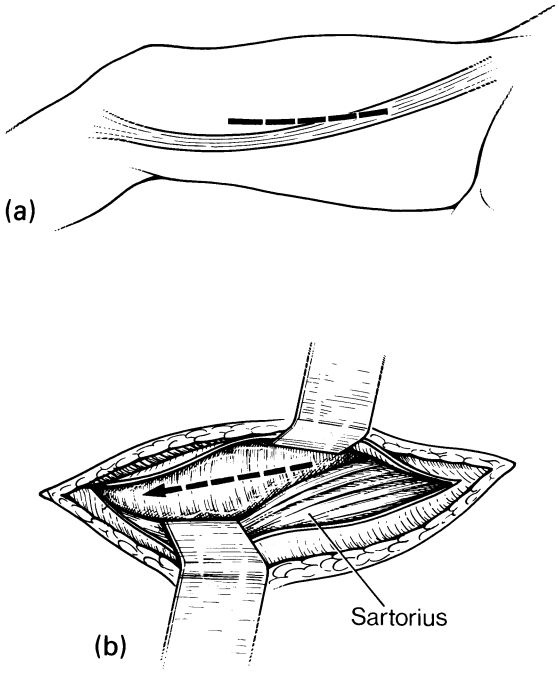


Figure 7.4(a, b) Exposure of the superficial femoral artery: (a) skin incision. (b) sartorius reflected medially

ischaemic for several hours. If the fracture is too unstable to perform a satisfactory vascular repair an intraluminal shunt may be inserted between the two ends of the artery after preliminary catheter thrombectomy and distal heparinization[10,11]. This manoeuvre allows ample time for skeletal fixation and for repair of any additional vein or nerve damage. In addition early restoration of limb perfusion aids in identifying non-viable soft tissue so that a more accurate debridement is possible. When all these matters have been dealt with, the artery is repaired and the shunt dismantled.

Arterial repair

Uncommonly the arterial injury may be no more than a punctiform laceration which after suitable trimming may be closed either directly or with a small patch (Figure **7.5a, b**).

In other patients the artery may occasionally appear intact from the outside but on opening the vessel a clean linear split in the intima is found. Providing the vessel is of reasonable size, suture repair of the intima may be sutured and the arteriotomy closed with a patch (Figure **7.6a, b**).

In the great majority of cases the arterial damage is more extensive, with or without loss of continuity and a resection is required. This should include 0·5 cm of macroscopically normal artery on either side of the traumatized zone in order to avoid the risk of postoperative re-thrombosis. If the resulting defect is limited and the two ends will come together without tension a direct reanastomosis may be performed. This may be carried out by a triangulation technique or by a running intraluminal suture posteriorly with interrupted sutures along the anterior half of the anastomosis (Figure **7.7a, b**). If the artery is small, or the injury occurs in childhood, interrupted

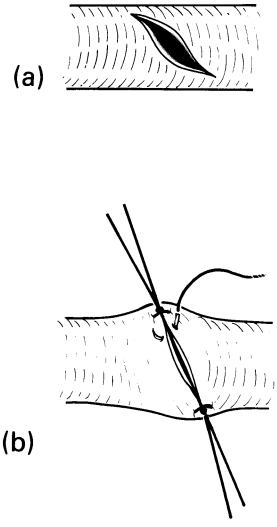


Figure 7.5(a, b) (a) Laceration trimmed. (b) Defect closed by direct suture

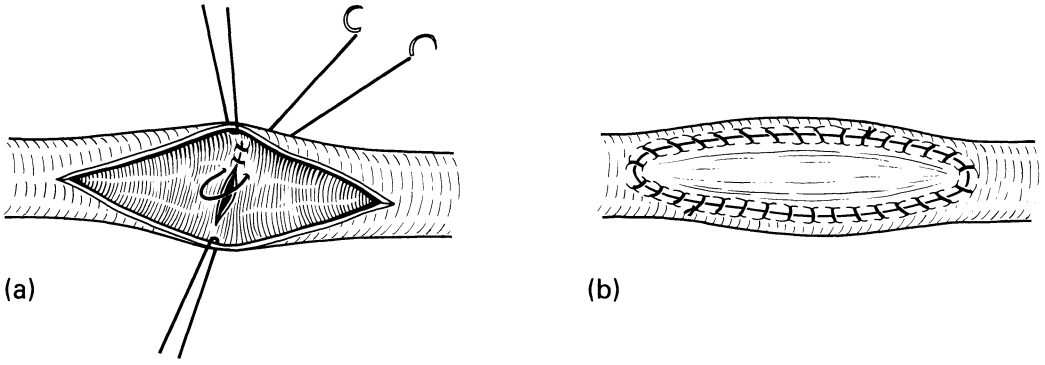


Figure 7.6(a, b) (a) Clean intimal split repaired with tack-down sutures. (b) Arteriotomy closed with a small vein patch

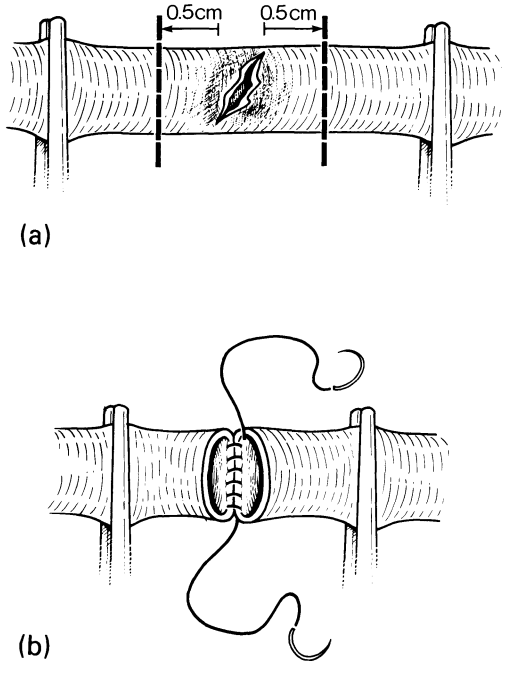


Figure 7.7(a, b) Resection of the damaged zone with 0·5 cm margin of macroscopically normal artery on either side. Direct end-to-end re-anastomosis

sutures should be used for the entire anastomotic circumference.

Where the two ends cannot be brought together without tension, a graft will be necessary since attempted re-anastomosis will inevitably lead to stenosis and premature occlusion. The usual graft material is autogenous saphenous vein. If there is additional venous injury, it is probably advisable to obtain the graft from the contralateral leg. Prosthetic grafts are quicker and have given good results even in open wounds according to some reports[12,13]. The graft may be interposed with appropriate spatulation (Figure **7.8a**) or inserted as an end-to-side bypass (Figure **7.8b**). This latter arrangement allows a wider anastomosis with less risk of subsequent stenosis than a direct end-to-end interposition.

After completing the repair an arteriogram should be obtained as a routine to verify the quality of the reconstruction and to confirm the absence of a distal embolus or a second traumatic lesion. It may also indicate a need for fasciotomy by identifying extrinsic compression of the vessels in the calf.

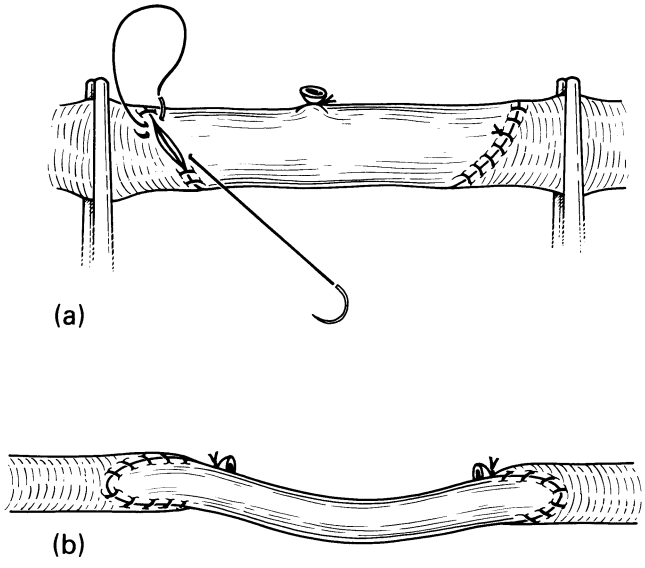


Figure 7.8(a, b) Resection with graft replacement: (a) interposition vein graft. (b) end-to-side vein bypass with supplementary ligation

Venous repair

An associated venous injury should be repaired if possible as this may increase the chances of limb salvage[14,15] and will avoid the late sequelae of chronic venous insufficiency.

If the vein can be repaired by lateral suture a good result is likely[16]. Replacement grafts are less satisfactory but may be worth attempting at particularly critical sites such as the popliteal fossa. Mismatch in size may require insertion of a venous compilation or PTFE graft (Figure **7.9b**). Alternatively an end-to-side bypass may be placed, using saphenous vein from the opposite leg (Figure **7.9c**).

Other injuries

Concomitant nerve damage is the most significant factor adversely affecting functional recovery in limb injuries[17]. If the nerve has been cleanly transected, which is unusual, a direct primary repair may be attempted. However, in many blunt injuries the nerve is macerated or a segment is missing. In these cases the nerve ends should be identified and secured to adjacent soft tissues to prevent undue retraction. This reduces the length of a subsequent nerve graft. Once the wounds are healed, repair by cable grafting may be undertaken. Major proximal nerve injuries in general have a rather poor prognosis and the combination

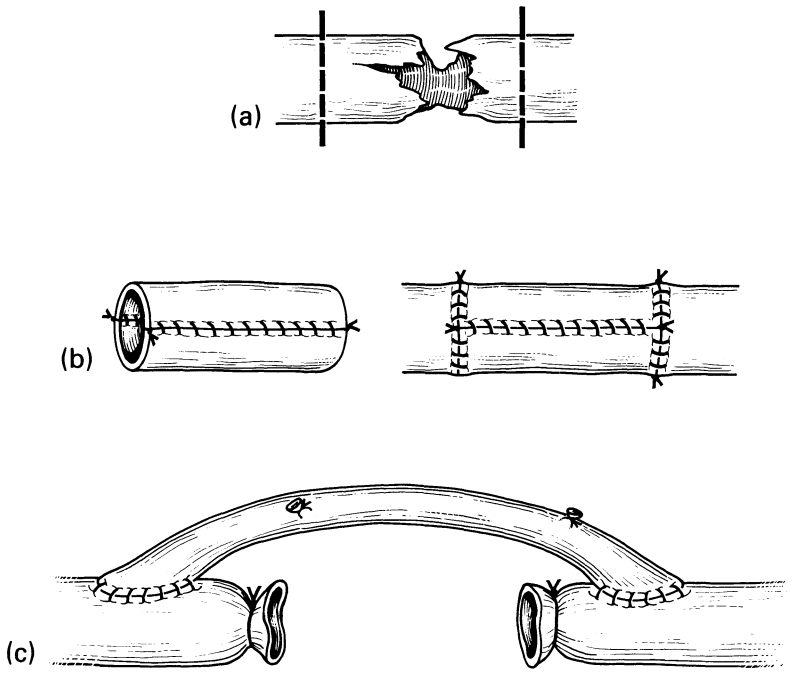


Figure 7.9(a–c) (a) Venous injury. (b) Resection of damaged segment and replacement with a venous compilation (panel) graft. (c) End-to-side vein bypass

of severe neurovascular, soft tissue and bony injury may be more appropriately treated by a primary amputation.

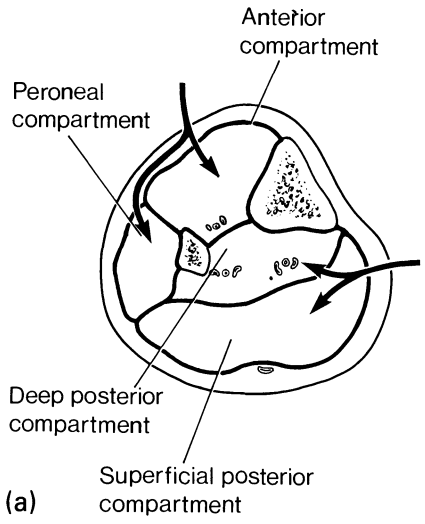
Soft tissue damage requires meticulous debridement with removal of all devitalized and contaminated tissue. Penicillin, broad-spectrum antibiotics and tetanus prophylaxis are given as a routine.

Usually the vascular repair can be covered by healthy muscle, in which case the skin can be left open if there is swelling or gross contamination of the wound. If there is more extensive soft tissue injury, a muscle slide, skin advancement or a myocutaneous flap may be necessary to provide cover for the vascular repair. Where this is not feasible an extra-anatomic bypass may be considered with direct in-line vascular reconstruction at a later date when wound healing is complete[18].

Fasciotomy

This should be considered in the majority of vascular injuries. It is particularly important when ischaemia is severe and surgery delayed. In such cases the leg may already be tense from a compartment syndrome and it may be helpful to perform a fasciotomy prior to revascularization[7]. Other specific indications for fasciotomy in arterial injuries include prolonged shock, severe soft tissue injury and coexistent venous injury[19].

Fasciotomy may be achieved via medial and anterolateral incisions[20]. Usually the skin and fascia are both incised but if only the latter is contributing to the constriction, limited skin incisions may be utilized. Decompression of the posterior calf compartments is achieved via a longitudinal incision just behind and parallel with the medial



border of the tibia (Figure **7.10a, b**). The superficial compartment is opened first, then the deep compartment which is directly accessible anterior to soleus in the distal part of the incision. If necessary the decompression may be extended into the sole of the foot (Figure **7.10c**). A second incision is then made down the anterolateral aspect of the leg and the anterior and peroneal compartments decompressed via separate fascial incisions (Figure **7.10a**). In severe cases the fasciotomy may be extended onto the dorsum of the foot.

An alternative fasciotomy technique is based on fibular resection[21]. This allows direct access to all four compartments through one incision (Figure **7.10d**). Fibulectomy–fasciotomy may be particularly appropriate when the fibula is already fractured or access to the peroneal artery is required. It may also be indicated if standard fasciotomy does not achieve full decompression in the face of massive soft tissue swelling[7].

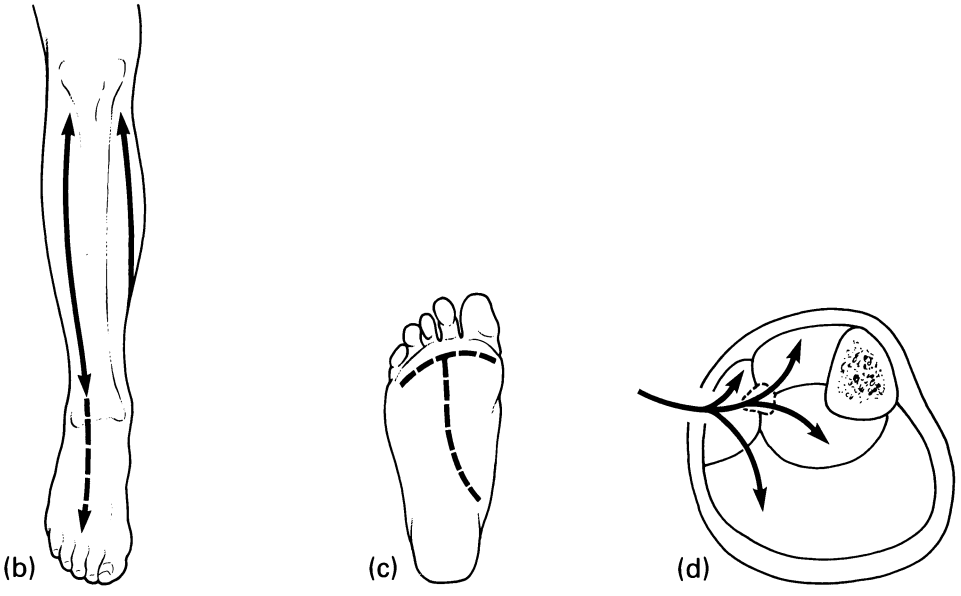


Figure 7.10(a–d) Fasciotomy techniques: (a) access to the fascial compartments via anterolateral and medial skin incisions. (b) extent of fasciotomy. (c) plantar extension. (d) Fibulectomy–fasciotomy with access to all four compartments of the leg

LATE REVASCULARIZATION

As in the case of embolic occlusion, traumatic arterial interruption may still be amenable to surgical correction many hours after the event providing collateral supply has maintained distal viability. The degree of collateral perfusion is difficult to predict and is governed by systemic arterial pressure, the extent of the arterial occlusion, coexistent trauma to the soft tissues carrying the collateral vessels and the site of the injury. The knee is a particularly dangerous site because of the relative scarcity of alternative channels if the main artery is interrupted. Venous injury may further impair perfusion by preventing venous return and raising compartmental pressure.

If the injury occurs in a limb without collateral supply, delay in revascularization may be disastrous. Muscle necrosis begins to occur after 4–6 hours[22] and this may progress to mass infarction. Less severe cases may exhibit patchy necrosis with replacement fibrosis resulting in a Volkmann's contracture. Systemic effects may occur when revascularization is undertaken in the severely ischaemic limb and include acidosis, hyperkalaemia and renal or cardiac impairment. Myoglobin pigment may be deposited in the renal tubules increasing the risk of renal failure (see Chapter 6).

Acid-base status, blood urea, creatinine, potassium and muscle enzyme levels should be monitored post-repair in these cases. In addition, various prophylactic measures should be adopted, including intravenous fluid loading and frusemide to promote a diuresis and bicarbonate to correct acidosis. Glucose–insulin or an exchange resin may be needed if there is hyperkalaemia while severe cases may require temporary dialysis. Wide fasciotomy is crucial in patients with advanced ischaemia since it will decompress the muslces and interrupt the vicious circle of oedema and anoxia. Mannitol may also be helpful in this respect[23].

TRAUMATIC FALSE ANEURYSMS

These usually arise after a penetrating injury though occasionally blunt trauma may be responsible. The initial consequence of an uncorrected partial transection injury is a pulsatile haematoma (Figure **7.11a**). This may enlarge over several days and may produce local pain and swelling or distal ischaemia as a result of compressing the adjacent arterial axis. Treatment should be undertaken urgently because of the risk of free haemorrhage. Once proximal and distal control has been achieved the haematoma is evacuated. This may reveal quite a small defect in the artery wall which can often be closed with one or two interrupted sutures.

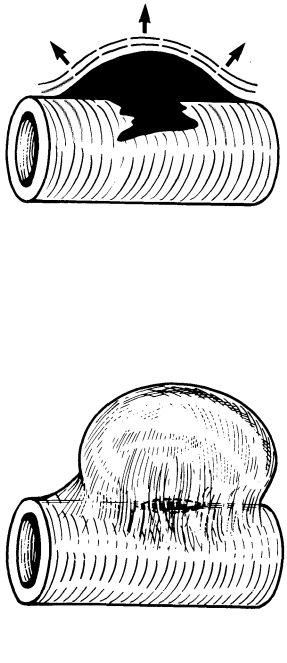


Figure 7.11(a, b) Consequences of unrepaired partial transection: (a) pulsatile haematoma. (b) false aneurysm

In other cases the periarterial haematoma liquefies and resorbs leaving a false aneurysmal sac (Figure **7.11b**). This may present weeks or months after a penetrating injury as a localized swelling or with pressure effects, particularly on adjacent nerves[24,25]. Unfortunately, at this stage correction of the arterial lesion may not restore full neurological function even with meticulous neurolysis. Recognition of partial transection injuries at their inception is hence of crucial importance and underlines the advisability of obtaining an arteriogram in all proximity penetrating wounds. Apart from compression effects, traumatic false aneurysms are also liable to rupture, particularly if infection is present.

Where a false aneurysm is suspected, arteriography should be undertaken to confirm the diagnosis and to allow a planned reconstruction. Surgical access may be difficult if there is post-traumatic scarring and often the easiest method of managing these lesions is to clamp the inflow at a distance and then to open the sac without attempting any local dissection. Endoluminal catheters will keep the field dry while the afferent, efferent and branch orifices are sewn off from within. Vascular continuity is then restored by an end-to-side bypass graft. If the false aneurysm is infected, the latter should be routed extra-anatomically through a separate clean field.

ARTERIOVENOUS FISTULAE

An arteriovenous fistula is a less common late complication of arterial injury (Figure **7.12**). It usually occurs as a result of simultaneous perforation of an artery and vein though it may arise following rupture of a false aneurysm into an adjacent vein. In some cases the fistula is recognized at the time of the injury by the presence of a bruit. However, in most cases the communication is initially surrounded by thrombus and the presence of a fistula is not recognized clinically until days or weeks later. The basis of treatment is to disconnect the fistula, followed by repair of the artery and often the vein as well (for details see Chapter 8).

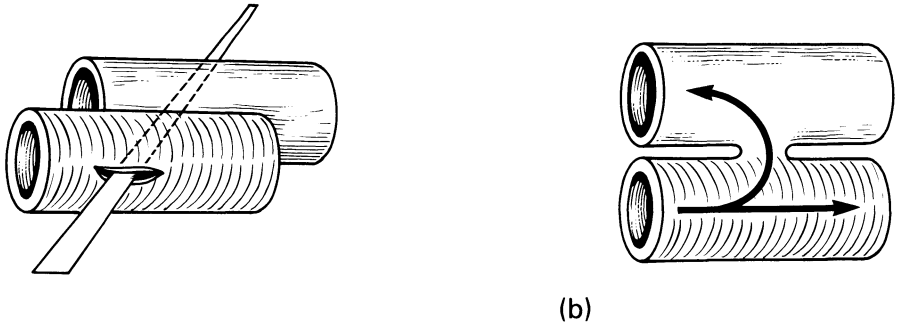


Figure 7.12(a, b) Traumatic arteriovenous fistula

REGIONAL CONSIDERATIONS

COMMON FEMORAL ARTERY

The particular feature of penetrating injuries in the groin is their possible involvement of a major arterial (and venous) bifurcation. Haemorrhage may be profuse and endoluminal catheters may be useful in achieving initial control. If possible the common femoral bifurcation should be preserved (Figure **7.13a–c**), otherwise a bifurcated graft (Figure **7.14a–c**), or more simply a branching arrangement (Figure **7.15**) may be utilized. Autogenous saphenous vein may be used as graft material though prosthetics, especially PTFE, may be preferred on the grounds of speed and better size match. Even in the presence of an open wound the risk of prosthetic sepsis seems to be surprisingly low[12,13]. In the case of the Seldinger catheter injury a relatively long segment of intima may be damaged, extending into the external iliac artery. Graft replacement may then be required, particularly in children.

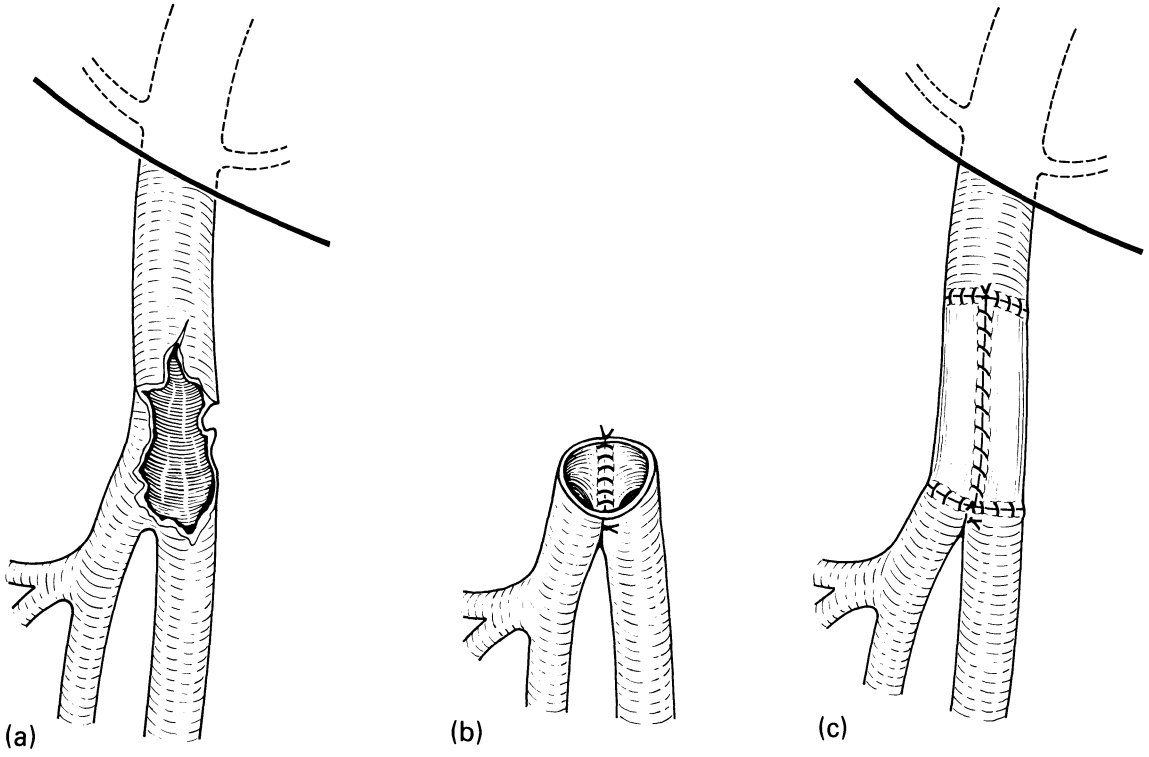


Figure 7.13(a–c) (a) Penetrating injury of the common femoral artery. (b) Femoral bifurcation reconstituted. (c) Venous compilation (panel) graft interposed

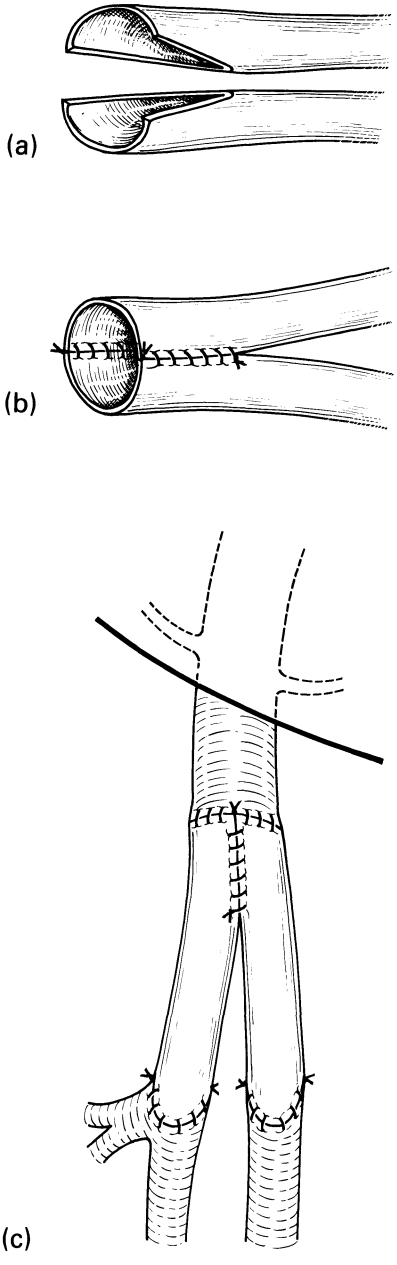


Figure 7.14(a–c) Common femoral bifurcation replaced with a bifurcated graft formed from two segments of autogenous vein

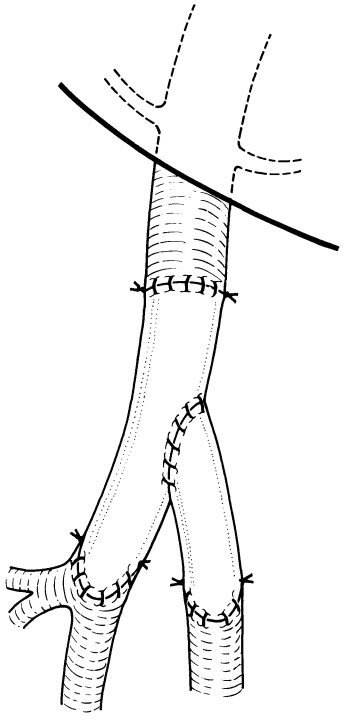


Figure 7.15 Alternative method of common femoral reconstruction using a branching PTFE arrangement

POPLITEAL ARTERY

Injuries at this site are particularly serious and carry a higher amputation rate than any other arterial injury. Blunt popliteal trauma in particular is likely to pose a threat to the limb[9,26]. Factors adversely affecting the surgical outcome include the severity of distal ischaemia due to the inherently poor collateralization, the delay in diagnosis, the frequency of additional injury to the popliteal vein, and the associated soft tissue and skeletal injury. Despite these difficulties, recent reports have indicated an improved outlook in patients with popliteal artery injuries[7]. Early wide fasciotomy and routine venous repair have been important factors in achieving this response.

Patients suspected of suffering popliteal vascular injury may be evaluated by hand-injection arteriography either in the emergency room or in the operating suite. This will help locate the exact site and nature of the injury. After prepping and draping the leg, wide fasciotomy should be undertaken as the first step to maximize collateral flow in the calf before revascularization[7].

The injured vessels are best exposed via a medial approach, since this can be easily extended up or down the leg in case bypass at a distance is required. In addition, the patient can remain supine, which is an important consideration where there are abdominal or other injuries.

If there is an associated tibial or femoral fracture, this should be rapidly stabilized using crosspins inserted above and below the fracture site. Where there is difficulty in achieving skeletal stability, a temporary heparin-bonded shunt should be utilized, as previously described. Dislocations should be reduced and the capsule and ligaments repaired as best possible followed by application of a long leg cylinder cast. In the worst cases the knee will require fusion.

Intraoperative systemic heparinization may be particularly helpful in popliteal injuries[27], but often only regional heparinization is possible, because of serious injuries elsewhere. In many cases the popliteal artery is damaged over a relatively long segment and graft replacement is preferable to attempting end-to-end re-anastomosis. The technique of end-to-side bypass at a distance is particularly suitable here, and if necessary the distal attachment can be taken well down the calf[28]. Completion angiography is then obtained as a routine.

As previously indicated, venous repair is particularly important at this level. This may be quite demanding because of the short wide venous tributaries from the soleus and gastrocnemius muscles. Infection has been an important contributor to the overall amputation rate in penetrating popliteal vascular injuries, and thorough wound debridement is essential. Soft tissue cover may be a particular problem with extensive wounds around the knee, and a myocutaneous flap may be necessary[9].

INFRAPOPLITEAL VESSELS

Arterial repair is usually necessary only when both the anterior and posterior tibial arteries are damaged, which is unusual. Vascular injuries below the knee are sometimes missed, particularly when there is associated skeletal trauma, and may subsequently give rise to a false aneurysm or arteriovenous fistula. Diagnosis depends on early use of arteriography whenever there is marked swelling and tenseness of a muscle compartment or a penetrating wound in proximity to one of the infrapopliteal vessels.

Arterial repair may be accomplished by end-to-end re-anastomosis using 7/0 interrupted sutures or by means of a vein graft. Optical magnification is helpful in performing the more distal repairs.

The infrapopliteal vessels are also at risk from extrinsic compression by fracture haematoma or compartmental oedema. This may lead to a Volkmann's contracture or loss of the limb even without an intrinsic arterial injury. Prompt fasciotomy and evacuation of haematoma is essential whenever this syndrome develops. The underlying fractures may then be managed by nailing or plating using the fasciotomy incisions for access.

VASCULAR INJURIES ASSOCIATED WITH FRACTURED PELVIS

These may put the patient's life at risk[29]. In some instances immediate exploration is necessary because a major vascular or visceral injury is suspected. If a retroperitoneal haematoma is then found this should be left undisturbed, and a postoperative arteriogram obtained (see below). However if the peritoneum is torn and there is active bleeding, some attempt at surgical control should be made. Manual compression at the pelvic brim and in the groin may achieve temporary control while direct dissection and clamping of the injured iliac vessels is achieved. If access to the external–internal iliac venous confluence is required this may be facilitated by division of the internal iliac artery. Vascular repair then follows standard lines. If bleeding is uncontrollable from deep within the pelvis it may be necessary to pack the pelvis and proceed to postoperative arteriography and embolization.

Those patients who exhibit signs of blood loss but do not require immediate laparotomy should undergo arteriography. Usually the source of bleeding is located in the internal iliac territory and there may be more than one vessel involved. Immediate embolization should be carried out and will successfully arrest the haemorrhage in most cases[30,31]. More than one embolization session may be required to achieve complete control. Continued bleeding in the presence of a negative arteriogram is an indication for venography since injury to the pelvic veins may be the primary cause.

ABDOMINAL AORTA

Blunt aortic injuries are very uncommon. Intimal rupture may cause embolization or lead to a dissection and acute occlusion. Other cases present later on with aortic stenosis or a traumatic aneurysm. Direct repair of an intimal disruption may be possible if the lesion is localized. More extensive injury will require prosthetic replacement.

Penetrating injuries usually present with circulatory collapse and urgent aortic control is required as part of the initial resuscitative effort. If the patient is moribund with no recordable blood pressure, a left seventh interspace thoracotomy should be rapidly performed in the emergency room and the descending thoracic aorta cross-clamped (Figure **7.16**). This incision is then subsequently extended into the abdomen to allow access to the injury site.

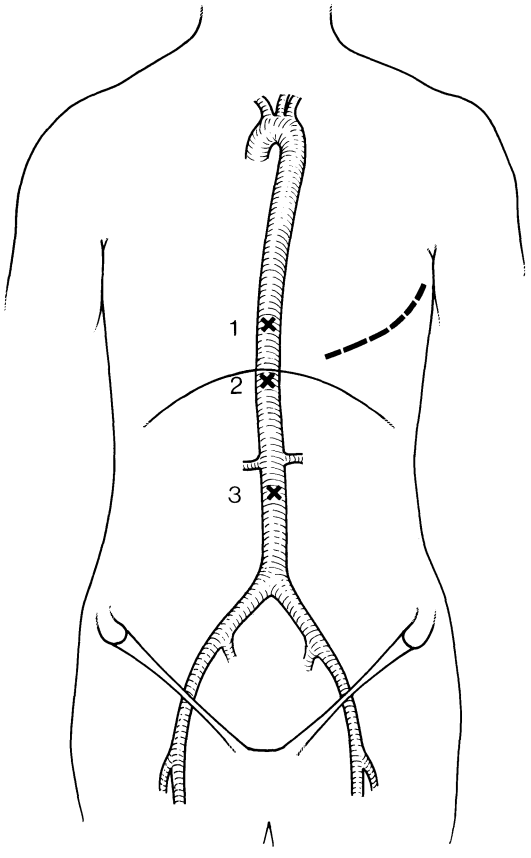


Figure 7.16 Clamp sites for controlling a penetrating aortic injury: **1.** descending thoracic aorta via seventh interspace thoracotomy. **2.** supracoeliac aorta. **3.** infrarenal aorta

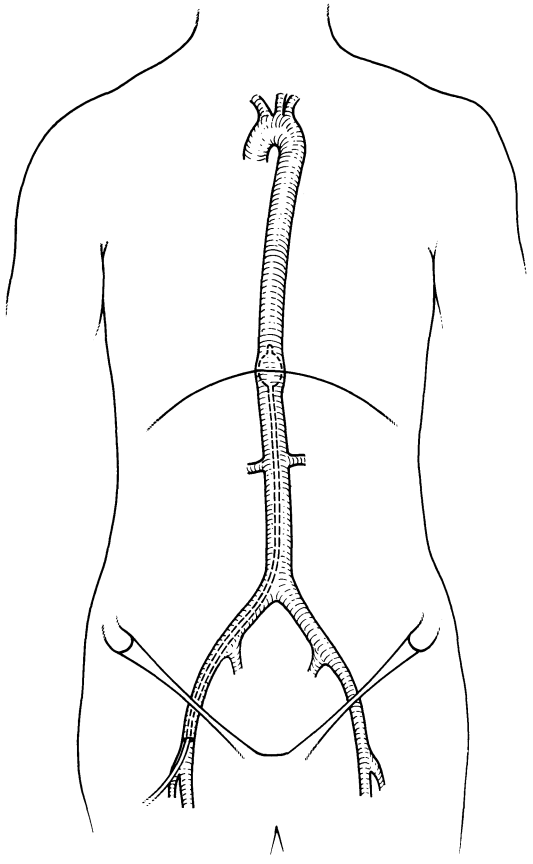


Figure 7.17 Alternative method of aortic control using a Fogarty catheter from the groin

An alternative method of control in the emergency room is to pass a large (8F) Fogarty catheter into the descending thoracic aorta either from the groin or the left arm (Figure **7.17**). Balloon inflation will then control the aorta while the patient is vigorously resuscitated en route for the operating room.

In less extreme cases a midline xiphopubic incision is made and initial control obtained by clamping the supracoeliac aorta (see Chapter 2). If the injury is subsequently found to be below the renal arteries this controlling clamp may then be transferred to an infrarenal position prior to repair. Where the aortic injury appears to be suprarenal, where the peritoneum of the left paracolic gutter should be incised and the spleen, pancreas, left colon and kidney reflected to the right (cf. thoracoabdominal aneurysm, Chapter 2). This exposes the entire abdominal aorta and its branches except for the right renal artery. If the patient is unstable, temporary manual compression at the hiatus may be instituted while the viscera are being reflected. The left crus is then sectioned adjacent to the aorta to allow room for clamp placement.

Once haemostasis is secured the aorta is repaired. A clean anterior laceration may be amenable to direct suture, though a ragged laceration will require trimming and insertion of a prosthetic patch (Figure **7.18**). If the operative field is contaminated (see below), autogenous material should be used for the repair. A perforating injury with a breach in the anterior and posterior wall may be managed by enlarging the anterior defect and repairing the posterior wall from within (Figure **7.19**). Anterior closure is then accomplished with a patch. A more extensive injury with loss of aortic tissue will require segmental prosthetic replacement.

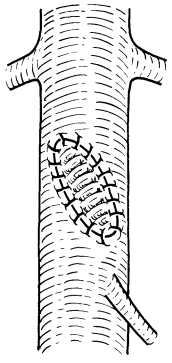


Figure 7.18 Anterior aortic laceration closed with a prosthetic patch

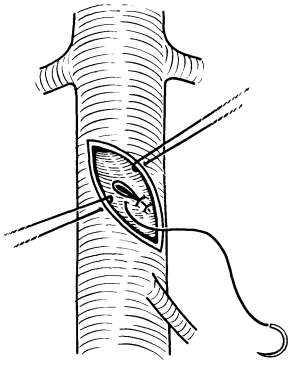


Figure 7.19 Through-and-through aortic perforating injury. Posterior defect repaired from within the aortic lumen

Associated injuries

Penetrating aortic injuries are frequently associated with damage to other major vessels[32]. These include the inferior vena cava, portal vein, superior mesenteric vein and vessels in the mesentery. Vena caval injuries are discussed below. Injuries to the portal vein or to the mesenteric veins can usually be controlled by compression and clamping. Subhilar injuries of the portal vein are more difficult and it may be necessary to insert balloon catheters into the right and left intrahepatic branches to achieve distal control[33]. The vein may then be repaired either by direct suture or by graft replacement using a venous compilation graft or a PTFE prosthesis.

Coexistent visceral injuries are also common at infrarenal aortic level (chiefly duodenum, small bowel and mesentery), and almost invariable whenever the suprarenal aorta is injured (stomach, pancreas, colon, liver). These injuries should be repaired at the outset to minimize contamination. The operative field is then irrigated with antibiotic solution and the repaired aorta wrapped in omentum. If the colon has been breached and there is peritoneal soiling, aortic repair may be inadvisable, particularly if it involves the use of prosthetics. Instead, the colon should be exteriorized and the aorta oversewn. The abdomen is then closed and an axillobifemoral bypass inserted after re-prepping and draping and a change of gown, gloves and instruments (see Chapter 14).

INFERIOR VENA CAVA AND HEPATIC VEINS

As with aortic injuries, these patients are often admitted in shock and endoluminal aortic control or emergency clamping of the thoracic aorta may prove necessary as part of the initial resuscitation. The abdomen is then opened via a xiphopubic approach and the site of the injury identified.

The subhepatic cava is exposed by reflecting the duodenum and hepatic flexure to the left (see Figure **7.21**). Bleeding may be difficult to control due to the large collateral flow from the lumbar veins and other tributaries. Local compression with sponge-holding forceps or conventional clamping may work, but often the best method is

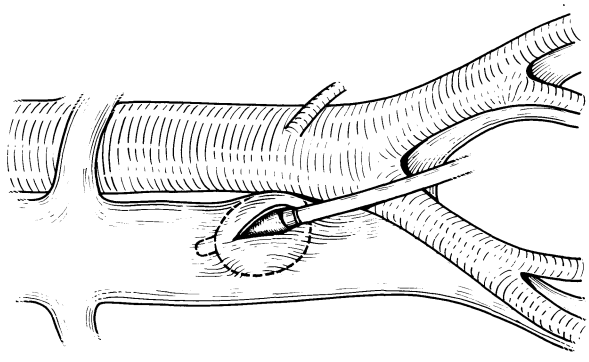


Figure 7.20 Control of vena caval injury using an endoluminal balloon catheter

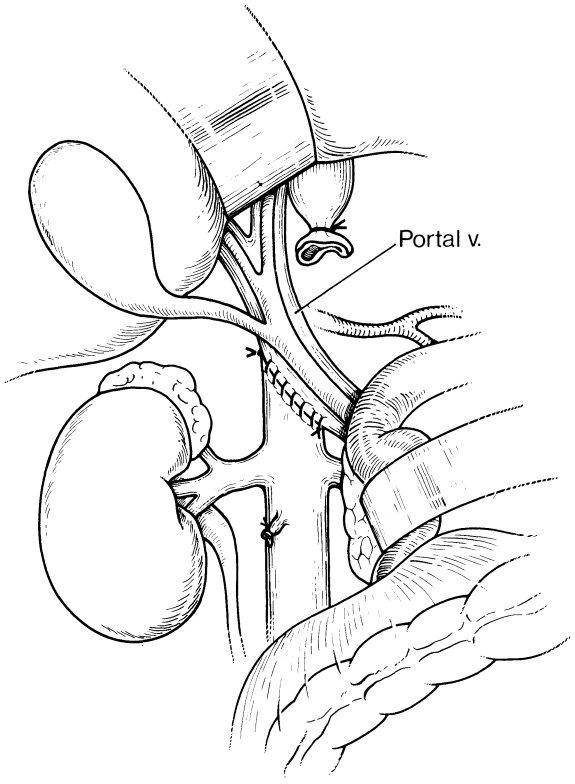


Figure 7.21 Extensive injury of the suprarenal vena cava managed by proximal caval ligation and reimplantation of the distal stump into the portal vein

to rapidly insert a balloon catheter into the defect and then apply traction (Figure **7.20**). If there is continuing difficulty, and particularly if the aorta is also injured, the right common iliac artery should be divided and the aorta rotated to the left. This will allow unrestricted access to the vena cava and iliac veins.

Limited injuries may be amenable to direct suture. Through-and-through injuries may be managed by repairing the posterior hole from within, rather than attempting to rotate the cava, which may result in damage to the lumbar veins. More extensive injuries will require segmental replacement using PTFE or a venous compilation (panel) graft. Access for direct repair may be difficult at suprarenal level and an alternative approach is to ligate the cava, and then reimplant the caudal stump end-to-side into the portal vein (Figure **7.21**).

Injury to the intrahepatic vena cava or hepatic veins can only be managed by completely isolating the liver from its blood supply. As soon as this injury is identified the xiphopubic laparotomy incision should be rapidly extended into the chest as a median sternotomy. The pericardium is opened and a tape passed around the inferior vena cava. A second tape is then passed around the cava immediately below the liver.

If the patient is profoundly shocked, it may be necessary to cross-clamp the cava at these sites

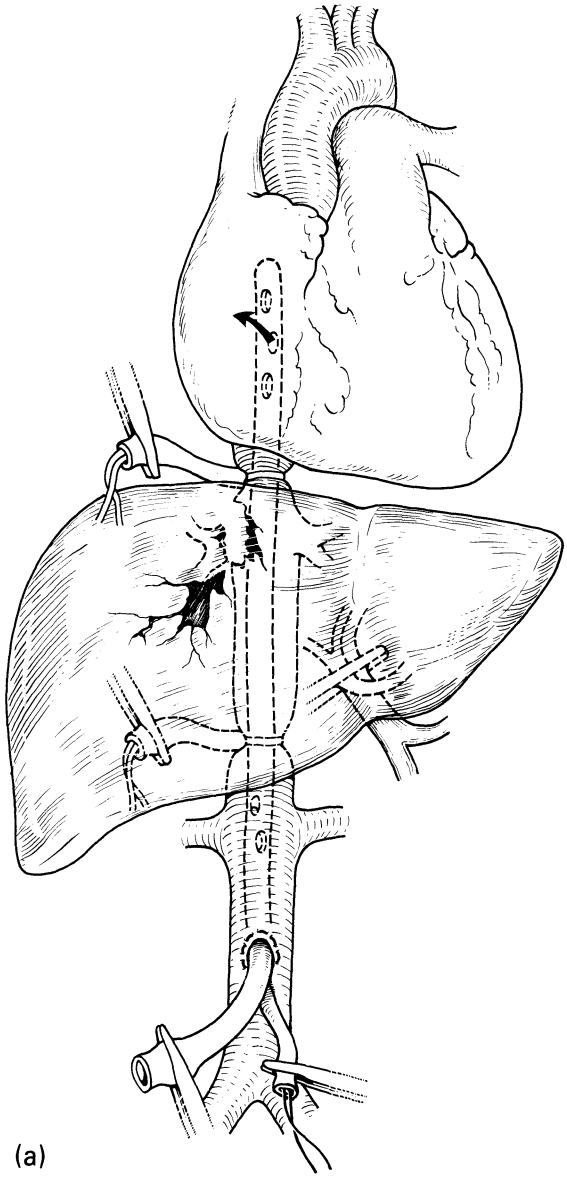


Figure 7.22(a, b) (a) Method of managing an intrahepatic caval or hepatic vein injury using an intraluminal shunt. In this case the shunt has been inserted antegradely from the infrarenal cava

and also the supracoeliac aorta while resuscitation is carried out. However, prolonged caval occlusion is inadvisable and an intraluminal shunt to maintain venous return should be inserted as soon as possible. A 30–34 F intercostal catheter is suitable and after side-holes have been cut at appropriate sites, this is inserted through a purse-string suture in the infrarenal vena cava (Figure **7.22a**). Retrograde insertion via the right atrium is an alternative approach[34,35], but there may be a slightly greater risk of air embolism with this method. The tapes are then tightened onto the shunt above and below the liver. Additional clamping of the portal triad completes the vascular isolation.

Often the right lobe of the liver is extensively fractured and splitting the liver down the interlobar fissure will expose the hepatic vein–caval confluence[34]. In other cases the liver may be appropriately rotated after dividing its peritoneal attachments. The caval–hepatic vein injury may then be repaired by lateral suture (Figure **7.22b**). Clamp control of the portal triad is released and the tapes slackened around the intraluminal shunt to test haemostasis. The shunt is then removed and the opening in the infrarenal cava closed over a partial occlusion clamp.

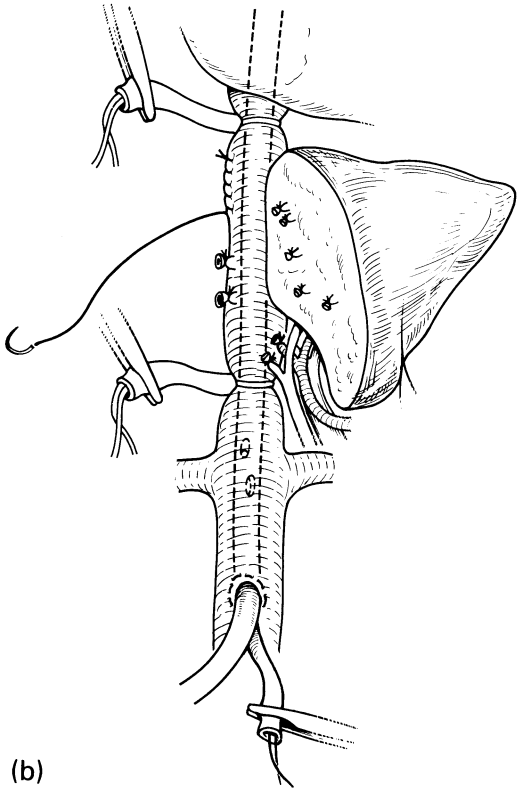


Figure 7.22 continued (b) The right lobe of liver has been removed and the laceration at the right hepatic vein–caval junction repaired by lateral suture

CAROTID ARTERY INJURIES

The great majority of carotid artery injuries result from penetrating trauma[36,37]. The common carotid artery is the most frequently affected segment. Most injuries are of the partial transection or perforating type. Occasionally an arteriovenous fistula may occur from simultaneous injury to the internal jugular vein. Blunt trauma may produce an intimal tear, dissection, occlusion or rarely, a false aneurysm[38].

In patients with a penetrating wound and a rapidly expanding haematoma in the neck, urgent intubation is necessary, followed by immediate exploration. Tracheostomy should be avoided if possible because of possible contamination of the wound and of the vascular repair. Stable patients with penetrating injuries at the base of the skull or at clavicular level and patients with suspected blunt carotid trauma should undergo arteriography.

Carotid repair should be attempted in virtually all cases even if a neurological deficit is present[36,37]. Many of the latter patients will fully recover after arterial repair, and the risk of haemorrhagic infarction seems more theoretical than real. Coma, providing it is of short duration, is also no longer regarded as a contraindication to surgery, though prognosis remains poorest in this group[39].

The technique of repair is usually straightforward and may involve lateral suture, patching, end-to-end re-anastomosis, or graft replacement. Occasionally a proximal internal carotid injury may be conveniently managed by transposition of the external carotid artery. An internal shunt is not necessary during carotid repair, though as with elective carotid surgery, systemic arterial pressure should be well maintained throughout the procedure.

Access to injuries near the base of the skull may be difficult. Division of the posterior digastric and detachment of the styloid process may be utilized. Other approaches include anterior dislocation of the jaw or section of the mandible (see Chapter 9). Where preoperative arteriography reveals a lesion extending up to the petrous bone or a long internal carotid artery occlusion, a preliminary extracranial–intracranial bypass may be advisable[40]. If direct repair then proves impossible, the internal carotid artery can be safely ligated.

Associated injuries are common with penetrating cervical trauma. The internal jugular vein may be repaired by lateral suture but can be ligated if necessary. Injury to the pharynx, oesophagus or trachea is more serious because of the risk of sepsis. Such injuries should be carefully identified and repaired and the wound closed with drainage. Arterial reconstruction should involve only autogenous techniques in these cases. Damage to the hypoglossal, phrenic or other nerves may also occur. Very occasionally a cleanly transected nerve may be primarily repaired, though in most cases this is not feasible.

SUBCLAVIAN ARTERY

Management of subclavian artery injuries is governed by the site of the injury and the clinical circumstances. Thus those patients who are admitted with a penetrating injury at the root of the neck and who are in profound shock or who have a rapidly expanding haematoma, external bleeding or bleeding into the chest, require urgent action. Intravenous lines are set up, intercostal drainage instituted for pneumothorax or haemothorax and the patient intubated. Explora-

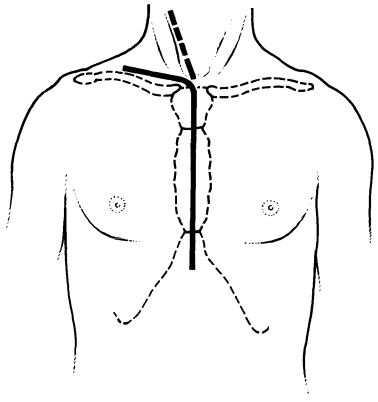


Figure 7.23 Median sternotomy with cervical extension offers speed and maximum flexibility in injuries at the root of the neck

tion is then undertaken to arrest the bleeding. Since the exact site of the injury is unknown, the surgical approach must be both rapid and sufficiently flexible to cover all eventualities. These aims are best fulfilled by a median sternotomy with appropriate extension into the neck (Figure **7.23**). Resection or division of the clavicle can be added to improve the exposure but are seldom necessary. This approach allows proximal control of all three supra-aortic trunks even in the midst of a haematoma. In addition, median sternotomy is the only approach which adequately displays the innominate veins and the jugular–subclavian venous confluence. Proximal control of the left subclavian artery may be less easy than the other trunks, but if the patient is placed on the table with the shoulders braced back and the table tilted towards the operator, access is usually satisfactory. Continued difficulty may need extension of the sternotomy into the left chest ('book' thoracotomy)[41].

Proximal control of the innominate or left subclavian artery origins will not arrest the bleeding because of the abundant collateral supply in this region, and local compression may be needed while the distal subclavian artery is exposed and clamped. The branches of the artery between the proximal and distal clamps are then successively controlled and the clamp positions readjusted in closer proximity to the injury. Any veins which impede access should be divided and ligated, apart from the subclavian and innominate veins which should if possible be preserved for repair.

The majority of patients with subclavian artery injuries are stable on admission or have mild hypotension which responds rapidly to intravenous fluids. These patients should undergo arteriography[42]. Advance knowledge of the site of the injury may greatly assist surgical planning. For example, if a proximal left subclavian artery injury is identified, an anterolateral thoracotomy may be chosen as this gives optimum exposure of this particular segment[43,44]. Equally, the site of the injury may be such that repair can be accomplished by a cervical approach alone without the need to open the chest[44].

Arterial repair

The principle objective in repairing proximal subclavian artery injuries is preservation of flow to the arm and to the vertebral artery. Several techniques are available. A limited injury may be managed by trimming and patch angioplasty (Figure **7.24a, b**). An injury involving loss of a greater part

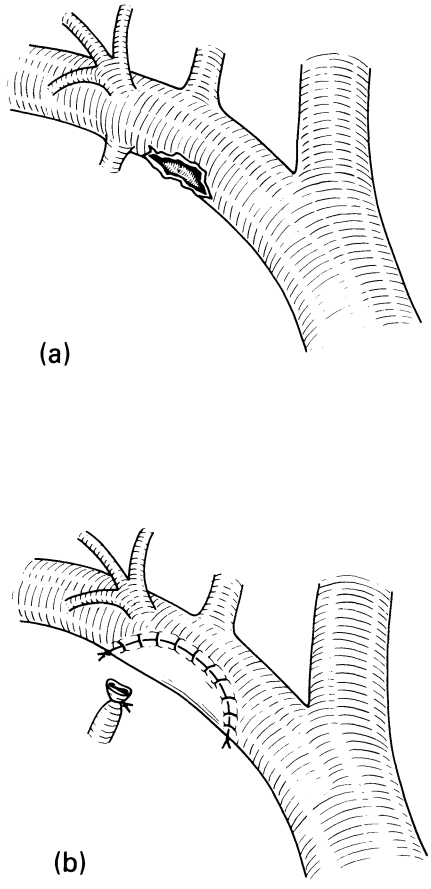


Figure 7.24(a, b) (a) Proximal subclavian artery injury of limited extent. (b) The injury site is trimmed and the defect closed with a vein patch

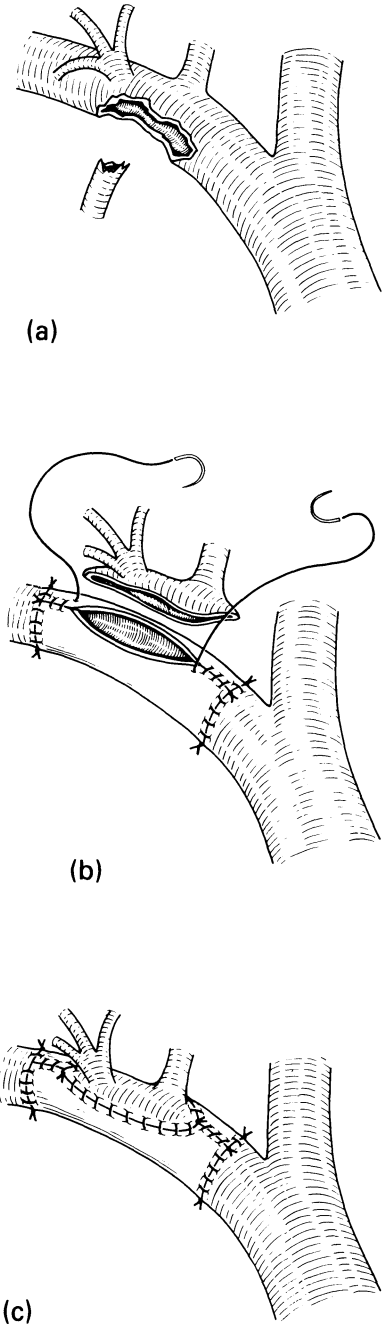


Figure 7.25(a–c) (a) A more extensive proximal subclavian artery injury. (b) The damaged segment is replaced by a venous compilation graft. (c) The vertebral and thyrocervical branches are reimplanted into the graft using a button technique

of the wall may be managed by graft replacement and reimplantation of the vertebral–thyrocervical trunk as a button (Figure **7.25a–c**). Alternatively, in order to avoid the suturing difficulties created by the subclavian branches, the traumatized zone may be excluded and the vertebral artery reimplanted into the adjacent common carotid artery. A carotid–distal subclavian or carotid–axillary bypass is then inserted to revascularize the arm (Figure **7.26**).

If the injury involves the distal subclavian artery, the classical approach is via a supraclavicular incision extended into the deltopectoral groove with division, or preferably, resection of the clavicle (see Chapter 11). The damaged segment is then replaced by a graft (Figure **7.27a, b**). This approach is particularly appropriate when the clavicle is already fractured. However, an alternative solution which preserves the shoulder girdle involves excluding the injured arterial segment, followed by insertion of a carotid–axillary bypass (or proximal subclavian–axillary bypass if anatomical conditions permit) (Figure **7.28**).

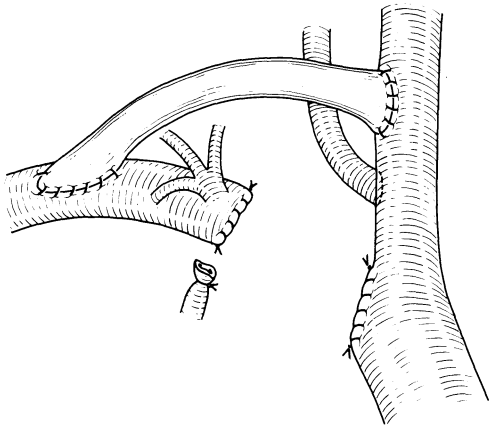


Figure 7.26 Alternative method of managing proximal subclavian artery injuries. The damaged segment is excluded and the vertebral artery reimplanted into the common carotid artery. The arm is revascularized with a carotid–distal subclavian graft

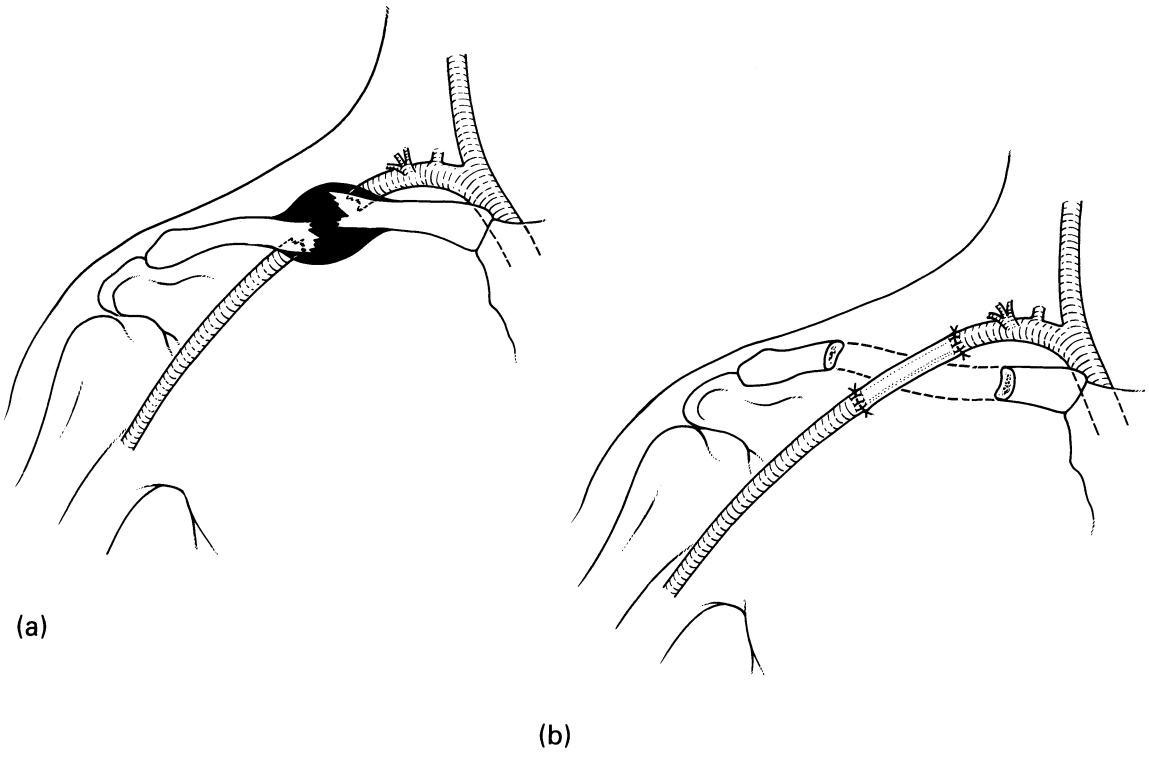


Figure 7.27(a, b) (a) Distal subclavian artery injury associated with a fractured clavicle. (b) Clavicular resection and direct graft (PTFE) replacement

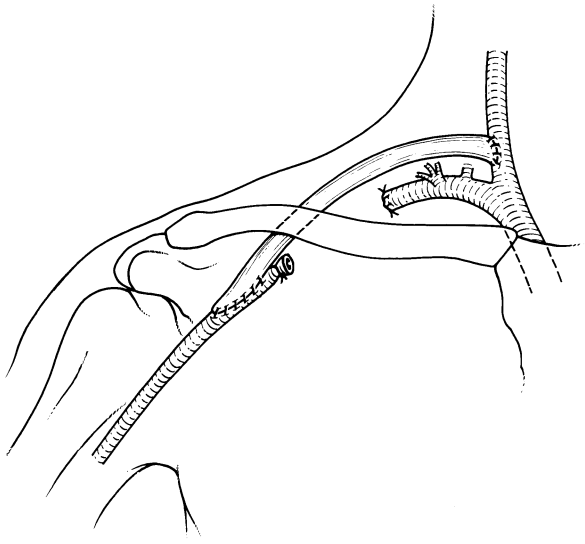


Figure 7.28 Alternative method of managing a distal subclavian artery injury with preservation of the clavicle. The damaged segment is excluded and a carotid–axillary graft inserted

Associated injuries

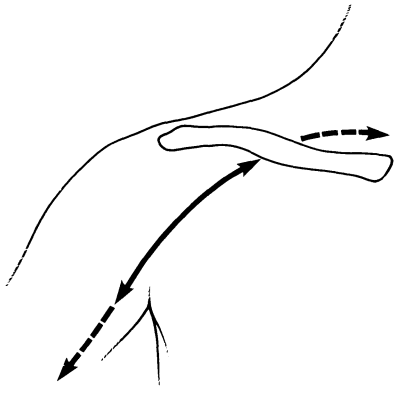
Penetrating cervicomediastinal trauma may involve structures other than the major arteries. Venous injuries are common and in most instances can be treated by ligation. However, the subclavian and innominate veins should if possible be repaired. A careful search should always be made for additional injury to the lung, pulmonary hilum, oesophagus, trachea or thoracic duct. A breach of the oesophagus or trachea is particularly dangerous because of the risk of mediastinitis and possible secondary haemorrhage. As previously described, autogenous methods of vascular reconstruction should be utilized in these circumstances, particularly exclusion with bypass at a distance.

Blunt subclavian artery trauma is unusual but may arise from one of two mechanisms each of which may have implications for adjacent structures. In the first instance the artery may be impaled by a fracture of the clavicle or first rib. Arterial repair should then be accompanied by resection of the adjoining bony fragments. The second mechanism involves a traction injury and occurs particularly in motorcyclists[45]. The artery is disrupted sometimes at both subclavian and axillary levels and there is a significant incidence of associated damage to the brachial plexus. The latter governs the prognosis for the limb. Primary nerve repair is seldom feasible but it may be possible to tack the nerve ends to adjacent soft tissues in order to reduce the length of any subsequent nerve graft. In severe traction injuries, particularly those involving the cervical nerve roots, little can be done and vascular repair may be inappropriate.

AXILLARY ARTERY

Injuries to the axillary artery do not usually pose particular problems. However, this is one of the sites where partial transection injuries may be easily missed if proximity wounds are not assessed arteriographically or explored. These patients may then present several weeks later with brachial plexus compression from an expanding false aneurysm[25,46]. A traumatic occlusion may also be overlooked in the absence of arteriography since distal pulses are often maintained as a result of the efficient collateral circulation around the shoulder.

Access for axillary artery repair is best achieved by a deltopectoral approach with section of pectoralis minor (Figure **7.29a**). If necessary the incision can be extended distally with division of the tendon of pectoralis major, thereby exposing the axillobrachial junction (Figure **7.29b**). Similarly, if proximal access is limited, the incision can be taken back into the supraclavicular fossa, with or without clavicular resection.



(a)

Figure 7.29(a, b) Exposure of the axillary artery: (a) incision in the deltopectoral groove. This may be extended proximally or distally as necessary

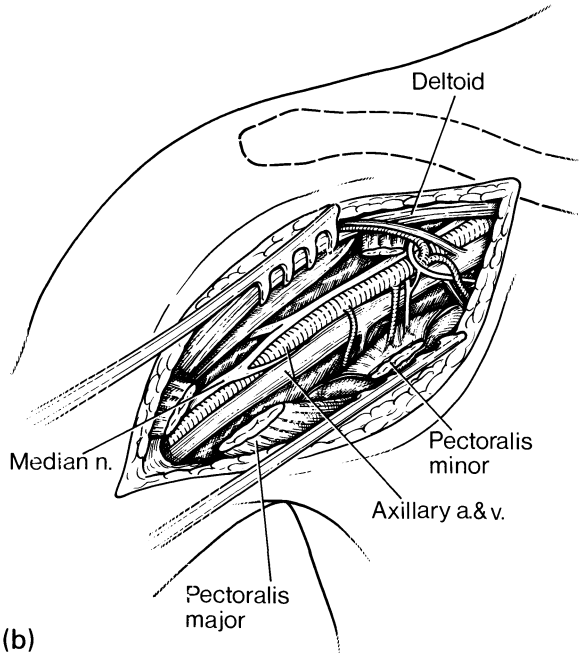


Figure 7.29 continued (b) pectoralis minor divided and the neurovascular bundle exposed. In this case the pectoralis major tendon has also been divided to allow access to the axillobrachial junction

Arterial repair may be accomplished by the methods previously described. Exclusion of the traumatized zone and end-to-side venous bypass will maximally preserve the collateral vessels in this region and is usually preferred if direct repair is not feasible. The axillary vein and brachial plexus are in close proximity and coexistent injuries to these structures are frequent. The former may be managed by lateral repair or ligation. Brachial plexus injuries may be repaired by secondary grafting or rarely, by primary suture. Those patients who present late with neurological compression from a false aneurysm may be managed by laying open the aneurysmal sac and end-to-side bypass grafting. A careful neurolysis should also be carried out, though even when the plexus has been thoroughly decompressed, neurological function may not fully return[25,46].

BRACHIAL ARTERY

This is one of the most commonly injured vessels in civilian practice. Injury may occur in association with fractures of the shaft of the humerus, supracondylar fractures and dislocations of the elbow or arise from a variety of penetrating agents. Cardiac catheterization also continues to account for a number of cases[47] though with the change from transbrachial to transfemoral route these are now less common.

The brachial artery may be exposed by an incision along the medial border of biceps (Figure **7.30a, b**). If necessary this can be extended into the antecubital fossa for access to the radial and ulnar arteries.

An associated fracture of the humeral shaft may be nailed or plated, unless it is comminuted or associated with a contaminated wound, in which

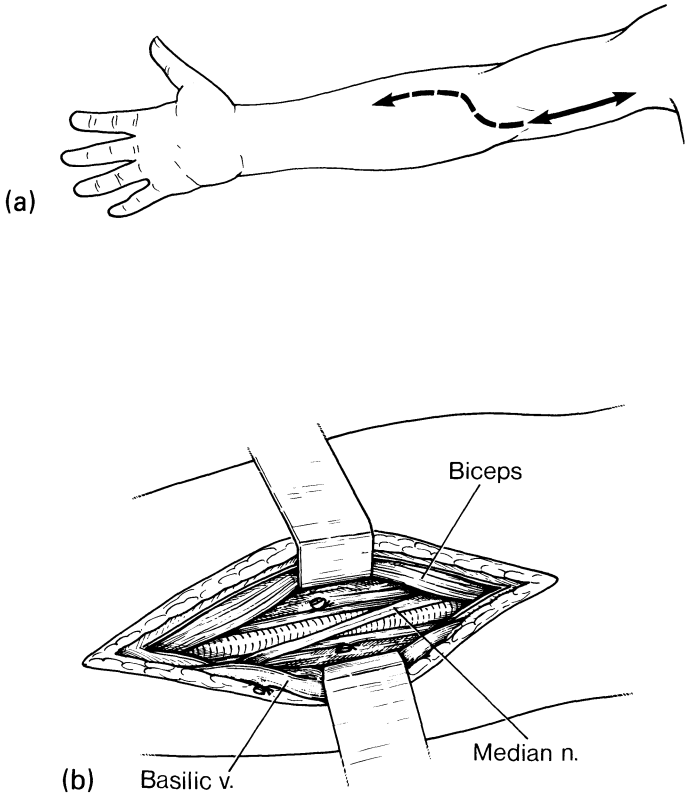


Figure 7.30(a, b) Exposure of the brachial artery: (a) incision along the medial border of biceps. This may be extended into the antecubital fossa if necessary. (b) brachial artery displayed with median nerve crossing anteriorly

case external fixation is employed. Supracondylar fractures in children may be conveniently stabilized using Kirschner wires inserted obliquely from either condyle. If there has been delay between injury and repair a temporary arterial shunt may be useful while the skeleton is stabilized and other soft tissue injuries corrected[11].

The most satisfactory method of vascular repair is resection with either end-to-end re-anastomosis or vein graft replacement. Lateral suture is not advisable with most injuries at this level apart from post-catheter injuries seen within hours of inception[47]. Soft tissue loss may be a problem, particularly in displaced fracture-dislocations, and a myocutaneous flap may be necessary to cover the arterial repair.

RADIAL AND ULNAR ARTERIES

Damage to either vessel alone may be treated by ligation if there is a fully intact palmar arch and viability of the hand is not in doubt. If this is not the case, or if both vessels have been injured, repair should be undertaken using interrupted 7/0 or 8/0 sutures and optical magnification.

FASCIOTOMY IN THE UPPER LIMB

As in the lower limb, fasciotomy should be undertaken whenever there is oedema or haematoma within the muscle compartments or post-ischaemic muscle swelling. Warning signs of a compartment syndrome include severe pain, particularly on wrist or finger extension, distal parasthesiae and paralysis. Prompt decompression is then essential to avoid a Volkmann's contracture. The flexor compartment may be decompressed by a longitudinal anterior incision extending into the palm if necessary (Figure **7.31**). An incision immediately adjacent and parallel with the ulnar crest may be used to decompress the extensor compartment.

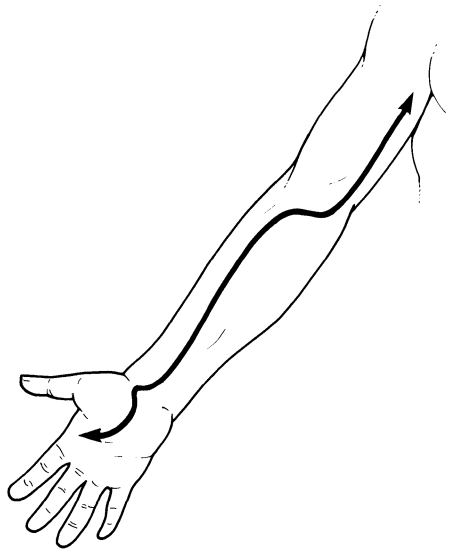


Figure 7.31 Anterior fasciotomy of the upper limb with extension into the hand

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8

Arteriovenous Fistulae

Arteriovenous fistulae are relatively uncommon vascular lesions. Two major varieties of arteriovenous fistula may be recognised, congenital and acquired. Congenital fistulae represent an arrest of the normal differentiation process with persistence of primitive plexiform connections between the arterial and venous anlage. In contrast, acquired fistulae usually consist of a single communication only between artery and vein. The most common aetiological factor in acquired arteriovenous fistulae is trauma, particularly of the penetrating type, though a minority of cases occur spontaneously as a result of prior disease of the artery wall.

ACQUIRED ARTERIOVENOUS FISTULAE

These lesions usually result from penetrating injuries caused by gunshot or knife wounds, metal or glass fragments etc. Occasionally blunt trauma may be responsible as, for example, when the vessels in the calf are lacerated by a fractured tibia or fibula, or when a carotid–cavernous fistula complicates a fractured skull. A small but important group of traumatic fistulae follow surgical or investigational procedures. These include Seldinger catheterization, Fogarty embolectomy, percutaneous angioplasty, renal or hepatic needle biopsy and orthopaedic operations such as intervertebral disc excision, menisectomy or hip replacement. Mass ligation of a vascular pedicle may also occasionally be responsible, e.g. following nephrectomy, thyroidectomy or splenectomy.

Spontaneous arteriovenous fistulae arise as a result of a local disease process which weakens the artery wall. Usually this is in the form of an atherosclerotic or mycotic aneurysm which ruptures into an adjacent vein (see aortocaval fistula Chapter 2). Rarer causes include neurofibromatosis, malignant infiltration etc.

A fistula may be detected immediately after injury but often a bruit or thrill is not apparent for several hours or days due to temporary occlusion of the communication by clot[1]. In some cases, a traumatic fistula may not come to clinical attention until several months or even years after the initial injury. Apart from a local bruit and thrill, other signs of a fistula include prominent superficial veins, which may or may not pulsate, a bluish discoloration of the skin, local warmth and distal oedema. Venous distension may be marked and reach aneurysmal proportions if there is a large arteriovenous communication. A false aneurysm may be additionally present in over half of the cases and is manifest by a pulsatile mass at the injury site. Distal ischaemia may occur from shunting of blood away from the extremity but is not common.

Complications of untreated fistulae may include chronic venous hypertension, cardiac dilatation or failure, bacterial endocarditis, endarteritis and increased limb growth if the fistula is

acquired before the epiphyses close. In addition, longstanding fistulae may cause progressive dilatation and eventual aneurysmal degeneration in the proximal arterial tree. Once established, these structural changes may advance despite surgical closure of the fistula[2,3].

INVESTIGATION

Arteriographic assessment or exploration of all penetrating wounds in proximity to known vessels will allow arteriovenous fistulae to be detected at their inception when treatment is relatively easy. In established cases, arteriography should be undertaken to define the site of the fistula, to exclude additional distal lesions, and to identify false aneurysm formation or degenerative changes in the proximal arterial tree. Rapid-change cassettes or preferably cineangiography are required in studying fistulae. Even so, fistula location may be difficult due to the rapid flow and selective catheterization with multiple views may be needed. Doppler assessment may also assist in preoperative mapping[4].

OPERATIVE MANAGEMENT

Although spontaneous fistula closure has been reported, it is rare[5]. Essentially all traumatic fistulae should be corrected, since even small ones may cause secondary pathological changes in the adjacent vessels in time. Ideally, repair should be undertaken at the time of injury, since dissection is still relatively easy and vascular repair can often be achieved by simple suture. However, if the patient does not present until several days after the injury, it is usually advisable to defer repair for 3 weeks or so until the soft tissues have healed and the traumatic reaction has settled.

Chronic traumatic fistulae may pose difficulties in exposure and in vascular control. There may be extensive post-traumatic fibrosis with loss of the normal cleavage planes, and nerves or other adjacent structures may be at risk. This difficulty may be compounded by the large number of dilated fragile veins in the vicinity of fistula. One solution is to expose and clamp the main vessels at a distance from the fistula and then gradually work towards the latter. However, clamping at a distance will not provide a bloodless field because of the marked collateral flow, and a pneumatic tourniquet may therefore be considered in some limb fistulae. If a tourniquet is used it should be released after the fistula has been closed, to confirm that no further arteriovenous communications remain.

Extensive fistulae at sites such as the groin, pelvis or root of neck will require alternative measures to diminish the risk of intraoperative haemorrhage. In the past, controlled hypotension or even total circulatory arrest with cardiopulmonary bypass have been employed[6,7]. Nowadays, preoperative embolization of nonessential branches may be utilized whenever surgery is likely to be haemorrhagic. In addition, balloon occlusion catheters can be placed in the fistula itself prior to surgery and may greatly facilitate surgical access[8]. If these techniques are coupled with endoarterial fistula closure, the risk of intraoperative venous haemorrhage may be largely avoided.

The type of vascular repair utilized depends on the configuration (Figure **8.1a–d**) and site of the fistula, the degree of secondary change in the adjacent vessels and the ease of surgical access. As a general principle both the artery and vein should be repaired in major limb fistulae, though with more peripheral lesions the venous component may be managed by ligation.

Arteriovenous fistula with intermediate canal

Rarely the artery and vein are connected by a narrow channel as a result of organization of an intermediate haematoma (Figure **8.1a**). Management is relatively simple and involves clamping the artery and vein either side of the fistula. The latter is then divided and the ends ligated or oversewn.

Latero-lateral fistula

More frequently the artery and vein communicate directly in side-to-side manner. An example of a latero-lateral fistula involving major limb vessels is shown in Figure **8.2a–e**. In this case a fistula has occurred between the superficial femoral artery

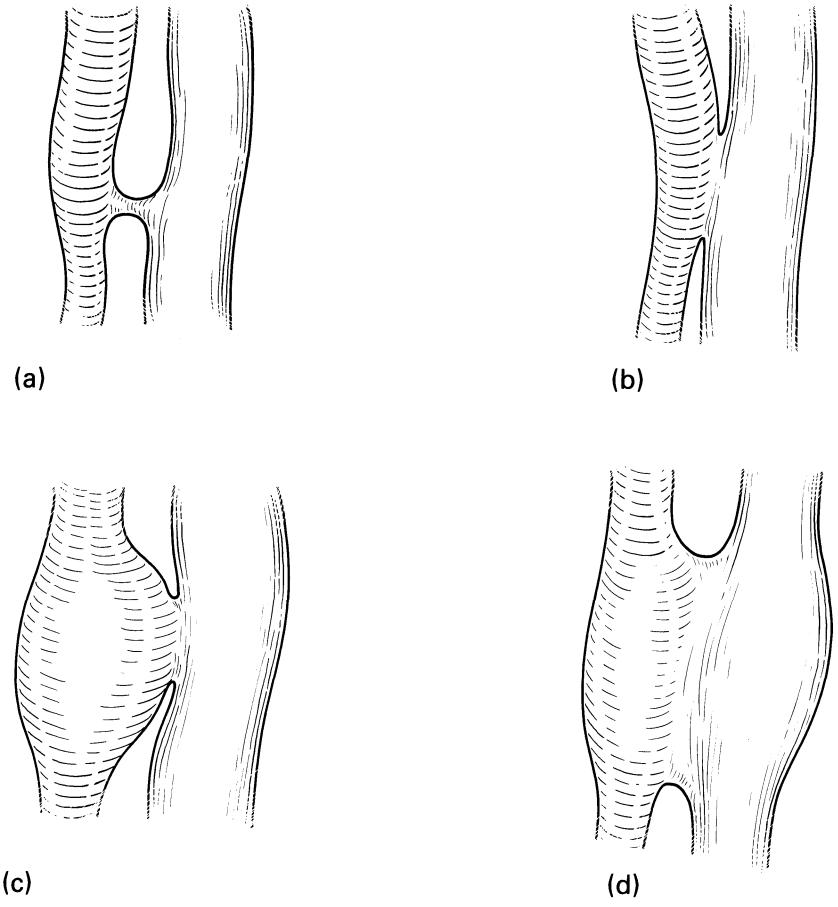


Figure 8.1(a–d) Morphological varieties of traumatic arteriovenous fistula: (a) fistula with intermediate canal. (b) latero-lateral fistula. (c) false aneurysm with AV fistula. (d) AV aneurysm

and vein as a result of a penetrating injury. Operation may be conducted under pneumatic tourniquet to avoid troublesome bleeding from the many dilated veins in the superficial tissues. A standard midthigh incision is utilized with reflection of sartorius (Figure **8.2a**). The arterial and venous axes are then mobilized and the fistula identified (Figure **8.2b**). If dissection is difficult, only the anterior aspects of the fistula need be exposed. The artery and vein are then clamped either side and the fistula sectioned with a scalpel. This should be done in such a way that the vein is maximally preserved, thereby avoiding the uncertainty of a venous reconstruction.

Occasionally both vessels are sufficiently well preserved to allow direct closure. However, in most cases the edges of the artery or vein must be trimmed and a patch inserted or a resection-anastomosis carried out (Figure **8.2c, d**). If the artery wall requires more extensive resection due to degenerative changes, graft replacement will be necessary (Figure **8.2e**). Following reconstruction, viable soft tissue should be interposed between the two vessels or a Dacron wrap used to separate the suture lines.

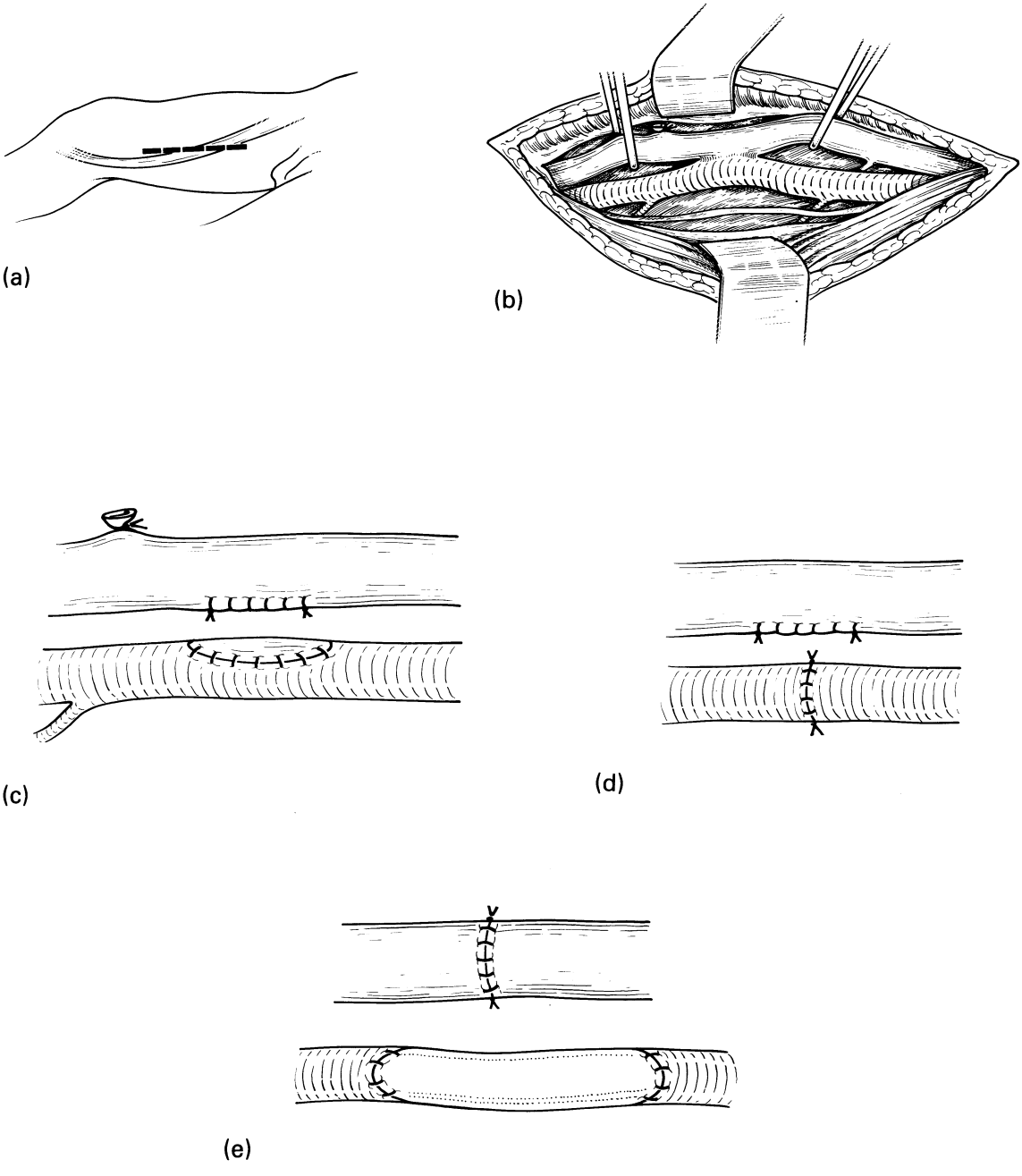


Figure 8.2(a–e) Latero–lateral fistula involving the superficial femoral vessels: (a, b) exposure of fistula site. (c) direct repair of the vein and patch angioplasty of the artery. (d) resection-anastomosis. (e) graft interposition (PTFE)

In some cases the latero-lateral communication is between a major artery and a branch vein. An example is a fistula between the external iliac artery and the deep circumflex iliac vein following Seldinger catheter injury (Figure **8.3a**). In this circumstance, rather than attempting to disconnect the two vessels externally, it is simpler to perform a transvenous repair of the fistula with sacrifice of the branch vein (Figure **8.3b**). This requires prior clamping of the arterial axis and of the vein branch either side of the fistula. Once the involved segment of vein has been isolated it is opened and the fistula oversewn. The vein wall is then used to provide a second layer closure (Figure **8.3c**).

The reverse arrangement applies where the fistula is between a major vein and a branch artery, e.g. popliteal vein–sural artery fistula following a penetrating injury at the knee (Figure **8.4a**). The fistula is isolated and after opening the branch artery a transarterial closure is accomplished (Figure **8.4b**).

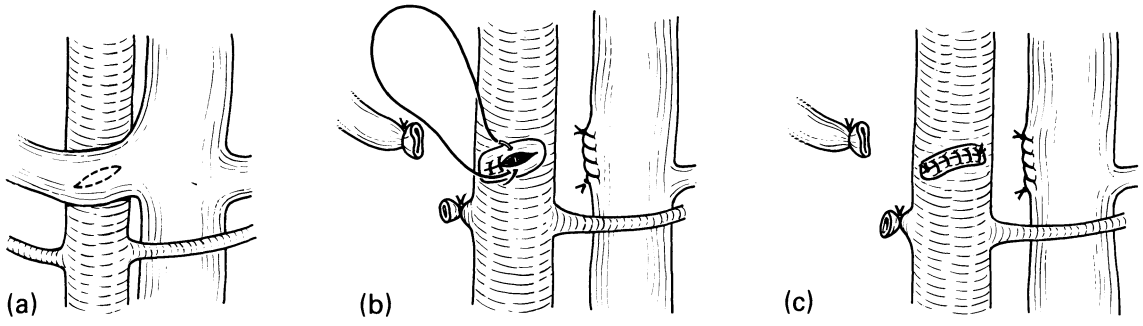


Figure 8.3(a–c) (a) Latero-lateral fistula between a major artery and a branch vein; e.g. external iliac artery–deep circumflex iliac vein. (b) The branch vein is isolated and opened to expose the fistula. (c) Endovenous closure of fistula using the vein wall to buttress the closure

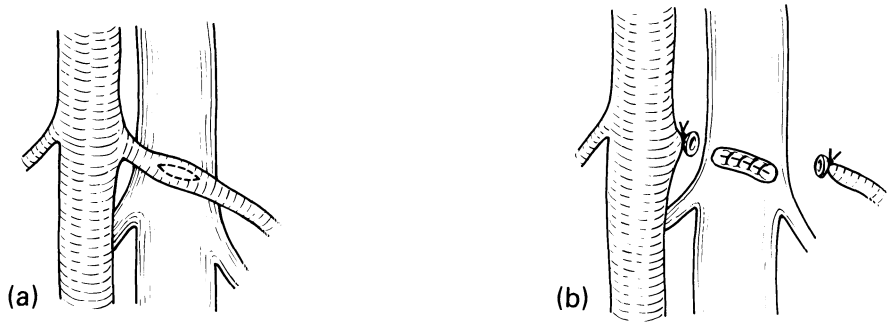


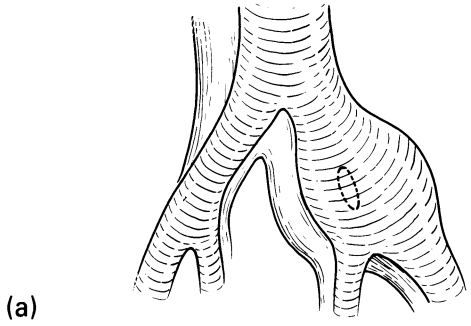
Figure 8.4(a, b) (a) Latero-lateral fistula between a major vein and a branch artery, e.g. popliteal vein–sural artery. (b) Branch artery sectioned and opened to allow endoarterial closure of the fistula

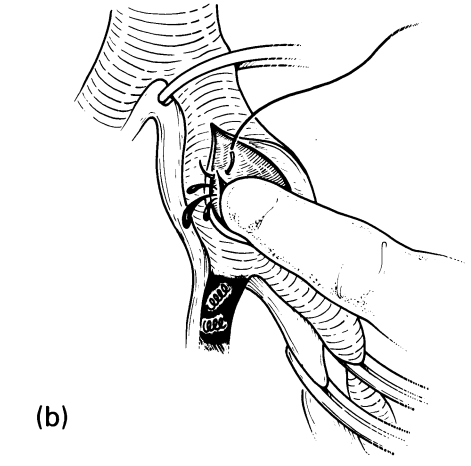
Arteriovenous fistula with false aneurysm

In some patients a false aneurysm is present in association with an arteriovenous fistula. Usually, the false sac is of modest size, but occasionally may dominate the clinical picture[9]. The most satisfactory method of managing these cases is to close the fistula from within the aneurysmal sac (see aortocaval fistula, Chapter 2). This avoids the need to dissect the venous axis and at the same time preserves venous flow. After the fistula has been closed, the aneurysmal segment is replaced with a graft.

An example of this type of injury is a common iliac fistula secondary to intervertebral disc surgery (Figure **8.5a**). In order to reduce operative dissection and the consequent risk of venous injury, it may be helpful to embolize the internal iliac artery on the affected side 24–48 hours preoperatively. Steel coils or a detachable balloon may be suitable for this purpose (Figure **8.5b**). At operation, only the common iliac and external iliac arteries therefore need to be controlled. The external iliac vein may also be clamped though the common and internal iliac veins are best left undisturbed since posterior dissection in the trauma zone may risk catastrophic venous haemorrhage.

The aneurysmal sac is then entered and the venous back-bleeding controlled digitally while the fistula opening is oversewn (Figure **8.5b**). Additional sutures may be placed at the internal iliac artery origin to reinforce the effect of the embolization. Arterial continuity is then restored by means of a graft between the common and external iliac arteries (Figure **8.5c**).





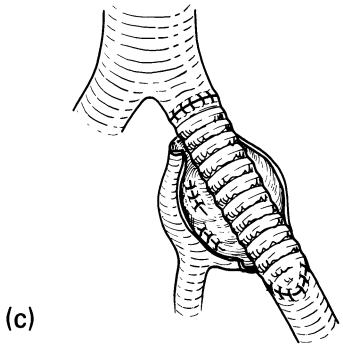


Figure 8.5(a–c) Common iliac AV fistula with false aneurysm secondary to intervertebral disc surgery: (a) lesion. (b) preliminary internal iliac artery embolization with steel coils. At operation the false aneurysmal sac has been opened after clamping the common and external iliac arteries and the external iliac vein. The fistula is then closed under digital control. (c) arterial continuity restored using a Dacron graft

Arteriovenous aneurysm

This variety of arteriovenous fistula arises from loss of a substantial part of the artery and vein wall. A communal sac thus develops into which the afferent and efferent orifices of the main artery and vein open, together with any additional collateral vessels (Figure **8.6a, b**). Restoration of vascular continuity usually requires graft replacement of both the artery and vein.

The arterial and venous axes should be initially controlled proximal and distal to the aneurysm. The latter is then opened and any collateral vessels oversewn from within the sac. Several options are then available for reconstruction. The artery and vein may be sectioned at each pole of the aneurysm and a graft inserted in end-to-end fashion (Figure **8.6c**). However, posterior dissection may risk damage to adjacent structures, such as the medial popliteal nerve, and an inlay technique or bypass at a distance with exclusion of the aneurysm may therefore be preferable (Figure **8.6d, e**). The latter method in addition provides optimum conditions for anastomosis and, in the case of a saphenous vein graft, will avoid the problem of mismatch in size between graft and recipient artery or vein.

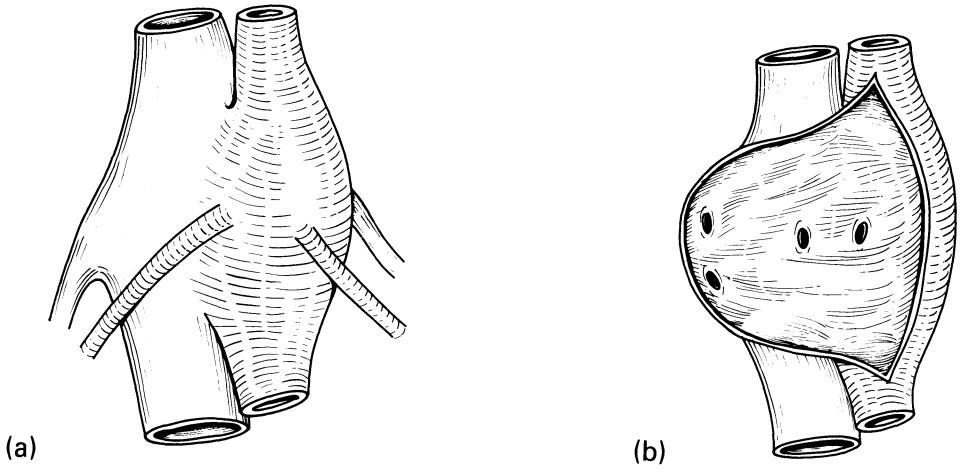


Figure 8.6(a–e) Popliteal arteriovenous aneurysm: (a) lesion. (b) sac opened prior to sewing off the afferent, efferent and collateral orifices

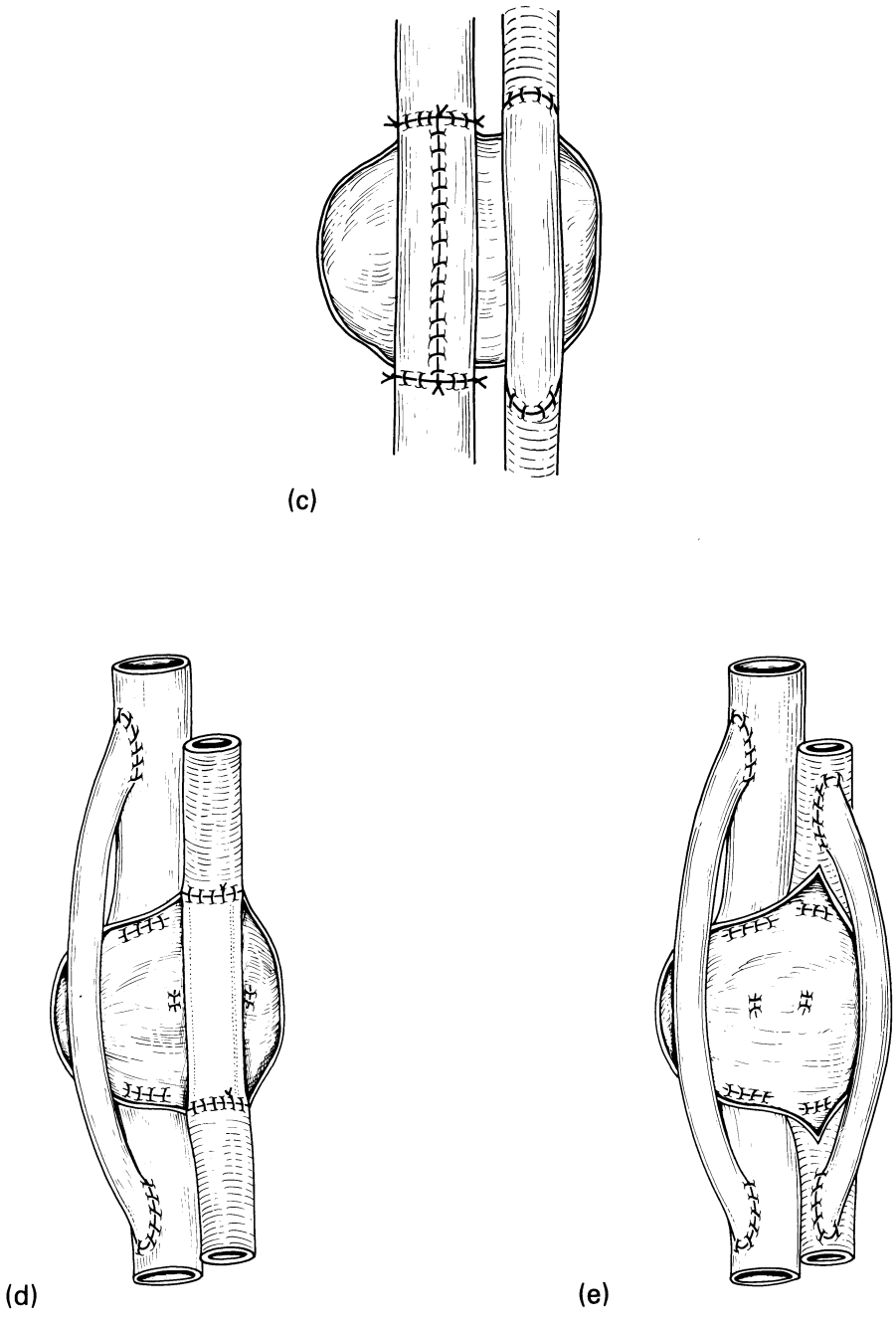
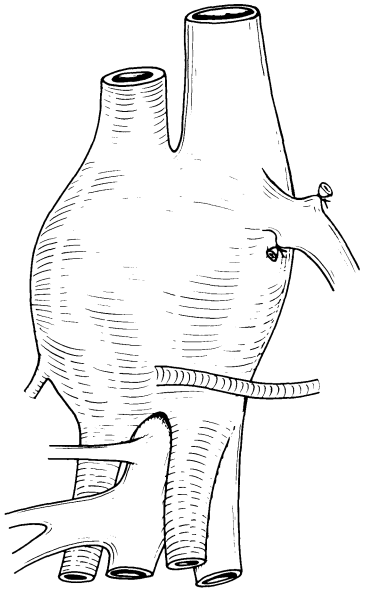
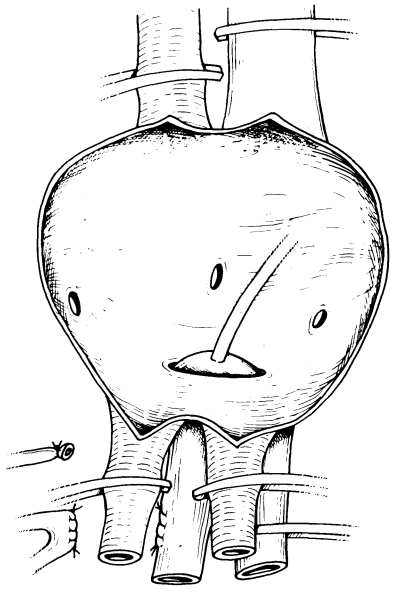


Figure 8.6 continued (c) reconstruction by interposition grafting. (d) artery repaired by inlay graft (PTFE). Venous continuity restored by end-to-side venous bypass. (e) aneurysm excluded with bypass at a distance

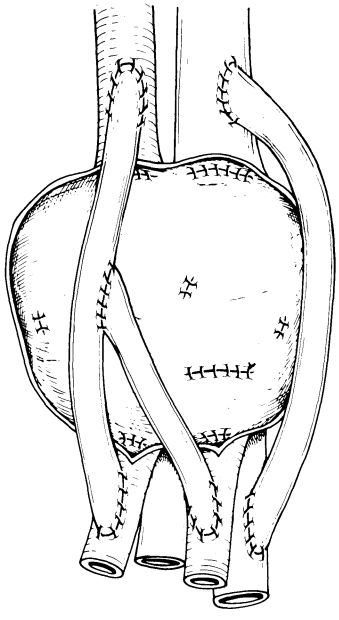


(a)

Additional difficulties may be encountered with arteriovenous aneurysms in the groin or those involving the subclavian vessels. In the former case, there is a need to initially control and then revascularize a major arterial and venous bifurcation (Figure **8.7a**). The common, superficial and if possible, the deep femoral arteries are isolated and clamped. The accompanying veins may also be clamped, though usually the deep femoral vein is more safely managed by endoluminal balloon occlusion once the sac has been opened (Figure **8.7b**). All the vessels opening into the sac are then oversewn and the arterial axis reconstructed with a branching PTFE or venous bypass graft (Figure **8.7c**). A vein bypass is then placed between the superficial and common femoral veins.



(b)



(c)

Figure 8.7(a–c) (a) Common femoral AV aneurysm. (b) The sac is opened after partial clamp control. The deep femoral vein is then controlled by an endoluminal catheter. (c) The aneurysm has been excluded and the arterial axis reconstructed by means of a branching venous bypass graft. An additional bypass has been placed between the superficial and common femoral veins

Arteriovenous fistulae involving the subclavian vessels may be a problem in terms of access and the multiplicity of the arterial and venous collaterals. Clamping the main arterial and venous axis at a distance will not provide a dry field and preoperative embolization of the subclavian artery branches (excluding the vertebral artery) should be used to reduce intraoperative haemorrhage. As previously described, temporary balloon occlusion of the fistula itself may also aid operative dissection[8].

Wide operative exposure is advisable and in the case of proximal subclavian fistulae, this may be best achieved by a median sternotomy, with or without medial resection of the clavicle. The external jugular and vertebral veins and any other venous branches are ligated and divided as the fistula is approached. After heparinization, clamps are applied to the arterial and venous axes either side of the fistula and to the vertebral artery. The sac is then opened on the arterial side and back-bleeding controlled digitally while the afferent, efferent and collateral orifices are oversewn. The vertebral artery may then be reimplanted into the adjacent common carotid artery and the upper limb revascularized by means of a carotid–distal subclavian (or axillary) bypass graft (see Figure **7.26**). If possible venous continuity should also be restored. An end-to-side saphenous vein bypass graft is probably the best method of reconstruction.

EMBOLIZATION

Although the treatment of traumatic fistulae is essentially surgical, embolization may be used as an alternative to surgery in some circumstances. This particularly applies to patients with local or general operative risk factors[10] and to fistulae which are located distally in the limb[1] or at surgically inaccessible or difficult sites. Examples of the latter include fistulae in the internal iliac territory[11], distal profunda[12] and visceral organs, particularly the kidney and liver[13]. In most instances embolization will result in occlusion of the involved artery. However in special instances such as carotid–cavernous or vertebral fistulae, it may be possible to close the arteriovenous communication with preservation of arterial flow[14,15].

A variety of agents are available for embolizing traumatic fistulae. Clearly the material chosen should be large enough to occlude the fistula without embolizing the lung. Gelfoam sponge has been widely used for smaller fistulae (2–3 mm), while larger fistulae may be closed with steel coils or detachable balloons, the latter being particularly suitable because of their flow-directed properties[16].

CONGENITAL ARTERIOVENOUS FISTULAE

Congenital arteriovenous fistulae result from arrest or misdirection of normal vascular differentiation in embryonic life. The vascular system begins as a meshwork of endothelial lakes in the primitive mesenchyme (endothelial stage). These then coalesce into a network of communicating capillary channels (retiform stage) from which the mature arteries and veins ultimately develop. Lack of differentiation at the endothelial stage may result in haemangiomas of the capillary or cavernous type while arrest of development in the retiform stage may produce arteriovenous fistulae or mixed haemangiomata, or both.

The clinical features of peripheral congenital arteriovenous fistulae depend on the degree of arteriovenous shunting and on whether the fistulae are distributed throughout the limb or only in part. A localized fistula with a large shunt, e.g. cirsoid aneurysm of the hand or foot, may be recognized by the presence of distended superficial veins which are often pulsatile, a compressible vascular mass, local warmth and a continuous bruit. Digital ulceration or necrosis may occur as a result of distal ischaemia. Lesions with a small degree of shunting may be manifest merely by an abnormal cluster of veins, local warmth or a soft tissue mass.

Diffuse fistulae usually cause overgrowth of the limb which is sometimes evident at birth. Skin angiomata, particularly of the port-wine variety, and coexistent skeletal abnormalities are common. The limb is warm and distal ulceration may occur. An additional lymphoedematous element is often present and may contribute to limb overgrowth. Superficial veins may become prominent in time, but these are more commonly associated with a congenital venous disorder than an arteriovenous fistula (Klippel-Trenaunay syndrome)[17,18]. Cardiac effects may occur with either local or diffuse congenital fistulae but are uncommon.

The most important investigation is arteriography aided by Doppler ultrasound. In the case of a large shunt fistula, blood flow is rapid and selective catheterization with rapid contrast injection is essential for proper definition. The most common arteriographic findings are dilatation and tortuosity of the afferent vessels, with a cluster of abnormal vessels at the site of the lesion and early opacification of the draining veins. Malformations with a small degree of shunting may opacify poorly or not at all on conventional arteriography. Where there is clinical or ultrasound suspicion of an arteriovenous element[19] superselective catheterization should then be undertaken to define the lesion[20]. Venography and lymphangiography may also be needed if there is associated venous dysplasia or a suspected lymphatic anomaly. Other investigations may include a CT scan to assess the extent of the lesion and cardiac output studies if there is clinical, ECG or radiological evidence of a cardiac effect. Peripheral arteriovenous lesions which extend into the root of the limb or primary trunk fistulae may require additional investigations such as pyelography, cystoscopy, proctocolonoscopy as well as full abdominal arteriography. Congenital fistulae of the limbs may coexist with arteriovenous malformations at distant sites (brain, lung, viscera) and these may necessitate further specific investigative measures.

MANAGEMENT

Congenital arteriovenous malformations are often difficult to treat and an initial conservative approach is advisable whenever possible. Elastic support may be useful in controlling discomfort and swelling of the limb in the diffuse type of fistula and may limit limb overgrowth if applied at a sufficiently early age. Where limb lengthening remains a problem a contralateral shoe raise or a carefully timed epiphysiodesis may be helpful.

Treatment of a more active nature may be necessary in patients with large-shunt fistulae which are causing pain, ulceration, haemorrhage or cardiac decompensation. Surgical excision may very occasionally be considered when an arteriovenous malformation is superficial and well localized[21,22]. More extensive lesions can sometimes be made operable by initial embolization[23,24,25]. One example where this combined approach may be possible is with a cirsoid aneurysm of the scalp (Figure **8.8a**). This lesion is usually supplied by the superficial temporal and occipital arteries and sometimes by branches of the contralateral external carotid artery. Embolization of the supply vessels may be achieved with Gelfoam sponge 24–48 hours prior to surgery. A scalp flap containing the lesion is then turned down and the latter excised by sharp dissection, taking care to preserve the blood supply to the flap (Figure **8.8b–d**).

By far the largest group of patients have extensive malformations which are not amenable to surgical excision. The results of partial excision have been unsatisfactory and ligation of afferent vessels alone is futile because of collateral regrowth[26]. Moreover, proximal ligation makes subsequent catheter access more difficult[21]. Fortunately many malformations can now be controlled by embolization alone. However, it is important to recognize that this is often palliative rather than curative as a consequence of the multiplicity of vessels supplying the malformation[27].

The basic principle in embolotherapy is to deliver the occlusive agent into the nidus of the lesion. The proximal access vessels should be preserved in case repeat embolization is required. At the present time, suitable materials for obliterating the nidus include small particle Ivalon or Bucrylate[28]. Silicon liquid may also be used in conjunction with proximal balloon occlusion to slow the flow[29]. Staged procedures are often advisable in order to reduce the risk of distal ischaemia. In addition, staging allows identification of any new feeding vessels that may develop.

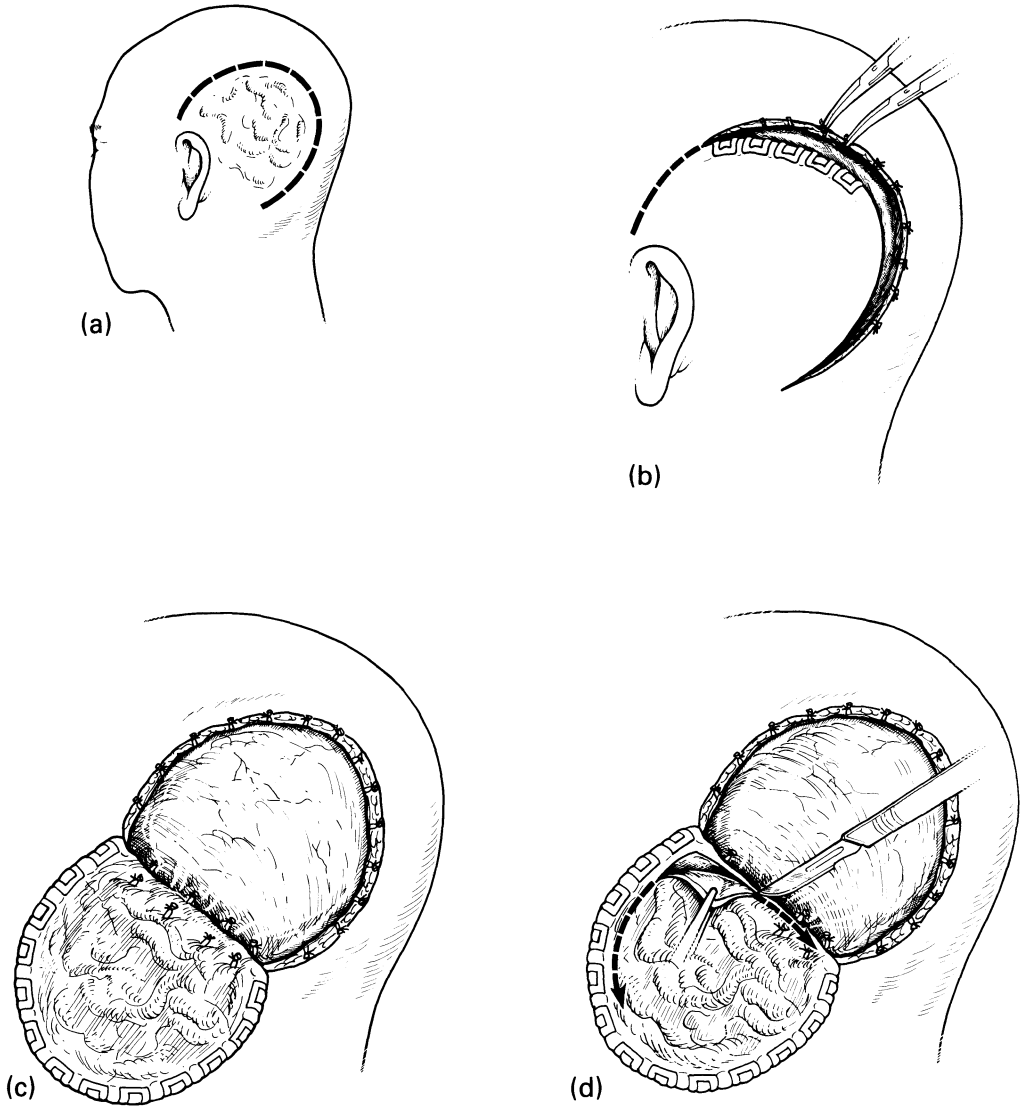


Figure 8.8(a–d) Surgical management of a localized congenital AV fistula, e.g. cirsoid aneurysm of scalp: (a) outline of the scalp incision to encompass the lesion. (b) haemostasis of the scalp flap using Raney clips. The vessels in the opposite edge are controlled by ligation and diathermy. (c) the scalp flap has been turned down in a plane superficial to the pericranium and the feeding vessels at the base have been ligated. (d) the galeal aponeurosis is incised inferiorly and the AV lesion is shaved off the deep aspect of the flap. Once the incision is completed, the scalp is closed in two layers with suction drainage

Occasionally it may be impossible to introduce a catheter into a feeding vessel, either because of tortuosity or an extremely distal position. Direct puncture of the lesion[30] or surgical exposure of the major feeders may then be considered[28].

Complications of embolotherapy have been relatively few but may include mass ischaemia and inadvertent reflux of embolic material. These may be minimized by cautious injection with the catheter advanced well into the supply vessel. Pulmonary embolization has not been a risk despite early fears to the contrary.

Failure to adequately control symptoms as, for example, when there is extensive bone involvement, may eventually require amputation. This approach may also be occasionally required when there is monstrous overgrowth of the limb with lymphoedema, recurrent cellulitis and ulceration.

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9

Surgery of the Carotid Bifurcation

Approximately 75% of patients with an ischaemic stroke syndrome have at least one obstructive arterial lesion at an extracranial site[1]. By far the most important of these sites is the carotid bifurcation and surgery of this area is considered in the present chapter. Reconstruction of the vertebral artery and aortic arch vessels is dealt with in Chapter 10.

CAROTID ENDARTERECTOMY

Atheroma at the carotid bifurcation typically involves the distal common carotid artery and proximal few centimetres of the internal carotid artery. In the great majority of cases, there is a clearly defined end point 1–3 cm beyond the internal carotid bulb where the abnormal intima thins out and is replaced by normal intima which is firmly adherent to the underlying media. These characteristics make endarterectomy a particularly appropriate form of treatment at this site. Occasionally the gross atheroma terminates in the internal carotid bulb but the distal intima is abnormally thick and easily separated, this abnormality extending to the base of the skull. Similar changes may also be present in the common carotid artery proximal to the bifurcation. A satisfactory endarterectomy end point may be difficult to achieve in these cases and bypass grafting may be a preferable method of reconstruction.

INDICATIONS FOR SURGERY

The principal indication for carotid endarterectomy is transient cerebral ischaemia, which may be hemispheric, retinal or vertebrobasilar in distribution. The chief importance of a transient ischaemic attack (TIA) is as a forerunner of a permanent stroke. Several reports have indicated that among patients with TIAs an average of 5–8% will develop cerebral infarction in each year of follow-up[2,3]. Surgery has been singularly effective both in abolishing these attacks and in reducing the risk of stroke[4,5].

Patients with a completed stroke may be considered for surgery if the deficit is mild and a threatening lesion is identified on arteriography. Although the long-term benefits of this policy have been questioned[6], with careful patient selection surgery does appear to offer protection against recurrent stroke[7].

Some patients who are neurologically unstable may also benefit from carotid artery surgery[8]. This applies not only to patients with crescendo TIAs but also to the more controversial group of patients who present with a fluctuating deficit or with a mild deficit which slowly progresses[9]. If arteriography reveals a severe stenosis or an intraluminal thrombus prompt intervention should be undertaken and may fully reverse the neurological impairment. Unstable patients with a complete occlusion may also be considered for urgent surgery providing there is no CT evidence of infarction and distal internal carotid patency is still preserved.

Chronic cerebral ischaemia is an additional indication for carotid endarterectomy. Symptoms may include impaired memory and mental acuity, postural light headedness, dimness of vision and difficulty with balance. These patients nearly always have occlusive lesions at more than one extracranial site and symptoms arise as a result of global hypoperfusion. Treatment may necessitate reconstruction of more than one vessel, though carotid bifurcation lesions should be regarded as a priority target (see Chapter 10).

The final group of patients in whom carotid artery surgery may be indicated are those with asymptomatic lesions[10]. These may be discovered during arteriography for symptomatic disease on the other side, during investigation of a mid-cervical bruit or as a result of non-invasive screening tests prior to cardiac or peripheral vascular surgery. If there is severe stenosis or ulceration, especially with intraplaque haemorrhage[11], surgery may be advisable in order to avoid embolization or progression to occlusion. Particularly strong indications apply if there is contralateral internal carotid occlusion[12] or severe bilateral stenotic disease, or if the patient is about to undergo major surgery with an attendant risk of hypotension.

CONTRAINDICATIONS TO SURGERY

Carotid artery surgery is essentially prophylactic and may not therefore be justified when life expectancy is limited or when there are significant operative risk factors[13,14]. In addition to these general reservations, there are specific neurological or arteriographic features which contraindicate a surgical approach. Thus surgery should not be considered in patients with an acute profound or rapidly progressing stroke. In many of these patients cerebral infarction will already have occurred and revascularization may lead to worsening of the neurological status[15]. Patients who develop a stroke immediately after carotid endarterectomy or during arteriography may be exceptions to this rule. Despite an apparently marked deficit, full recovery may be possible providing the artery is reopened within 1–2 hours (see below)[16,17]. Severe generalized intracranial disease is also a contraindication to carotid bifurcation surgery. Isolated siphon disease was formerly cited as a specific contraindication but it now seems clear that carotid endarterectomy can be safely and successfully accomplished in the presence of quite marked siphon stenosis[18,19].

TIMING OF OPERATION

The timing of carotid endarterectomy may be important. Patients with crescendo TIAs or a fluctuating deficit require emergency work-up. If a significant bifurcation lesion is identified, immediate surgery may be indicated to prevent a major stroke. Similar urgent action may be required if routine arteriography reveals either a very tight stenosis with only a trickle of antegrade flow, or an intraluminal thrombus.

Conversely patients with a recent minor deficit or TIA but evidence of infarction on CT scan should have surgery delayed for at least 4 weeks. If the patient is hypertensive it is advisable to delay operation even longer in view of the increased risk of intracerebral haemorrhage in this group[20].

The presence of bilateral carotid bifurcation disease may also influence surgical timing. As a general rule, such lesions are best dealt with on a staged basis with an interval of 2–3 weeks in between. Simultaneous correction is feasible but there may be difficulty with the airway or with swallowing as a result of local oedema or cranial nerve palsies. The risk of postoperative hypertension is also greater in a single-stage bilateral procedure. If arteriography reveals that one carotid artery is providing cross-flow to the opposite hemisphere, the lesion on the subordinate side is corrected first. In the absence of such evidence the most severely stenosed vessel is selected first. If both stenoses are equally severe, operation should be commenced on the symptomatic side.

DIAGNOSTIC STUDIES

Numerous non-invasive methods are available for assessing carotid bifurcation disease both before and after surgery. Oculoplethysmography, phonoangiography, Doppler imaging and ultrasonography have all been utilized. B-mode scanning alone or in combination with spectral analysis (duplex scan) is particularly useful and will provide information not only on luminal diameter but on the presence of ulceration and plaque composition[21,22]. This information correlates closely with the pathological findings[23] and may have a direct bearing on the stroke risk[11].

Digital subtraction angiography via central venous injection may also be used as a screening test though definition is not comparable to conventional arteriography[24]. Intra-arterial digital subtraction may offer considerable improvements in this respect and, providing small catheters are utilized, may be equally applicable on an outpatient basis[25].

All patients who are identified as having surgical lesions should undergo detailed arteriography of the extracranial and intracranial vasculature. Transfemoral arch aortography is preferred with selective catheter studies as appropriate. Conventional cut film arteriography may be supplemented by digital subtraction in order to reduce catheter time and contrast volume.

Where possible, all patients should also undergo CT scanning prior to surgery. This may establish whether or not infarction has occurred in the recently symptomatic case. In addition, routine CT scanning may detect a small but significant number of completely silent infarcts[26].

ASSOCIATED RISK FACTORS

Particular attention should be paid to controlling hypertension prior to surgery and to evaluating cardiac status. Arrhythmias and congestive failure may be improved by medical treatment and coronary artery disease carefully assessed. Carotid endarterectomy in patients with symptomatic coronary artery disease carries a high risk of perioperative myocardial infarction[27] and patients with severe or unstable angina, a positive ECG stress test or radionuclide studies should undergo coronary arteriography. Those with significant operable disease may then be managed by simultaneous carotid and coronary revascularization[28,29]. Percutaneous coronary angioplasty may be used in this setting as an alternative to coronary artery bypass grafting. Whether or not coronary revascularization is indicated, all patients with a clinical history of ischaemic heart disease or hypertension should be intensively monitored perioperatively and appropriate measures taken to optimize cardiac function and pressure (see Chapter 2).

CEREBRAL PROTECTION DURING CAROTID SURGERY

Local or regional anaesthesia is used in some centres on the grounds that clamp intolerance can be readily detected in the conscious patient[30]. However, considerably more patients are intolerant of clamping under these circumstances[31] than suffer a postoperative deficit in non-shunted series under general anaesthesia. The clinical relevance of trial clamping must therefore be questioned.

Most surgeons prefer to conduct the operation under general anaesthesia. This reduces cerebral metabolic demands, provides better oxygenation and control of the airway, and allows easier intraoperative blood pressure regulation[32]. In addition, the patient and surgeon are more comfortable and the operative exposure superior, particularly if access to the distal internal carotid artery is required. Balanced anaesthesia is utilized with thiopentone or etomidate induction followed by nitrous oxide-oxygen, fentanyl and a muscle relaxant. This technique provides maximum blood pressure stability[33]. Neither hypercarbia nor hypocarbia have been clearly shown to be beneficial[34] and pCO_2 should be maintained within normal limits throughout the procedure.

An intraluminal shunt is advocated by many authorities either on a routine basis[3] or selectively, according to stump pressure[35] or EEG change[36]. However, stump pressure is an unreliable indicator of cerebral perfusion[37] and may

not correlate closely with post-operative stroke rate[38]. Electroencephalogram changes more accurately reflect cerebral blood flow, but these may be influenced by a number of intraoperative variables apart from clamping[39]. Moreover, EEG changes can quite clearly occur after clamping without any subsequent adverse clinical effect[40]. In our view, most postoperative deficits are due to embolization or thrombosis in the endarterectomy zone rather than to clamp intolerance and the risks of the latter have been much overrated. Practical evidence in support of this view is provided by the fact that a similar low postoperative stroke rate (1–2%) has been observed in shunted and non-shunted series alike[3,41].

It is absolutely essential that a meticulous endarterectomy is carried out and a shunt inevitably obscures the operative zone, particularly at the critical distal edge. In addition, intimal damage may occur on shunt insertion while thrombus or air may enter the system augmenting the risk of embolization. In view of these risks and the lack of clear evidence for the role of clamp intolerance in postoperative stroke, we prefer to operate without a shunt. Reliance is placed instead on carefully controlled general anaesthesia with strict maintenance of arterial pressure at or slightly above the patient's usual preoperative level. This can nearly always be achieved by colloid infusion and the natural response to surgery. Pressor agents should not be needed though it may be advisable to keep an aramine infusion readily available.

Excessive sedation should be avoided both during anaesthesia and in the postoperative period. Myocardial infarction and cardiac arrhythmias are other major causes of perioperative hypotension and these may be best prevented by careful preoperative preparation, close regulation of cardiac haemodynamics and administration of anti-arrhythmic drugs as necessary. An additional possible cause of intraoperative hypotension is stimulation of the carotid sinus nerve during dissection of the carotid bifurcation. Usually this can be corrected by blocking the nerve locally with 1% lignocaine though a small systemic dose of atropine may also be needed in some cases.

TECHNIQUE OF CAROTID ENDARTERECTOMY

The patient is placed in a supine position with the head turned 45° away from the side of the operation and the neck slightly extended (Figure **9.1a**). An oblique skin incision is made in the line of the anterior border of sternomastoid. A more transverse incision, which is commonly advocated, provides a slightly better cosmetic result but access to the distal internal carotid artery is diminished and for this reason is not preferred.

The incision is deepened through platysma and the subcutaneous fat to the anterior border of sternomastoid (Figure **9.1b**). The external jugular vein entering the upper limit of the operative field may be divided, though the accompanying great auricular nerve should be preserved to prevent troublesome anaesthesia of the ear. The deep fascia is then opened and the common facial vein identified. This is the key to displaying the carotid bifurcation (Figure **9.1c**). Once this vein has been divided the internal jugular vein and sternomastoid can be retracted laterally, thereby exposing the arterial axis. Care should be taken not to include the vagus with the internal jugular vein as retraction may induce a vocal cord paresis. The ansa hypoglossi nerve overlying the carotid bifurcation may be displaced medially away from the vascular axis, but often can be sacrificed.

Dissection in the region of the carotid bifurcation should be avoided prior to clamping because of the risk of embolizing loose atherothrombotic material lying within the arterial lumen. Instead, the internal carotid artery should be traced distally and encircled at the most cephalic extent of the exposure. The posterior belly of digastric and the hypoglossal nerve are routinely displayed during this stage (Figure **9.1c, d**). If the carotid bifurcation is high, it may be necessary to divide a small branch of the occipital artery which passes backwards to sternomastoid creating a sling around the hypoglossal nerve. Once this vessel has been divided the nerve can be displaced superiorly away from the future clamp site (Figure **9.1d**).

The common carotid artery is then encircled proximal to the bifurcation at a point where there is relatively little atherosclerosis. Mobilization of the common carotid artery within its investing

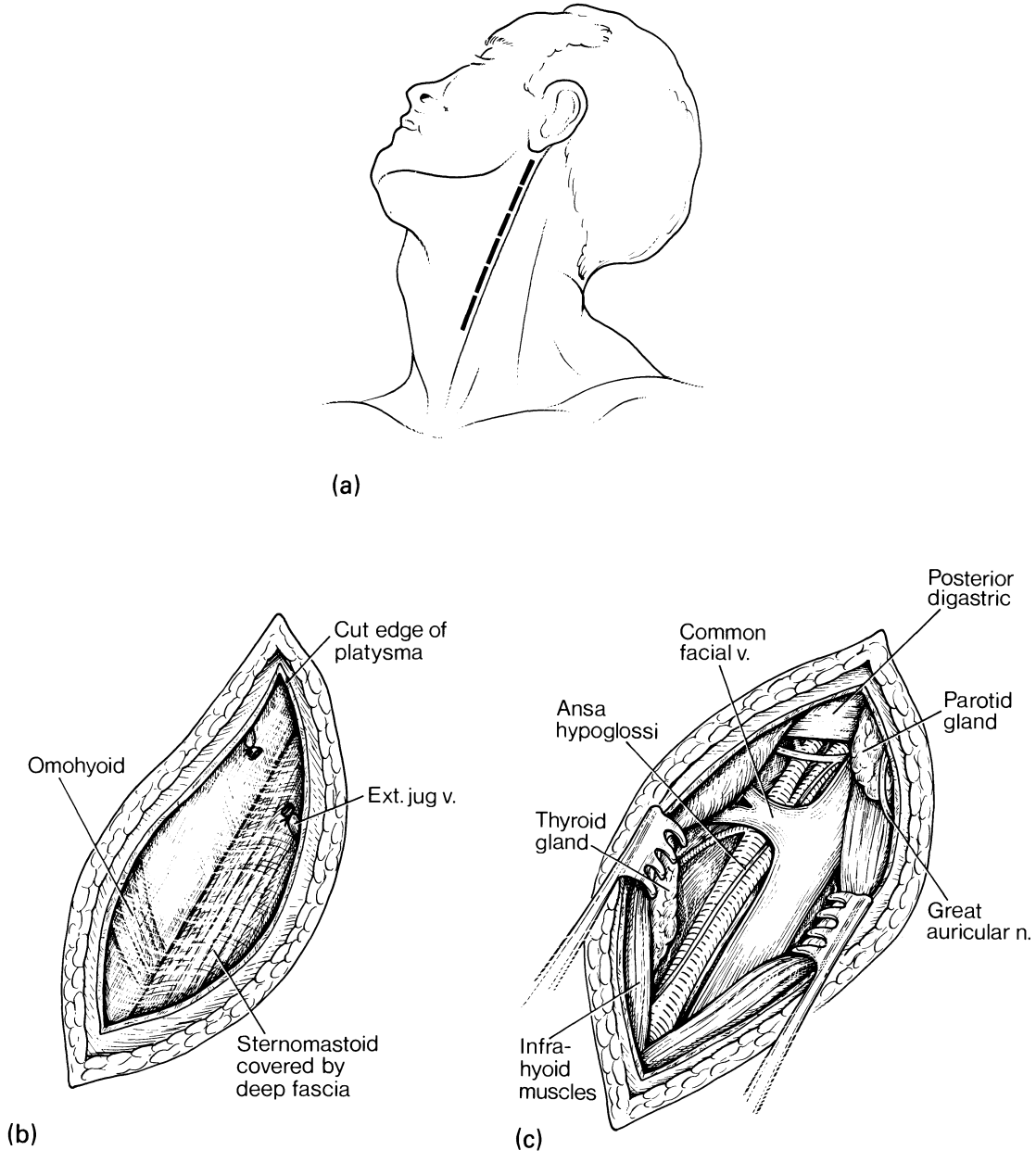


Figure 9.1(a–d) Exposure of the carotid bifurcation: (a) skin incision. (b) platysma incised. (c) deep fascia opened to reveal the common facial vein overlying the carotid bifurcation

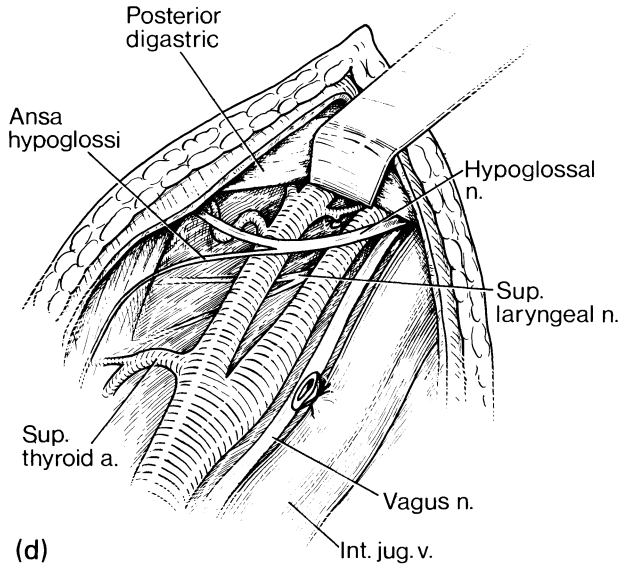
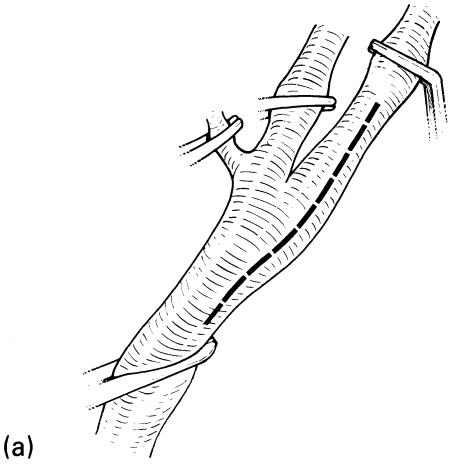


Figure 9.1 continued (d) exposure of the internal carotid artery completed

sheath should avoid vagal nerve damage during subsequent clamp placement. The external carotid artery is then cleared commencing at its anterior border and proceeding as far as the superior thyroid branch. During this stage the superior laryngeal nerve may be encountered posteriorly and should be safeguarded.

Heparin (1 mg/kg. body weight) is given by the anaesthetist and clamps applied, first to the internal carotid artery, then the external, and finally the common carotid artery. The dissection of the posterior and lateral aspects of the bifurcation can now be safely completed without risk of embolization. Once this has been done the arterial axis is rotated anteriorly, to facilitate the endarterectomy. An arteriotomy is then made along the lateral face of the distal common carotid artery and extended into the internal carotid artery using Pott's angled scissors (Figure **9.2a**). Restricting the arteriotomy to the internal carotid bulb is commonly advocated, and has the advantage of permitting subsequent direct closure. However, such an incision provides a less than perfect view of the critical distal intimal edge and we therefore prefer as a routine to lay open the internal carotid artery beyond the bulb and then to close the arteriotomy with a vein patch.

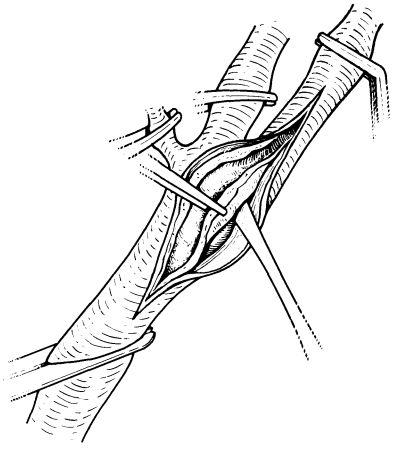
Separation of the atheromatous core is conveniently begun at the site of maximal disease in the distal common carotid artery using a blunt-nosed dissector (Figure **9.2b**). An external endarterectomy plane is chosen if possible (see Chapter 1). The atheromatous core is developed proximally in the common carotid artery and is then sharply divided where it thins out near the inferior limit of the arteriotomy. If the intimal edge is excessively thick or loose here, with a risk of subsequent mural thrombus deposition, the arteriotomy should be extended proximally to allow a more extensive clearance. If a satisfactory proximal end point cannot be established, it may be advisable to abandon the endarterectomy in favour of a bypass technique.



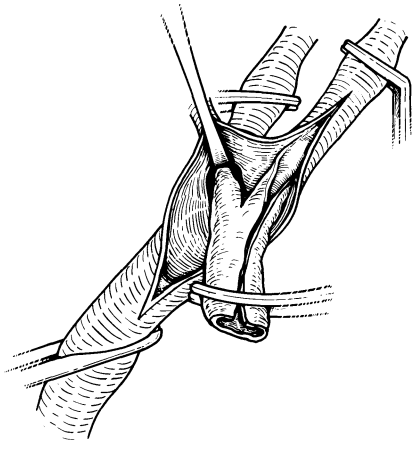
(a)

The atheromatous core is then developed distally into the external and internal carotid arteries. It may be helpful to bisect the atheroma longitudinally and then concentrate on clearing each vessel in turn. Removal of the atheroma from the external carotid artery (Figure **9.2c**) is inevitably something of a blind procedure, but gentle traction on the specimen and minimal use of the dissector should ensure that a clean distal separation is achieved as the artery is progressively everted into the common carotid lumen. Occasionally there may be a tongue of atheroma extending into the external carotid artery beyond the controlling clamp. Momentary release of the latter should then allow clearance to be completed. The lumen of the external carotid artery is carefully inspected and irrigated and any remaining debris removed with forceps.

Achievement of a satisfactory end point in the internal carotid artery is the most important step in the entire operation. Continued gentle traction on the specimen, and use of the dissector in



(b)



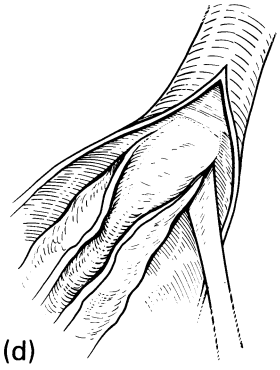
(c)

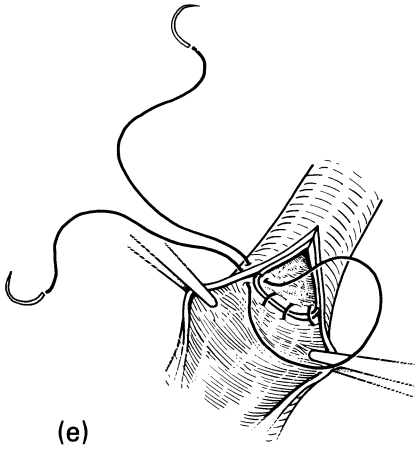
Figure 9.2(a–h) Technique of bifurcation endarterectomy: (a) arteriotomy. (b) atheromatous core developed in the distal common carotid artery. (c) eversion endarterectomy of the external carotid origin

a progressively more superficial plane, should result in a natural separation where the atheroma feathers out beyond the bulb (Figure **9.2d**). The remaining distal edge should then be securely adherent and of minimal thickness. If this is not the case, additional intimal stitches will be required (Figure **9.2e**). However, this is not ideal and if there is continuing concern regarding either the thickness or security of the distal intimal edge it is preferable to exclude the endarterectomy zone and insert a bypass. The latter manoeuvre may also be required if the endarterectomy plane has been taken too deeply leaving a roughened surface (see below).

Once the atheromatous specimen has been detached, several minutes should be spent meticulously removing all loose shreds of media and fibrin strands in a circumferential manner (Figure **9.2f**). Repeated lavage with heparin–saline will assist in identifying these loose tags. The arteriotomy is then closed with a patch (Figure **9.2g**) which avoids any possibility of suture line stenosis, particularly at the apex. In addition, a patch secures the intima and produces an over-pass effect (see Chapter 1). Patching may also protect against recurrent stenosis due to intimal hyperplasia in the endarterectomy zone.

Autogenous saphenous vein is the preferred material for patching as it sews in to the delicate





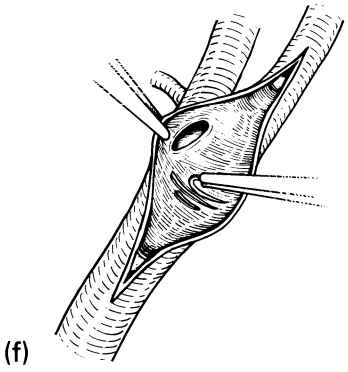


Figure 9.2 continued (d) critical end point in the internal carotid artery. (e) tack-down sutures to secure distal intimal edge (not ideal). (f) meticulous removal of all loose shreds

endarterectomized vessel more easily than prosthetic material. A suitable length is harvested from the ankle or distal leg, preserving the rest of the vein for possible use as a coronary or femoro–distal bypass. Usually only half the width of the vein is required since a larger patch may result in aneurysmal dilatation. The patch is then sewn in with a running 6/0 prolene suture from each extremity (see Chapter 1). It is particularly important in carotid patching to inspect as much as possible of the interior of the suture line before completion so that any deficiencies can be corrected with supplementary intimal stitches. Each of the clamps is then momentarily released and the lumen well rinsed with heparin–saline before completing the suture line.

The internal carotid clamp is then removed and replaced by manual compression (Figure **9.2h**). This allows sufficient back-bleeding to expel the air from the operated segment. Gentle massage will assist this expulsion while the internal carotid artery is held occluded. The external carotid clamp is released next, followed by the common carotid clamp. This order of clamp release will ensure that any remaining air or debris in the operated segment is shunted into the external carotid circulation (Figure **9.2h**). Finally, the internal carotid artery is released and a gauze or cotton-wool compress applied to the suture line. Any persistent leaks are subsequently corrected with supplementary 7/0 or 8/0 stitches. The heparin is then partially reversed with protamine (0·5 mg/kg. body weight) and the wound closed with suction drainage.

Intraoperative arteriography has been utilized in some centres prior to wound closure but this is not without hazard[42] and there may be uncertainty regarding the significance of some of the defects initially shown[43]. Because of these difficulties a completion arteriogram is not obtained as a routine, reliance being placed instead on full visualization of the endarterectomy zone and a meticulous clearance in the manner described.

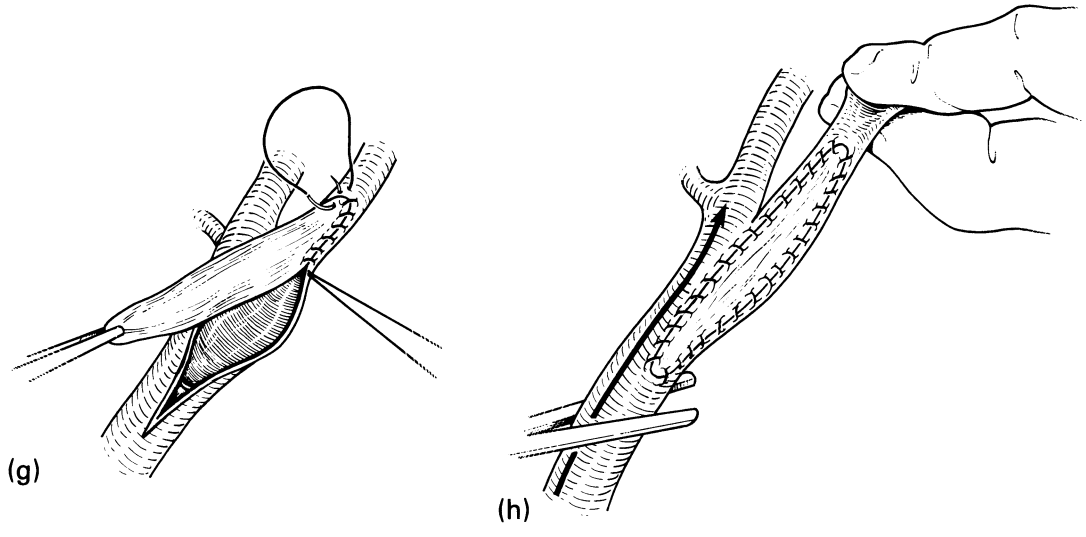


Figure 9.2 continued (g) arteriotomy closed with a vein patch. (h) preliminary flush into the external carotid territory before releasing flow into the internal carotid artery

EARLY POSTOPERATIVE COMPLICATIONS

Cervical haematoma

A haematoma may develop within the first few hours of surgery. Predisposing factors include heparin overdosage, previous antiplatelet therapy and postoperative hypertension. An expanding haematoma may cause airway obstruction and as soon as a swelling starts to appear the patient should be returned to the operating room. The haematoma is then evacuated and any obvious open vessel ligated. It is uncommon for the suture line to be the source of postoperative bleeding but if this is the case, additional 8/0 sutures or alternatively 6/0 sutures backed by Teflon pledgets, may be required. These can be inserted without reclamping the carotid axis (see Chapter 1).

Blood pressure abnormalities

Careful monitoring of blood pressure is particularly important in the early postoperative period. Postoperative hypotension may occur as a result of overstimulation of the sinus nerve following removal of the rigid stent-like atheromatous core from the artery wall[44]. Oversedation or cardiac causes may also be contributory, as previously described. If severe, hypotension may give rise to thrombosis at the endarterectomy site or produce a deficit in other territories harbouring previously well compensated lesions[45]. Prompt correction is hence essential and can usually be achieved by fluid replacement alone, without the need for a vasopressor infusion.

Hypertension may be more of a problem. The exact mechanism is uncertain but surgical interference with the sinus nerve may be partly to blame[44]. Postoperative hypertension is particularly likely in those patients who have poorly controlled hypertension prior to surgery. Cerebral haemorrhage is the main risk, though there may also be an increased incidence of postoperative ischaemic events[46]. These possibilities underline the importance of adequate blood pressure control prior to carotid surgery. If postoperative hypertension still develops (systolic pressure >200 mmHg), prompt treatment with a nitroprusside or hydralazine infusion should be commenced.

Nerve palsies

Some of the superficial branches of the cervical plexus may be divided when making the initial incision. This is of little consequence, though the great auricular nerve should if possible be preserved to avoid anaesthesia of the lobe and posterior surface of the ear.

Cranial nerve injuries occasionally occur. The superior laryngeal nerve which runs deep to the internal and external carotid arteries may be damaged during clamping of either vessel. This may result in fatiguability of the voice and impaired phonation at high pitch. Vagal injury may occur from inadvertent lateral traction during initial display of the bifurcation or from inadequate isolation of the common carotid artery prior to clamping. Postoperative hoarseness and loss of an effective cough mechanism may be an indication of such an injury. A hypoglossal nerve palsy is less common and may present with deviation of the tongue or in severe cases with difficulty in speaking and chewing. As previously described, this hazard may be avoided by dividing the branch artery to sternomastoid and displacing the hypoglossal nerve superiorly before the internal carotid artery is clamped. The glossopharyngeal nerve may rarely be damaged and then only in a high (retrostyloid) exposure. Anaesthesia of the palate and attendant swallowing problems may result. Finally, retraction at the inferior margin of the mandible may occasionally produce a temporary drooping of the corner of the mouth due to palsy of the mandibular branch of the facial nerve.

Neurological deficit

The occurrence of a neurological deficit or the aggravation of an existing deficit is obviously a major concern following carotid endarterectomy. The main causes are either embolization or thrombosis in the endarterectomy zone. Less commonly, a deficit is due to reperfusion injury, cerebral haemorrhage, or thrombosis of the contralateral internal carotid artery or of an intracranial vessel. Ischaemia as a result of clamping is seldom, if ever, the cause of post-operative stroke.

Embolization of loose atherothrombotic

material may occur during preliminary surgical exposure if attempts are made to mobilize the bifurcation before the distal internal carotid artery has been clamped. In other cases, air or debris may be embolized from the operative zone on clamp release. Postoperative embolization may occur if thrombus is deposited on the suture line or on an overthick intimal edge. Other postoperative emboli may be due to previously underestimated arterial lesions proximal or distal to the carotid bifurcation or arise from the left heart following an arrhythmia or myocardial infarction.

Thrombosis in the operative zone may occur from factors common to all endarterectomy procedures. These include an error in the cleavage plane (overdeep, uneven or too superficial), a distal intimal flap, a loose overthick proximal intimal edge, or a stenotic suture line. Perioperative hypotension and hyperviscosity states favour thrombosis.

A reperfusion injury may develop during the first postoperative week and is characterized by severe unilateral headache and paroxysmal epileptiform discharge. A previous deficit may reappear or become aggravated post-ictally and in severe cases there may be intracerebral haemorrhage. The syndrome is uncommon but may be due to paralysis of normal autoregulation leading to hemispheric hyperperfusion[36]. Reperfusion injury is difficult to avoid but mannitol and anticonvulsant therapy may abort the full syndrome, if begun as soon as headache occurs.

Cerebral haemorrhage is a complication which is particularly likely in hypertensive patients who have had a cerebral infarct in the recent past[20]. Avoidance depends on strict regulation of blood pressure before and after surgery and on allowing an adequate period of time after infarction before attempting revascularization.

Management of patients with a post-endarterectomy deficit

All patients who are noted to have a deficit on recovery from anaesthesia and those who develop a deficit in the early postoperative period (up to 72 hours) should be returned to the operating room for re-exploration. Although a more conservative approach has been suggested in patients with a minor deficit[47] this may leave *in situ* thrombosis or a potentially embologenic lesion uncorrected. Thrombosis cannot be distinguished reliably from embolization on a time interval basis and it is safer to embark upon a negative exploration than to miss the chance of reversing a deficit by thrombectomy. Providing due care is taken with anaesthetic management, re-exploration can be carried out without any additional hazard to the patient[48].

Others have suggested using non-invasive techniques or arteriography to document internal carotid occlusion prior to re-exploration[49,50]. However, the former may be difficult or unreliable in the immediate postoperative period, while conventional or digital arteriography inevitably incurs some delay, which may prejudice the chances of a successful response to thrombectomy.

At re-exploration the artery may be found to be grossly normal with good pulsation. The likely cause of the deficit may then be assumed to be embolization. An operative arteriogram should be obtained to exclude mural thrombus or a technical error which might produce further embolic episodes. If arteriography is normal no further action is necessary.

In cases of thrombosis of the endarterectomy zone, the artery appears distended with a bluish discoloration and absent pulsation. After heparinization the patch should be reopened and local thrombus removed. A Fogarty catheter is then passed distally to the base of the skull and gently withdrawn. This should retrieve any remaining thrombus and result in a good back-bleed. The endarterectomy zone is then examined for a technical error. If a distal intimal flap is present, one approach is to extend the arteriotomy, excise the loose intima and secure the distal edge with tack-down sutures. The arteriotomy is then reclosed with a patch. However, in most patients it is safer to exclude the endarterectomy zone and insert a common–internal carotid vein bypass (see below). This is also the best method of correcting errors in the endarterectomy cleavage plane. An overthick or loose proximal intimal edge can usually be managed by extending the endarterectomy proximally until a more satisfactory end point is reached, though a bypass may again be warranted if there is a continuing problem.

All patients who develop a post-endarterec-

tomy deficit should be placed on dexamethasone or other anti-oedema agents post-operatively. Repeat CT scans may be helpful thereafter in following progress.

LATE POSTOPERATIVE COMPLICATIONS

Recurrent stenosis

The incidence of symptomatic recurrent stenosis following carotid endarterectomy is of the order of 1–2%[51,52]. However, routine follow-up with non-invasive testing would suggest that the true incidence of restenosis/occlusion is probably much higher[53,54]. Restenosis within the first 2 years of surgery is usually due to intimal hyperplasia whereas later cases are mainly the result of atherosclerotic progression[51].

Patients with symptomatic recurrence or severe asymptomatic disease require further treatment. Although most reported cases have been managed surgically, percutaneous transluminal angioplasty may be a feasible alternative in some patients with intimal hyperplasia[55]. The typical lesion in this condition is a smooth, fibrous narrowing and dilatation may be attempted with relatively little risk of embolization. However this approach is not advisable in patients with atherosclerotic re-stenosis because of the more friable nature of the arterial pathology.

Surgical access may be difficult due to postoperative scarring and there is an increased risk of cranial nerve injury, particularly to the hypoglossal nerve. The common carotid artery can usually be clamped without difficulty but distal internal carotid control may require use of an endoluminal balloon catheter. Re-do endarterectomy has been utilized in some patients with atherosclerotic recurrence, while patients with intimal hyperplasia have been managed by patching, with or without intimal excision. However, these options are not particularly satisfactory and most cases of re-stenosis are best managed by a common–internal carotid bypass.

Aneurysmal dilatation

This is essentially a complication of patch graft angioplasty[56]. Most reported cases have been false aneurysms secondary to prosthetic patch insertion and have resulted either from low grade infection or technically inadequate suture placement[57] (see Chapter 14). True aneurysmal dilatation may also occur particularly if an overlarge saphenous vein patch has been inserted or if the patch material has been taken from the jugular or cephalic veins.

Post-endarterectomy aneurysms contain mural thrombosis and their most serious risk is cerebral embolization. Thrombotic occlusion or rupture are other possible complications. In view of these possibilities all cases should be repaired. As in the case of recurrent stenosis, access may be difficult and the internal carotid artery may be best controlled by endoluminal catheter. The aneurysm is then either resected or excluded and a carotid bypass graft inserted. If overt infection is present, *in situ* bypass may risk secondary haemorrhage and an extracranial–intracranial bypass with exclusion of the infected zone may be a safer option. Since exclusion of the bifurcation will involve loss of the external carotid artery, intracranial revascularization may require a saphenous vein graft from the common carotid artery to a cortical vessel[58].

THE OCCLUDED INTERNAL CAROTID ARTERY

In a number of patients presenting with TIAs or amaurosis fugax, subsequent investigation reveals an internal carotid artery occlusion on the symptomatic side. These patients may be managed in a variety of ways. In general, attempts at reopening a chronic internal carotid artery occlusion are not worthwhile[59]. However, if distal internal carotid patency has been preserved down to the petrous canal, local endarterectomy and thrombus extraction may be highly effective even if some considerable time has elapsed since occlusion[60,61]. If there is doubt about distal patency, rapid sequence CT scanning[62] may assist in deciding for or against a local approach.

Other methods rely on increasing the collateral supply to the ischaemic hemisphere. These may include contralateral carotid endarterectomy, vertebro-subclavian reconstruction and correction of an ipsilateral external carotid artery sten-

osis. If none of these indirect measures is appropriate, consideration should be given to direct revascularization by extracranial–intracranial bypass.

In some cases, continuing symptoms after internal carotid artery occlusion are due to embolization via the external carotid–ophthalmic artery pathway[63] rather than to hemispheric hypoperfusion[64]. These microemboli may arise from a stump at the internal carotid origin or from ulcerated atheroma in the distal common carotid artery. Remodelling of the bifurcation with local endarterectomy may provide effective symptomatic relief in these cases.

EXTERNAL CAROTID ENDARTERECTOMY

This is principally indicated in patients with internal carotid artery occlusion who have significant stenosis at the external carotid origin or ulcerated atheroma in the distal common carotid artery with or without an internal carotid 'stump'. External carotid endarterectomy may also be undertaken to maximize inflow into the superficial temporal artery prior to extracranial–intracranial bypass. However, in a number of these patients, correction of the external carotid lesion alone may be sufficient to relieve symptoms, making direct cerebral revascularization unnecessary.

Technique

The carotid bifurcation is exposed as previously described and the external carotid artery dissected out beyond the atheromatous disease. This may necessitate dividing the posterior digastric and reflecting the hypoglossal nerve in a cephalic direction.

After heparinization, clamps are applied to the distal external carotid artery, the branch vessels and the common carotid artery. The common–external carotid segment is then incised anteriorly and an open endarterectomy achieved (Figure **9.3a**). The thrombosed internal carotid artery is amputated and the origin oversewn flush with the arterial axis to eliminate a cul-de-sac effect (Figure **9.3b, c**). The lumen is thoroughly irrigated and any remaining loose shreds of media removed, after which the arteriotomy is closed with a patch (Figure **9.3d**).

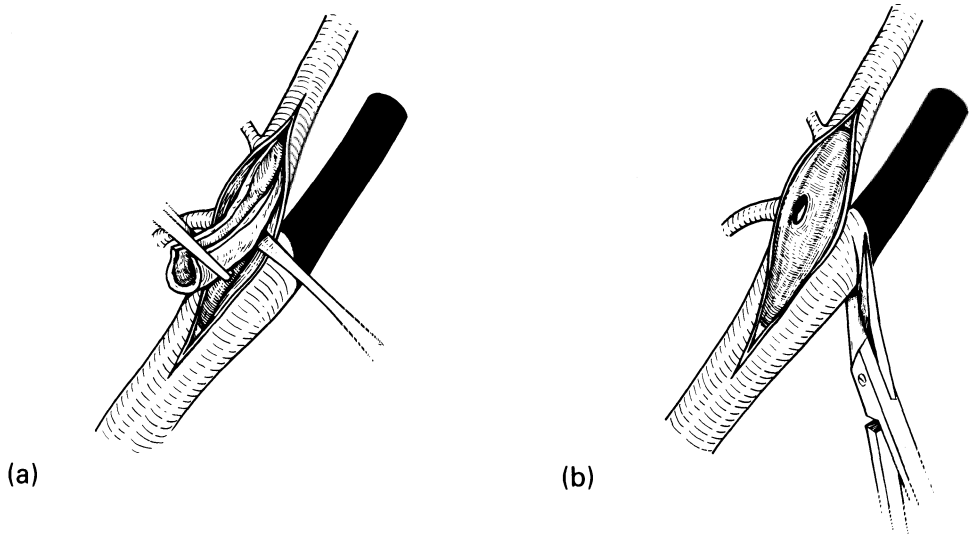


Figure 9.3(a–d) External carotid endarterectomy: (a) open endarterectomy of the distal common–external carotid segment. (b) occluded internal carotid artery amputated

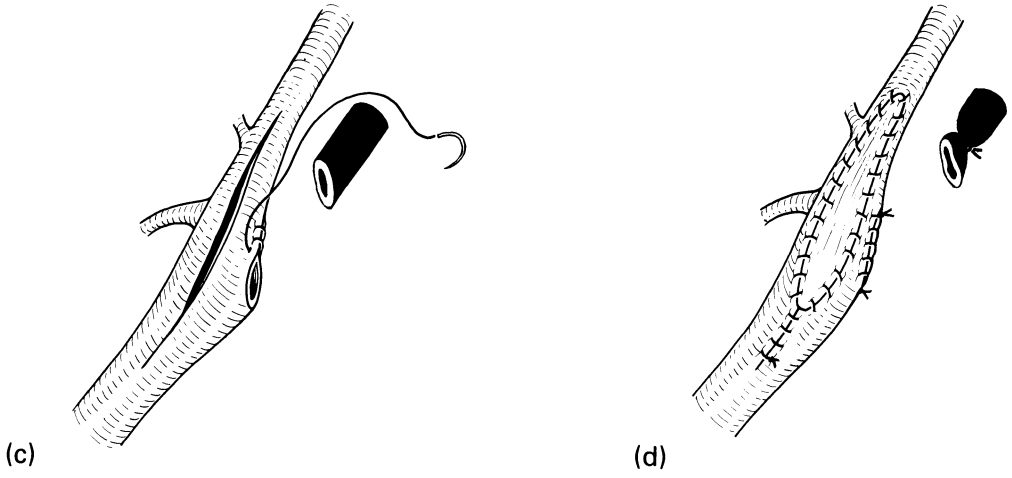


Figure 9.3 continued (c) bifurcation remodelled. (d) arteriotomy closed with a vein patch

INTERNAL CAROTID ARTERY BYPASS

INDICATIONS

Revascularization of the internal carotid artery may be achieved by bypass grafting as an alternative to endarterectomy. In view of the small but significant incidence of restenosis and occlusion after endarterectomy some consideration should perhaps be given to wider use of bypass grafting as a form of primary treatment even when endarterectomy is technically feasible. Specific indications for bypass placement include bifurcation atheroma with diffuse intimal thickening proximal and distal to the main pathological zone (Figure **9.4**), internal carotid stenosis with additional coiling or kinking (Figure **9.5**) and post–endarterectomy complications such as early thrombosis, re-stenosis or aneurysmal dilatation. A miscellaneous group of conditions may also occasionally require bypass grafting e.g. certain cases of fibromuscular dysplasia, internal carotid dissection, traumatic lesions, post-irradiation and other forms of arteritis, aneurysms, carotid body tumours and malignant invasion.

TECHNIQUE

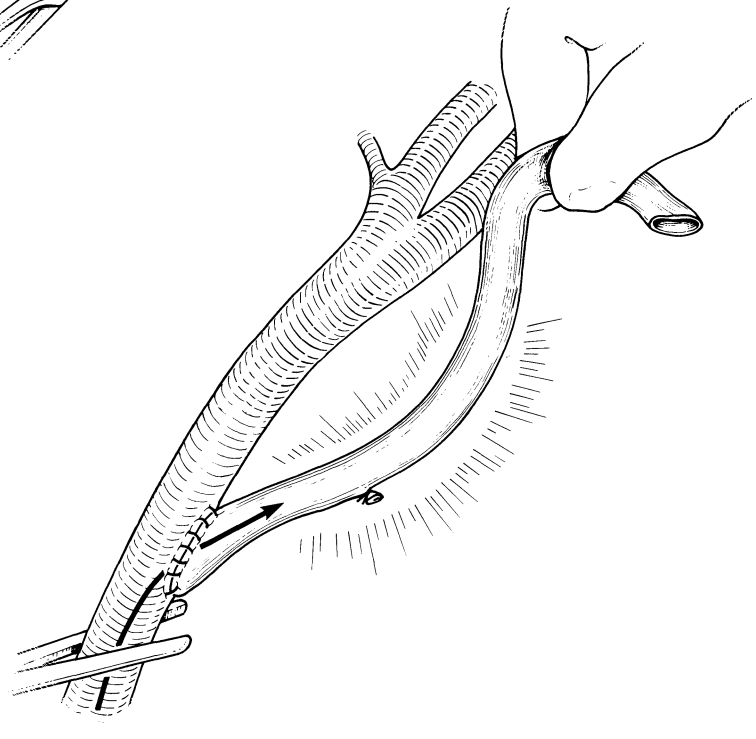
The carotid bifurcation is exposed as previously described. The proximal graft attachment should be to a relatively undiseased segment of the common carotid artery and this may require exposure of the artery to below the level of the omohyoid muscle. A segment of long saphenous vein is then harvested from the distal thigh. If possible this should be avalvular (see Chapter 5) and should include a branch vein with which to enlarge the heel of the proximal anastomosis (Figure **9.4a**). If autogenous saphenous vein is unavailable, PTFE may be an acceptable substitute.

After heparinization, the common carotid artery is cross-clamped and the graft attached end-to-side at the selected site. On completion any air or debris within the lumen is evacuated via the unattached end of the graft before flow is restored in the common carotid artery. The graft is then filled with heparin–saline and exposed to arterial tension (Figure **9.4b**). This manoeuvre is helpful in assessing graft length and avoiding torsion.

Distally the graft may be attached in end-to-end or end-to-side fashion. The latter will avoid all risk of late anastomotic stenosis and should be

particularly utilized where the distal internal carotid intima is thickened or abnormal. End-to-end anastomosis is less satisfactory in these cases because of the risk of intimal splitting on suture insertion (see Chapter 1). Once an end-to-side anastomosis has been completed the internal carotid artery is sectioned immediately proximal to the anastomotic heel, and the graft and distal internal carotid artery flushed via the cut stump (Figure **9.4c**). The latter is then ligated, converting the anastomosis into an effective end-to-end configuration (Figure **9.4d**). If there is additional external carotid stenosis or distal common carotid ulceration, the internal carotid origin should be amputated and a local common–external carotid endarterectomy carried out. This can be done under direct vision with full control of the distal end point and is one of the bonuses of a bypass

(a)



(b)

Figure 9.4(a–d) Internal carotid bypass for bifurcation atheroma with diffuse intimal thickening: (a) pathology and site of proximal graft attachment. A vein branch has been used to widen the heel of the graft. (b) after completing the proximal anastomosis, the vein graft is exposed to arterial tension in order to gauge the length

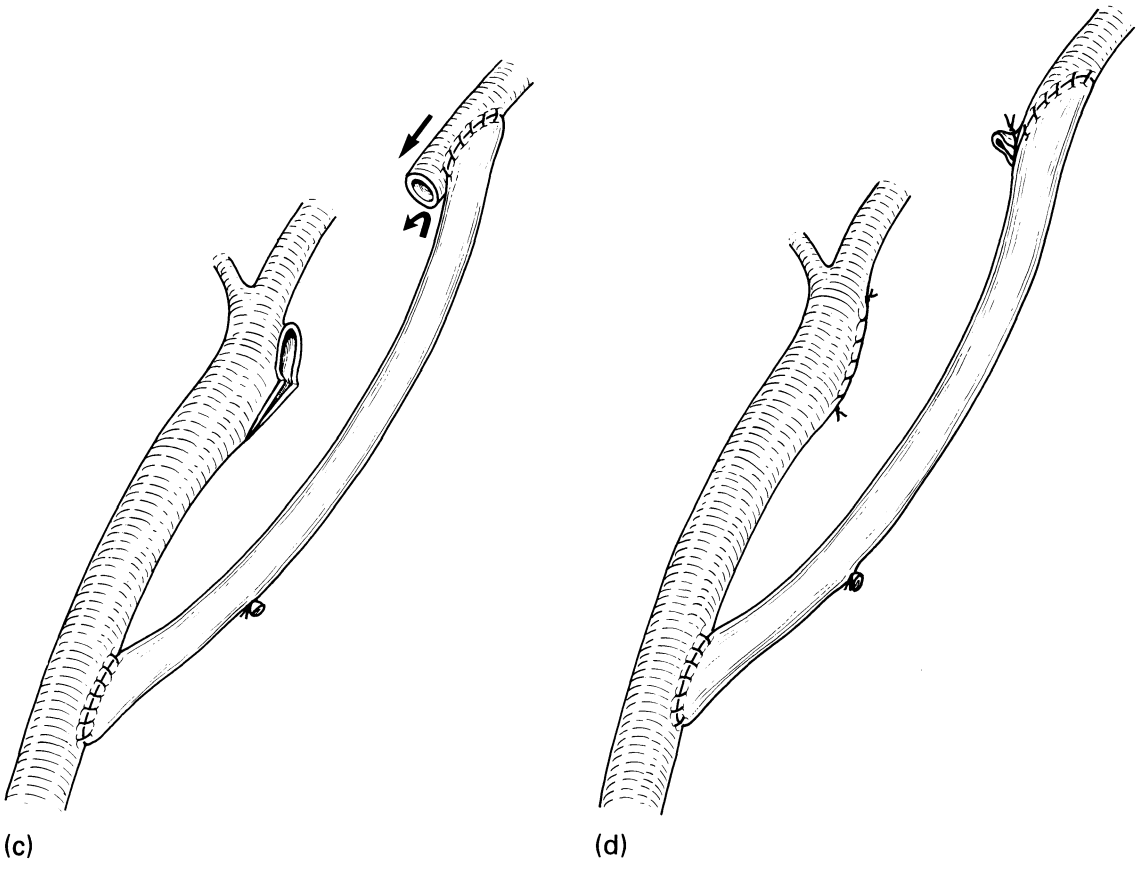


Figure 9.4 continued (c) distal end-to-side anastomosis completed. The internal carotid artery and the graft are then flushed via the cut end. (d) a ligature is placed adjacent to the heel of the distal anastomosis. The bifurcation is then closed after endarterectomy of the external carotid origin

technique compared with a conventional bifurcation endarterectomy.

If the graft is being inserted for other conditions such as kinking or coiling (Figure **9.5a**), it may be more appropriate to resect the proximal internal carotid artery and attach the graft distally by direct end-to-end anastomosis. Depending on the level of section relative to the bifurcation, it may be helpful to transpose the distal internal carotid stump anterior to the hypoglossal nerve prior to anastomosis. The graft and distal artery are then suitably spatulated and the anastomosis completed with either a continuous prolene suture posteriorly and interrupted sutures anteriorly or with interrupted sutures throughout, according to vessel size.

If the vein graft is without valves, flushing may be conveniently achieved via the amputated origin of the internal carotid artery (Figure **9.5b**). Where the graft is valved this will not be possible and, instead, the last two or three anterior sutures of the distal anastomosis should be left untied. Antegrade and retrograde flushing is then achieved via this site after which the sutures are tightened and finally tied with flow running through the graft.

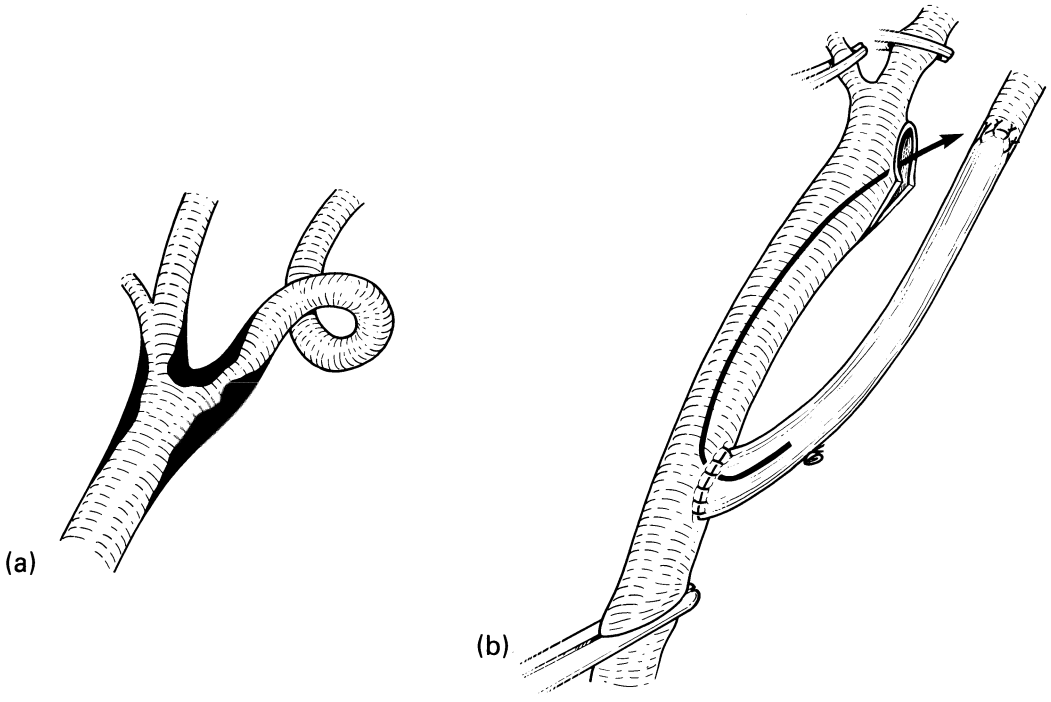


Figure 9.5(a, b) Bifurcation atheroma and coiling of the internal carotid artery: (a) pathology. (b) the redundant length of internal carotid artery has been excised and a vein graft placed between the common and internal carotid arteries. In this case the vein segment is without valves, allowing retrograde flushing via the transected internal carotid origin

In very distal bypass grafts, e.g. for dissecting or traumatic aneurysm, the distal anastomosis should be completed first. Endoluminal control may be necessary if there is insufficient space for clamp placement. Interrupted 7/0 prolene sutures are inserted at a distance from inside-out along the posterior half of the anastomotic circumference (Figure **9.6**). The graft is then slid down onto the artery and the sutures tied. The remainder of the anastomosis is completed with interrupted sutures again passed alternatively through the graft and artery. These are initially held with forceps until the suture line is complete and then tied. The integrity of the anastomosis is tested by temporary back-bleeding and by rinsing with heparin–saline. The graft is then cut to length while maintaining slight longitudinal tension and anastomosed end-to-side to the common carotid artery. Careful flushing and staged declamping are carried out as previously described before flow is finally released into the distal internal carotid artery.

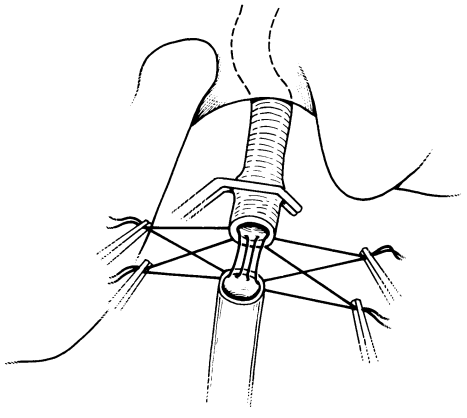


Figure 9.6 Technique of high internal carotid anastomosis.

OTHER LESIONS AT THE CAROTID BIFURCATION

KINKING, COILING AND TORTUOSITY OF THE INTERNAL CAROTID ARTERY

Elongation of the extracranial portion of the internal carotid artery may give rise to three types of morphological abnormality: tortuosity, coiling or kinking[65] (Figure **9.7**).

Tortuosity is a not uncommon consequence of atheroma and hypertension. Unless there is additional bifurcation disease the condition is usually asymptomatic and surgical correction unnecessary. Mild tortuosity associated with internal carotid stenosis can be corrected at the time of carotid endarterectomy by taking slightly wider bites of the artery than of the patch during closure. More severe degrees of tortuosity may require one of the options described below.

Coiling is often bilateral and symmetrical and occurs in young subjects, suggesting a congenital basis. The typical site of involvement is the distal cervical (retrostyloid) segment.

Kinking is often a feature of fibromuscular dysplasia and may also sometimes occur in atherosclerosis. Additional wall disease will augment the haemodynamic effects of the kink. Both coiling and kinking may on their own produce cerebral ischaemic events, particularly in relation to head rotation[66]. If no other cause for the symptoms can be identified, these lesions should be surgically corrected.

An essential principle in surgical correction is full mobilization of the affected segment, and this may require proximal extension of the usual bifurcation exposure. The wall of the kinked or coiled segment may be thin and pathological so that mobilization should proceed with care. Often the loops of the artery are held to each

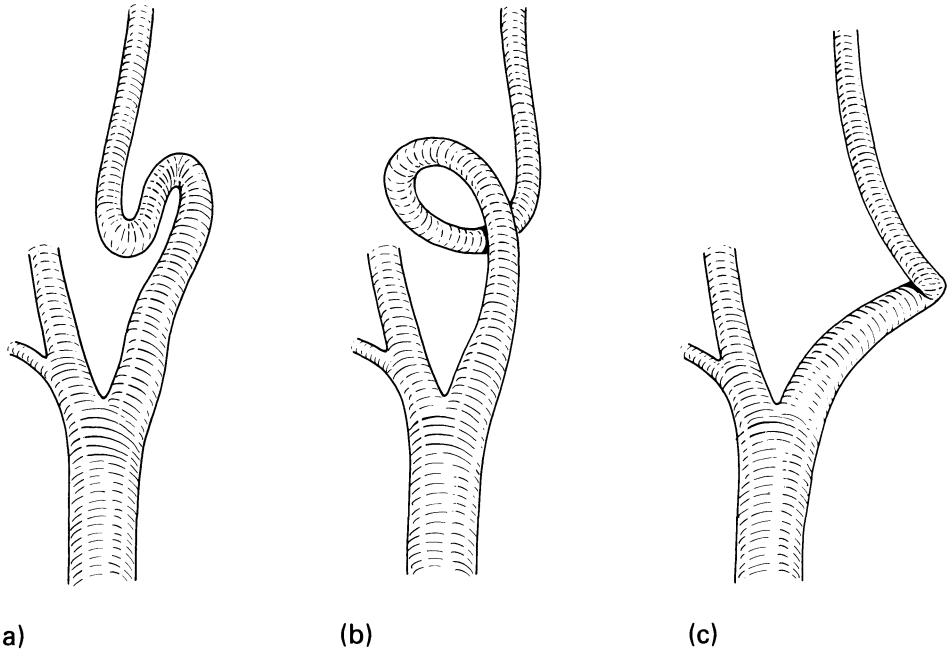
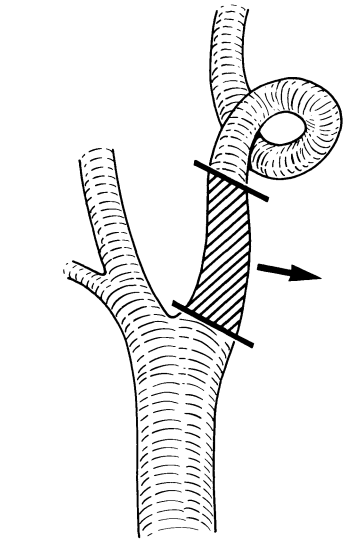


Figure 9.7(a–c) Elongation of the extracranial internal carotid artery: (a) tortuosity (b) coiling (c) kinking

other and to the surrounding tissues by fibrous bands which must be divided before the artery can be straightened out. Once the artery has been freed it is sectioned proximally and held under slight longitudinal tension. A metal bougie is then passed up the vessel thereby restoring normal alignment.

A number of options are then available for dealing with the excess length of artery. The most direct solution is resection with end-to-end re-anastomosis (Figure **9.8a, b**). However, to avoid redundancy the anastomosis must be constructed under a certain amount of tension which in a pathological vessel may lead to intimal splitting and premature stenosis. Insertion of a patch may help to offset this tendency (Figure **9.8c**). A



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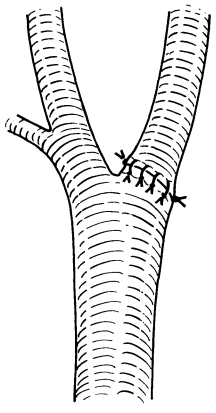

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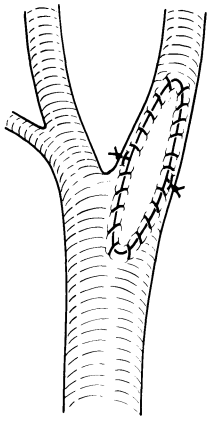


(c)

Figure 9.8(a–c) Coiling of the internal carotid artery: (a) a segment of the internal carotid artery is excised. (b) end-to-end anastomosis with interrupted sutures. (c) anterior vein patch

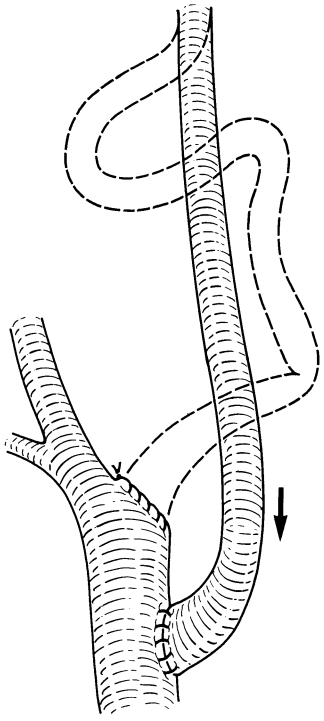


Figure 9.9 Reimplantation of the internal carotid artery

second alternative is to reimplant the internal carotid artery at a more proximal site (Figure **9.9**). However, there may then be similar difficulties in anastomosing the thin-walled and perhaps dysplastic internal carotid artery to the thicker-walled common carotid artery. A third option which avoids the need to suture the internal carotid artery is to resect a portion of the common carotid artery, thereby lowering the bifurcation (Figure **9.10a, b**). The external carotid artery will require to be divided during this process. This may be of little consequence if the contralateral artery is intact and the reconstructed common–internal carotid segment free from coexistent atheroma. However, the most satisfactory method of correcting internal carotid coiling or kinking is probably by a common–internal carotid bypass graft. This is particularly appropriate when there is additional bifurcation atheroma (see Figure **9.5**). Anastomosis of the vein graft to the distal internal carotid artery is usually without problem while at the same time external carotid perfusion is preserved.

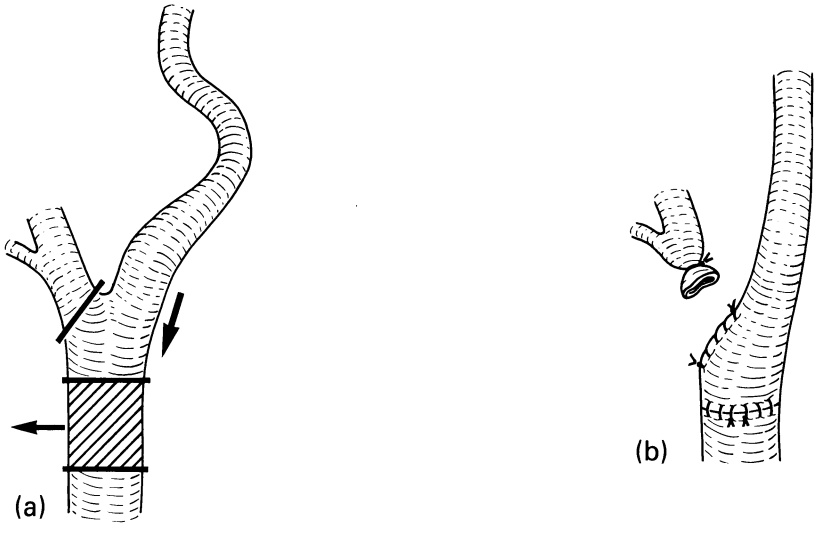


Figure 9.10(a, b) Resection–anastomosis of the common carotid artery

FIBROMUSCULAR DYSPLASIA

This is a condition of unknown aetiology which predominantly affects middle-aged females[67]. The usual site of involvement is the internal carotid artery at C1–2 vertebral level. Four main histological types of fibromuscular dysplasia have been identified: intimal fibroplasia, medial fibroplasia, medial hyperplasia and perimedial dysplasia[68]. By far the commonest type is medial fibroplasia, in which the media is replaced by ridges of fibroproliferative tissue with intervening areas of medial thinning. This gives rise to a 'string of beads' appearance on arteriography (Figure **9.11a**). In addition, the artery is elongated and there may be kinking. Less common arteriographic variants include diffuse tubular narrowing, focal stenosis, macroscopic aneurysm and dissection[69]. Intracerebral aneurysms may coexist in a number of cases and there may be fibromuscular dysplastic lesions elsewhere, particularly in the renal arteries.

Fibromuscular dysplasia may present with hemispheric TIAs, amaurosis fugax or a completed stroke. A bruit may be audible and many patients are aware of a buzzing or roaring noise in the ear. Non-localizing symptoms such as headache, dizziness and syncope may also occur. Although the natural history of carotid fibromuscular disease is largely unknown, the condition is undoubtedly associated with a risk of stroke[70]. Spontaneous dissection may supervene while in other cases the disease may gradually progress and terminate in occlusion.

In view of this background, corrective measures seem advisable when localizing symptoms are present or there is severe arteriographic disease. Patients with non-localizing symptoms can probably be kept under observation. Although carotid revascularization may be thought risky in patients with intracerebral aneurysms, this fear appears to be largely unfounded[71].

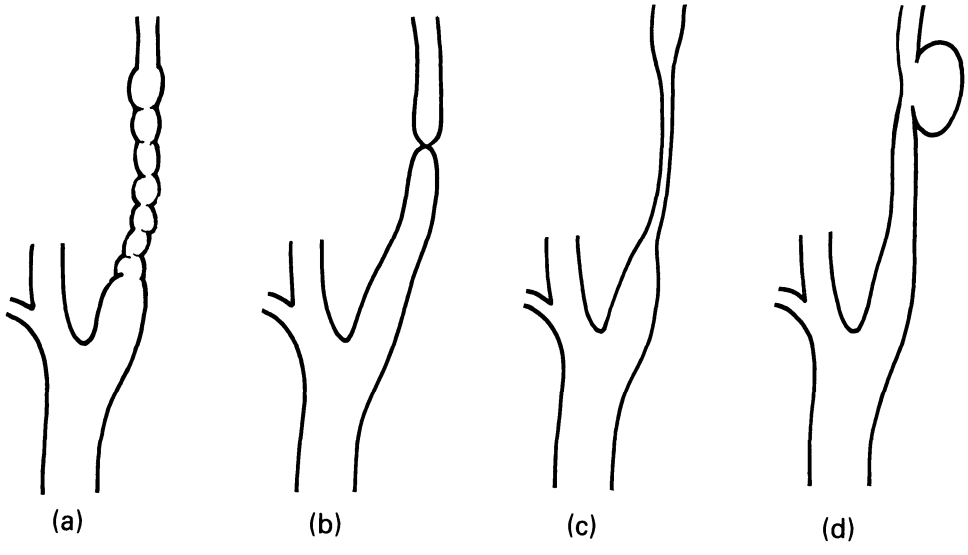


Figure 9.11(a–d) Arteriographic variants of fibromuscular dysplasia: (a) segmental stenoses and dilatations (b) isolated annular stenosis (c) long tubular stenosis (d) dissecting aneurysm

Where the disease is limited to the surgically accessible internal carotid artery, resection with vein graft replacement may be the treatment of choice. However, in most cases the disease extends more distally towards the base of the skull and graduated intraluminal dilatation is a better option[72]. Full mobilization of the internal carotid artery up to the level of the styloid process is an essential preliminary step. If the artery is not straightened out there is a serious risk of perforation during subsequent instrumentation. The patient is then heparinized and a transverse arteriotomy made in the distal common carotid artery or the internal carotid bulb (Figure **9.12**). A 2 mm lubricated dilator is then passed gently up the internal carotid artery to the base of the skull. Some resistance may be encountered at each stenosis but gentle pressure should ensure onward passage of the dilator. Progressively larger dilators are used up to 5 mm size. On completion the largest dilator should slide smoothly up to the base of the skull without resistance. The distal artery is back-bled to flush out any debris and after rinsing the lumen the arteriotomy is closed with interrupted sutures.

More recently, percutaneous transluminal angioplasty has been applied to the management of fibromuscular dysplasia[73]. Although there may be a theoretical risk of embolization, this may be less than in atheromatous disease and in skilled hands angioplasty may be an alternative to surgery, particularly when there are operative risk factors.

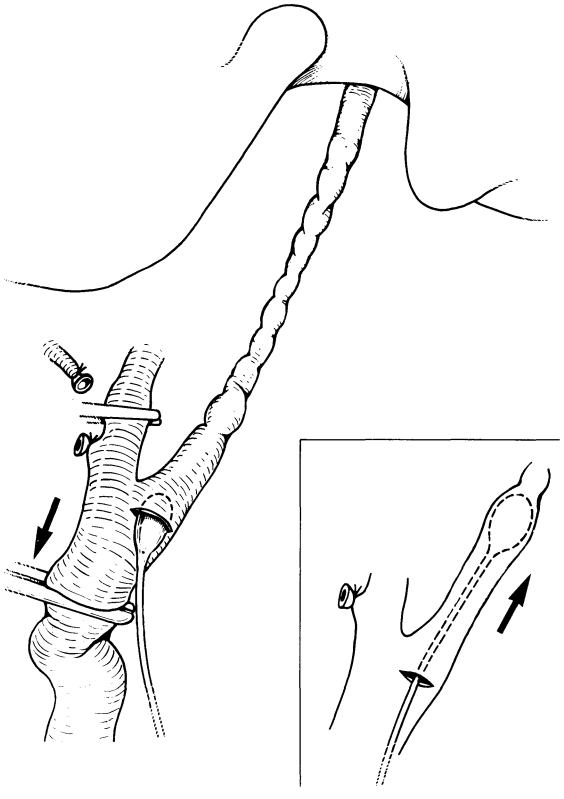


Figure 9.12 Graduated intraluminal dilatation. Caudal traction straightens out the internal carotid artery prior to passage of dilators (inset)

SPONTANEOUS DISSECTION OF THE INTERNAL CAROTID ARTERY

Spontaneous dissection of the extracranial internal carotid artery, though rare, is one of the most important causes of stroke in young subjects. Dissection may occur as a result of degenerative changes in the media with increased mucoid deposition[74] or it may be associated with more specific arterial pathology such as fibromuscular dysplasia, atherosclerosis, cystic medial necrosis or arteritis. Blunt cervical trauma may also initiate internal carotid dissection[75]. Indeed, minor and perhaps unnoticed injury may be implicated in a significant number of apparently spontaneous dissections.

The presenting symptoms include hemispheric or retinal TIAs, ipsilateral headache, facial or neck pain and a subjective bruit. Physical signs may include a hemiparesis or visual defect, a cervical bruit and a Horner's syndrome. Occasionally vagal or hypoglossal nerve palsies may occur if the internal carotid artery is involved near the base of the skull[76].

Arteriography typically reveals a normal proximal internal carotid artery with a narrowing just beyond the bulb which extends to the base of the skull ('string sign'). Less commonly there may

be segmental narrowing or intimal irregularity beyond the bulb, an aneurysmal outpouching near the base of the skull or a postbulbar occlusion (Figure **9.13a–c**).

Management is governed by the clinical presentation and the arteriographic findings. An initial non-operative approach may be advisable in many patients since spontaneous improvement may occur in time, with resorption of intramural thrombus and restoration of normal patency[77]. Antiplatelet drugs or anticoagulants may be prescribed to reduce the risk of embolization in the interim period providing there is no evidence of recent infarction. Drugs such as propanolol or hydralazine may also be useful in arresting the dissection process[75].

Those patients who have recurrent or persistent ischaemic symptoms or who have arteriographic evidence of either serious intimal disruption or progressive aneurysmal expansion should undergo surgery. The treatment of choice for accessible dissections is resection and vein graft replacement. If necessary, the distal false lumen can be obliterated by sewing the inner septum to the outer wall prior to distal graft attachment. Dissections which give rise to intimal disruption or aneurysmal outpouching near the base of the skull may be difficult to manage. A cervical approach may still be possible in some cases after mandibular osteotomy or styloid resection (see internal carotid aneurysms). Where the lesion is too distal for cervical repair, extra-cranial–intracranial bypass with internal carotid ligation may be undertaken.

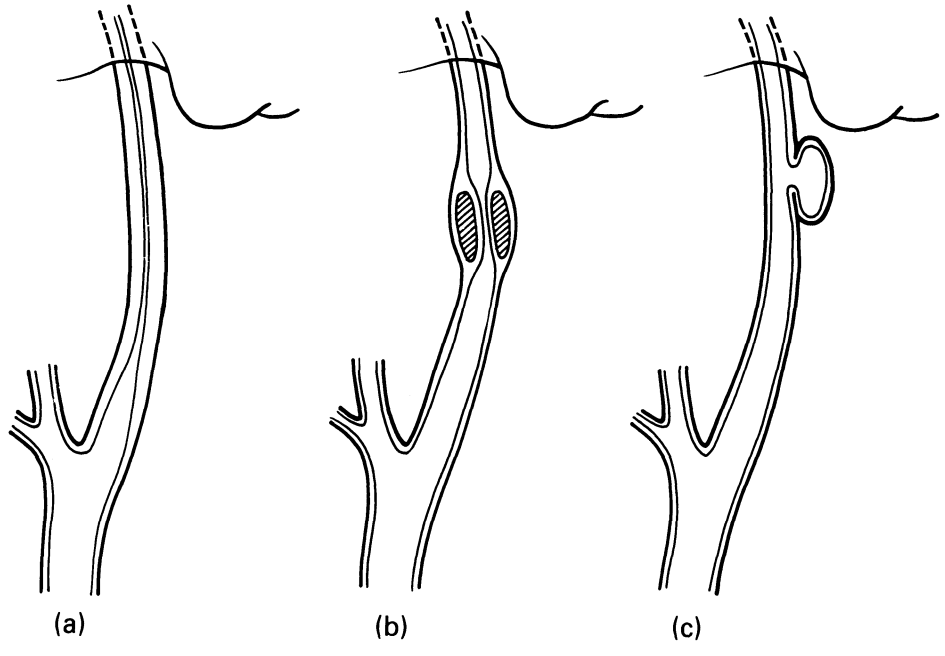


Figure 9.13(a–c) Spontaneous dissection of the internal carotid artery: (a) postbulbar narrowing with extension to the base of the skull ('string sign'). (b) segmental narrowing. (c) aneurysmal outpouching

EXTRACRANIAL CAROTID ARTERY ANEURYSMS

Aneurysms of the extracranial carotid artery are distinctly uncommon. Most cases are due to atherosclerosis, trauma or previous carotid surgery[78,79]. Rarer causes include bacterial infection, fibromuscular dysplasia, arteritis, dissection and congenital anomalies. Morphologically three types of aneurysm may be recognized: a fusiform variety which occurs at the bifurcation; saccular, which may arise from the common or internal carotid artery; and dissecting aneurysm, which typically affects the distal internal carotid segment (Figure **9.14a–c**).

Carotid artery aneurysms usually present as a symptomless swelling in the neck, though in a number of cases a transient ischaemic attack or stroke may first draw attention to the lesion. Sudden stroke either from embolization or thrombosis is the main risk posed by carotid artery aneurysms though local compression and rupture may also occur. In view of these possible complications surgery is advisable in virtually all cases.

Atherosclerotic aneurysms at the carotid bifurcation may be approached by a standard cervical incision. These lesions contain a variable amount of atherothrombotic debris and it is essential to avoid handling the aneurysm until the distal internal carotid artery has been clamped.

After heparinization, clamps are applied to the internal, external and common carotid arteries and the aneurysm is opened. Thrombus is evacuated and the external carotid origin oversewn. A

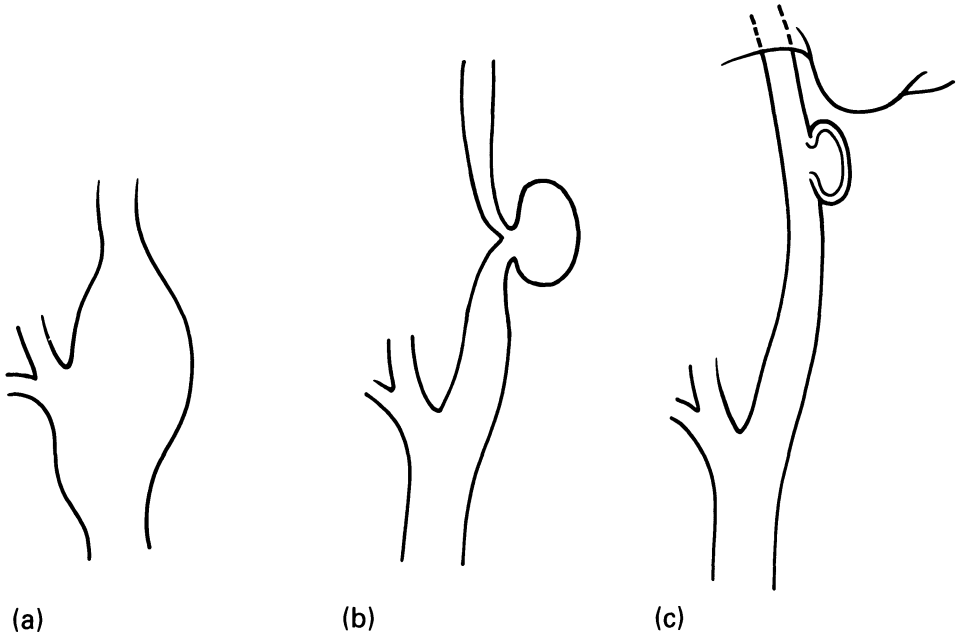


Figure 9.14(a–c) Varieties of extracranial carotid artery aneurysm: (a) fusiform (b) saccular (c) dissecting

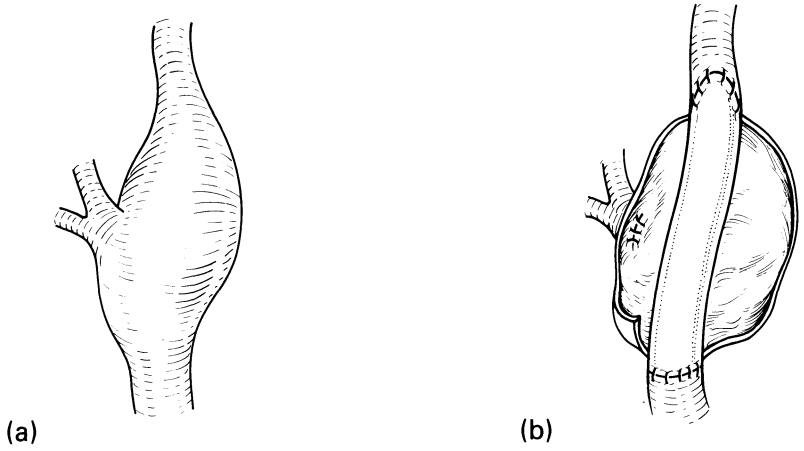


Figure 9.15(a, b) Atherosclerotic fusiform aneurysm at the carotid bifurcation: (a) extent of lesion. (b) the sac has been laid open and a PTFE graft inserted

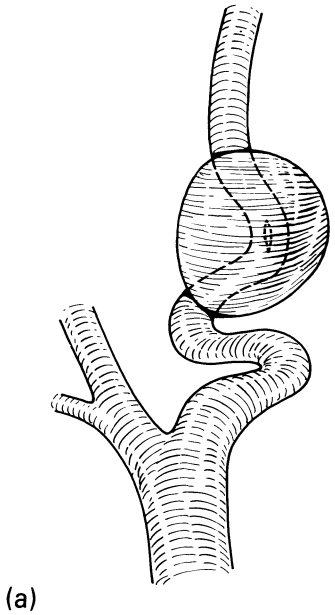
PTFE graft or a segment of saphenous vein is then interposed between the common and internal carotid arteries (Figure **9.15a, b**). If the internal carotid artery is occluded, the external carotid artery is mobilized beyond the aneurysm and a common–external carotid bypass is inserted. In either case, the distal anastomosis should be suitably spatulated to allow for any discrepancy in size between the graft and the distal artery. If there is sufficient redundant sac this may be wrapped around the prosthesis on completion.

Saccular aneurysms of the internal carotid artery can often be managed by excision and direct re-anastomosis since the adjacent arterial axis is usually tortuous and the neck of the aneurysm small (Figure **9.16a, b**). To avoid stenosis, the vessel ends should be spatulated and anastomosis performed with fine interrupted sutures. An anterior vein patch may also sometimes be useful.

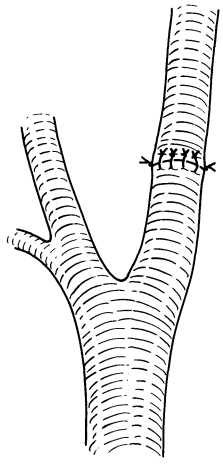
Distal internal carotid artery aneurysms present special problems in access. One approach is to extend the incision used for bifurcation exposure upwards and backwards onto the mastoid process. The sternomastoid insertion is then partially detached, carefully preserving the spinal accessory nerve, and the posterior belly of digastric is divided. If necessary, the tip of the mastoid may be detached to enlarge the operative field. After reflecting the parotid gland superiorly, the styloid process is sectioned at its base and displaced anterolaterally along with its attached muscles and ligaments. This exposes the internal carotid artery and internal jugular vein at the base of the skull. The glossopharyngeal, vagus, accessory and hypoglossal nerves lie in close proximity and will require gentle reflection to isolate the artery.

Alternative methods of exposing the distal cervical portion of the internal carotid artery include mandibular osteotomy with outward rotation of the posterior fragment[77], anterior dislocation of the mandible[80] and burring of the mastoid and inferior part of the auditory canal[81].

Distal control of high carotid aneurysms may be achieved by endoluminal balloon catheter or



(a)



(b)

Figure 9.16(a, b) Saccular aneurysm of the internal carotid artery: (a) lesion (b) excision with end-to-end re-anastomosis

via a stent impacted in the carotid foramen[82]. The aneurysm is then replaced with a vein graft. The distal anastomosis should be completed first, using interrupted sutures and a parachute technique as previously described. Proximally the graft is attached end-to-side to the common carotid artery with oversewing of the internal carotid origin.

Aneurysms in contact with the carotid canal and which cannot be mobilized from the neck may be treated by extracranial–intracranial bypass with exclusion of the aneurysmal zone.

CAROTID BODY TUMOURS

Tumours of the carotid body present as a slowly enlarging cervical mass. Some patients with large tumours may have additional local pain, dysphagia from pressure on the pharynx or hoarseness from vagal involvement. At first the tumour lies in the angle between the external and internal carotid arteries, but ultimately engulfs one or both vessels, particularly the internal carotid artery. However, even with large tumours, a subadventitial dissection plane is usually preserved between the tumour and the rest of the artery wall, permitting surgical removal.

In a small number of patients, the tumour has malignant characteristics with penetration of the media and invasion into surrounding structures, particularly the internal jugular vein and the hypoglossal or vagus nerves. Metastases to regional lymph nodes or bones may also occur. These features are a more reliable indication of malignancy than the histology, which is often little different from the vast majority of tumours which pursue an entirely benign course[83].

Arteriography or B-mode scanning are diagnostic and will in particular distinguish carotid body tumours from metastatic lymph nodes overlying the carotid bifurcation. Occasionally displacement or distortion of the carotid vessels or hypervascularity may suggest the presence of an invasive type of tumour. Arteriography is also of value in excluding coexistent bifurcation atheroma and in delineating the status of the circle of Willis and the contralateral carotid axis.

Surgical removal is advisable in all cases unless there are prohibitive medical conditions. If these lesions are not removed they will eventually kill the patient by local compression or extension into the base of the skull[84]. Removal is best undertaken at an early stage since large tumours pose a greater risk of cranial nerve injury, intraoperative haemorrhage and neurological deficit[85].

Operation should be carried out with the possible need for carotid arterial reconstruction in mind. Prepping and draping should therefore include the distal thigh as well as the neck in case a saphenous vein graft is required. A standard approach is made to the carotid bifurcation. In most cases the internal jugular vein can be separated from the tumour and retracted posteriorly once the common facial vein has been divided. Where the tumour is adherent to the internal jugular vein an en bloc resection should be carried out. The internal and external carotid arteries are then mobilized beyond the tumour, if necessary by transecting the posterior digastric muscle. Prior identification of the hypoglossal nerve may avoid inadvertent injury, though with large tumours the nerve may be hidden at this stage of the procedure.

Dissection in the subadventitial plane is then commenced at the lower pole of the tumour (Figure **9.17a**). This plane is developed along the lateral aspect of the internal carotid artery and the tumour progressively split away from the arterial axis (Figure **9.17b**). A similar maneouvre is carried out along the anteromedial surface of the external carotid artery after dividing the superior thyroid branch. In some cases it may be helpful to divide the external carotid artery itself to allow access to the deep aspect of the tumour. However, care should then be taken to avoid undue rotation of the tumour about its longitudinal axis, as this may occlude the internal carotid artery.

Once the internal and external carotid arteries have been freed, the tumour should remain attached only at the bifurcation (Figure **9.17c**). Adherence tends to be particularly marked at this site and the carotid body artery must be divided before tumour separation can be completed (Figure **9.17d**). After removal of the tumour the internal carotid artery may go into spasm. This may be corrected by briefly clamping the arterial axis and gently passing a dilator up the internal carotid artery via a short proximal arteriotomy.

If the internal carotid artery is damaged during

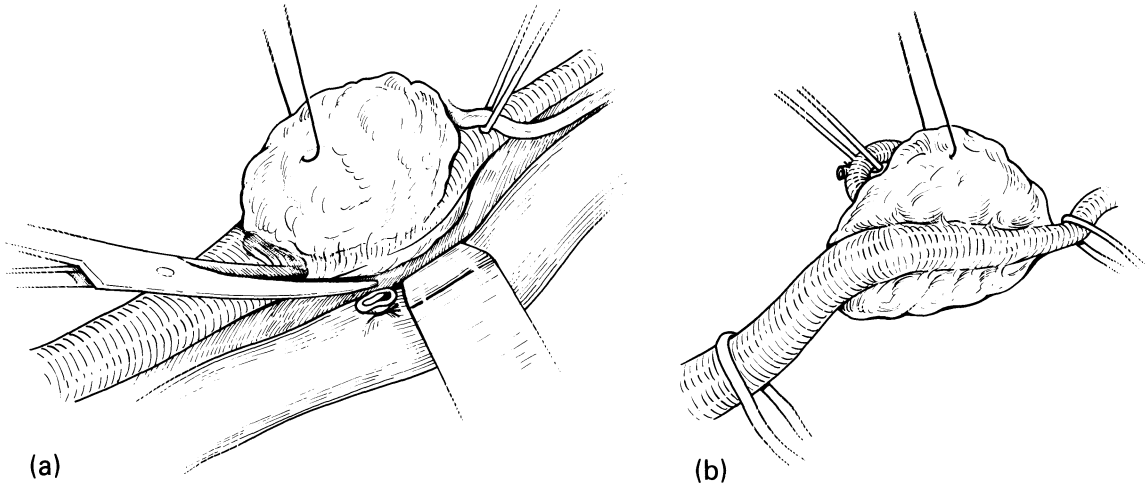


Figure 9.17(a–d) Excision of a carotid body tumour: (a) subadventitial dissection commenced on the anterior surface of the common carotid artery. The hypoglossal nerve has been looped beforehand. (b) the tumour is progressively split away to expose the lateral surface of the internal carotid artery

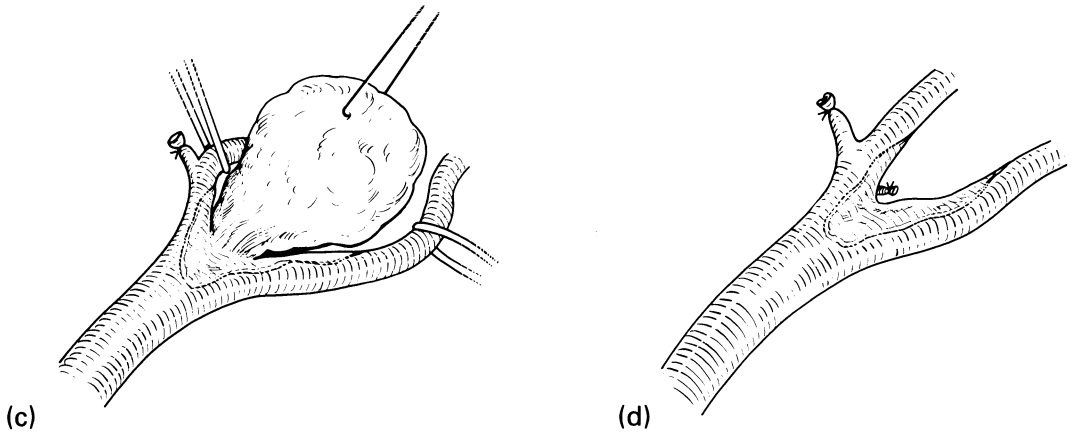


Figure 9.17 continued (c) the external carotid artery has been freed leaving the tumour attached only at the bifurcation. (d) the carotid body artery is ligated and tumour removal completed

tumour separation, clamps should be applied above and below and tumour removal completed. The artery is then re-anastomosed or repaired with a patch or vein graft, as appropriate.

Invasive tumours require a more aggressive approach. After initial dissection to achieve vascular control the patient should be heparinized and clamps applied to the internal and external carotid arteries beyond the tumour and to the common carotid artery proximally. In the case of large tumours it may be difficult to apply clamps distally and endoluminal control may be necessary. An en bloc resection of the tumour and carotid bifurcation is then carried out together with any metastatic lymph nodes that are present. This excision will require sacrifice of the vagus and hypoglossal nerves if these are involved in the tumour. Vascular continuity is then restored by means of a common–internal carotid vein graft.

Tumours which are invading the base of the skull should be regarded as inoperable. Partial excision is of no real benefit and although radiotherapy may be utilized in some cases, the response may be rather limited[85].

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10

Aortic Arch and Vertebral Revascularization

VERTEBRAL ARTERY SURGERY

Although vertebral artery reconstruction is not as widely undertaken as carotid bifurcation surgery, it nevertheless may play a significant role in the treatment of cerebrovascular disease. The chief objective of operations on the vertebral artery is the relief of symptoms of vertebrobasilar insufficiency. These most commonly consist of vertigo, visual disturbances such as bilateral blurring or diplopia, ataxia and drop attacks. Sensory disturbances or motor loss may occur on one side of the body or there may be bilateral alternating attacks.

The most important vertebral artery pathology is atherosclerosis. This typically produces a smooth non-ulcerated stenosis involving the ostium or first few millimetres of the vessel with normal intima beyond. A variable degree of atherosclerosis is present in the subclavian artery adjacent to the vertebral ostium. Occasionally the distal vertebral artery or basilar trunk may be stenosed either alone or in tandem with ostial disease, while a minority of patients have diffuse lesions throughout the vertebral artery. As with atheromatous disease elsewhere, thrombosis may supervene in the presence of a severe ostial stenosis. This may not necessarily render the condition inoperable, since distal vertebral patency may be maintained by collateral flow from the ascending cervical, deep cervical and occipital arteries.

Other vertebral artery lesions which may cause vertebrobasilar ischaemia include kinking[1], fibromuscular dysplasia[2], arteritis[3] and spontaneous dissection[4]. Symptoms may also occur from extrinsic compression of the vertebral artery by cervical spine osteophytes, fibrous bands or sympathetic nerve fibres[5]. However, in many of these cases ostial atheroma is also present and may contribute significantly to symptomatology.

Patients with vertebrobasilar ischaemia should undergo complete arteriographic evaluation of the extracranial and intracranial circulation. Arch injection with selective catheterization or intra-arterial digital subtraction may be utilized[6,7]. Vertebral ostial lesions are easily overlooked on AP films and oblique views are essential. Even so, it is sometimes difficult to visualize the vertebral origin and indirect evidence of a significant stenosis should be sought, such as post-stenotic dilatation, notching of the adjacent subclavian wall, or delayed basilar opacification. Cervical collateral

vessels may also be present if the stenosis is very severe or there is proximal vertebral artery occlusion.

Many patients with vertebral artery stenosis have additional disease at the carotid bifurcation or in the brachiocephalic trunks[8,9]. Correction of these lesions should take priority, though particularly severe vertebral artery stenoses should be corrected concomitantly because of the risk of perioperative occlusion. Carotid endarterectomy will relieve hindbrain ischaemic symptoms in most,[10,11] but not all,[12,13] cases providing the internal carotiod lesion is severe. The small number of patients who remain symptomatic after carotid artery surgery and who have demonstrable vertebral artery narrowing may then be considered either for percutaneous transluminal angioplasty (see Chapter 15) or direct vertebral artery surgery.

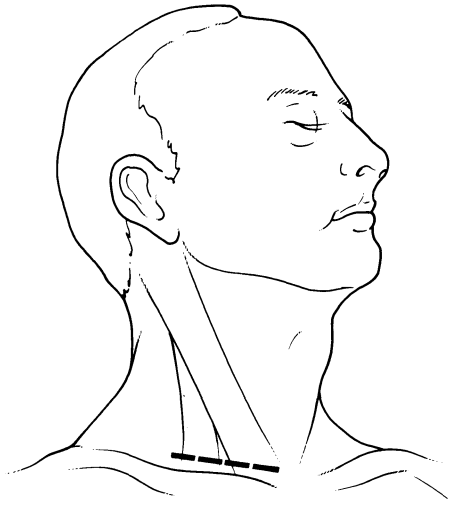
In the absence of carotid bifurcation disease, vertebrobasilar symptoms usually develop only if flow is restricted in both vertebral arteries[14]. This may result from bilateral vertebral ostial stenoses, unilateral stenosis and contralateral hypoplasia or occlusion, and various combinations of vertebral and proximal subclavian disease. An isolated unilateral vertebral artery stenosis is rarely symptomatic. The occurrence of symptoms in such cases should prompt a careful search for a second lesion in the distal vertebrobasilar circulation or for the congenital anomaly in which the vertebral artery opens into the posterior inferior cerebellar artery instead of the basilar trunk. Where there is obvious bilateral vertebral ostial narrowing, surgical priority should be given to the most severe stenosis and to the dominant artery.

The value of vertebral artery surgery in asymptomatic disease is controversial since the stroke potential of such lesions is largely unknown. Although clinical experience would suggest that the stroke risk is low, vertebral occlusion has been associated with a significant incidence of hindbrain infarction in autopsy series[15]. Our preference, therefore, is to undertake prophylactic reconstruction of vertebral ostial stenoses when they are either very severe (90%) or associated with bilateral internal carotid artery occlusions.

EXPOSURE OF THE VERTEBRAL ARTERY

The patient is placed in the supine position with a sandbag beneath the cervicodorsal spine and the head turned to the opposite side (Figure **10.1a**). A horizontal skin incision is made just above and parallel with the medial half of the clavicle. The platysma and both heads of sternomastoid are then divided to expose the triangular area bounded by the infrahyoid muscles medially, the omohyoid muscle above and laterally, and the clavicle below. The investing layer of the cervical fascia is then incised and the internal jugular vein mobilized longitudinally. Adequate mobilization of the latter may require division of the omohyoid muscle and the middle thyroid vein. The common carotid artery is similarly mobilized and by appropriate retraction the jugulocarotid interval is developed (Figure **10.1b**).

A layer of lymphoadipose tissue is then encountered overlying the vertebral vessels. On the left, the thoracic duct may be identified as a single channel but often multiple lymphatics are present, which must be carefully ligated and divided. A similar procedure is carried out on the right side.



(a)

Figure 10.1(a–c) Exposure of the vertebral artery: (a) skin incision

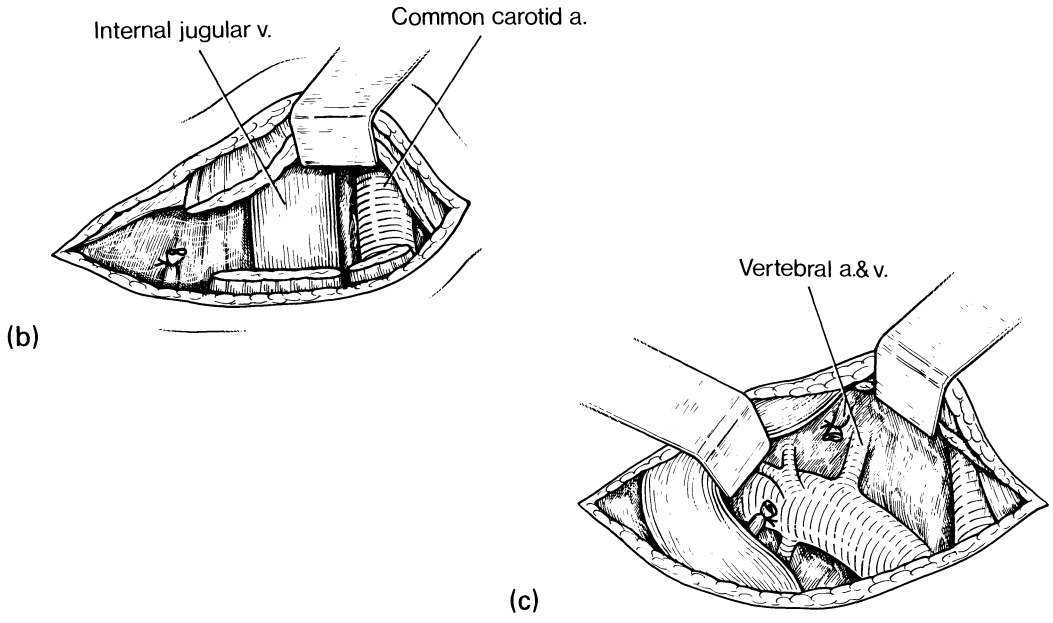


Figure 10.1 continued (b) sternomastoid divided. (c) the vertebral artery is exposed in the interval between the internal jugular vein and the common carotid artery

Deep to the lymphatic layer is the vertebral vein, which is the key to exposing the vertebral artery. Once this vein has been divided, or preferably resected, the artery can be located immediately behind (Figure **10.1c**). The latter is then traced down to its origin on the posterosuperior aspect of the subclavian artery. On the left the origin is invariably lower than on the right and a more extensive dissection is required.

The subclavian artery is then mobilized either side of the vertebral origin, though if a reimplantation of the vertebral artery into the common carotid artery is to be carried out, this may not be necessary. Silastic loops should be passed around the subclavian artery with care since this vessel is notoriously fragile. On the left, the costocervical trunk sometimes arises very close to the vertebral origin and may need to be divided to avoid avulsion during encirclement. Usually the thyrocervical trunk and the internal mammary artery can be preserved, though they should be divided if access is limited. The same comments apply to scalenus anterior.

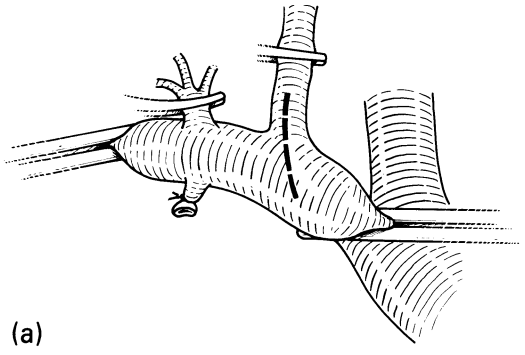
If an endarterectomy is planned, it is sufficient to clear the proximal 2–3 cm only of the vertebral artery. However, if a reimplantation is envisaged the artery must be fully mobilized up to the intertransverse canal. This additional dissection is necessary to ensure that the artery will lie in a smooth curve to its attachment site on the common carotid artery. The distal artery is encircled by sympathetic fibres passing between the stellate and middle cervical ganglia and these will need to be divided during this distal display. A mild Horner's syndrome may result. In addition it is often necessary to divide the inferior thyroid artery which crosses the operative field.

VERTEBRAL ENDARTERECTOMY

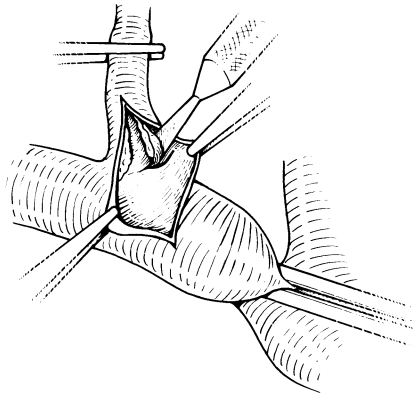
After heparinization, a microvascular Heifetz clip is applied to the vertebral artery well clear of the diseased segment. This type of clip is preferred since heavier clamps are liable to damage the intima of this delicate vessel. The subclavian artery is then clamped a suitable distance away from the vertebral origin and the other branches controlled as necessary. A curved transverse incision is then made in the subclavian artery and extended into the vertebral artery beyond the stenotic zone (Figure **10.2a**). A cleavage plane is developed at the edge of the ostial plaque and continued into the wall of the vertebral artery (Figure **10.2b**). A transmedial plane should be used if possible (see Chapter 1). At the distal end point the atheromatous lesion should separate as a thinly feathered tip, leaving an adherent edge of minimal thickness.

The lumen is then irrigated with heparin–saline and after checking the security of the subclavian intima, the arteriotomy is closed with a small vein patch (Figure **10.2c**). The vertebral back-bleed is used to expel air from the operated segment and antegrade flow is released firstly into the arm and then into the vertebral artery.

An alternative method of endarterectomy utilizes a trans-subclavian eversion technique (Figure **10.3a–c**). A longitudinal or slightly curved subclavian arteriotomy is made opposite the vertebral origin. The subclavian intima is incised circumferentially around the vertebral ostium and the plaque developed in a centripetal fashion into the vertebral artery. The latter is progressively everted into the subclavian lumen until the end point is reached, when the plaque should cleanly detach (Figure **10.3b**). After the usual precautions of flushing and irrigation, the subclavian arteriotomy is closed directly, or with a small patch if the edges are particularly friable (Figure **10.3c**).



(a)



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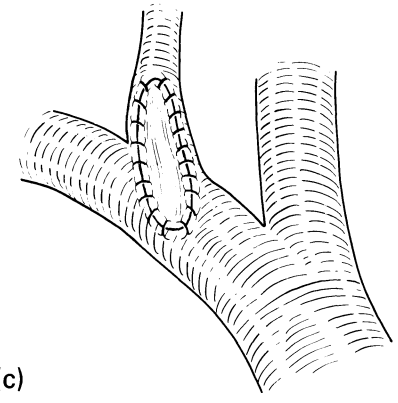


(c)

Figure 10.2(a–c) Open endarterectomy of the vertebral artery: (a) arteriotomy. (b) plaque separation commenced at the vertebral ostium. (c) the endarterectomy has been completed and a vein patch inserted

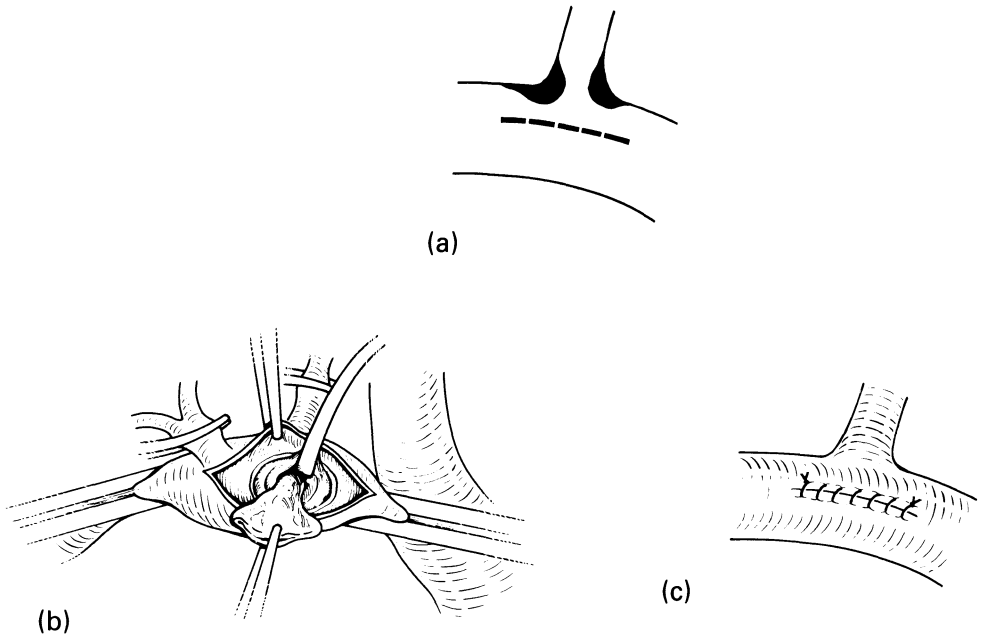


Figure 10.3(a–c) Trans-subclavian endarterectomy: (a) arteriotomy. (b) eversion endarterectomy of the vertebral orifice. (c) direct closure

This method has the advantage of avoiding an incision in the vertebral artery, but control of the distal end point is less certain than with open endarterectomy. In addition, security of the subclavian intima may cause problems necessitating a formal endarterectomy even though the vessel is not grossly diseased.

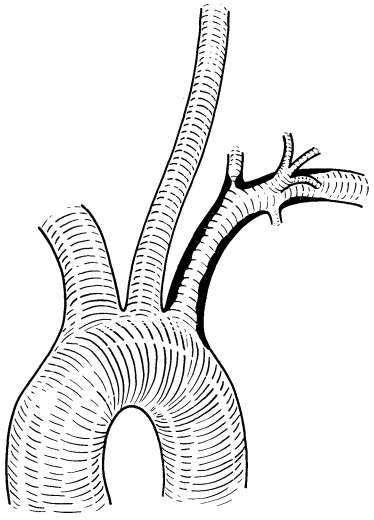
REIMPLANTATION OF THE VERTEBRAL ARTERY

Reimplantation of the vertebral artery into the common carotid artery is an alternative method of managing patients with vertebral ostial stenosis[16]. The subclavian artery is a relatively difficult vessel with which to work, and in many patients vertebral transposition is more satisfactory than endarterectomy. Reimplantation is particularly indicated when the vertebral origin is low, as is commonly the case on the left (an aortic arch origin is an extreme example), or when vertebral ostial stenosis is associated with kinking. Patients with vertebral stenosis and diffuse subclavian atherosclerosis may also be best managed by this approach since endarterectomy is particularly likely to leave an unsatisfactory distal intimal edge in the subclavian artery (Figure **10.4a**).

Although vertebro–carotid reimplantation requires simultaneous carotid and vertebral artery clamping, this is quite safe in the great majority of patients. However, certain circumstances may be identified where this method is best avoided. These include the presence of a contralateral internal carotid occlusion with a normal ipsilateral carotid axis, an ipsilateral internal carotid occlusion with limited hemispheric perfusion via the external carotid artery, and thirdly a severe ipsilateral siphon stenosis.

TECHNIQUE

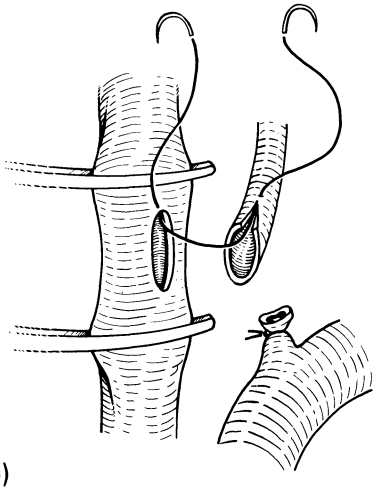
The vertebral artery is exposed in the jugulocarotid interval and then fully mobilized up to the intertransverse canal, as previously described. A suitable length of adjacent common carotid artery is then cleared for anastomosis. This may require partial transection of the infrahyoid muscles which overlap the vessel from in front.



(a)

After heparinization, a Heifetz clip is placed on the vertebral artery distally and the vessel is then clamped and divided immediately beyond the stenosis. The proximal stump is ligated and the distal end placed against the common carotid artery in order to select the optimum anastomosis site. It may be helpful to place the patient's head in various positions of flexion or rotation to ensure that the transposed vessel will be free from kinking or angulation. Rarely the atherosclerotic process extends beyond the vertebral ostium and the distal undiseased segment is too short to reach the common carotid artery comfortably. An autogenous vein graft should then be interposed between the two vessels. This is also a useful fallback measure following a technically unsatisfactory vertebral endarterectomy.

Once the anastomosis site is selected, the common carotid artery is cross-clamped and the clamps rotated anteriorly to present the posterolateral surface for anastomosis. An ellipse of artery wall (or alternatively a small triangular segment with the base anteroinferiorly) is excised here and a corresponding cutback made in the



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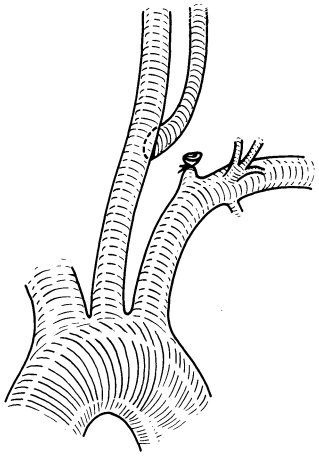


(c)

Figure 10.4(a–c) Vertebro–carotid reimplantation: (a) vertebral ostial stenosis with diffuse subclavian thickening. (b) an arteriotomy is made on the posterolateral wall of the left common carotid artery; the transposed vertebral artery is spatulated prior to anastomosis. (c) completed reimplantation

anteromedial wall of the vertebral artery (Figure **10.4b**). An end-to-side anastomosis is then constructed using a continuous intraluminal suture of 6/0 or 7/0 prolene posteriorly and interrupted sutures along the anterior wall (see intraluminal anastomosis, Chapter 1). Occasionally a small vein patch may be used to enlarge the anastomosis. The common carotid artery is nearly always rather thick-walled and delicate suturing is required during vertebral artery attachment. Optical magnification may also be helpful.

Before completing the suture line the clamps are flushed and the lumen well irrigated with heparin–saline. Air is then evacuated using the vertebral artery back-bleed while the last sutures are tied. Antegrade flow is released into the vertebral artery and then into the carotid axis (Figure **10.4c**).

In an alternative transposition technique, the vertebral artery is divided beyond the stenosed ostium and reimplanted at an adjacent site on the subclavian artery[9]. This method avoids the risk of carotid embolization from a faulty vertebro–carotid suture line or from an inadequate flushing technique, but suffers from the disadvantage that the subclavian artery itself is often diseased and liable to disease progression especially on the left side. In addition, unless the vertebral origin is low or there is additional tortuosity, the transposed vessel may be rather short in length.

SUBCLAVIAN–VERTEBRAL BYPASS

The vertebral artery may also be revascularized by means of a vein bypass graft placed end-to-side between the postvertebral subclavian artery and the vertebral artery beyond the stenotic zone[17]. Distal anastomosis is rather difficult and graft length may be difficult to gauge. As a result, this method is only used when reimplantation is not feasible.

PERCUTANEOUS TRANSLUMINAL ANGIOPLASTY

Vertebral ostial atheroma, unlike that at the carotid bifurcation, is nearly always a nonulcerative fibrous lesion. As a result, transluminal dilatation may be undertaken with little risk of cerebral embolization[18] (see Chapter 15). However, this approach may sometimes be limited by technical considerations, particularly the small vessel size and angle of origin. Relatively few patients have so far been managed by transluminal dilatation, but this method has obvious appeal in patients with risk factors.

REVASCULARIZATION OF THE DISTAL VERTEBRAL ARTERY

Involvement of the intertransverse portion of the vertebral artery by extrinsic compression, trauma, fibromuscular dysplasia or dissection may very occasionally necessitate a distal vertebral artery reconstruction. This is best achieved by means of a vein bypass graft from the common carotid artery attaching to the vertebral artery between C1 and C2 vertebrae.

Access to the distal cervical portion of the vertebral artery is provided by a pre-sternomastoid incision extending back onto the mastoid process. The internal jugular vein is mobilized and held forwards while sternomastoid is retracted backwards to expose the transverse process of C1 vertebra. This landmark may be easily recognized by palpation. The muscles attaching to the lower border of the transverse process are then divided and the vertebral artery identified as it ascends from the transverse process of C2 vertebra. The anterior ramus of C1 nerve lies in front of the artery and may need to be divided to obtain an adequate site for graft attachment.

A segment of saphenous vein is then harvested from the distal thigh and anastomosed to the exposed vertebral artery. End-to-side graft attachment is technically easier than end-to-end at this level. The anastomosis should be undertaken with optical magnification using fine continuous or interrupted sutures. The graft is then brought down to mid-cervical level and after careful estimation of length, attached end-to-side to the common carotid artery.

SUBCLAVIAN ARTERY RECONSTRUCTION

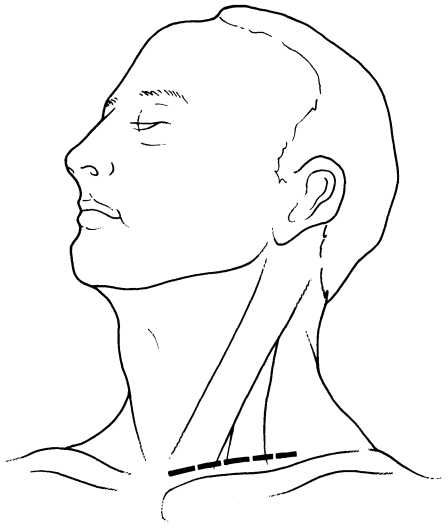
Occlusive disease in the subclavian arteries is relatively common and in most surgical series reconstruction for left subclavian stenosis or occlusion outnumbers other brachiocephalic reconstructions by a considerable margin. On the left side, atherosclerosis may develop at any point from the aortic arch to the vertebral origin. On the right, the principal lesion is usually much shorter and located at the vessel origin. The vertebral artery may be additionally involved on either side though it is uncommon for disease to extend significantly into the postvertebral segment.

Many patients with subclavian occlusive disease are asymptomatic and never require surgical treatment. Others develop hindbrain or upper limb ischaemia or symptoms in both territories. When cerebral symptoms are present, there are often additional lesions at the carotid bifurcation or in the contralateral vertebrosubclavian axis. Subclavian lesions produce hindbrain symptoms by directly impairing antegrade vertebral perfusion. Although a radiological subclavian steal is often demonstrable in these cases this is not thought to have much clinical relevance[19,20]. Upper limb symptoms include heaviness or ache in the arm on use, coldness, numbness and paraesthesiae. More severe distal ischaemia manifest by digital pain and necrosis is very uncommon and usually seen only with ulcerative or aneurysmal subclavian lesions which have given rise to embolization (see also Chapter 11). Patients who have had previous reconstructive surgery may also develop severe ischaemia due to loss of collaterals around the shoulder.

CAROTID–SUBCLAVIAN BYPASS

This is a useful method of revascularizing left subclavian lesions providing the vertebral artery is open. Transthoracic subclavian endarterectomy has given durable results in some hands but cervical bypass is simpler and is attended by minimum morbidity and mortality[21,22]. An essential prerequisite is that the proximal common carotid artery should be free from occlusive disease and the graft inflow therefore unrestricted. In addition the carotid bifurcation should be widely patent to avoid a possible carotid steal. Graft material may be PTFE, Dacron or autogenous vein. Vein grafts have been subject to premature occlusion in some reports and this has been attributed to kinking with head or shoulder movement[19,20]. However, our own experience has been more favourable and both vein and PTFE grafts are in current usage.

Access may be achieved via a horizontal supraclavicular incision in which platysma and both heads of sternomastoid are divided (Figure **10.5a**). The pre-scalene fat pad is then dissected from below upwards to expose scalenus anterior and the phrenic nerve. The latter is looped and held aside while scalenus anterior is sectioned low down near its insertion into the first rib (Figure **10.5b, c**). The usual site of graft attachment is distal to the main subclavian branches and the artery should therefore be gently mobilized as far distally as possible behind the clavicle. The dorsal scapular artery may arise from this segment and



(a)

Figure 10.5(a–f) Carotid–subclavian bypass: (a) skin incision

should be divided to improve mobility (Figure **10.5c**).

The internal jugular vein is then freed longitudinally so that it can be subsequently retracted in either direction. The thoracic duct and other lymphatic vessels usually need to be ligated and divided during this stage. The carotid sheath is then opened, taking care to preserve the vagus nerve, and a segment of the common carotid artery freed for anastomosis.

After heparinization, the common carotid artery is cross-clamped as low down as possible and an arteriotomy is made on the lateral aspect. A vein or PTFE graft is then attached in end-to-side fashion with the tip directed inferiorly (Figure **10.5d**). The proximal and distal artery segments are then carefully flushed via the unattached end of the graft to avoid embolization. The graft is exposed to arterial tension and usually passed behind the internal jugular vein for distal anas-

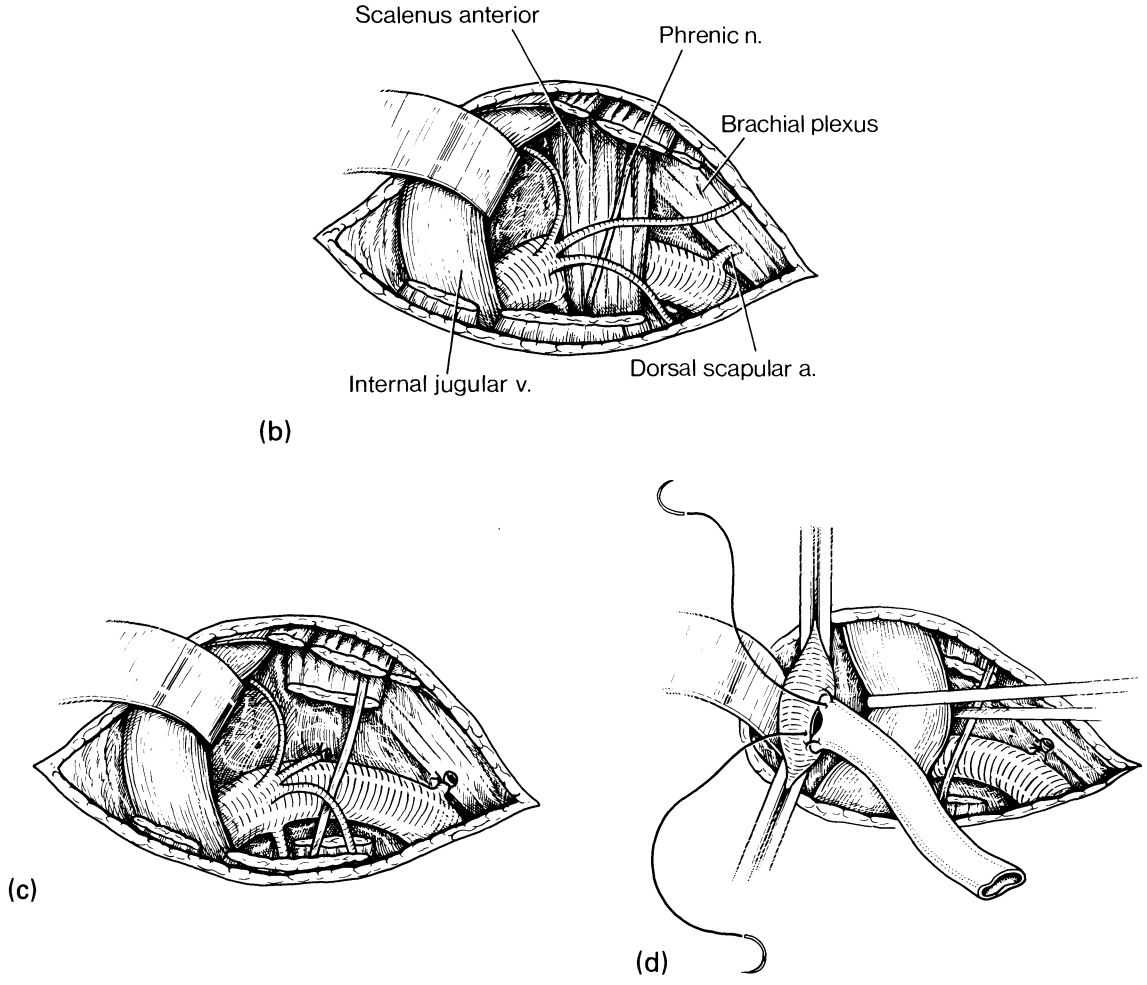
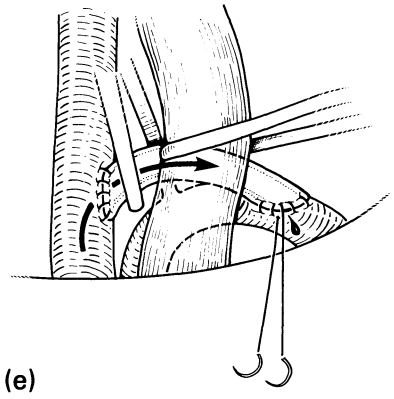


Figure 10.5 continued (b) sternomastoid divided. (c) the subclavian artery is exposed by sectioning scalenus anterior. The dorsal scapular artery has been ligated to improve mobility. (d) a PTFE graft is then anastomosed to the left common carotid artery

tomosis. Graft length is best estimated by placing the patient's head in different positions. A clamp is then placed across the graft origin and the graft attached end-to-side to the superior aspect of the distal subclavian artery. The usual precautions of flushing and rinsing are observed before the suture line is completed. Flow is then released firstly into the arm and then into the vertebral artery (Figure **10.5e**).

If the graft is being inserted for an embologenic ulcerative lesion in the proximal left subclavian artery, the latter should be excluded by proximal ligation once the bypass is in place. Alternatively such cases may be managed by subclavian transposition rather than by a bypass technique (see below).

In some patients the subclavian artery is too diseased for satisfactory graft attachment. Distal anastomosis to the axillary artery may then be preferable[23]. Exposure of the axillary artery is relatively simple via a short infraclavicular incision (see axillofemoral bypass, Chapter 3). The graft is anastomosed to the common carotid artery and then passed behind the clavicle for attachment to the proximal axillary artery[24]. If necessary the vertebral artery may be revascularized by reimplantation into the common carotid artery (Figure **10.6**).



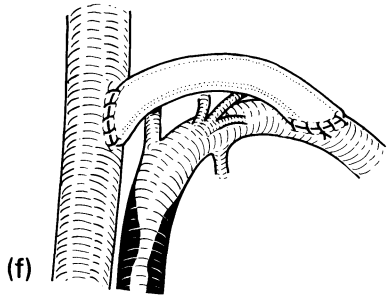


Figure 10.5 continued (e, f) the graft is passed behind the internal jugular vein for subclavian attachment

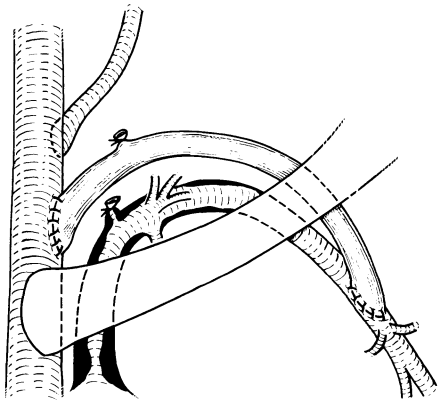


Figure 10.6 Carotid–axillary bypass with vertebral reimplantation for diffuse vertebrosubclavian atherosclerosis

SUBCLAVIAN–CAROTID REIMPLANTATION

Reimplantation of the subclavian artery into the common carotid artery is an alternative method of managing proximal subclavian disease[25]. The main advantages of this method compared with carotid–subclavian bypass are that graft materials are avoided and coexistent vertebral ostial stenosis can be concomitantly corrected. However, it does require a more extensive dissection than a bypass technique and cannot be used when the vertebral origin is low, because of the risk of kinking.

Exposure is similar to that for carotid–subclavian bypass. The internal mammary artery and the thyrocervical trunk may be divided to provide adequate mobility. The subclavian artery is then progressively mobilized back towards the aortic arch using narrow-bladed retractors to displace the carotid sheath medially and the mediastinal pleura laterally. By staying directly on the surface of the artery a surprising length can be mobilized from the neck without recourse to clavicular manipulation or resection. Dissection within the periarterial sheath also avoids the risk of damaging the recurrent laryngeal nerve which is closely related to the posterior aspect of the vessel. The left superior intercostal vein crosses the subclavian artery origin and should also be avoided. The prevertebral subclavian artery is normally without branches, though rarely an intercostal vessel may arise from this segment and this possibility should be kept in mind during proximal mobilization.

Once the subclavian dissection is complete, the carotid sheath is opened at the base of the neck and a segment of common carotid artery prepared for anastomosis. Systemic heparin is then given and a small curved clamp placed across the left subclavian artery as close as possible to the aortic arch. Additional clamps are placed on the verte-

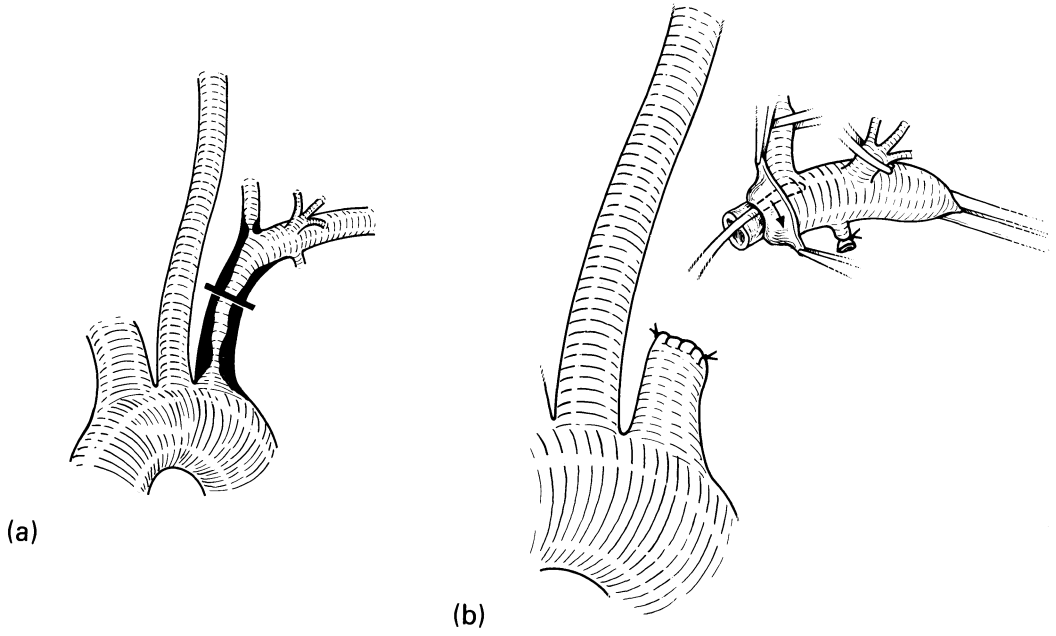


Figure 10.7(a–d) Left subclavian–carotid reimplantation: (a) site of subclavian transection. (b) eversion endarterectomy of the distal subclavian stump

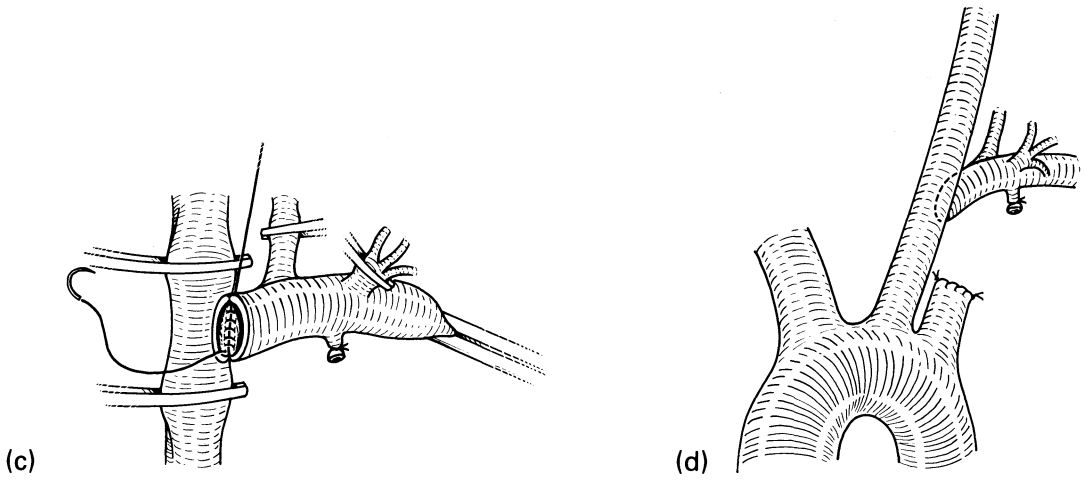


Figure 10.7 continued (c, d) end-to-side anastomosis to the left common carotid artery

bral and distal subclavian arteries. The subclavian artery is then transected and the proximal stump carefully oversewn. If there is obvious plaque extending from the distal cut edge into the vertebral origin, an eversion endarterectomy should be carried out (Figure **10.7b**). Usually distal plaque separation is easy but if there is doubt regarding the end point in either the vertebral or the distal subclavian artery, the vertebrosubclavian junction should be laid open and the intima secured (Figure **10.8a–c**).

The subclavian artery is then passed behind the internal jugular vein and a suitable anastomosis site selected on the common carotid artery. This site should be carefully chosen to avoid kinking or angulation of the vertebral artery. A 10–15° posterior rotation of the subclavian stump so that the vertebral origin is directly posterior instead of posterosuperior may further improve the lie. The subclavian stump is then trimmed with a slight inferior bevel ready for anastomosis.

The common carotid artery is then cross-clamped and the clamps rotated anteriorly to present the posterolateral surface for anastomosis. An ellipse is excised from this wall and the subclavian artery anastomosed in end-to-side fashion taking particular care to avoid a turning-in effect at the corners (Figure **10.7c**). On completion, back-bleeding from the subclavian artery is used to expel any air in the operated segment. Flow is then released firstly into the arm and then into the cerebral vessels.

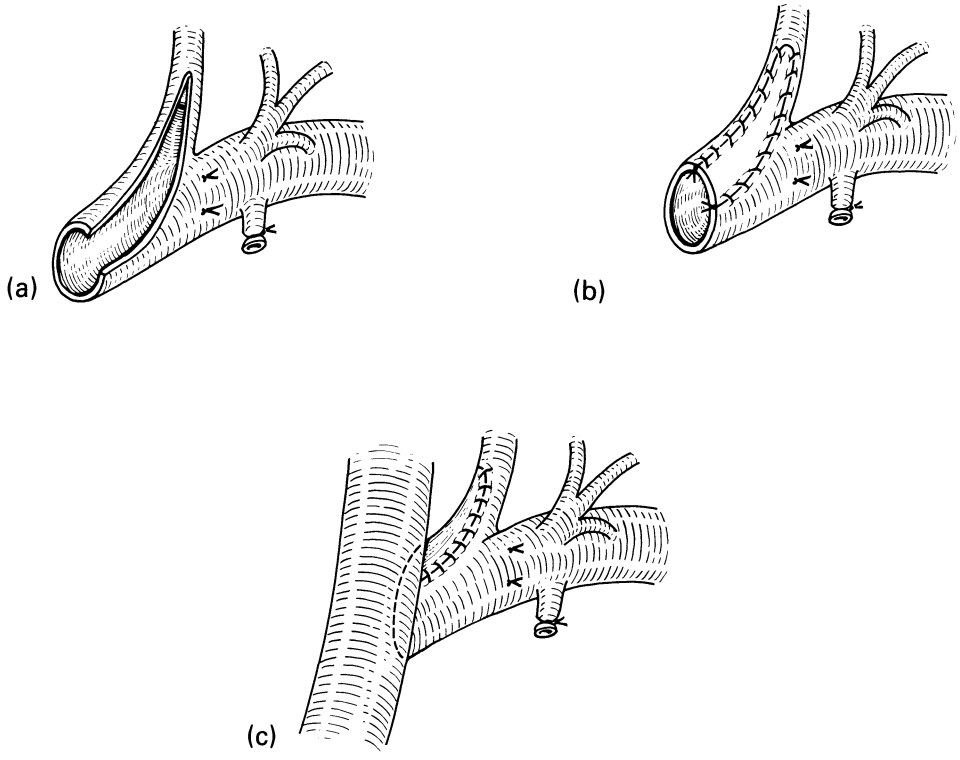


Figure 10.8(a–c) Subclavian–carotid reimplantation: variant (a) the subclavian artery is transected and opened longitudinally into vertebral origin to allow an open endarterectomy. (b) vein patch inserted. (c) completed reconstruction

SUBCLAVIAN ENDARTERECTOMY

The entire length of the right subclavian artery is accessible from the neck and endarterectomy is hence a feasible option on this side.

Exposure is similar to that described for vertebral endarterectomy. Care must be taken to avoid damaging the recurrent laryngeal nerve during dissection of the prevertebral subclavian segment. If the innominate bifurcation is unduly low, access may be improved by manipulating or resecting the medial portion of the clavicle, though this is rarely necessary.

After heparinization, clamps are applied to the distal innominate, common carotid, vertebral and subclavian arteries. A longitudinal arteriotomy is then made in the prevertebral subclavian artery (Figure **10.9b**). This may be extended proximally into the innominate bifurcation if necessary. Similarly, if there is additional vertebral ostial stenosis the arteriotomy may be extended distally to allow removal of this lesion as well. The obstructing

lesion is then removed by open endarterectomy, taking care to avoid encroaching onto the common carotid origin. At the distal limit of the endarterectomy tack-down sutures may sometimes be required to control the subclavian intima. The arteriotomy is then closed, either directly or with a patch (Figure **10.9c**).

CROSSOVER GRAFTS

Subclavian revascularization may also be achieved by subclavian–subclavian or axillo–axillary bypass[26,27]. These crossover grafts avoid the need to clamp, or place anastomotic lines in, the carotid axis and are relatively simple to construct. However, their greater length and unfavourable reversed angle of take-off may reduce durability. In addition, problems may arise if median sternotomy is subsequently required for coronary artery bypass or further brachiocephalic reconstruction. A specific indication for their usage may be in patients with subclavian occlusion and severe bilateral carotid bifurcation disease (Figure **10.10**). Preliminary insertion of a crossover graft may increase vertebral perfusion and make subsequent carotid endarterectomy safer.

The technique of subclavian–subclavian bypass is relatively straightforward. The donor and recipient arteries are exposed via a short supraclavicular incision on either side. The clavicular head of

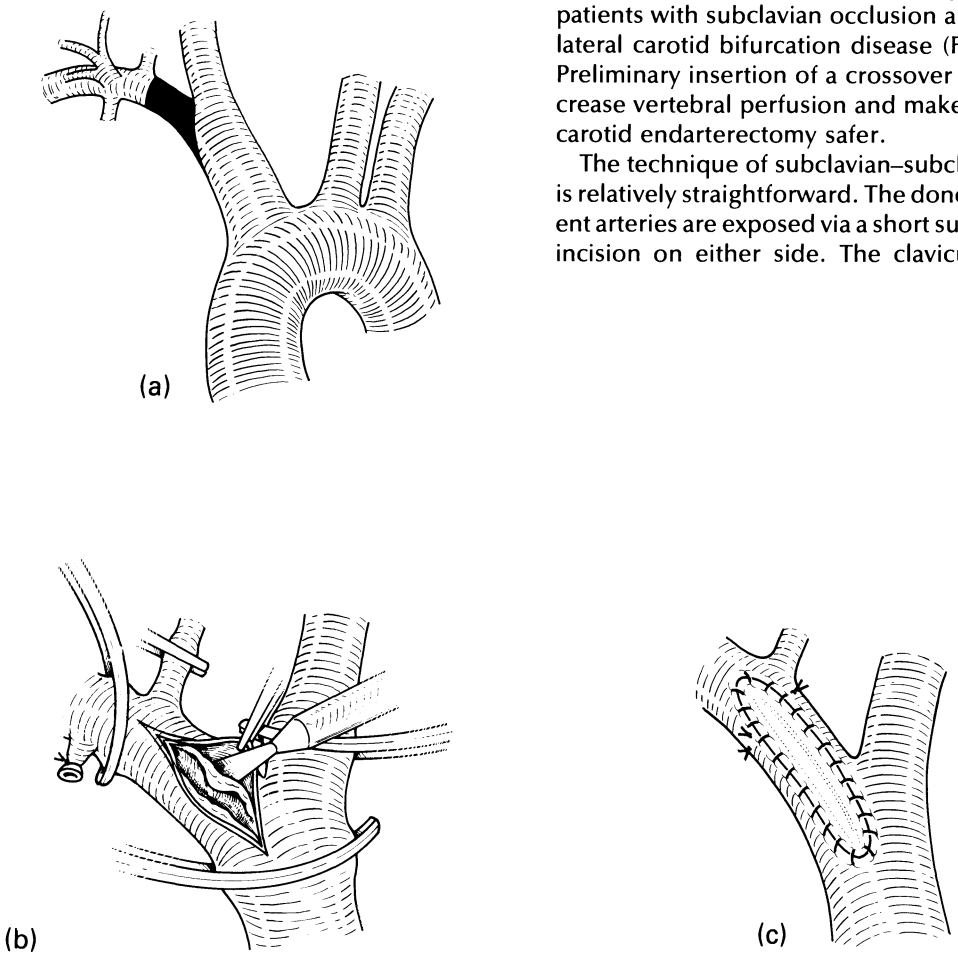


Figure 10.9(a–c) Right subclavian endarterectomy: (a) pathology. (b) open endarterectomy. This can be extended into the vertebral or distal innominate arteries as appropriate. (c) closure with a PTFE patch

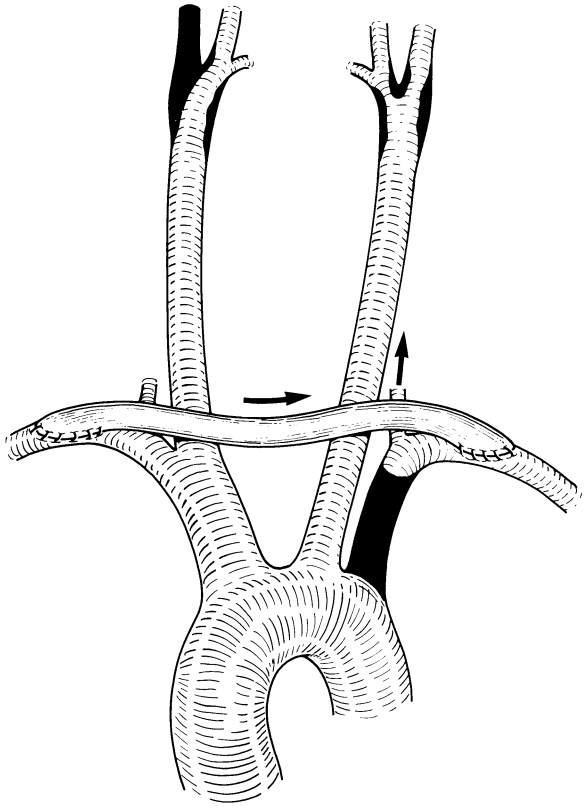


Figure 10.10 Left subclavian occlusion corrected by a crossover graft. The resulting increase in vertebral perfusion is useful as a prelude to carotid bifurcation surgery

sternomastoid and scalenus anterior are divided. A tunnel is then developed digitally from one side of the neck to the other, passing behind sternomastoid but anterior to the carotid vessels. A PTFE or vein graft is anastomosed to the donor subclavian artery and then passed through the tunnel for anastomosis on the opposite side.

Axillo–axillary bypass has been advocated as an alternative to cross-subclavian grafting since the axillary artery is more readily accessible and easier to work with than the subclavian artery[27]. However, the graft has to pass subcutaneously over the sternum and this relative lack of protection may render the graft liable to compression or erosion[28].

TRANSLUMINAL ANGIOPLASTY

The subclavian artery is the most suitable of the brachiocephalic vessels for percutaneous transluminal angioplasty[18]. Subclavian stenoses are usually non-ulcerative and relatively fibrous and may be dilated with little risk of embolization. Occlusions may also be attempted but with less chance of success. Although the place of angioplasty remains to be decided, it may be particularly useful in poor risk patients, those with postoperative stenoses, and patients with multivessel disease where it may reduce the complexity of the surgical reconstruction.

INNOMINATE ARTERY RECONSTRUCTION

Atherosclerosis principally affects the proximal segment of the innominate artery. In some cases the lesion is essentially an aortic arch atheroma which extends into the origin of each of the arch vessels, while in other cases the proximal innominate artery is involved but the origin is spared. Uncommonly, the distal segment only is affected, with or without extension into the right subclavian artery.

As with subclavian disease, isolated lesions are often asymptomatic and in symptomatic patients multiple arch involvement or additional carotid bifurcation or vertebral artery lesions should be anticipated. Symptoms usually result from cerebral hypoperfusion and may involve either the vertebrobasilar or carotid territories, or both. Vertebrobasilar symptoms have already been described. Hypoperfusion in the carotid system may cause unilateral visual dimming, impaired mentation and postural dizziness. In contrast, ulcerative innominate lesions may give rise to amaurosis fugax, transient ischaemic attacks or a frank stroke. The mechanism in these cases is embolization rather than hypoperfusion and the innominate artery may then be the only arch vessel involved.

The treatment of choice is direct reconstruction either by means of an aorto–innominate bypass graft or endarterectomy. Direct reconstruction is more durable than transcervical bypass and median sternotomy is very well tolerated even by quite elderly patients[14,21]. Multiple arch lesions or coexistent coronary artery disease requiring bypass are additional specific indications for a thoracic approach.

Innominate endarterectomy has yielded good results in some hands[29] but the usual proximal nature of the disease with extension into the aortic arch may make this hazardous in most patients. Attempts to remove an orifice lesion completely may initiate an aortic dissection or distal embolization, while a more cautious procedure may risk re-stenosis. For these reasons aorto–distal bypass is now preferred for all innominate lesions other than those located in the distal segment.

ASCENDING AORTO–DISTAL INNOMINATE BYPASS

A sternal-splitting incision is utilized with extension into the neck to allow exposure of the proximal subclavian and common carotid arteries as appropriate (Figure **10.11a**). After reflecting the thymic remnant and anterior mediastinal fat pad, the left innominate vein is identified and progressively cleared. Division of the inferior thyroid, internal mammary and left superior intercostal veins will allow the innominate vein to be retracted freely in either direction. The innominate artery is then isolated and the dissection extended to display the proximal right subclavian and right common carotid arteries. The pericardium is then incised vertically and the ascending aorta exposed (Figure **10.11b**).

A partial occlusion clamp is applied to the ascending aorta sufficiently far away from the innominate origin to avoid the risk of fracturing atheromatous plaque (Figure **10.11c**). Sometimes a partial occlusion clamp may be cumbersome

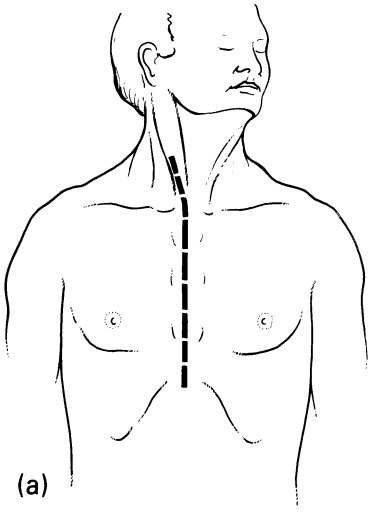


Figure 10.11(a–d) Technique of aorto–distal innominate bypass: (a) access via a median sternotomy extended into the neck

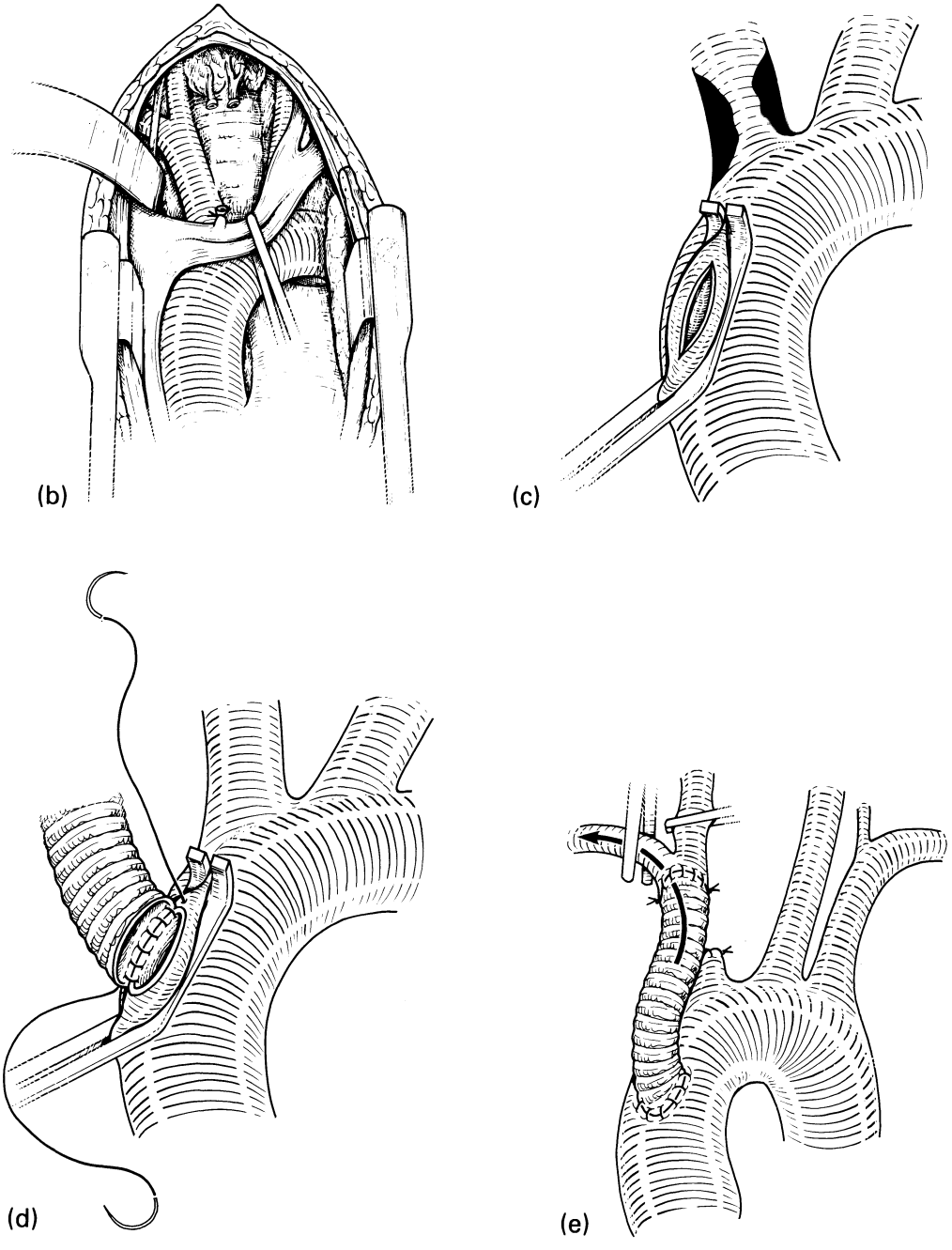


Figure 10.11 continued (b) the left innominate vein is retracted to expose the innominate artery. (c) a partial occlusion clamp is applied to the ascending aorta. (d) the aortic anastomosis is commenced (Dacron graft). (e) completed reconstruction. Flow is released firstly into the arm and then into the carotid axis

and two small curved clamps with the tips touching may be used instead. Controlled hypotension is helpful prior to clamping the aorta, in order to reduce the risk of wall damage and to avoid possible clamp displacement. A 2 cm aortotomy is then made in the excluded portion and an 8 mm or 10 mm prosthetic graft anastomosed in end-to-side fashion at this site (Figure **10.11d**). A flanged graft will allow slightly deeper suture placement if the aortic wall is friable (see Figure **12.3**).

Once the anastomosis is complete, a rubber-shod clamp is placed across the graft adjacent to the suture line and the aortic clamp removed. Systemic heparin is then given and clamps applied to the proximal innominate artery and each of the branches. The innominate artery is sectioned beyond the occlusive zone and the proximal stump oversewn. In order to produce a larger anastomosis the distal innominate stump may be spatulated, if necessary with a cutback into the right subclavian origin. The graft is then cut to length and attached in end-to-end fashion. On completion, air is evacuated from the graft using the subclavian back-bleed, and flow released firstly into the arm and then into the carotid axis (Figure **10.11e**).

In some patients there is significant plaque extension into and beyond the innominate bifurcation, precluding distal innominate graft attachment. One method of managing these cases is to place a graft from the ascending aorta to the common carotid artery with an extension limb to the subclavian artery (Figure **10.12**). Where the innominate artery is stenosed rather than occluded, distal end-to-side bypass attachment should be accompanied by proximal ligation to avoid any possibility of postoperative embolization.

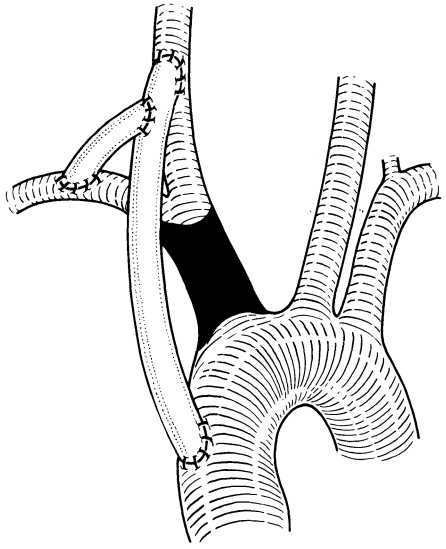
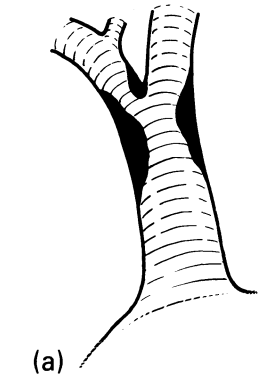
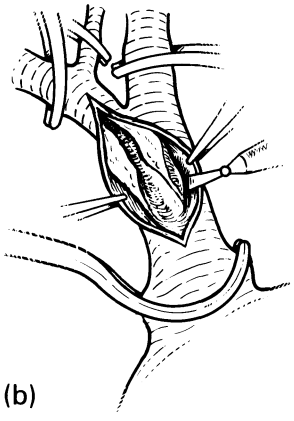


Figure 10.12 Innominate bypass (PTFE): variant necessitated by extension of atheroma into the right subclavian origin

INNOMINATE ENDARTERECTOMY

Atherosclerotic disease which principally affects the distal innominate artery may be appropriately managed by endarterectomy. Access is by a cervico–sternotomy as previously described. After heparinization, clamps are applied to the proximal innominate artery and to each of its branch vessels. A longitudinal arteriotomy is then made in the distal innominate artery. This may need to be extended into the right subclavian artery to achieve full clearance (Figure **10.13b**). An open endarterectomy is then carried out transecting the intima proximally at a site of minimal disease. The distal intimal edge in the subclavian artery may need to be controlled with tack-down sutures. A similar arrangement may be used at the origin of the common carotid artery, though a section–eversion procedure may provide more certain control here (Figure **10.13c**). The innominate arteriotomy is then closed either directly or with a patch.





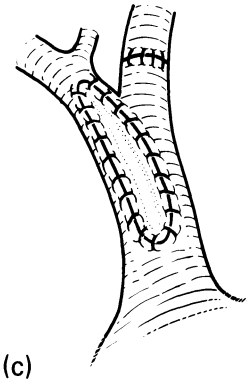


Figure 10.13(a–c) Distal innominate endarterectomy: (a) atherosclerosis localized to the innominate bifurcation. (b) open endarterectomy of distal innominate–subclavian segment. (c) the intima at the common carotid origin has been controlled by section–eversion. The arteriotomy is then closed with a PTFE patch

TRANSCERVICAL BYPASS

In poor risk cases or in patients who have previously undergone sternotomy, a crossover bypass may be considered as a means of achieving innominate revascularization (Figure **10.14**). This of course depends on finding a disease-free arch vessel to act as source. If the left subclavian artery is unsuitable, or cannot be corrected by angioplasty, a crossover graft from the left common carotid artery or a femoro-axillary bypass may be considered[30].

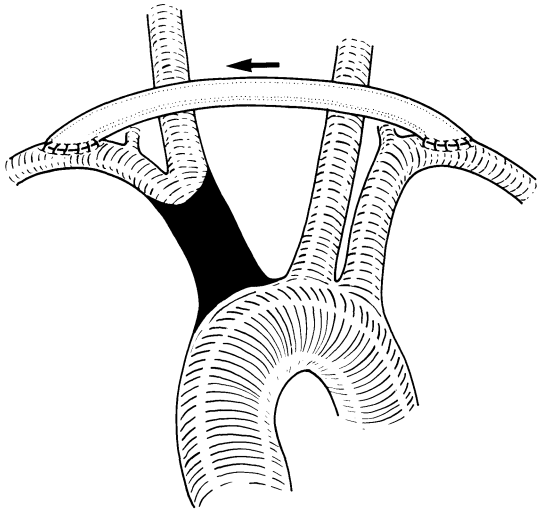


Figure 10.14 Innominate artery revascularized by means of a cross-subclavian PTFE graft

COMMON CAROTID ARTERY RECONSTRUCTION

The most frequent site of atherosclerosis in the common carotid arteries is at their distal extremity, as part of a bifurcation lesion. Less commonly a significant stenosis may be encountered at the left common carotid origin. Mid–carotid stenoses are rarely encountered in atherosclerotic disease, but may occur after irradiation and in Takayasu's disease or other forms of arteritis[31]. Thrombosis may supervene in either retrograde or antegrade fashion depending on the site of the primary lesion.

Common carotid lesions may present with amaurosis fugax, transient ischaemic attacks, a completed stroke, or chronic hypoperfusion symptoms. Surgery is indicated for symptomatic patients with either stenosis or occlusion. In the latter cases extension of the occlusion into the internal carotid segment should not contraindicate a surgical approach since distal external carotid patency is almost always retained as a result of collateral flow. External carotid revascularization by subclavian–carotid bypass or other means is thus feasible in most of these cases and will be sufficient to provide symptomatic relief[32].

Asymptomatic patients with common carotid stenosis may merit surgery because of the stroke risk[14,33]. These lesions may progress to occlusion and thrombus may then extend into the internal carotid artery with resulting cerebral infarction. Similarly, asymptomatic patients with common carotid occlusion and a patent internal carotid artery should also undergo surgery since distal thrombus propagation may still occur. Demonstration of retained internal carotid patency is clearly critical in deciding whether operation is worthwhile in these latter cases, and if arteriography is equivocal, rapid sequence CT scanning should be utilized[34].

COMMON CAROTID THROMBOENDARTERECTOMY

Occlusion of the right common carotid artery may be satisfactorily managed by thromboendarterectomy, since the entire length of the occluded segment is accessible from the neck.

Exposure is achieved via an extended incision along the anterior border of sternomastoid (Figure **10.15b**). After displaying the carotid bifurcation the common carotid artery is mobilized proximally down to its origin. Heparin is given and a routine bifurcation endarterectomy carried out (Figure **10.15c**). If the internal carotid artery is already occluded, the external carotid artery only is opened (see Chapter 9).

Retrograde thrombosis from the carotid bifurcation usually leaves a patent cul-de-sac at the common carotid origin (Figure **10.15a**). As a result, a curved clamp can be placed proximal to the thrombus column without the need to mobilize the distal innominate artery. Marker stitches are inserted for subsequent realignment and the common carotid artery is then sectioned just beyond the clamp. If the artery is found to be markedly atherosclerotic at this site, it may be advisable to abandon the proposed endarterectomy in favour of a subclavian–carotid bypass graft (see below). Outside these circumstances, a central core is developed in the distal cut edge of the common carotid artery and a progressive eversion thromboendarterectomy then carried out up to the carotid bifurcation (Figure **10.15d**).

Once the atherothrombotic core has been removed the vessel may be turned inside-out via the bifurcation arteriotomy to verify that clearance is complete (cf. external iliac endarterectomy, Chapter 3). Normal alignment is then restored and the common carotid artery re-anastomosed. After briefly releasing the proximal clamp to test the security of the anastomosis, the common carotid lumen is irrigated with heparin–saline and the carotid bifurcation closed with a vein patch (Figure **10.15e**). Distal flow is then restored to the external and internal carotid arteries in turn, after taking the usual precautions to evacuate air and debris.

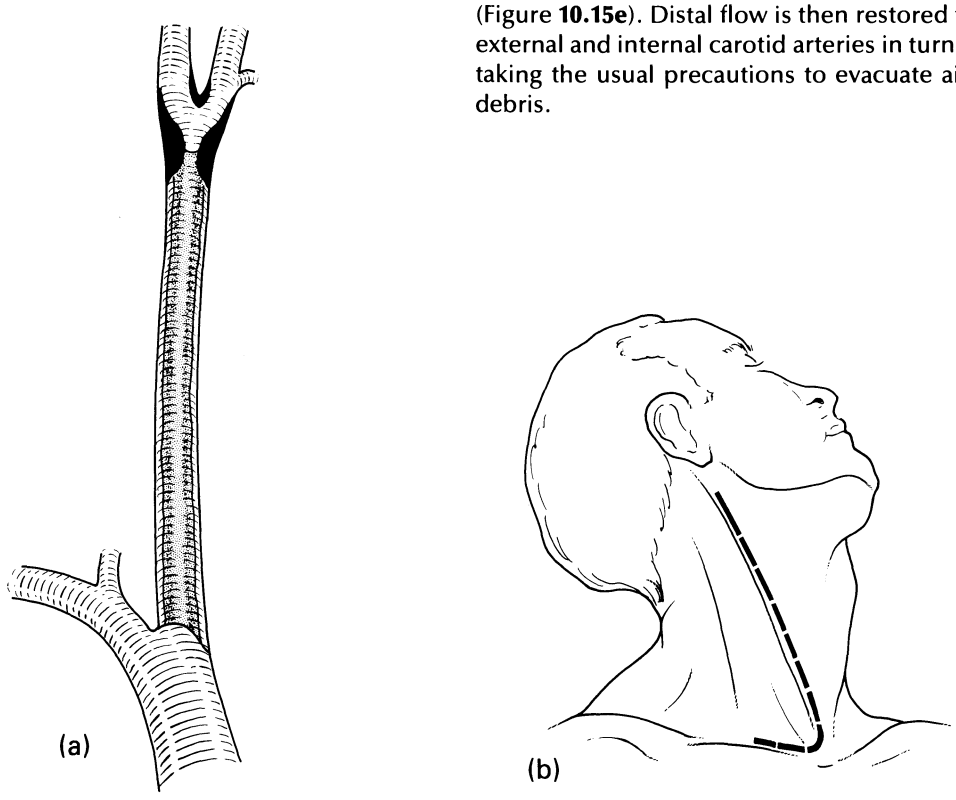


Figure 10.15(a–e) Right common carotid occlusion: (a) bifurcation atheroma with retrograde thrombosis. (b) skin incision.

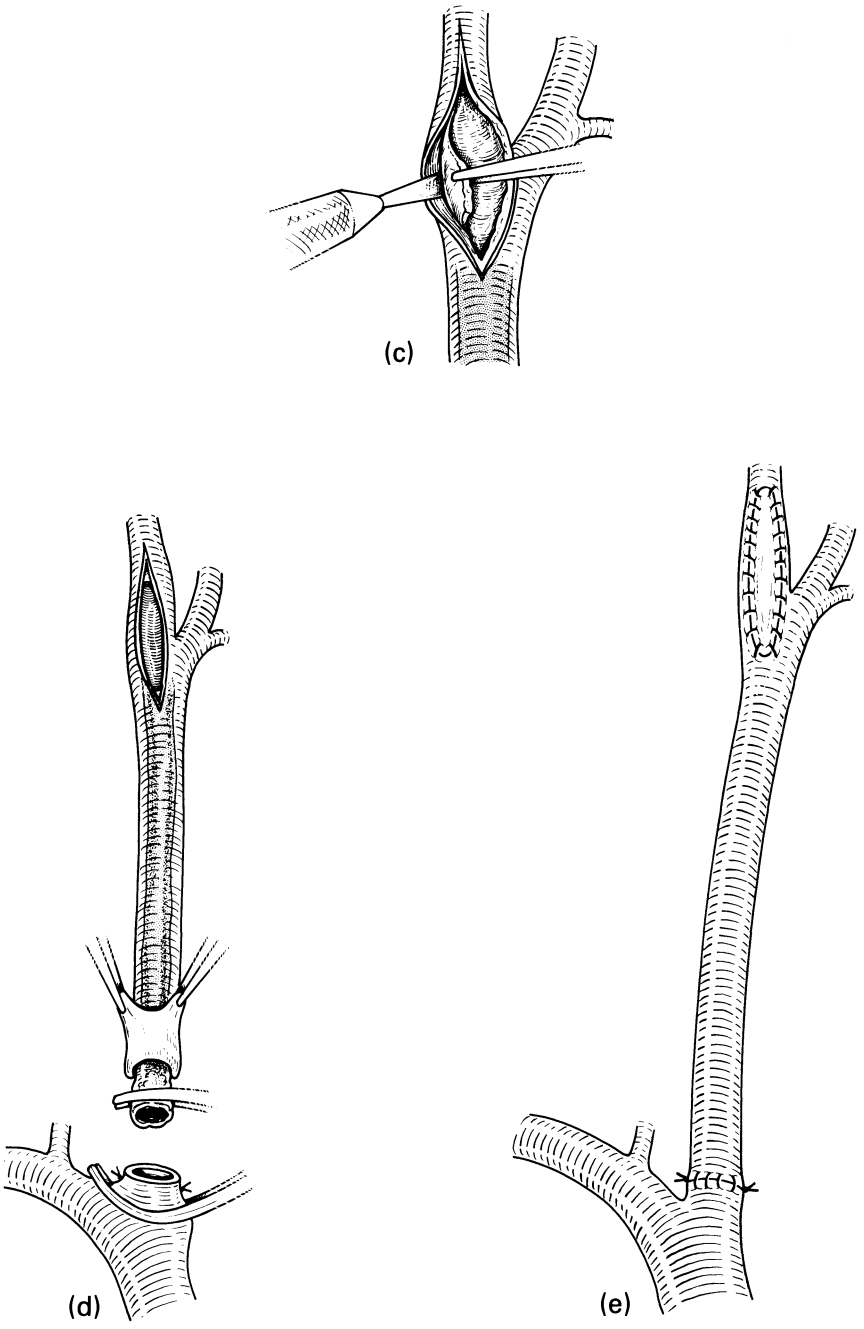
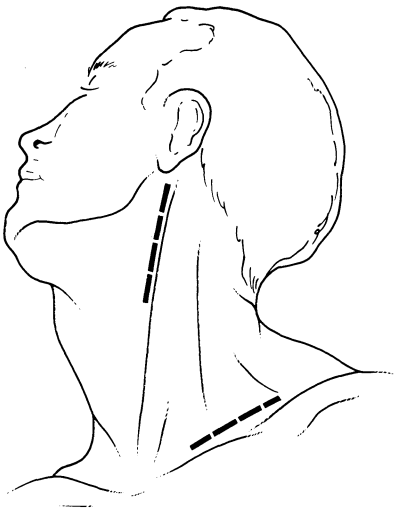


Figure 10.15 continued (c) bifurcation endarterectomy. (d) section and eversion thromboendarterectomy of the common carotid artery. (e) re-anastomosis of the common carotid artery. The bifurcation is closed with a vein patch

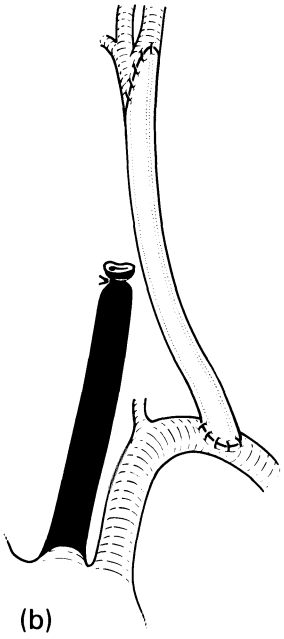


(a)

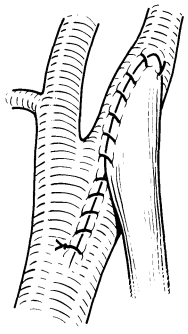
SUBCLAVIAN–CAROTID BYPASS

This is a relatively simple method of revascularization which is equally applicable to right or left common carotid occlusive disease. An important prerequisite is that the donor subclavian axis should be disease-free. In the case of a stenotic lesion at the left common carotid origin with patent distal vessels, a short graft may be placed between the subclavian and common carotid arteries at the base of the neck (see carotid–subclavian bypass).

In most patients a longer bypass is required. The carotid bifurcation and subclavian artery are first exposed through separate incisions (Figure **10.16a**). A PTFE or vein graft is attached end-to-side to the subclavian artery and passed deep to sternomastoid into the carotid bifurcation exposure. An endarterectomy of the bifurcation is then carried out and the graft attached to produce a patch effect at the internal carotid origin (Figure **10.16b, c**). If the internal carotid artery is occluded, an outflow tract is developed in the external carotid artery by endarterectomy and the graft attached end-to-side at this site (Figure **10.16d, e**).



(b)



(c)

Figure 10.16(a–e) Subclavian–carotid bypass: (a) skin incisions. (b) a PTFE graft is anastomosed to the left subclavian artery and then to the endarterectomized carotid bifurcation. (c) alternative method of distal graft attachment.

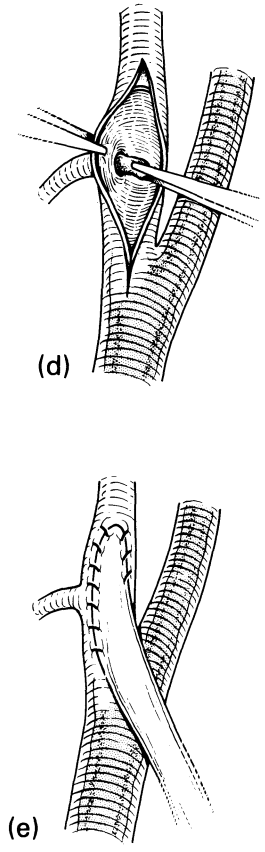


Figure 10.16 continued (d–e) additional internal carotid occlusion: the graft is attached to the external carotid artery after a local endarterectomy

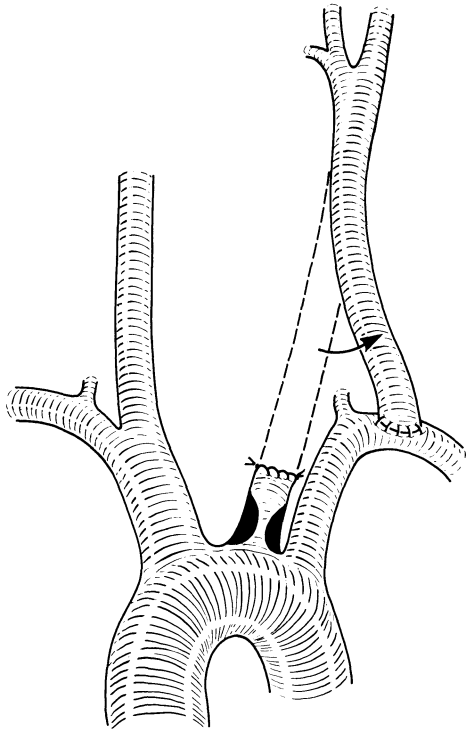


Figure 10.17 Left carotid–subclavian reimplantation

CAROTID–SUBCLAVIAN REIMPLANTATION

An alternative method of managing left common carotid ostial stenosis, which avoids the use of graft materials, is carotid–subclavian reimplantation (Figure **10.17**). Left common carotid occlusion may also be corrected by this manoeuvre if an eversion thromboendarterectomy is carried out prior to reimplantation.

AORTO–CAROTID BYPASS

A direct bypass from the ascending aorta to the distal common carotid artery may be considered in patients with left common carotid occlusive disease when there is no suitable source of revascularization available in the neck. Aorto–carotid bypass is usually then combined with reconstruction of other vessels (see Figure **10.20**).

MULTIPLE ARCH LESIONS

As previously indicated, single brachiocephalic vessel involvement is often asymptomatic due to the profuse collateralization possible in this region. Patients with symptoms tend to have multiple arch vessel lesions, additional carotid bifurcation or vertebral artery disease, or ulcerative lesions which give rise to emboli. Multiple arch vessel involvement may produce transient hemispheric or vertebrobasilar attacks, global hypoperfusion or upper limb ischaemia. Often symptoms are present in more than one territory[35,36]. Reconstruction must be individualized but the innominate and common carotid arteries should be regarded as priority targets. Proximal left subclavian occlusive disease has a relatively benign prognosis and in the absence of significant upper limb ischaemic symptoms may not merit surgical correction.

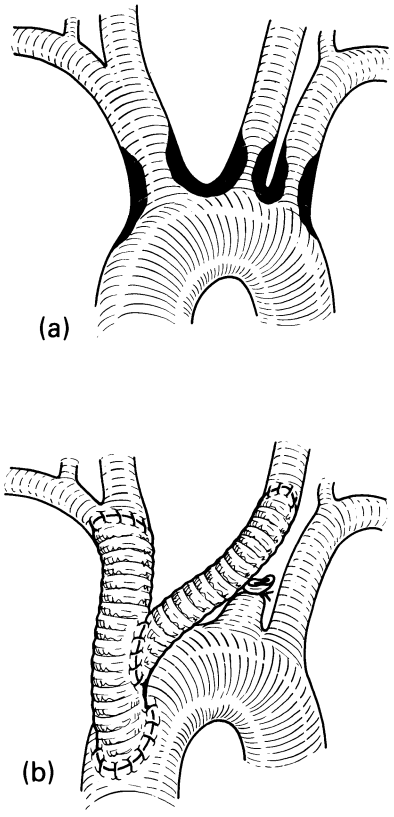


Figure 10.18(a, b) Multiple arch atheroma. Reconstruction by aorto–distal innominate Dacron bypass with a side-limb to the left common carotid artery. The left subclavian lesion has not been corrected

The preferred method of managing multiple arch vessel disease is by in-line bypass grafting via a cervico-sternotomy. An 8 mm or 10 mm prosthetic graft is attached to the ascending aorta and led to either the distal innominate or left common carotid artery. Separate side-arms may then be added to revascularize the remaining vessels (Figures **10.18, 20**). Single tube grafts are used in preference to a preformed bifurcation graft in order to reduce the bulk of prosthetic material in the superior mediastinum with its attendant risk of venous compression[37].

In patients who are unfit for a direct thoracic approach or who have previously undergone sternotomy, a transcervical bypass may be utilized providing one of the arch vessels is spared (Figure **10.19**). In the absence of a suitable donor artery in the neck, a subcutaneous femoro-axillary bypass may be considered with crossover extensions as appropriate[38]. Unfortunately, many patients with extensive brachiocephalic occlusions have similar disease in the aortoiliac segment, so that in practice this latter approach has limited potential.

TAKAYASU'S DISEASE

This is a non-specific panarteritis of unknown aetiology which usually, but not always, affects young female patients. The disease is principally located in the aortic arch vessels but may also be encountered in the descending thoracic or abdominal aorta and visceral vessels and at other sites such as the pulmonary artery[39,40] (see also Chapter 13). Involvement of the left common carotid and postvertebral subclavian arteries is a particularly characteristic pattern of arch involvement (Figure **10.20**).

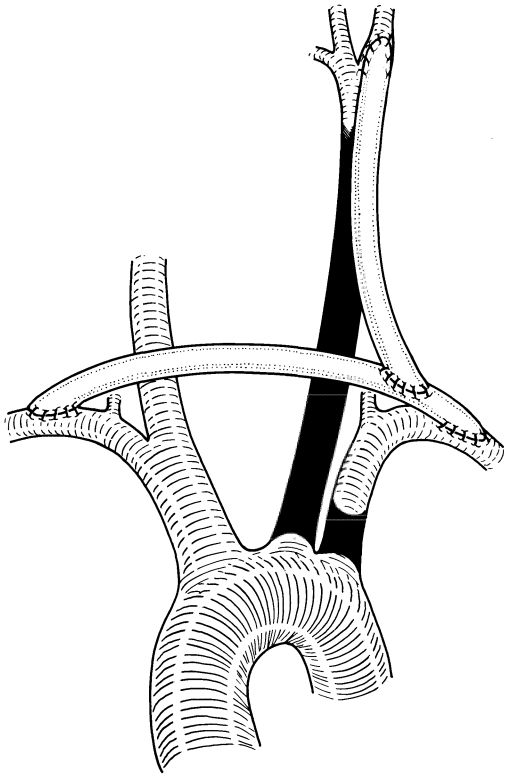


Figure 10.19 Revascularization of the left common carotid and subclavian arteries using transcervical bypass

Although steroids have been advocated in the early acute inflammatory stage of the disease they are of no benefit in the chronic phase of arterial occlusion[40]. Endarterectomy techniques are rarely feasible because of the absence of a cleavage plane and surgical reconstruction is therefore based on the use of bypass grafts (Figure **10.20**). Multiple and innovative grafting procedures are often necessary in this condition[3]. Where the disease pattern is one of short segmental stenoses rather than occlusion, percutaneous transluminal angioplasty may sometimes be of value[41].

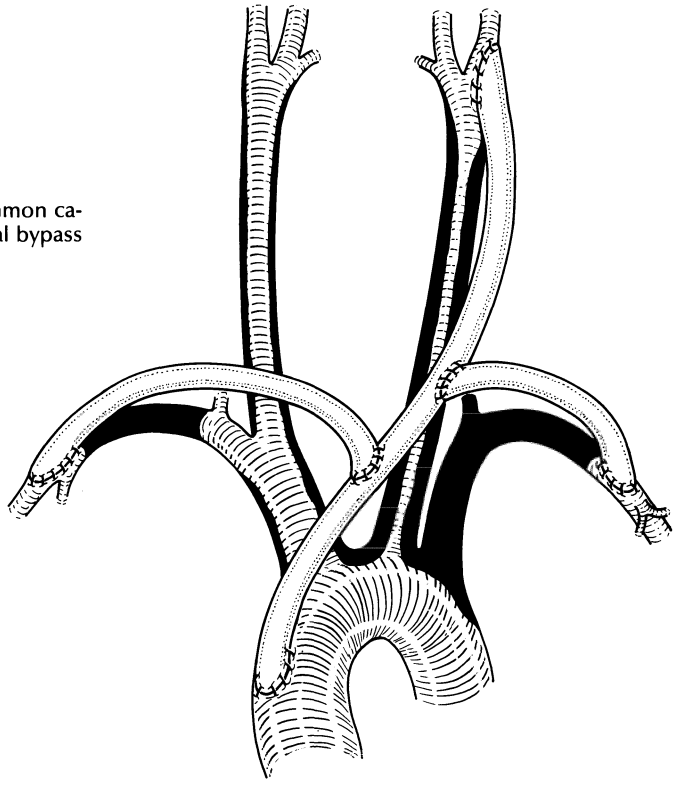


Figure 10.20 Takayasu's arteritis. In this patient an aorto–carotid PTFE graft has been placed with extension limbs to each axillary artery

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11

Thoracic Outlet Syndrome and Upper Limb Revascularization

THORACIC OUTLET SYNDROME

Thoracic outlet syndrome is the term used to describe a group of symptoms caused by extrinsic compression of the brachial plexus or subclavian vessels at the thoracic outlet. In the great majority of cases symptoms are neurological with pain in the arm and paraesthesiae or weakness, these features resulting from C8 or T1 root compression[1]. Pain may also occur in the head, neck, rhomboid and pectoral areas if the upper part of the plexus is compressed[2]. Vascular symptoms from arterial or venous compression are distinctly uncommon, accounting for no more than 5% of cases in several large series[1,3].

Several factors alone or in combination may be responsible for the neurovascular symptoms. Thus the neurovascular bundle may be compressed between the clavicle and first rib, as a result of a naturally low-lying shoulder girdle or from loss of muscle tone. Women have lower shoulder girdles than men and this may make them more susceptible to thoracic outlet compression. More specific aetiological factors include congenital fibromuscular bands which may cross the thoracic outlet tenting up the brachial plexus[4], and abnormalities of the scalene muscles, particularly a broad or aberrant insertion, interscalene connecting bands and post-traumatic fibrosis[3,5]. Other soft tissue elements such as the pectoralis minor tendon have been implicated by some authors[6]. Bony lesions may also be at fault particularly in patients with vascular symptoms. These include cervical ribs, an unusually broad or hypoplastic first rib and fractures or exostoses of the first rib or clavicle.

MANAGEMENT

In some cases it may be difficult to decide whether the symptoms are due to thoracic outlet compression or to some other cause. Even when the diagnosis seems clear, surgery may not always provide full symptomatic relief and for both these reasons an initial non-surgical treatment policy may be the wisest course in most cases. Patients with a clear local abnormality such as a cervical rib or those with arterial or venous symptoms are an exception and should be operated upon at an early stage. For the remainder, a programme of exercises to improve posture and strengthen the shoulder girdle muscles should be instigated. This may lead to symptomatic improvement in many of the milder cases. Those with residual severe symptoms should undergo surgery.

There is at present no agreement on the optimum method of achieving neurological decompression. First rib resection has been the most widely utilized method, particularly via the transaxillary route, but is not universally successful[7]. Scalenectomy and other soft tissue release procedures have also been employed, either alone[3] or in combination with first rib resection[8].

TRANSAXILLARY RESECTION OF FIRST RIB

The technique essentially follows the lines described by Roos[1]. The patient is placed in the lateral position with slight posterior tilt towards the surgeon (Figure **11.1a**). The upper limb, shoulder and thorax are prepped and draped for axillary exposure, leaving the arm free. Access to the first rib depends on the ability of the assistant to elevate the shoulder by applying upward traction on the upper arm. This manoeuvre opens up the costoclavicular space and pulls the neurovascular bundle away from the first rib (Figure **11.1b**). Traction is best applied with the patient's elbow flexed and should be intermittent rather than continuous in order to minimize the risk of injury to the brachial plexus.

A horizontal skin incision 8–10 cm long is made at the lower border of the axillary hair line over the third rib. This incision is then deepened to the fascia over serratus anterior. From here the dissection proceeds proximally towards the apex of the axilla in the areolar tissue plane between the chest wall and the axillary fat. The intercostobrachial nerve emerging from the second interspace should be identified and preserved. After dividing the superior thoracic vessels at the first interspace, the fascial roof of the axilla bridging

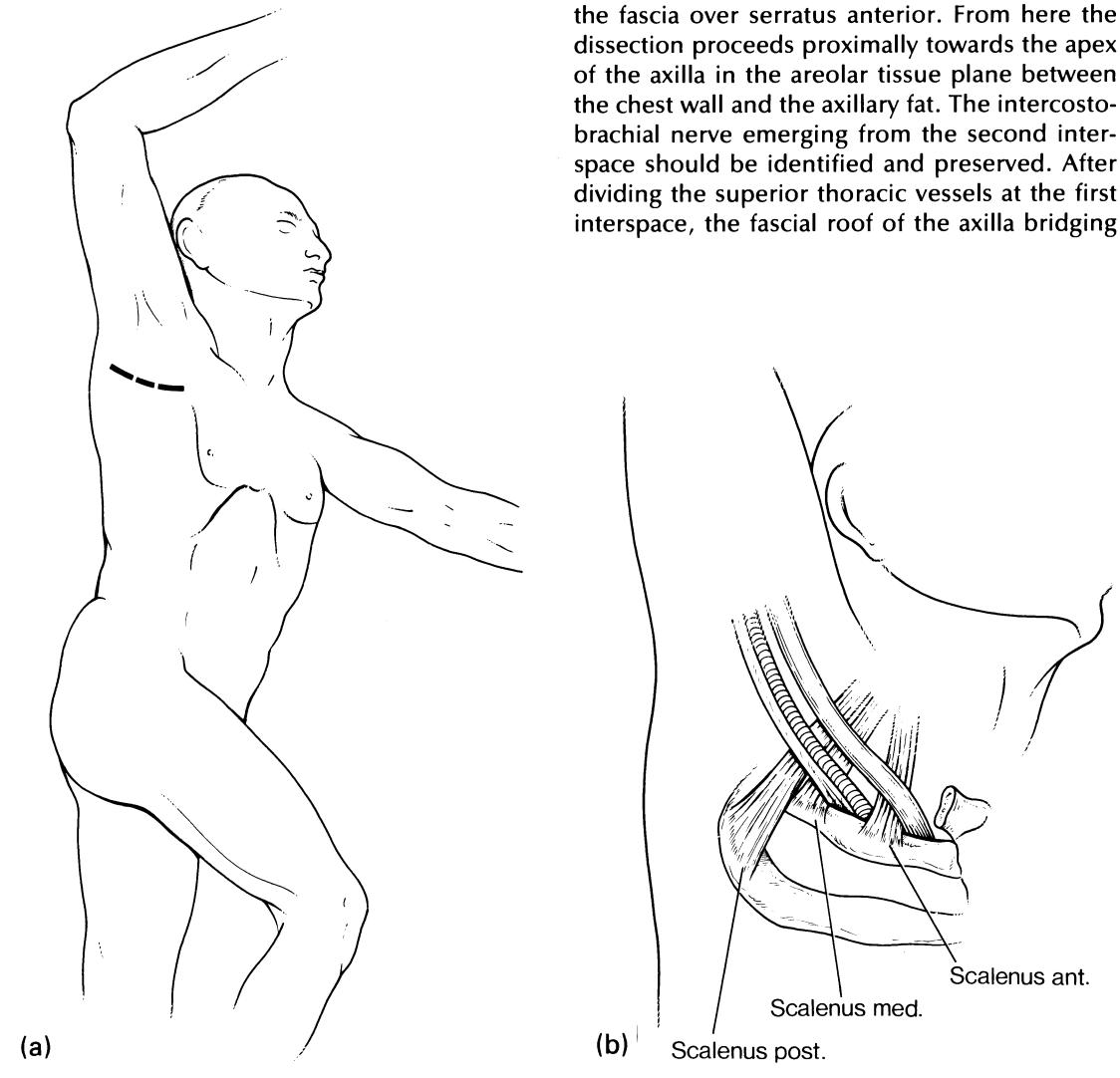


Figure 11.1(a–g) Transaxillary resection of the first rib: (a) operative position and skin incision. (b) neurovascular bundle pulled away from the first rib by traction on the arm.

between the chest wall and the upper limb is opened to expose the anterior portion of the first rib.

Traction is then applied to the arm, and scalenus anterior is identified at the upper border of the first rib with the subclavian vessels either side (Figure **11.1c**). Scalenus anterior is then separated from the artery with a right-angled forceps and sectioned at its attachment to the first rib. Retraction of the pectoral muscles will then expose the anterior segment of the rib and the costal cartilage. The tendon of subclavius is isolated beneath the head of the clavicle and cautiously divided, taking care to avoid the adjacent subclavian vein. The only remaining muscle attached to the upper border of the rib is scalenus medius, and this is now pushed off the rib using a blunt raspatory (Figure **11.1d**).

The intercostal muscles are then similarly detached from the lower and outer aspects of the rib. It is important to leave the periosteum on the rib during this manoeuvre since subperiosteal rib resection may lead to postoperative scarring and possible nerve entrapment. Once the lower border of the rib has been cleared, Sibson's fascia and other soft tissues are detached from the deep aspect with the raspatory. The pleura is then dropped back from the operative zone.

The rib is then sectioned at the costochondral junction and drawn laterally with bone-holding forceps to distance it from the neurovascular bundle. With the arm fully elevated the T1 root is now identified as it passes superiorly to join the C8 root above the neck of the first rib (Figure **11.1e**). Any anomalous bands attaching to this segment of the rib should be divided together

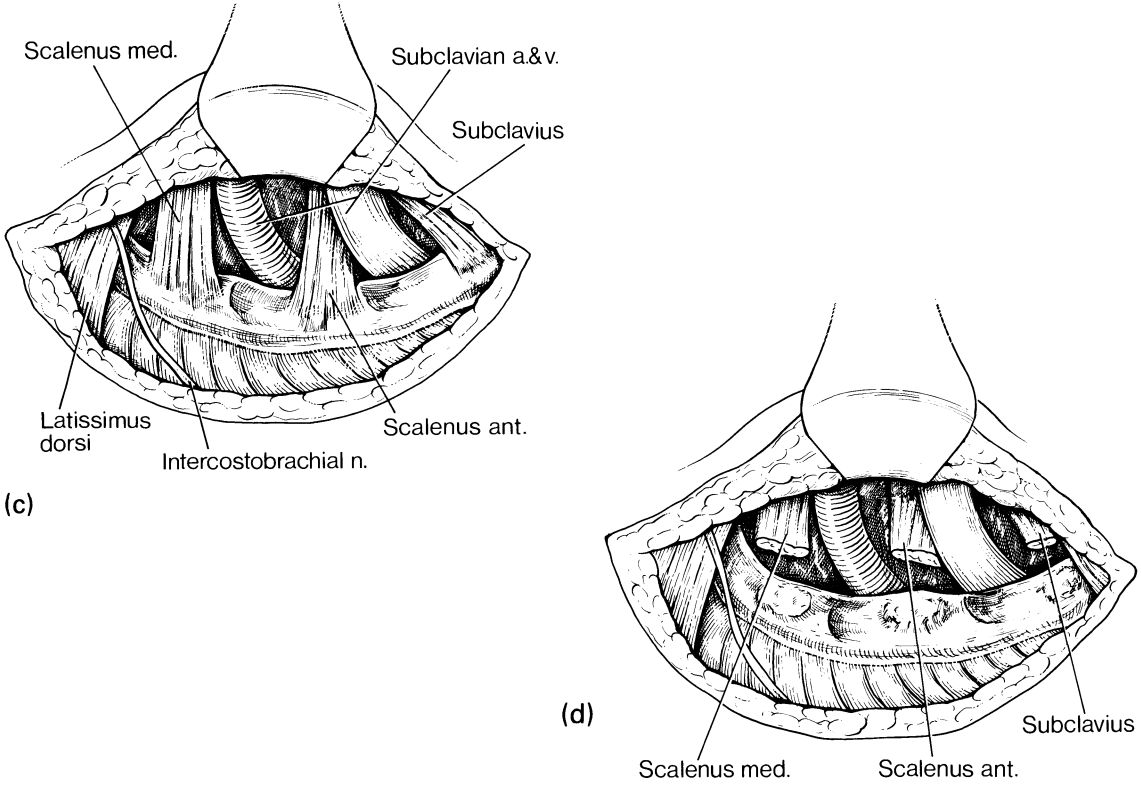
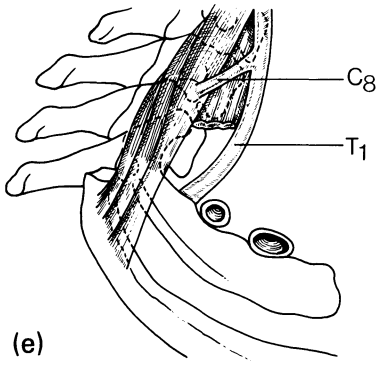


Figure 11.1 continued (c) exposure of the first rib, scalene muscles and subclavian–axillary vessels. (d) scalenus anterior, scalenus medius and subclavius are detached from the upper border of the rib

with the remaining fibres of scalenus medius. The T1 root is then gently displaced medially while an angled rib shear is set as far posteriorly as possible (Figure **11.1f**). The rib is then sectioned and removed. If necessary the stump can be further shortened to within 1–2 cm of the vertebral transverse process using rongeurs. It is important to leave a smooth stump here since sharp bony spicules may lacerate the plexus. Similarly, the costal cartilage at the anterior section site should be smoothed to avoid any risk of venous laceration.

A careful check is then made to ensure that there are no remaining fibrous bands around the plexus or vessels which might cause recurrent compression. Scalenus anterior should be pulled down from between the artery and vein and resected to a higher level to prevent reattachment to the rib bed or plexus.

The axilla is then filled with saline to evacuate air and debris and to test that the pleura is intact. If appearances are satisfactory, the wound is closed with a subcuticular stitch and suction drainage.



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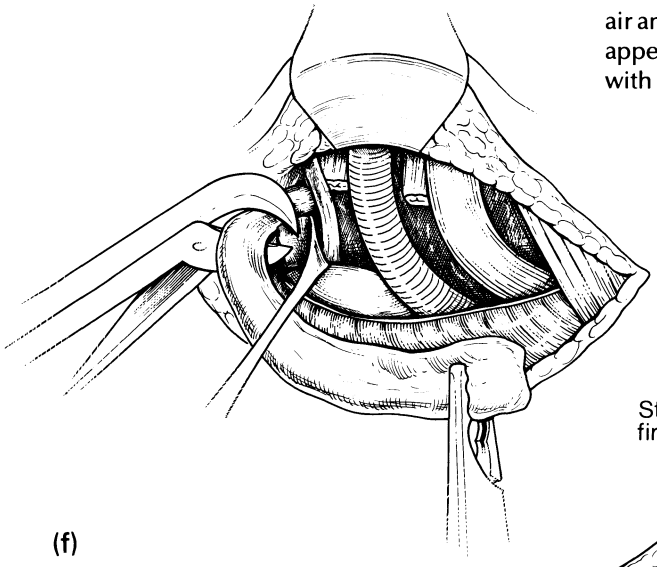

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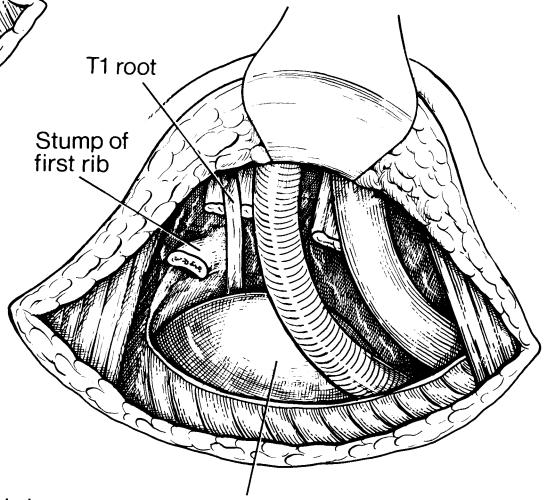


(g)

Figure 11.1 continued (e) relationship of C8 and T1 roots to the first rib in the operative position. (f) the rib has been disarticulated at the costochondral junction and is held aside to allow posterior section. T1 root is protected by a retractor. (g) extraperiosteal resection of the first rib completed. Additional soft tissue bands at the inner border of the rib have been divided to completely decompress the neurovascular bundle

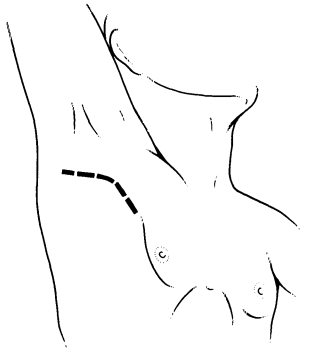
MODIFICATION OF THE ROOS TECHNIQUE

The operative exposure afforded by the transaxillary route is narrow and deep and in order to improve access the following modification of the standard technique may be utilized.

The patient is placed in the same operative position as above. A skin incision is then made at the lower limit of the axillary hair line and extended anteriorly for 4–5 cm in the line of the axillo–mammary skin crease (Figure **11.2a**). The dissection is commenced along the lateral wall of the axilla where the axillary vein is identified. The axillary fat is reflected inferiorly from the vein as the latter is cleared progressively towards the apex of the axilla (Figure **11.2b**). Usually the subscapular vessels can be preserved, though the lateral thoracic and superior thoracic vessels will require division. Pectoralis major is then retracted medially and the tendon of pectoralis minor divided to allow access to the anterior half of the rib (Figure **11.2c**).

The remainder of the procedure follows the lines already described. Scalenus anterior and subclavius are divided and scalenus medius detached from the upper rib surface (Figure **11.2d**). The lower and inner borders of the rib are then cleared using the raspatory. The rib is sectioned at the costochondral junction and held laterally while the posterior dissection is completed (Figure **11.2e**). With the T1 root clearly in view and protected, the rib is sectioned as close as possible to the vertebral transverse process.

Postoperative extrapleural haematoma may be a problem and may lead to sclerosis and nerve entrapment. In order to avoid this sequence of events, it is advisable as a routine to open the pleura after rib removal. An intercostal catheter is then inserted and left in place for 24–48 hours.



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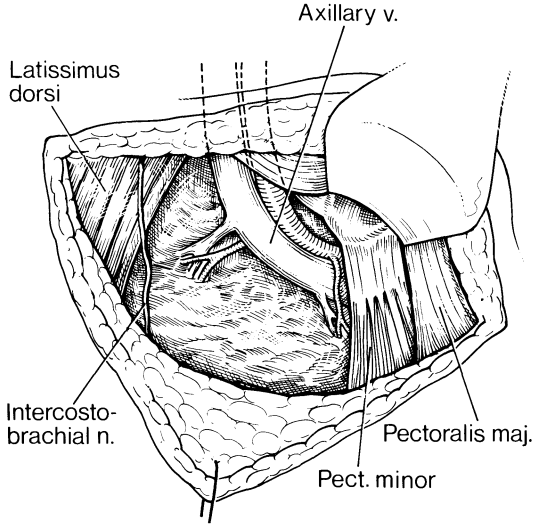
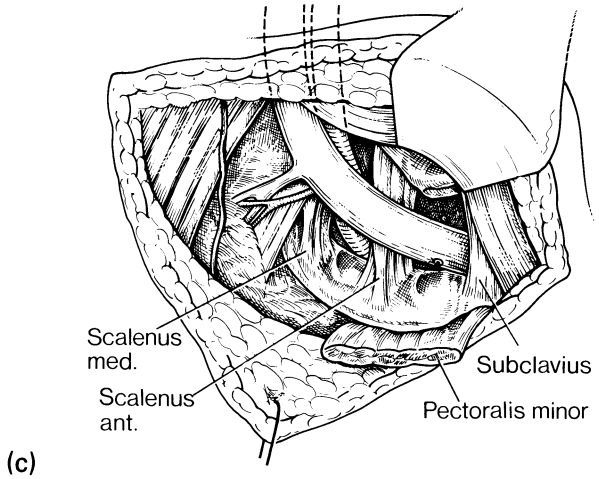


(b)

Figure 11.2(a–e) Modified technique of transaxillary rib resection: (a) skin incision. (b) initial exposure of the axillary vein and its branches



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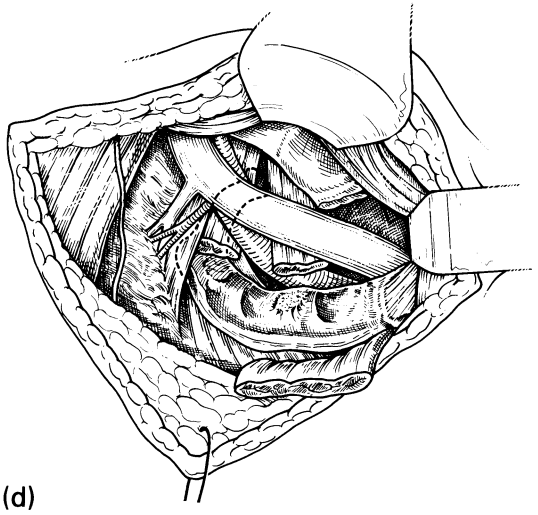

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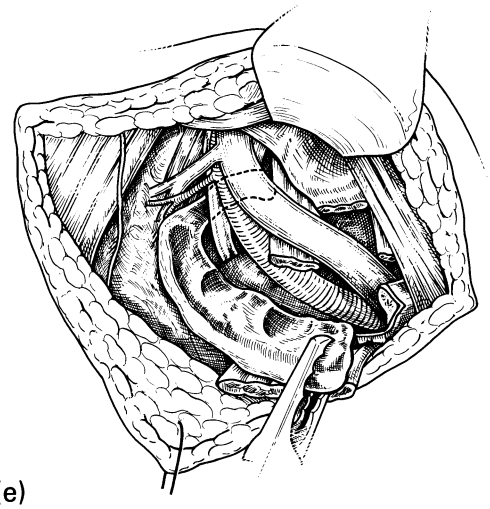


(e)

Figure 11.2 continued (c) the pectoralis minor tendon is divided and the costoclavicular space opened by upward traction on the arm. (d) scalenus anterior and medius are detached from the first rib. (e) the rib is sectioned anteriorly and drawn laterally away from the T1 root. The rib is then resected as far posteriorly as possible

SUPPLEMENTARY PROCEDURES

Additional procedures which can be carried out at the time of transaxillary first rib resection include excision of a cervical rib, and upper limb sympathectomy. Venous thrombectomy may also be possible using this approach (see below). However access is inadequate for arterial reconstruction and the arterial complications of the thoracic outlet syndrome are best managed by alternative methods.

Cervical ribs may be fully developed to articulate with the first rib or they may be incomplete, ending in the soft tissues of the neck. Others are represented by no more than an elongation of the transverse process of C7 vertebra. In either of the last two instances there may be a fibrous band passing from the bony protuberance to the first rib, tenting up the brachial plexus (Figure **11.3**). Such bands are readily removable at the time of first rib resection. If necessary, incomplete cervical ribs may then be further shortened with rongeurs once the first rib has been removed.

Complete cervical ribs usually pass under the nerve roots to join the first rib adjacent to the scalene tubercle (Figure **11.4a**). Often the extremity of the cervical rib is enlarged, forming a false joint, though in other cases the union is a synostosis. The cervical rib and first rib cannot be safely removed in one piece and the simplest method of managing these cases is to resect the extremity of the cervical rib and an adjacent portion of the first rib (Figure **11.4b**). The C8 and T1 nerve roots are then reflected medially while the stump of the cervical rib is followed back towards the vertebral transverse process and rongeured. This allows unimpeded access to the remaining segment of the first rib, which is then removed as previously described.

Upper extremity sympathectomy may also be undertaken at the time of first rib resection. The pleura is displaced inferiorly once the first rib has been removed and the sympathetic chain sectioned below the fourth thoracic ganglion. The chain is dissected up to the lower third of the stellate ganglion and removed at this level along with the ramus to the T1 root.

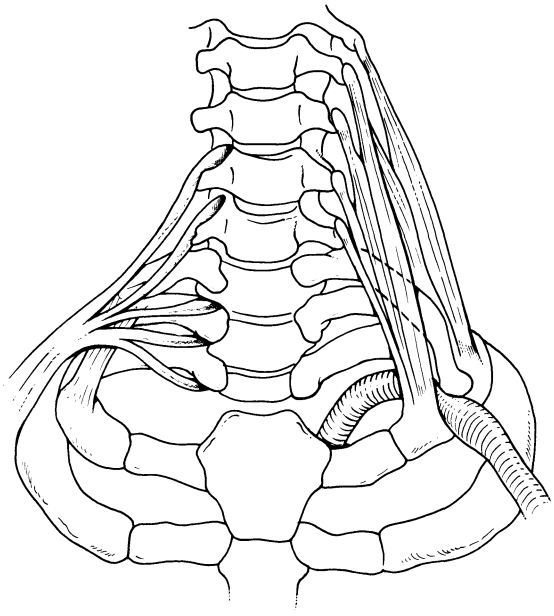
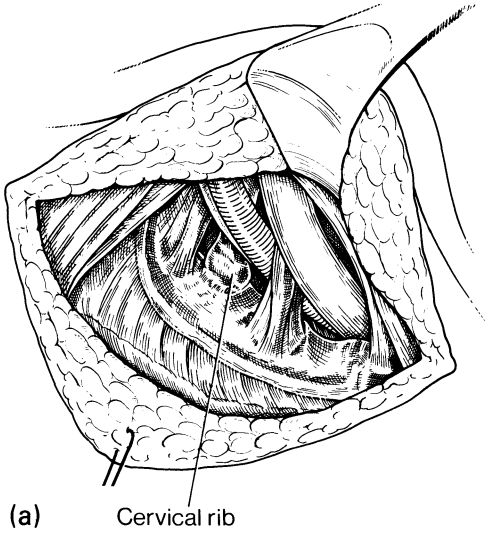


Figure 11.3 Neurovascular compression due to cervical rib. A fully developed cervical rib is shown on the left. A short cervical rib with fibrous prolongation is shown on the right



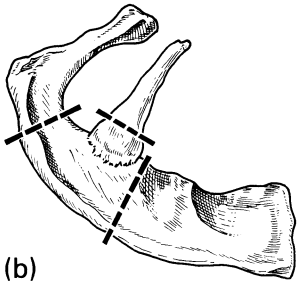


Figure 11.4(a, b) Associated cervical rib: (a) operative exposure showing a fully developed cervical rib with the artery and T1 root passing superiorly. (b) sites of initial bone section

COMPLICATIONS OF TRANSAXILLARY RIB RESECTION

Rarely the subclavian vein may be damaged during division of the subclavius tendon or anterior disarticulation of the rib. Direct repair is usually straightforward once the rib has been removed, though precautions should be taken to avoid air embolism. Injury to the axillary artery is also a possibility if a branch vessel is avulsed during initial exposure. Direct repair should again be feasible.

Postoperative complications include an extrapleural haematoma or effusion which may require drainage or aspiration. These complications may be avoided by routinely opening the pleura and inserting an intercostal catheter, as previously described. Anaesthesia or neuralgia affecting the inner aspect of the upper arm is a more troublesome complication and may be due to stretching or stripping the intercostobrachial nerve. Usually these symptoms spontaneously improve, though rarely nerve section or blockade may be required. Damage to the long thoracic nerve may occur during division of scalenus medius and gives rise to a winged scapula. Again, this is usually temporary.

The most serious complication is brachial plexus injury and this may be more common than has been reported[7]. Traction with the arm in a relatively abnormal position must be at least partly to blame. In addition, the T1 root may be caught by the rib shear or be damaged by instrumental retraction. The risk of neurological injury may be minimised by limiting the period of arm traction and by avoiding instrumental retraction of the plexus. Most important of all, the T1 root should always be kept fully in view during posterior rib manipulation.

ALTERNATIVE APPROACHES FOR FIRST RIB RESECTION

A number of routes other than the transaxillary approach have been utilized for first rib resection. Supraclavicular excision has yielded good results in some series[9] and has the advantage that the anatomy can be fully exposed without the need to maintain the arm in an abnormal or strained position. In addition, cervical ribs can be easily removed and scalenectomy achieved via this approach (see below). However, removal of the first rib from beneath the plexus and vessels may be awkward, even when performed in piecemeal fashion, and there is at least a theoretical possibility of vessel or nerve damage.

The infraclavicular route offers a quick method of removing the anterior two thirds of the rib[10]. However, access to the posterior segment is limited, so that decompression may be incomplete. In addition, cervical ribs cannot be removed through this approach.

The posterior or parascapular route is one of the oldest methods and undoubtedly provides good access to the posterior segment of the rib[11]. However, it requires extensive muscle division and suffers from the additional disadvantage that access to the supraclavicular fossa is difficult, should vascular control be needed.

SCALENECTOMY

Transaxillary first rib resection may be followed by persistent or recurrent symptoms in at least 10% cases[1,7] and in some series a much higher figure has been reported[3,8]. A major factor in symptomatic recurrence is reattachment of the severed scalenus anterior to the rib bed and to the brachial plexus with local scarring. The operation of scalenectomy has provided symptomatic relief in most of these cases and as a result this procedure has been increasingly included in the primary treatment plan. Scalenectomy may be particularly appropriate in patients with upper plexus symptoms while those with symptoms involving C8 or T1 roots may be satisfactorily managed by first rib resection[2]. Patients with both upper and lower plexus symptoms require combined scalenectomy and first rib resection.

Technique

Scalenectomy is accomplished by a transverse supraclavicular incision. After dividing the clavicular head of sternomastoid and the external jugular vein, the pre-scalene fat pad is dissected up and hinged laterally to expose scalenus anterior. If the omohyoid muscle limits the exposure it should be divided (see Figure **11.6g**). The phrenic nerve is freed and displaced medially while scalenus anterior is divided at its attachment to the first rib. The proximal stump of the muscle is then followed upwards and divided as close as possible to the vertebral transverse processes.

Scalenus medius is then also sectioned at the first rib, taking care to preserve the long thoracic nerve which emerges from its lateral border. If a cervical rib is present, this can be excised under direct vision. Additional fibrous bands around the nerve roots and trunks should be carefully removed to ensure full decompression. The prescalene pad is then spread over the exposed nerve roots to prevent postoperative adhesions and the wound closed with suction drainage.

If rib resection is to be combined with scalenectomy, one approach is to perform an initial transaxillary resection of the first rib as previously described. The patient is then turned to the supine position and a separate supraclavicular incision made for scalenectomy. If necessary the posterior rib stump can be further shortened with rongeurs during this stage. Alternatively, rib resection and scalenectomy can be undertaken by a single supraclavicular incision[8] or via a combined supra- and infraclavicular approach (see below). These alternatives have the advantage that they can be accomplished without the need to change the operative position of the patient.

ARTERIAL COMPLICATIONS OF THE THORACIC OUTLET SYNDROME

Arterial complications are nearly always associated with a local bony abnormality, particularly a complete cervical rib. Other bony causes may include an anomalous first rib and either fracture callus or exostosis of the first rib or clavicle.

The initial arterial lesion may consist of a fibrotic thickening in the wall of the distal subclavian artery where the vessel is compressed between the cervical rib and the lateral border of scalenus anterior. Intimal damage, post-stenotic dilatation or a frank aneurysm may eventually occur. Mural thrombosis may be associated with any of these changes and provides a constant threat of embolization.

Symptoms are predominantly due to embolization rather than to stenosis or occlusion of the subclavian artery[12]. Occasionally attention is drawn to the condition by the presence of a pulsatile supraclavicular mass. Most emboli are small and lodge initially in the hand with the production of episodic pallor, paraesthesiae and coldness suggestive of Raynaud's syndrome. Often these symptoms go unrecognized until a major ischaemic event occurs[13]. Repeated embolization results in progressive occlusion of the distal arterial tree with loss of pulses, in some cases back to the subclavian artery. As the collateral flow is progressively impaired, severe distal ischaemia may occur with digital pain, necrosis of the finger tips or more extensive gangrene[14]. Lesions on the right side may pose the additional remote threat of embolic stroke from retrograde propagation of thrombus into the common carotid artery.

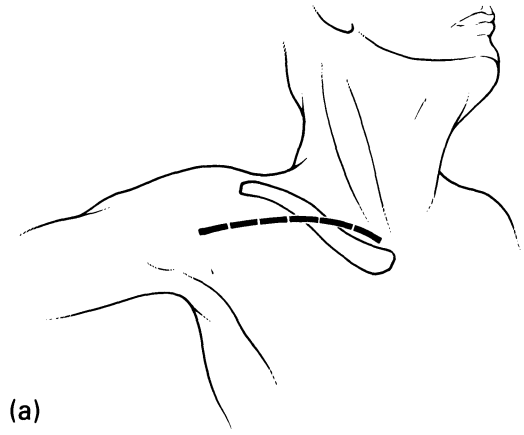
Once the distal arterial tree has become extensively occluded it is difficult to restore full function, and indeed in the worst cases amputation may be necessary[14,15]. Early recognition of this condition is hence of vital importance and arteriography should be undertaken whenever there is the slightest suspicion of an arterial lesion at this level. This may include patients who present with what appears to be unilateral Raynaud's syndrome, and asymptomatic patients with a cervical rib and a prominent supraclavicular pulse or bruit[16]. Pulse obliteration during arm elevation is not in itself an indication for arteriography, since this response occurs in a large number of normal subjects[1].

The subclavian artery may pursue a rather tortuous course as it exits from the thoracic outlet and multiplanar arteriography may be helpful in defining the arterial lesion. The arteriographic changes may sometimes be minimal, but even mild dilatation is significant in symptomatic cases, since intimal injury and mural thrombus will almost invariably be found at operation[13,14]. Occasionally if there is an appropriate bony abnormality at the thoracic outlet and clinical or radiological evidence of distal embolization, exploration of the subclavian artery may be indicated despite a completely negative subclavian arteriogram.

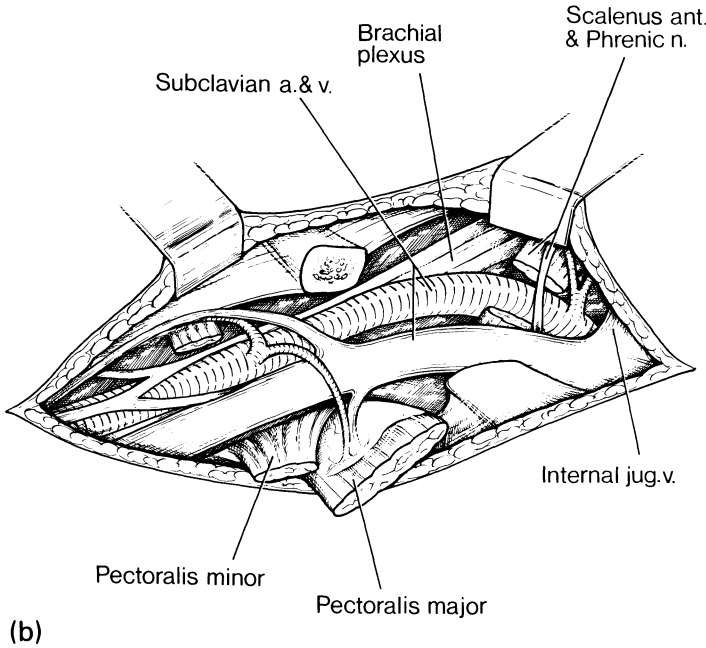
Transclavicular approach

The classical approach for decompression and arterial repair is via a supraclavicular incision extending out into the deltopectoral groove with resection or division of the clavicle (Figure **11.5a, b**). Clavicular resection is preferable to division as the latter may be followed by problems with bony union which may jeopardize the vascular repair[17]. Scalenus anterior is divided and the cervical rib and first rib resected after which the artery is repaired. This approach provides excellent exposure and can be readily extended distally if access to the axillary artery is required. It remains the method of choice for patients with previous clavicular fracture complicated by excessive callus or non–union.

Clavicular resection has cosmetic and functional disadvantages in some patients and alternative approaches may therefore be considered. One method is to perform initial transaxillary resection of the first rib followed by a separate supraclavicular exposure for arterial repair. However this requires changing the position of the patient on the table with an attendant risk of sepsis, and a combined supra- and infraclavicular approach is therefore preferable.

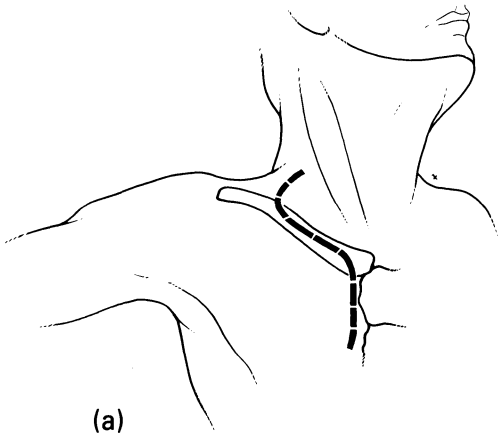


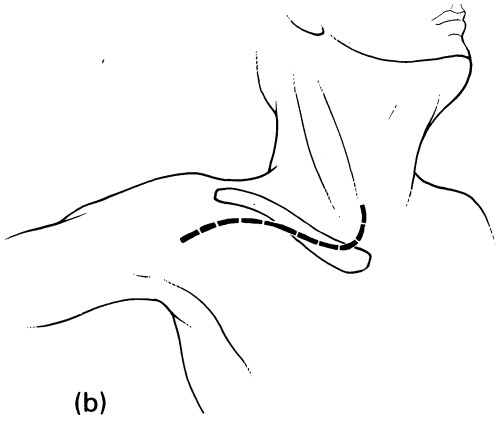
(a)

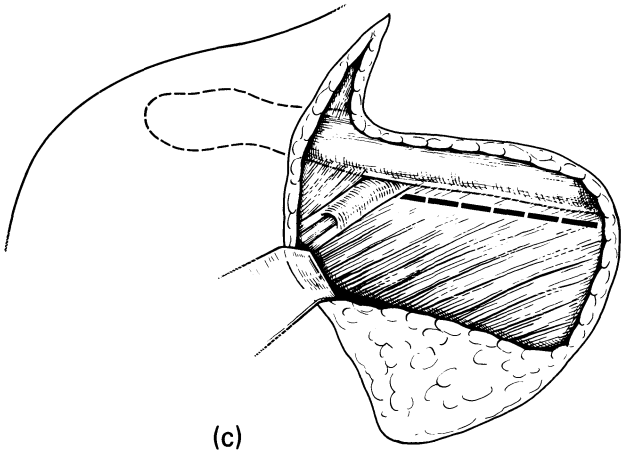


(b)

Figure 11.5(a, b) Classical approach for dealing with the arterial complications of the thoracic outlet syndrome: (a) supraclavicular incision extended into the deltopectoral groove. (b) subclavian–axillary vessels exposed by partial claviculectomy







Resection of first rib using a combined supra- and infraclavicular technique

The patient is placed in the supine position with a sandbag under the cervicodorsal spine and the head turned to the opposite side. An S-shaped incision is then made which follows the anterior border of trapezius before curving horizontally along the clavicle to descend anteriorly over the first three costal cartilages (Figure **11.6a**). A reverse S incision (Figure **11.6b**), which is slightly less aesthetic, may be used in patients with extensive distal occlusion when access to the axillo–brachial segment is required for thromboembolectomy or graft attachment (see below).

The infraclavicular dissection is commenced first. Pectoralis major is detached from the upper sternum and clavicle, taking care not to strip periosteum from the latter (Figure **11.6c, d**). Pectoralis minor is then sectioned and the clavipectoral fascia opened to expose subclavius and the anterior segment of the first two ribs. The axillary

Figure 11.6(a–j) Combined supra- and infraclavicular approach for first rib resection: (a) skin incision. (b) reverse incision when access to the axillo–brachial segment is required. (c) pectoralis major is detached from the clavicle

vein is identified and cleared distally in the line of the deltopectoral groove. The pectoral branches of the acromiothoracic artery and the medial pectoral nerve are then divided to allow full reflection of the musculocutaneous pectoral flap. Proximally the subclavius is resected and the vein freed up behind the clavicle. The axillary artery and brachial plexus are then similarly freed (Figure **11.6e**).

The next stage involves supraclavicular exposure of the subclavian artery. The clavicular head of sternomastoid and the external jugular vein are divided and the pre-scalene fat pad hinged laterally to expose scalenus anterior and the phrenic nerve (Figure **11.6f, g**). The latter is reflected medially and scalenus anterior sectioned near its attachment to the first rib. The subclavian artery and brachial plexus are then freed from their investing fibrous tissue in continuity with the axillary exposure.

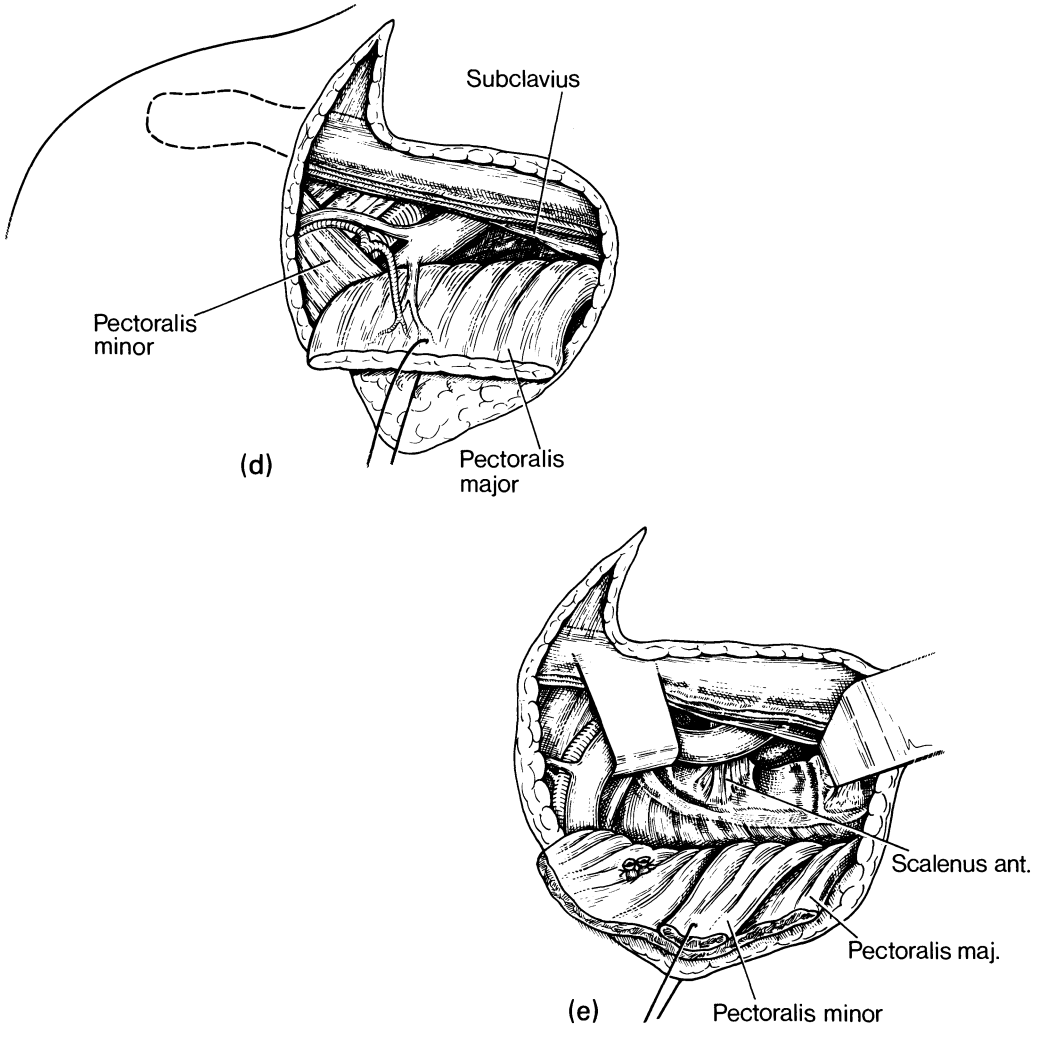
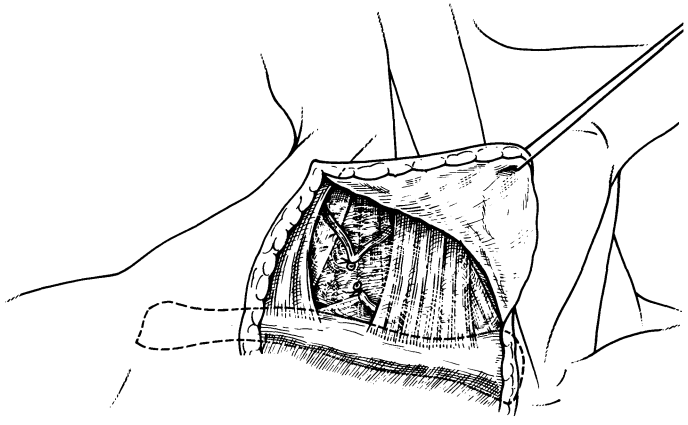


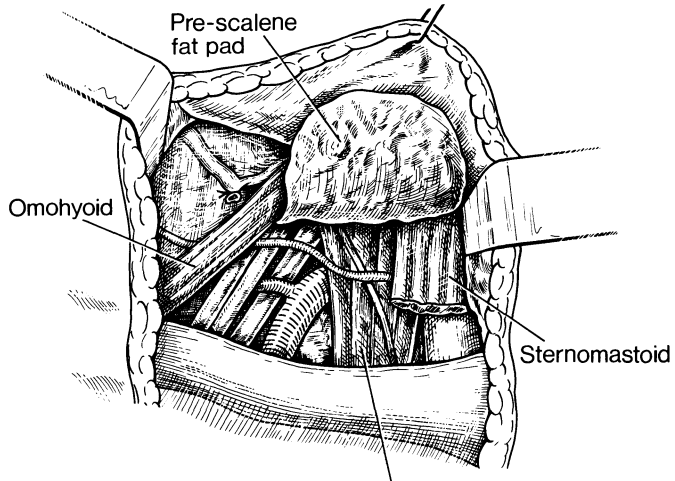
Figure 11.6 continued (d) exposure of the proximal axillary vessels. (e) the axillary vessels are held aside to show the first rib and the insertion of scalenus anterior

The infraclavicular dissection is now resumed and the intercostal muscles are detached from the lower border of the first rib. The rib is disarticulated at the costochondral junction and scalenus medius partially detached from its upper surface. The rib is then sectioned as far back as possible without attempting to reach the posterior segment.

The rest of the rib is then removed via the supraclavicular exposure. Access to the rib stump may be achieved by reflecting the brachial plexus laterally and the subclavian artery medially, after dividing the suprascapular branch (Figure **11.6j**). The remaining fibres of scalenus medius are then detached from the rib and after gently holding the T1 root aside, the rib is sectioned as close as

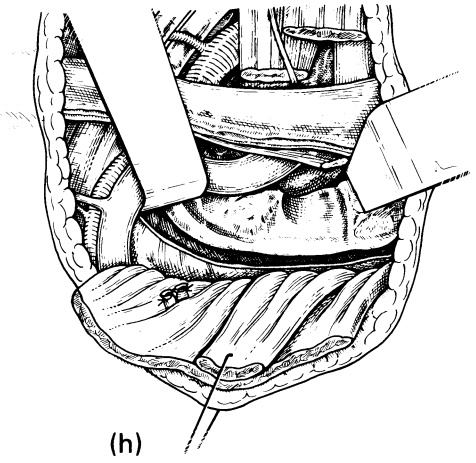


(f)



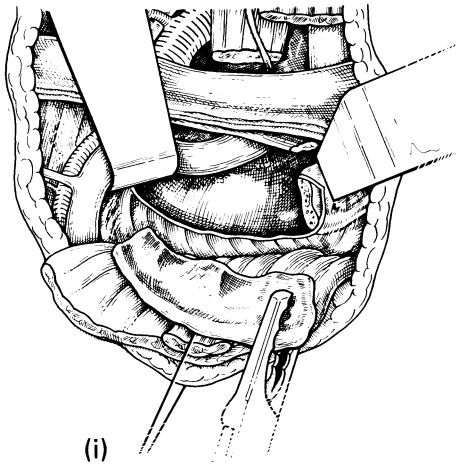
(g)

Figure 11.6 continued (f) the supraclavicular fossa is opened. (g) the pre-scalene fat pad is hinged laterally to expose scalenus anterior, which is then divided



possible to the transverse process. If necessary, rib removal can be completed by working around the outside of the plexus, the T1 root being reflected medially and arteriorly along with the other trunks.

If a complete cervical rib is present, the tip should be excised or disarticulated from the first rib via the infraclavicular exposure. The remainder of the cervical rib is then followed above the clavicle and resected back to the transverse process of C7 vertebra. In order to avoid injury the C8 and T1 nerve roots should be displaced medially during this stage. The posterior remnant of the first rib is then removed as previously described.



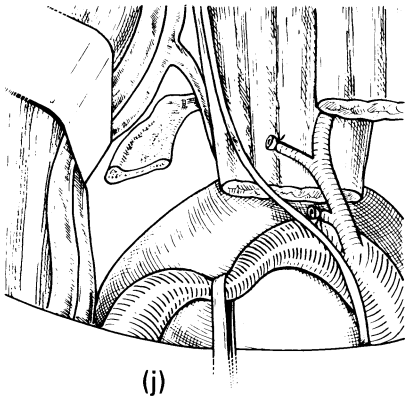


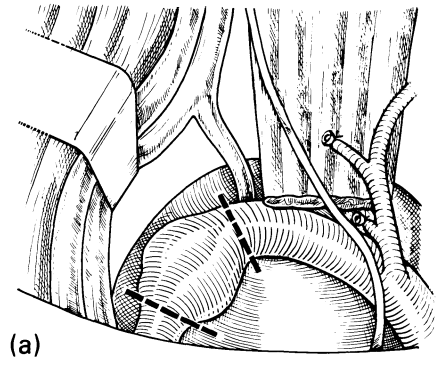
Figure 11.6 continued (h) the infraclavicular dissection is resumed with detachment of the intercostal muscles from the first rib. (i) the anterior portion of the rib is removed. (j) further shortening of the first rib stump via the supraclavicular exposure

Arterial repair

In most patients with a post-stenotic dilatation or aneurysm secondary to a cervical rib, the subclavian artery pursues an abnormally high curve in the neck. In consequence there is often sufficient length of artery to permit resection of the stenosis-aneurysm and direct re-anastomosis (Figure **11.7a, b**).

Where the arterial lesion is more extensive, graft replacement will be required. PTFE or autogenous saphenous vein is the preferred graft material at this site. Proximally the graft may be attached end-to-end to the subclavian artery just distal to the thyrocervical trunk. If autogenous vein is utilized there may be some disparity in graft–artery size and either a spatulated end-to-end anastomosis with interrupted sutures should be undertaken, or an end-to-side bypass utilized with exclusion of the abnormal zone. The graft is then passed behind the clavicle and attached end-to-end to the distal subclavian stump or, preferably, end-to-side to the axillary artery beyond the acromiothoracic branch (Figure **11.8**). A completion arteriogram should then be obtained to confirm the technical adequacy of the reconstruction and to assess the status of the distal tree.

In patients who present with a major embolic event, a catheter thromboembolectomy should be attempted via the subclavian artery with, or without, a supplementary brachial arteriotomy. This may be helpful in recent cases but when there is longstanding occlusion of the brachial or radio-ulnar vessels full distal patency is unlikely to be restored[14,15]. Instead, attention should be concentrated on restoring antegrade flow to the profunda brachii as this is sufficient to relieve distal ischaemia in most cases. Additional sympathectomy may also be considered when there is extensive distal occlusion.



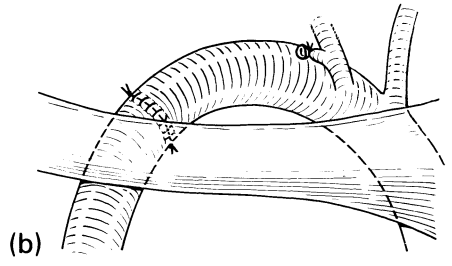


Figure 11.7(a, b) Post-stenotic aneurysm: (a) extent of arterial resection. (b) end-to-end re-anastomosis

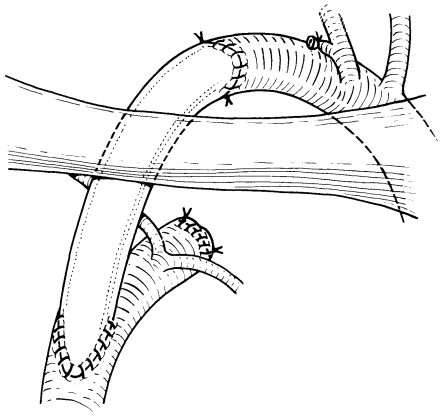


Figure 11.8 Graft replacement (PTFE) for a more extensive arterial lesion. The distal anastomosis is to the axillary artery beyond the acromiothoracic branch

VENOUS COMPLICATIONS OF THE THORACIC OUTLET SYNDROME

Venous complications are uncommon in patients with the thoracic outlet syndrome, though they are not as rare as arterial complications[1]. Venous compression may be due to natural narrowing of the costoclavicular space or to specific factors such as first rib or clavicular fracture callus, bony exostoses or an anomalous band.

In some patients, a syndrome of incomplete subclavian vein obstruction exists which is characterized by intermittent swelling or heaviness of the arm, particularly after overhead use or carrying heavy objects. Diagnosis depends on venography, which typically reveals venous compression at the thoracic outlet when the arm is in the abducted position[18]. Symptoms may be relieved by transaxillary first rib resection together with division of any additional bands which may be present around the subclavian vein.

Other patients present with acute subclavian vein thrombosis. This may occur spontaneously, or after strenuous muscular activity ('effort thrombosis') or prolonged hyperabduction of the arm[19]. Preceding symptoms of venous compression are usually absent. The onset of acute thrombosis may be marked by sudden pain and swelling of the limb with cyanosis, oedema and accompanying limitation of movement. Dilated superficial venous collaterals may develop around the shoulder within the first few days. It is important to recognize that not all cases of spontaneous subclavian vein thrombosis are due to thoracic outlet compression. Other causes include pulmonary or mediastinal tumours, polycythaemia, congestive cardiac failure and occult neoplasm.

If the patient is seen within 2 or 3 days of onset and venography reveals a short segmental occlusion with distal patency, a surgical approach is justified in order to avoid the late sequelae of venous occlusion and to prevent pulmonary embolism[20]. The basis of treatment is venous thrombectomy with decompression of the thoracic outlet. The latter may be best achieved by first rib resection, though others have preferred claviculectomy[21]. The previously described modification of the Roos transaxillary approach is particularly useful here, and will allow access to the entire axillary–subclavian venous segment.

After heparinization, proximal and distal venous control is obtained with Silastic loops and a longitudinal opening made in the axillary vein. The occluding thrombus, which should be limited in extent, is then directly extracted and any loose residual clot removed by suction or with a Fogarty catheter. Once patency has been restored the lumen is rinsed with heparin–saline and the venotomy closed with 7/0 prolene. Particular care must be taken with haemostasis in view of the heparinization. Bleeding from the posterior rib stump in particular may be a problem and may give rise to a postoperative haematoma. Following thrombectomy the patient may be placed on subcutaneous heparin and a low-molecular weight dextran infusion to help maintain venous patency.

Patients who are seen several days after the onset of venous thrombosis or those who are found to have extensive venographic occlusion are best managed conservatively since attempted thrombectomy is likely to be followed by rethrombosis. Even with the aid of a distal arteriovenous fistula, long-term patency is unlikely. Intravenous streptokinase therapy may be considered, but this is not without risks and may not be justified for what is still a relatively benign condition. Most patients are therefore initially managed by arm elevation and a heparin infusion. Oral anticoagulant therapy may be commenced 3 or 4 days later.

Although the initial symptoms of venous occlusion usually improve over a period of days or weeks, a significant number of patients have persistent or recurrent symptoms. These include swelling and discomfort of the arm, particularly on exercise, and recurrent episodes of superficial thrombophlebitis. Such patients should be evaluated by means of venography. This may show occlusion of the main venous channel with a number of collateral veins in the vicinity of the thoracic outlet. Late claviculectomy or first rib resection is said to improve collateral flow in these cases and has been advocated by some authors[12,20]. However, if patency has been retained in the distal veins, a venous bypass graft may be considered in conjunction with first rib resection. Autogenous saphenous vein should be used for

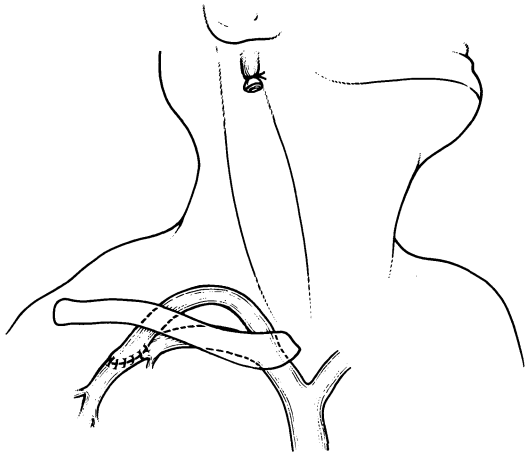


Figure 11.9 Chronic subclavian vein thrombosis. The internal jugular vein has been sectioned just below the parotid gland and tunnelled behind the clavicle for attachment to the axillary vein

this type of reconstruction. The graft is attached end-to-side to the axillary or brachial vein according to patency, and tunnelled behind the clavicle for attachment to the proximal subclavian or internal jugular vein. Graft patency may be promoted by creating a temporary distal arteriovenous fistula[22].

An alternative method of reconstructing a localized subclavian vein occlusion is an *in situ* jugular vein bypass[23] (Figure **11.9**). The internal jugular vein is mobilized in the neck and divided just below the parotid gland. The proximal stump is then turned down behind the clavicle for end-to-side attachment to the axillary vein.

OCCLUSIVE ARTERIAL DISEASE OF THE UPPER LIMB

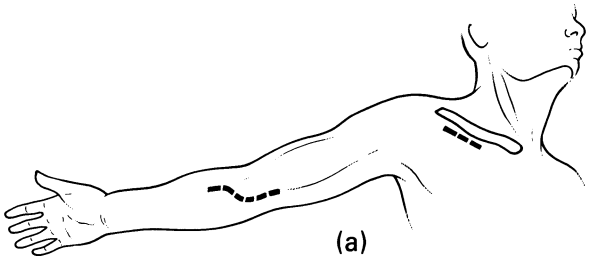
Ischaemia of the upper limb requiring operative treatment is relatively uncommon. Atherosclerosis, the most common cause of ischaemia in the leg, seldom involves the arm vessels, while the well-developed collateral routes around the shoulder and elbow are often sufficient to prevent ischaemic symptoms when occlusive conditions supervene.

PROXIMAL DISEASE

Atherosclerosis and trauma are the most important surgical causes of chronic upper extremity arterial insufficiency[24,25]. Rarer proximal arterial lesions include Takayasu's disease and other forms of arteritis, fibromuscular dysplasia, irradiation damage and congenital atresia[26,27].

Patients with occlusion of the subclavian, axillary or brachial arteries usually present with fatigue or ache on using the arm. More severe distal ischaemia with rest pain or digital necrosis is uncommon, unless previous surgery has impaired collateral routes or there have been repeated episodes of embolization from a proximal ulcerative or aneurysmal lesion (see above). Additional symptoms of vertebrobasilar insufficiency may be present in patients with innominate or proximal subclavian occlusive disease.

Direct reconstructive surgery is nearly always feasible in patients with proximal upper limb occlusive disease, since the disease processes tend to be segmental with retained distal patency. Methods of dealing with innominate and subclavian disease are described elsewhere (see Chapter 10). Axillobrachial occlusions are best managed by a bypass procedure (Figure **11.10a, b**). The choice of anastomotic sites depends on the length of the occlusive process. Usually these sites can be exposed by limited incisions and the bypass tunnelled either anatomically or subcutaneously between the two exposures. Autogenous saphenous vein is the preferred graft material, though if this is unavailable, the basilic vein or possibly PTFE may be considered.



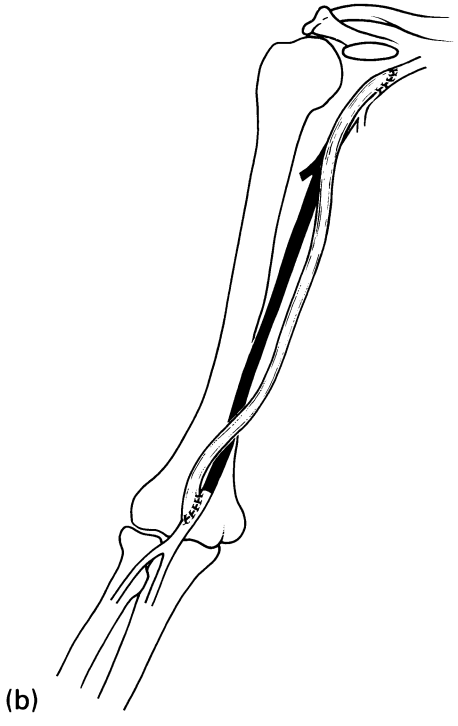


Figure 11.10(a, b) Upper limb revascularization. In this case, the brachial artery is occluded at the profunda origin. An axillo–distal brachial venous bypass graft has been placed utilizing two short exposures

Particulary good long-term results have been reported with upper limb bypass grafts when compared with similar procedures in the lower limb. This may be due to the relative lack of host vessel disease progression and to the apparent freedom from vein graft degenerative change[25,27].

DISTAL DISEASE

Conditions which may cause occlusion of the vessels in the forearm or hand include atherosclerosis, Buerger's disease, immunological and connective tissue disorders, and occupational trauma[28,29]. Digital occlusion due to emboli from a proximal arterial lesion must be carefully excluded since management options are entirely different (see above). The presenting features of distal occlusive disease may include coldness, Raynaud's colour changes, pain and digital necrosis.

Detailed arteriography is essential in evaluating distal occlusive disease and may entail selective brachial injection as well as a full arch study. Magnification views and intra-arterial vasodilation may be useful adjuncts[30]. In addition, intensive laboratory screening with particular emphasis on identifying immunological abnormalities should be undertaken[31].

Most patients with severe digital ischaemia due to distal disease can be managed conservatively. The natural history is often one of short periods of exacerbation followed by long periods of remission with mild residual symptoms. Avoidance of cold is helpful and abstinence from tobacco essential. A variety of vasodilator or sympatholytic agents may also be employed from time to time. Those patients who develop digital necrosis may require local debridement or formal amputation if the gangrene is more extensive.

Selected patients with localized occlusion of the distal radial–ulnar arteries or palmar arch may be managed by resection of the thrombosed segment and vein graft replacement using microsurgical techniques[32,33]. This approach is best reserved for occlusions due to non-immunological disorders with a good distal run-off.

Cervicodorsal sympathectomy[34] or the more

recent technique of digital sympathetic denervation[33] may also be considered in patients with severe distal ischaemia. However, long-term results of sympathectomy have generally been disappointing with an appreciable incidence of recurrent ischaemia. This is particularly true of patients with diffuse arteritis, and sympathectomy should be avoided in these cases[35].

UPPER LIMB ANEURYSMAL DISEASE

Aneurysms of the upper limb arteries are very uncommon. The most important causes are atherosclerosis, trauma, thoracic outlet compression and infection[36]. Those that are due to atherosclerosis are frequently associated with similar lesions elsewhere, particularly in the infrarenal abdominal aorta[37].

They may present with an asymptomatic pulsatile mass, distal ischaemia due to embolization or acute thrombosis, or with pain in the shoulder or arm from local expansion. False aneurysms secondary to penetrating trauma characteristically produce motor or sensory impairment in the arm as a result of brachial plexus compression[38]. As with aneurysms elsewhere, rupture may also occur and this is particularly likely in false aneurysms of traumatic or infective origin where the wall of the sac is thin.

Atherosclerotic subclavian artery aneurysms may be managed by the combined supra- and infra-clavicular approach previously described. Direct graft replacement may be possible, or alternatively the aneurysm may be sewn off from within, and an end-to-side bypass inserted at a distance. If the proximal subclavian artery is unsuitable for bypass attachment, the common carotid artery may be used as source (see Figure **7.26**). Vertebral perfusion should be preserved in all cases, and if the vertebral artery is involved in the aneurysm it should be reimplanted either into the graft or preferably into the common carotid artery (see Chapter 10).

Axillary artery aneurysms may be exposed via a deltopectoral incision with division of pectoralis minor and where necessary, pectoralis major (see Figure **7.29**). The preferred method of treatment is to oversew the aneurysm from within, followed by an end-to-side bypass using autogenous vein. Mycotic aneurysms, e.g. of the brachial artery require interruption of the arterial axis above and below the infected zone and local debridement[39]. Because of the good collateral supply in the arm, immediate revascularization may not be routinely necessary. However, if distal viability is in doubt an extra-anatomic bypass should be inserted through a separate clean field.

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12

Surgery of the Visceral Arteries

In comparison with vascular reconstruction in other territories, operations for mesenteric arterial disorders are uncommon. However, such disorders may be responsible for chronic invalidism or lethal gut infarction, and their prompt recognition and correction remains a matter of considerable importance.

This chapter describes the presentation and management of the acute and chronic forms of intestinal ischaemia. A brief account of visceral artery aneurysms is also included.

CHRONIC INTESTINAL ISCHAEMIA

The predominant pathology underlying chronic intestinal ischaemia is atherosclerosis. Rarer causes include fibromuscular dysplasia, arteritis, Buerger's disease, neurofibromatosis and developmental hypoplasia associated with abdominal aortic coarctation.

Atherosclerosis may affect the origin or proximal segment of all three visceral arteries. In most cases the lesion extends no more than 1–2 cm beyond the aortic ostium, though occasionally a more extensive lesion may be encountered in the superior mesenteric trunk. The distal tree is usually uninvolved, so that reconstructive surgery is a practical possibility in most cases.

If thrombotic occlusion supervenes in the coeliac artery, the distal portion retains its patency due to the large collateral flow in the terminal branches. Thrombosis in the superior mesenteric artery is more extensive, with clot propagation as far as the middle colic branch approximately 6–8 cm distal to the aortic origin. Both of these patterns of occlusion may influence surgical management.

Although symptoms of chronic intestinal ischaemia may occur as a result of occlusive disease in a single visceral artery, this is uncommon. Involvement of at least two of the three main vessels is nearly always necessary before symptoms occur, due to the abundant collateral circulation in this region[1]. Indeed, such is the potential for collateralization that many patients with occlusion of two or even all three primary gut arteries may remain entirely asymptomatic. As a result, chronic intestinal ischaemia remains an uncommon entity in clinical practice, in contrast to the prevalence of visceral artery atherosclerosis in autopsy series[2].

Symptoms of chronic intestinal ischaemia usually develop in patients beyond the fifth or sixth decade, often women. However, younger patients are also not infrequently affected and this may cause particular diagnostic difficulty. The cardinal feature is abdominal pain, which typically is central or epigastric, coming on 30–45 minutes after a meal and lasting for 1–2 hours. The pain is consistently reproducible and closely related to the amount of food ingested. In the occasional patient, the pain is not specifically related to meals but is of a more constant nature. Weight loss is almost invariable and is due to a fear of eating rather than to malabsorption. Bloating and diarrhoea are quite common and occasionally there may be nausea and vomiting. An abdominal bruit is present in over half of the cases and most patients exhibit signs of atherosclerosis elsewhere[3,4].

The true basis for the above symptoms is often not recognized and many patients undergo extensive and inconclusive abdominal investigation before an aortogram is eventually obtained. Failure to diagnose this condition may not only mean continued invalidism but may expose the patient to the risk of visceral infarction. The number of patients who exhibit this progression is unknown, though retrospective surveys of patients admitted with mesenteric infarction reveal prodromal symptoms in a high proportion[5,6].

The most important investigation is aortography. This should be obtained by a transfemoral Seldinger technique with selective catheter studies as appropriate. Lateral and occasionally oblique views are essential to show the visceral artery origins and the collateral pathways. When either the superior mesenteric or the coeliac artery is narrowed or occluded, a dilated pancreatico-duodenal arcade may become apparent. Superior mesenteric occlusion may also stimulate the development of a meandering mesenteric collateral from the inferior mesenteric artery or, if this is also occluded, from the internal iliac territory[7]. Due note should also be taken of coexistent renovascular or aortoiliac disease as lesions at these sites will influence the revascularization technique.

Surgery is clearly indicated in patients with intestinal angina both to relieve symptoms and to prevent progression to infarction. Operation may also be indicated in asymptomatic patients when two or all three primary gut arteries are severely obstructed, providing there are no serious operative risk factors. Such patients are heavily dependent on collateral supply and the chance occurrence of hypotension or an arrhythmia, e.g. following myocardial infarction, may precipitate mesenteric infarction. Although the risk of such a course of events is probably small in practice, gut infarction is so highly lethal that an active policy of revascularization would appear justifiable whenever severe multivessel disease is encountered.

Prophylactic gut revascularization is particularly critical in patients undergoing aortic or renovascular surgery. Failure to recognize and correct visceral artery disease at the time of aortic surgery has led to fatal postoperative gut infarction in our experience and that of others[8]. A borrowing–lending mechanism has been suggested as a factor in this response[9], though interruption of critical collateral pathways during the aortic or renal artery procedure or perioperative hypotension are of greater importance in most cases. Apart from preventing immediate postoperative gut infarction, concomitant visceral revascularization may avoid the need for a potentially difficult reoperation, should intestinal angina develop as a result of atherosclerotic progression months or years later.

Once a decision has been taken on the need for mesenteric revascularization, the question arises as to the number of arteries which should be reconstructed. Although single vessel reconstruction will relieve chronic intestinal ischaemia in most cases[10], there is a serious risk of symptomatic recurrence from graft failure or atherosclerotic progression[3,11]. It is therefore advisable to reconstruct as many of the visceral arteries as possible at one time so that if one of the reconstructions fails the patient is still protected.

BYPASS GRAFTS

Bypass grafting is the preferred method of visceral artery reconstruction in most situations. The infrarenal aorta has been the most frequently utilized graft source in the past. However, the infrarenal aortic wall is often diseased or liable to disease progression, while the usual retrograde trajectory of these grafts, with reverse flow at one or both ends, may carry at least theoretical haemodynamic disadvantages. An additional difficulty in the case of bypass from the infrarenal aorta to the superior mesenteric artery is the potential for buckling with change in position of the small bowel mesentery.

Because of these problems, other methods have been utilized. The best of these appears to be antegrade bypass from the distal thoracic aorta. The aorta here is nearly always well preserved and the short antegrade course to the visceral arteries may result in particularly good durability. In addition, if an infrarenal aortic reconstruction is required at a later date, this can be undertaken without particular difficulty. Rarely, the supracoeliac aorta is unsuitable for bypass attachment. In this case, an antegrade bypass may be placed from the descending thoracic aorta via a thoracoabdominal approach.

Apart from the aorta, the common or external iliac artery may be used as graft source for visceral artery revascularization. This avoids the problem of an unsatisfactory or difficult aortic attachment and is particularly useful when an aortic prosthesis has been placed some time beforehand[12]. However, the graft course is still retrograde and in addition there may be a risk of late failure from atherosclerotic progression in the iliac axis.

Autogenous saphenous vein, Dacron or PTFE have been used as graft material for visceral artery reconstruction. Although saphenous vein sews in well to the delicate mesenteric vessels, it has had limited durability in this position and prosthetics are generally preferred[12,13].

Technique of antegrade supracoeliac aorta–visceral artery bypass

Access may be achieved by either a thoracoabdominal or a midline xiphopubic incision. The latter produces less morbidity and is usually adequate unless the operative field is particularly deep, in which case extension into a median sternotomy may be necessary. A sternal retractor is secured at the upper end of the laparotomy incision and an approach made to the supracoeliac aorta through the lesser omentum. The left lobe of the liver is reflected to the right after division of the triangular ligament, and the oesophagus is held over to the left. This exposes the diaphragmatic hiatus, which is then split to allow the distal thoracic aorta to be displayed (Figure **12.1**)

In 80% of cases, the anatomical arrangement is such that the superficial layer of the right crus can be directly separated from the left crus once the median arcuate ligament has been divided (Figure **12.2a**). Longitudinal section of the deeper crossover fibres of the right crus then exposes the anterior surface of the distal thoracic aorta. In the remaining 20% of cases, the right crus is reinforced with fibres from the left side and this bun-

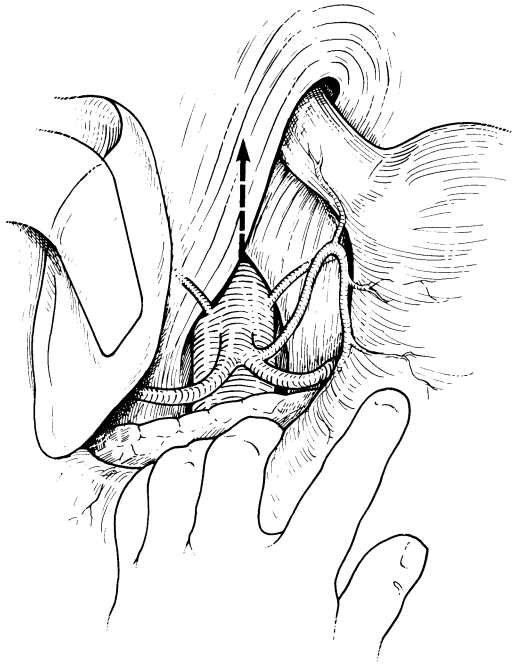


Figure 12.1 Trans-abdominal approach to the descending thoracic aorta. The arrow indicates the line of section of the right crus

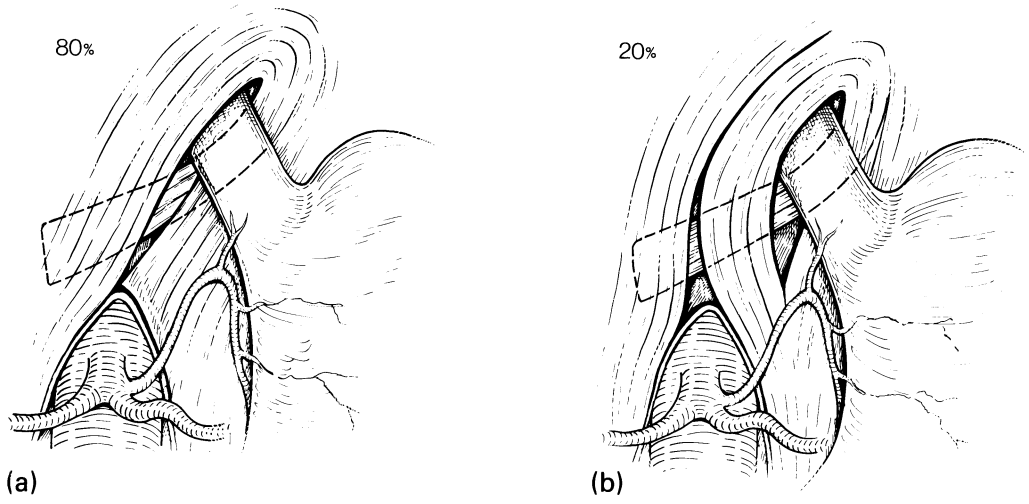


Figure 12.2(a, b) Variation in the disposition of the diaphragm fibres at the aortic hiatus

dle must also be divided before reaching the aorta (Figure **12.2b**). The inferior phrenic arteries are then divided and a 6–10 cm segment of the aorta cleared above the coeliac artery.

The next stage is to expose a suitable segment of each visceral artery for distal graft attachment. Uncommonly, the operative procedure may be confined to the coeliac artery, if this is the sole vessel involved or if the superior mesenteric artery is diffusely diseased or calcific. An end-to-end distal anastomosis to the coeliac stump may then be utilized (Figure **12.3**). Alternatively, coeliac revascularization may be achieved by end-to-side bypass attachment to either the hepatic or splenic artery. This will avoid the need to dissect the coeliac origin, while at the same time ensuring a wide distal anastomosis at a site free from intimal thickening. Usually the common hepatic artery is the most suitable branch and a segment of this vessel should be isolated at the upper border of the pancreas.

The superior mesenteric artery may be exposed in its interduodenopancreatic portion. This requires elevation of the transverse mesocolon and division of the ligament of Treitz. The duodenojejunal flexure is displaced inferiorly and the superior mesenteric vessels displayed by developing the interval between the pancreas and the third part of the duodenum (Figure **12.4**). The superior mesenteric artery is then separated from the adjacent vein in preparation for graft attachment.

Two segments of PTFE or knitted Dacron are then sewn together to form a branched graft. If Dacron is used, it is advantageous to cut one of the limbs from a standard bifurcation graft so as to produce a flange for aortic attachment (see Figure **12.3**). Instead of two graft segments sewn together, a preformed bifurcation graft of suitable size, e.g. 12 mm×6 mm may be used, though this gives less flexibility with regard to the disposition of each limb.

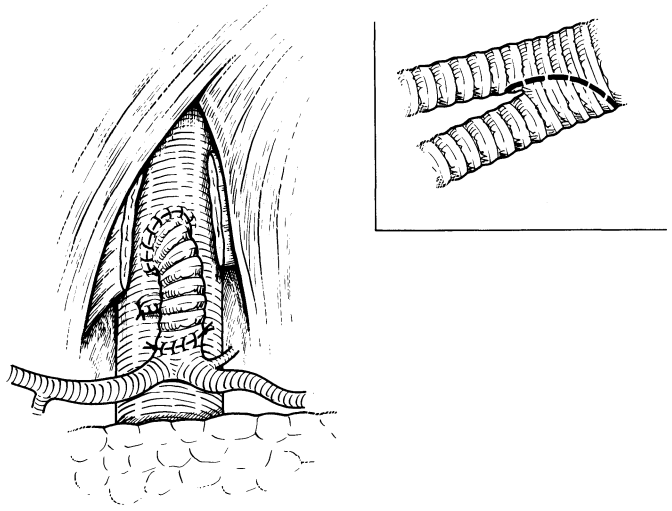


Figure 12.3 Variation in the technique of antegrade aortocoeliac bypass: the graft is attached end-to-end to the distal coeliac trunk. Inset shows the use of a bifurcation graft to provide a flange for aortic attachment

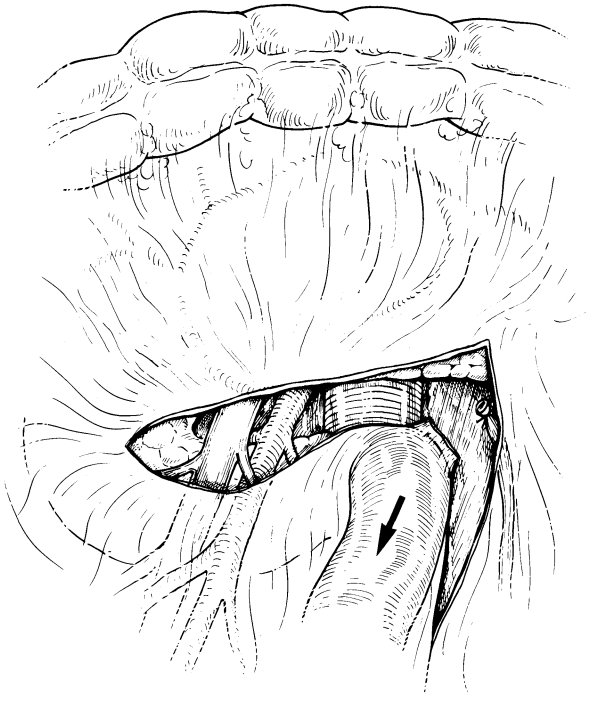


Figure 12.4 Exposure of the superior mesenteric artery in its interduodenopancreatic segment. The ligament of Treitz and the inferior mesenteric vein have been divided to allow the interval between the pancreas and the third part of the duodenum to be developed

The patient is heparinized and the distal thoracic aorta cross-clamped above and below the proposed anastomosis site. Partial aortic occlusion has the advantage of preserving distal perfusion, but suture placement is more difficult and this method is not recommended. Pharmacological and fluid adjustments may be necessary to avoid left heart strain during the cross-clamp period (see Chapter 2). An elliptical aortotomy 2 cm in length is then made above the coeliac origin and the first graft limb attached in end-to-side fashion. If the aortic wall is unduly friable, Teflon or Dacron pledgets may be used to reinforce the superior and inferior angles of the suture line (see Chapter 1).

Once the aortic attachment is complete, any debris within the aortic lumen is flushed via the unattached end of the graft and aortic flow restored. The superior mesenteric limb is clamped at its origin and the coeliac limb cut to length under tension and anastomosed end-to-side to the common hepatic artery. Flow is then started in the coeliac territory (Figure **12.5b**).

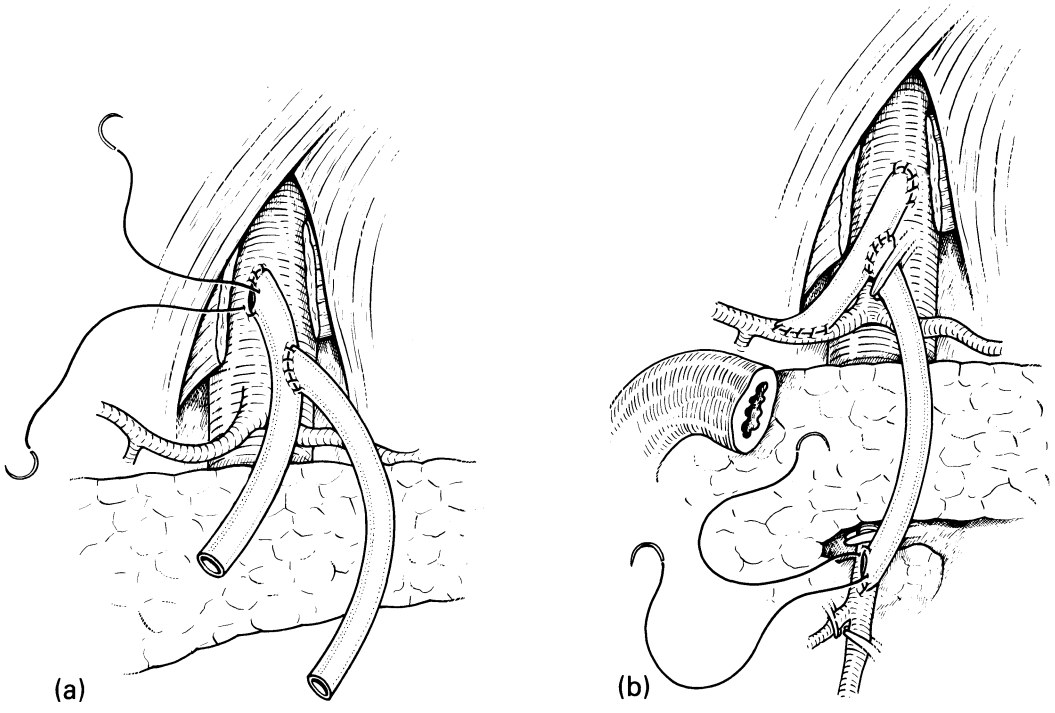


Figure 12.5(a–c) Antegrade aortocoeliac and aortomesenteric bypass: (a) two PTFE segments have been sewn together and the first limb is being attached to the supracoeliac aorta (clamps omitted for clarity). (b) coeliac revascularization completed. The second graft limb is passed in front of the pancreas and through the base of the transverse mesocolon to the superior mesenteric artery

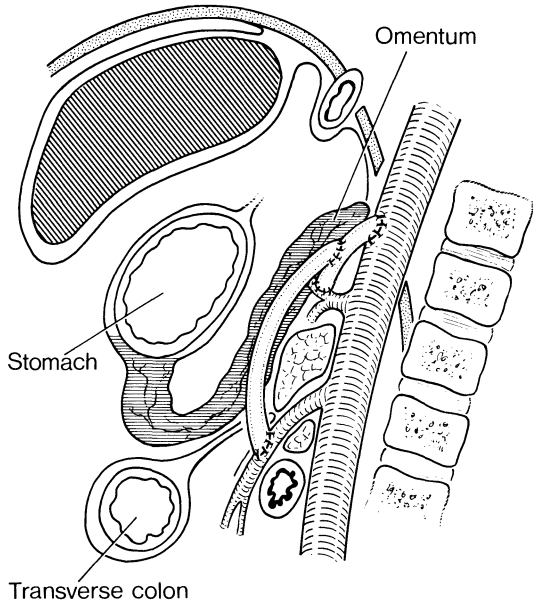


Figure 12.5 continued (c) lateral view of completed reconstruction. The superior mesenteric graft has been wrapped in omentum to isolate it from the gastrointestinal tract

The second graft limb is then led down either in front of or behind the pancreas to the superior mesenteric exposure. In the former case, a window should be cut in the base of the transverse mesocolon to allow the graft to enter the infracolic compartment. The graft is then cut to the appropriate length and attached end-to-side to the superior mesenteric artery near the first jejunal branch. If the proximal vessel is completely occluded, a spatulated end-to-end anastomosis may be preferable. Where a pre-pancreatic course has been adopted, the graft should be wrapped in omentum to isolate it from the gastrointestinal tract (Figure **12.5c**). Before closing the abdomen a fundoplication may be added to prevent possible postoperative gastro-oesophageal reflux secondary to division of the right crus.

Antegrade visceral artery bypass has proved to be a satisfactory method of reconstruction in most patients. Apart from the depth of the operative field and the minor hazard of opening the right pleura, the main potential problems relate to manipulating and clamping the supracoeliac aorta. The effects of supracoeliac clamping on cardiac haemodynamics and renal function are described elsewhere (see Chapters 2 and 13). Ascending aortic thrombosis is a specific risk in patients with severe distal aortoiliac stenosis. Adequate systemic heparinization and a short cross-clamp time should avert this complication. Embolization of the visceral or renal arteries is an additional problem which may occur during initial aortic mobilization, or on clamping or declamping, if loose atherothrombotic material is present in the suprarenal aortic lumen. Identification of such material on the preoperative arteriogram may be an indication for transaortic endarterectomy rather than a bypass technique (see below).

Combined aortic and visceral artery reconstruction

In many patients significant visceral artery atherosclerosis is found in association with aortoiliac aneurysmal or occlusive disease. These cases may be managed by a combined prosthetic reconstruction (Figure **12.6a–h**).

The infrarenal aorta is first exposed in the usual way. The common hepatic artery is then dissected out at the upper border of the pancreas and a retropancreatic tunnel developed by blunt dissection between the two exposures. The superior mesenteric artery may be conveniently approached in its intramesenteric portion by reflecting the duodenojejunal flexure to the right and incising the posterior peritoneum of the mesentery (Figure **12.6b**).

After systemic heparinization, the aorta is transected below the renal arteries and an aortic prosthesis is attached in end-to-end fashion. Supracoeliac control may be used if the juxtarenal aorta is unsuitable for clamping (Figure **12,6c, d**).

A 6 mm or 8 mm tube graft is then run from the body of the prosthesis to the common hepatic artery via the retropancreatic tunnel (Figure

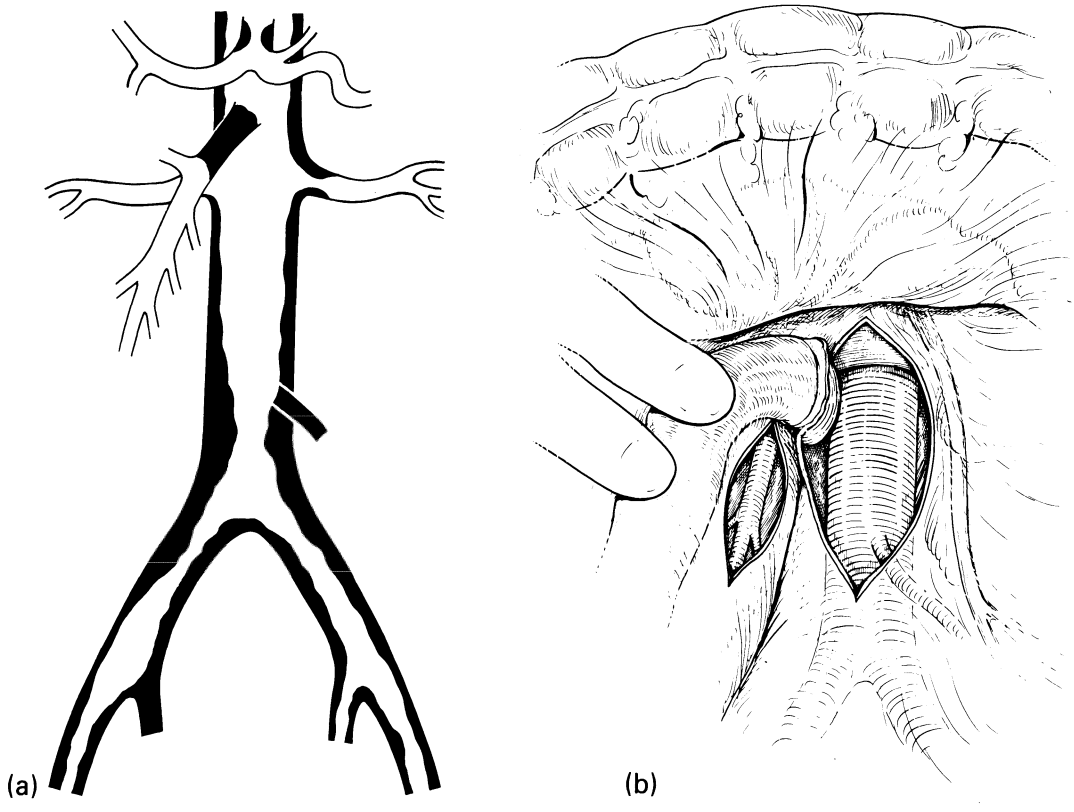


Figure 12.6(a–h) Combined aortic and visceral artery bypass for occlusive disease: (a) pathology. (b) exposure of the superior mesenteric artery via a posterior incision in the mesentery

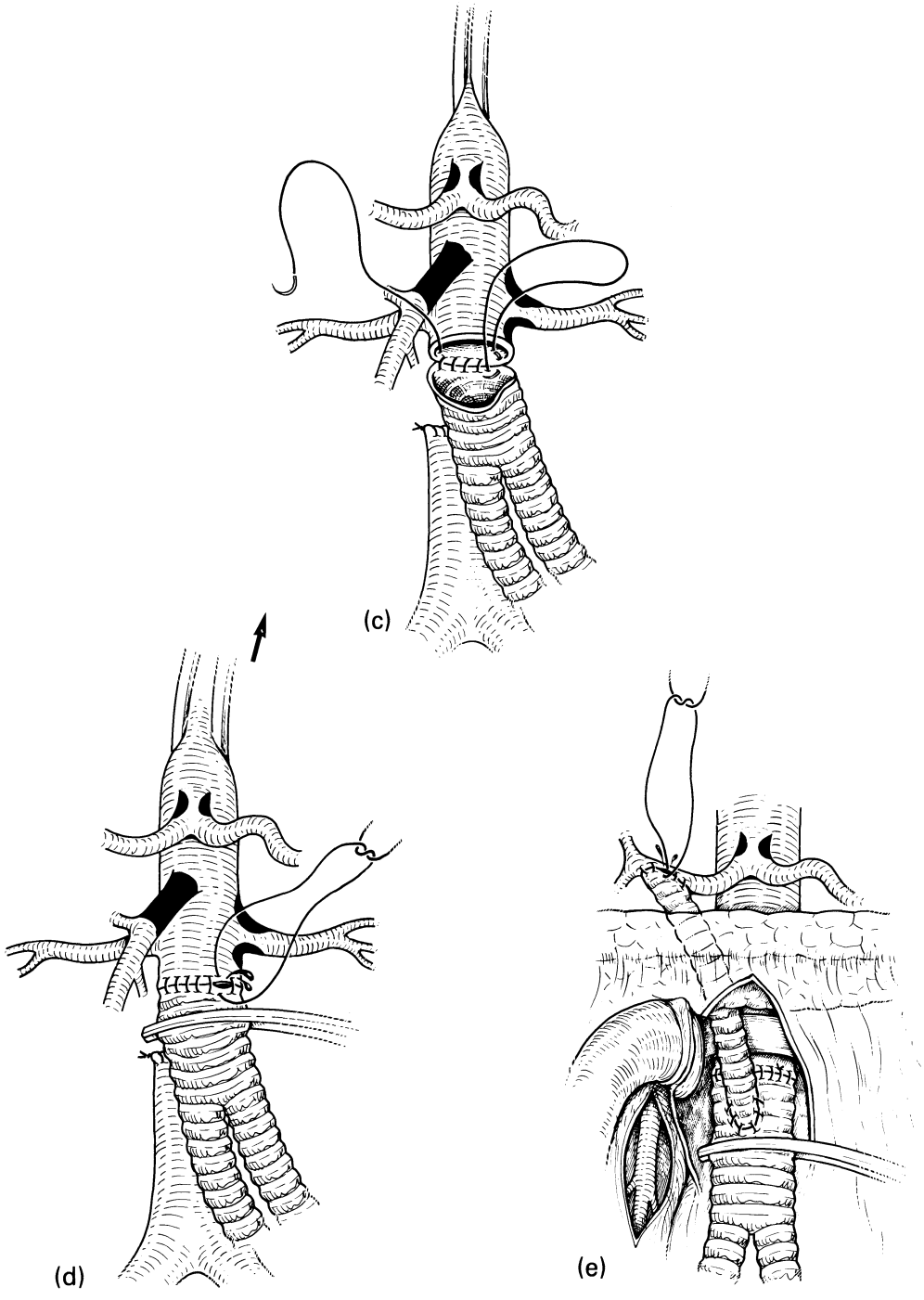
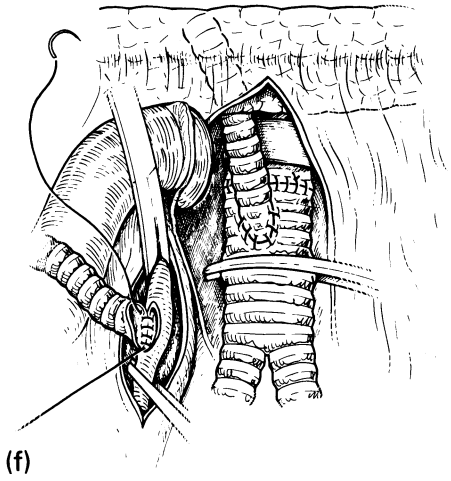


Figure 12.6 continued) (c, d) the aortic prosthesis is inserted under supracoeliac control. (e) a side limb is led from the aortic prosthesis to the common hepatic artery via a retropancreatic tunnel

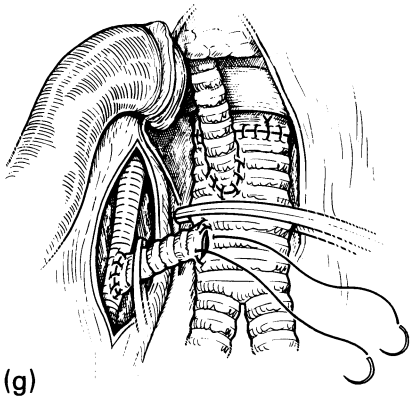


(f)

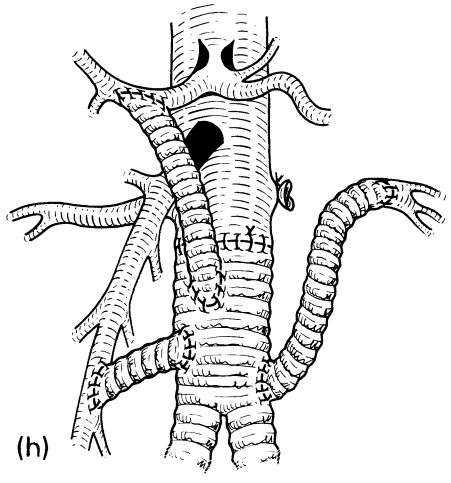
12.6e). Once coeliac flow has been established a second graft is led from the aortic prosthesis to the superior mesenteric artery (Figure **12.6f, g**). This limb must be kept very short to prevent kinking with movement of the small bowel mesentery. An alternative which avoids this difficulty is to loop the graft superiorly above the left renal vein for antegrade attachment to the superior mesenteric artery in its interduodenopancreatic segment[14].

Additional grafts may then be attached to the renal arteries if there is coexistent renovascular disease (Figure **12.6h**). Finally, the aortic prosthesis is attached distally to the iliac or femoral arteries, as appropriate.

If the infrarenal aorta is unsuitable for graft attachment, or if access to this segment is limited by previous surgery, the supracoeliac aorta may be used as source for the entire reconstruction. An aortofemoral prosthesis is first attached end-to-side to the supracoeliac aorta and additional limbs are then run antegradely from this to the visceral arteries. The limbs of the primary graft are then attached distally to the femoral arteries.



(g)



(h)

Figure 12.6 continued (f, g) the superior mesenteric limb is attached. (h) completed reconstruction. In this example an additional left renal artery bypass has also been carried out

ENDARTERECTOMY

One of the earliest methods of visceral artery reconstruction involved open endarterectomy of the proximal diseased segment. However, in most hands this method has proved unsatisfactory since attempts to clear the vessel origin completely may result in aortic dissection or embolization, while a more limited procedure may risk immediate occlusion or early re-stenosis.

Transaortic endarterectomy is an alternative technique which may avoid these difficulties. Its advantages compared with other reconstruction methods are that both the coeliac and superior mesenteric arteries can be cleared under one relatively short period of aortic occlusion and that graft materials are not needed. However, transluminal eversion endarterectomy, is inevitably something of a blind procedure, with less than full control of the distal end point in the branch vessel. Hence it should only be attempted when the disease is strictly localized to the aortic ostium or extreme proximal segment of both branch vessels.

Diffuse thickening in the visceral arteries or pre-aneurysmal degeneration of the aortic wall are contraindications to endarterectomy, since a satisfactory end point in the visceral vessel may be difficult to achieve. Complete occlusion rather than stenosis of the superior mesenteric artery is also difficult to manage by a transaortic technique, because the thrombotic process usually extends too far along the vessel to allow clearance from within the aortic lumen. Transaortic endarterectomy involves a more extensive exposure and longer operating time than a bypass technique, and age or operative risk factors may be additional contraindications to this approach.

Technique of transaortic visceral artery endarterectomy

Access may be achieved by either a left thoracoabdominal incision[13,15] or a retroperitoneal exposure through the bed of the eleventh rib[16,17]. In the technique described by Stoney et al. the left kidney remains *in situ* and a retroperitoneal approach is made to the proximal abdominal aorta and visceral artery origins[13] (Figure **12.7a**). Others have preferred to reflect the kidney anteriorly and to open the aorta along the posterolateral wall[15,17]. In the former method the patient is heparinized and clamps are applied to the aorta and to the renal and visceral arteries. A trapdoor aortotomy is then made around the coeliac and superior mesenteric origins. The aortic intima is detached from the undersurface of this trapdoor and the visceral artery origins approached individually. Traction is applied to the specimen and a cleavage plane developed in the wall of each visceral artery as the latter is prolapsed into the aortic lumen (Figure **12.7b**). Providing a sufficiently superficial plane is followed, the specimen should break free at the end point, leaving a thin adherent intimal edge.

Where the aorta is more extensively diseased,

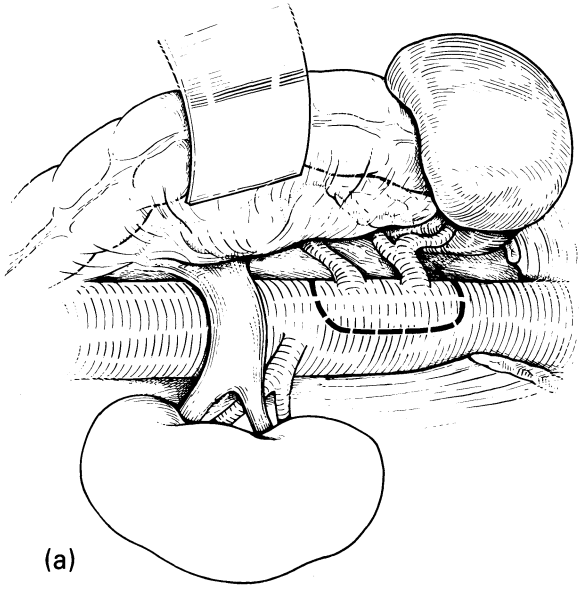


Figure 12.7(a–c) Transaortic endarterectomy of the visceral arteries: (a) left thoracoabdominal retroperitoneal approach. The aortotomy is outlined

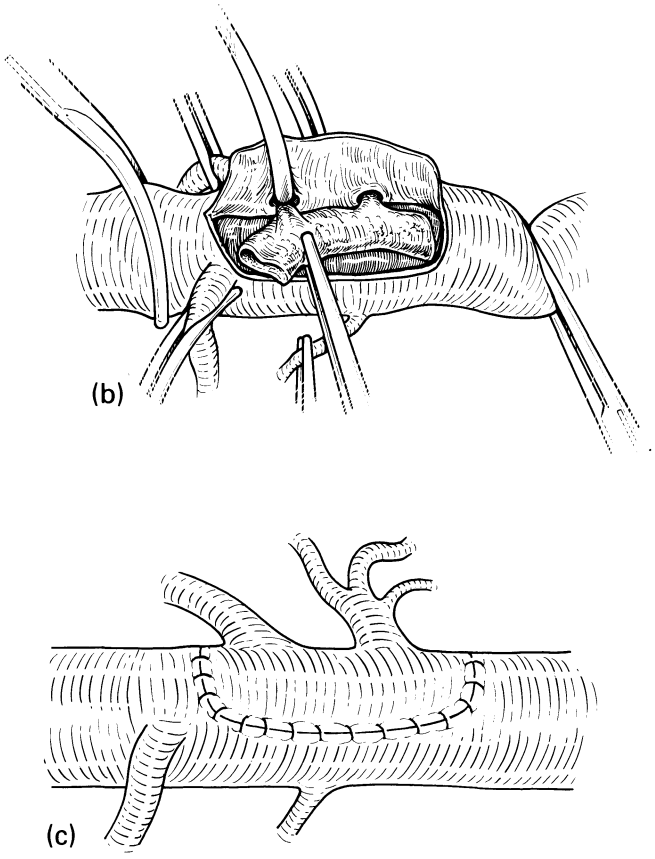


Figure 12.7 continued (b) aortic trapdoor elevated anteriorly to allow eversion endarterectomy of the coeliac and superior mesenteric orifices. (c) aortotomy closed

a cylinder of intima should be removed from the full aortic circumference, in continuity with the plaque at the visceral artery origins. Tack-down sutures may then be required to control the distal aortic intimal edge. The clamps are momentarily released to flush out residual debris and after rinsing with heparin-saline, the aortotomy is closed with a running stitch (Figure **12.7c**). Flow is then released into the distal aorta and into the renal and visceral arteries.

If necessary the initial aortotomy may be extended distally to allow an additional endarterectomy of the renal artery origins (see Chapter 13). Coexistent infrarenal occlusive or aneurysmal disease can also be managed from this approach, but with less facility than via the customary anterior transperitoneal route.

Inferior mesenteric endarterectomy

Stenosis of the inferior mesenteric artery origin may be corrected by simple patching[3]. However, eversion endarterectomy may be a more satisfactory option, either via a trapdoor aortotomy as above, or by excising a complete disc of aortic wall around the vessel origin. Once the atherosclerotic intima has been removed, the disc is reimplanted into the aorta (or into a prosthesis if a concomitant aortic replacement is being undertaken).

REIMPLANTATION

This may be a suitable method of revascularizing isolated superior mesenteric occlusive disease[18]. Reimplantation is not feasible in the case of the coeliac artery because the trunk of the vessel is too short.

The superior mesenteric artery is exposed between the pancreas and the third part of the duodenum and then mobilized back towards the aorta with progressive cephalic retraction of the pancreas. If the inferior pancreaticoduodenal artery limits mobility it may be divided, though the middle or right colic artery should be carefully preserved.

Systemic heparin is given and the superior mesenteric artery sectioned as close as possible to the aorta and immediately distal to the main atheromatous lesion. Any thrombus present should be extracted from the distal stump, after which the artery is trimmed and spatulated ready for aortic attachment. The aorta is cross-clamped and an ellipse of aortic wall excised caudal to the left renal vein. The stump of the superior mesenteric artery is then sewn in here using double-armed interrupted sutures passed from within outwards (Figure **12.8a**).

Reimplantation is a relatively simple autogenous technique but its use may be limited by the anatomical disposition of the superior mesenteric branches and by the state of the aortic wall. If there is an insufficient length of artery proximal to the middle colic branch or if an anomalous hepatic artery arises from this segment, direct reimplantation becomes impossible and a prosthetic (or vein) graft should be interposed between the aorta and the superior mesenteric stump (Figure **12.8b**). This graft must be kept short to avoid any risk of kinking. Atherosclerosis in the aortic wall may also limit direct reimplantation since fine suturing then becomes difficult. A prosthetic interposition may help, particularly if the aortic end is cut with a flange. Where the aortic disease is more marked, the superior mesenteric artery may be reimplanted into a supracoeliac aortofemoral bypass (Figure **12.9**).

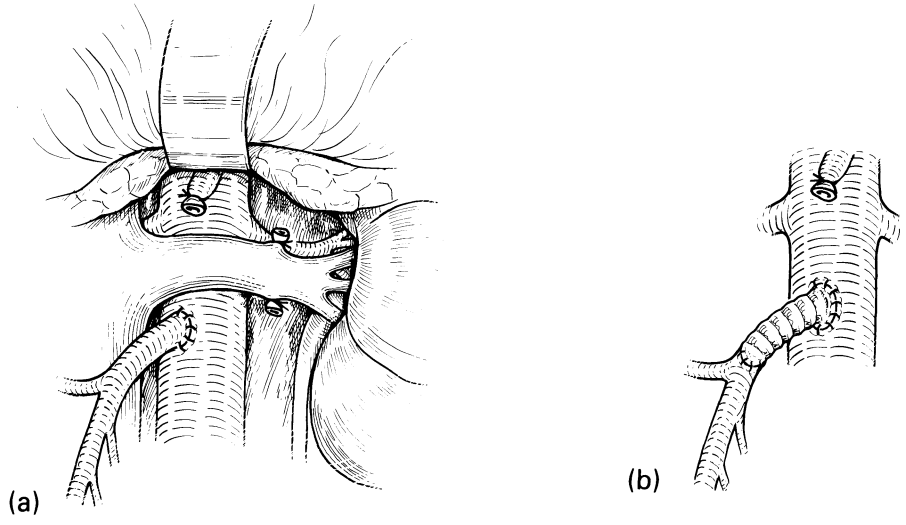


Figure 12.8(a, b) Reimplantation of the superior mesenteric artery: (a) direct reimplantation caudal to the left renal vein. (b) variant: reimplantation using an interposition graft (Dacron)

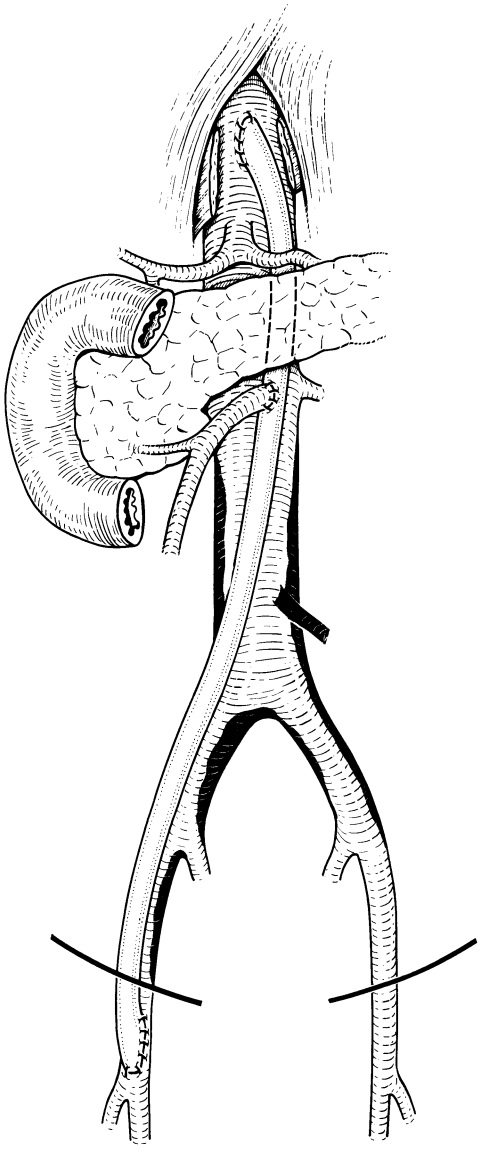


Figure 12.9 Alternative method of managing combined aortoiliac and visceral artery occlusive disease: a supracoeliac aortofemoral bypass (PTFE) has been inserted and the superior mesenteric artery reimplanted into the graft

TRANSLUMINAL ANGIOPLASTY

Percutaneous transluminal angioplasty is a recently available alternative to surgery in the management of chronic intestinal ischaemia. Patients with stenotic disease rather than complete occlusion of the visceral arteries are suitable, and good results have so far been obtained both radiologically and in terms of symptomatic relief[19,20]. As yet, the durability of transluminal angioplasty in the mesenteric territory is unknown, though clearly the method has particular appeal in the poor risk surgical case. Some limitation in the response to renal angioplasty has been observed when the narrowing is due to aortic atheroma rather than to intrinsic disease within the renal artery[21], and the same may apply to visceral artery atherosclerosis.

COELIAC ARTERY COMPRESSION SYNDROME

Extrinsic compression of the coeliac artery by the median arcuate ligament may occasionally be responsible for abdominal pain and other symptoms. Although controversy surrounds this condition, it does seem to represent a definite clinical entity and, providing patients are carefully selected, coeliac decompression may achieve symptomatic relief[22,23].

The syndrome tends to occur in younger patients, mostly women, and the chief complaint is abdominal pain. This may be related to meals, but often is of a more constant nature. Additional features such as diarrhoea, nausea and vomiting, an epigastric bruit and weight loss may occasionally be present. Diagnosis is made by excluding other possible causes of the pain and by demonstrating extrinsic compression of the coeliac artery on lateral aortography. Typically, the artery is seen to pursue a sharp downward bend at the site of compression, often with post-stenotic dilatation.

The basis of treatment is to divide the median arcuate ligament followed by clearance of investing fibres from around the coeliac origin. If secondary changes have occurred in the vessel wall in the form of stenosis or post-stenotic dilatation, an additional arterial reconstruction will be necessary. This may involve an aortocoeliac graft or an aortohepatic bypass with exclusion of the diseased zone.

ACUTE INTESTINAL ISCHAEMIA

Acute intestinal ischaemia remains a difficult clinical problem with a high mortality[24,25]. In cases of mesenteric infarction diagnosed before death, about half are due to acute superior mesenteric arterial occlusion, with embolization and thrombosis occurring in roughly equal proportions. Non-occlusive ischaemia and mesenteric venous thrombosis account for most of the remainder[26].

SUPERIOR MESENTERIC ARTERY EMBOLIZATION

Although embolic occlusion of the coeliac or inferior mesenteric artery may occur, this is rare, and for practical purposes only the superior mesenteric artery need be considered. Embolization is marked by the sudden onset of central abdominal pain, usually colicky in nature and characteristically out of all proportion to the physical signs. Often the pain is followed by vomiting or diarrhoea due to reflex gastrointestinal emptying. Initial examination may reveal a soft abdomen with perhaps minimal tenderness centrally or to the right of the umbilicus. Later on, if the condition goes undiagnosed, infarction supervenes with progressive abdominal distension and tenderness, sometimes bloody diarrhoea and systemic toxic effects. The white blood cell count is markedly raised at an early stage in most but not all patients.

The identification of an embolic source is an important point in the diagnosis. Likely sources include myocardial infarction, valvular disease, ventricular aneurysm or an arrhythmia, particularly atrial fibrillation. Rarely a thoracic aortic aneurysm or ulcerative plaque may be responsible. About a third of cases have an antecedent history of embolization elsewhere and this may be an additional help in establishing the diagnosis.

A chest X-ray may show evidence of an underlying cardiac (or thoracic aortic) cause for the embolus. Initial plain films of the abdomen are characterized by the absence of intestinal gas, though this is soon succeeded by non-specific small bowel distension. The essential investigation is arteriography and this should be obtained as a matter of urgency whenever a patient with an embologenic cardiac lesion suddenly develops abdominal pain. Although this policy may result in a number of negative arteriograms, this is more than offset by the possibility of diagnosing the condition at the pre-infarction stage.

Transfemoral arteriography is utilized and an initial aortic injection carried out to evaluate the proximal segment of the visceral arteries and to exclude renal or splenic emboli, which may also cause abdominal pain. This should be followed by selective catheterization to determine the site of the embolus and to exclude non-occlusive ischaemia or thrombosis (see below). Characteristically, an embolus produces a sharp cut-off (meniscus sign) within the artery several centimetres distal to the aortic origin. In some cases the distal mesenteric tree is opacified as a result of collateral flow or contrast escape around the embolus.

Embolectomy

Most major emboli are best managed by surgical means. If cardiac function is impaired or there is hypovolaemia or electrolyte imbalance, these matters should be urgently corrected while arrangements are being made for surgery.

Access is achieved via an anterior midline incision. The most frequent site of embolic occlusion is at or just distal to the middle colic origin, where the superior mesenteric trunk narrows (Figure **12.10**). Occlusion here results in ischaemia of most of the small bowel and a variable amount of the proximal colon. Typically, the first jejunal loop retains a normal vascularity, while the distal bowel may be rather grey and inert or showing signs of more advanced ischaemia. Elevation of the transverse mesocolon and palpation deep within the root of the mesentery will reveal a pulse in the proximal superior mesenteric artery. These findings are in contrast to those of superior mesenteric artery thrombosis, where the entire small bowel is usually ischaemic and a pulse cannot be palpated in the root of the mesentery.

Unless the bowel is already extensively infarcted, embolectomy should be attempted. The superior mesenteric artery is first exposed below the duodenum by elevating the transverse mesocolon and drawing the first jejunal loop down-

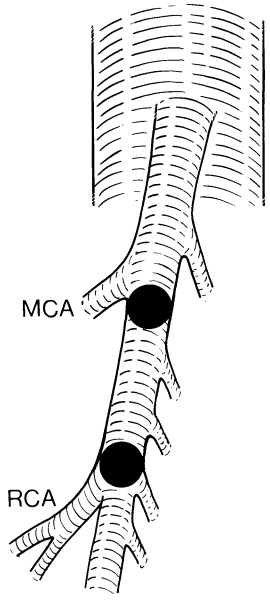


Figure 12.10 The two most common sites of superior mesenteric embolization. MCA = middle colic artery; RCA = right colic artery

wards and to the left (Figure **12.11a**). This puts the root of the mesentery under tension. Incision of the peritoneum at this point then exposes the superior mesenteric vessels immediately adjacent to the origin of the middle colic artery (Figure **12.11b**).

The superior mesenteric artery is separated from the accompanying vein for 3–4 cm and a longitudinal arteriotomy is made over the embolus. The latter may extrude spontaneously or be lifted out with forceps. Proximal and distal clearance is then completed using a 3F Fogarty catheter. If there is extensive distal propagated clot, or the embolus has fragmented, a supplementary distal arteriotomy may be required to allow catheterization of the branch vessels. Great care must be taken with these catheter manipulations, otherwise damage may occur to the edges of the arteriotomy or to the fragile distal mesenteric tree. Once the vessels have been cleared of

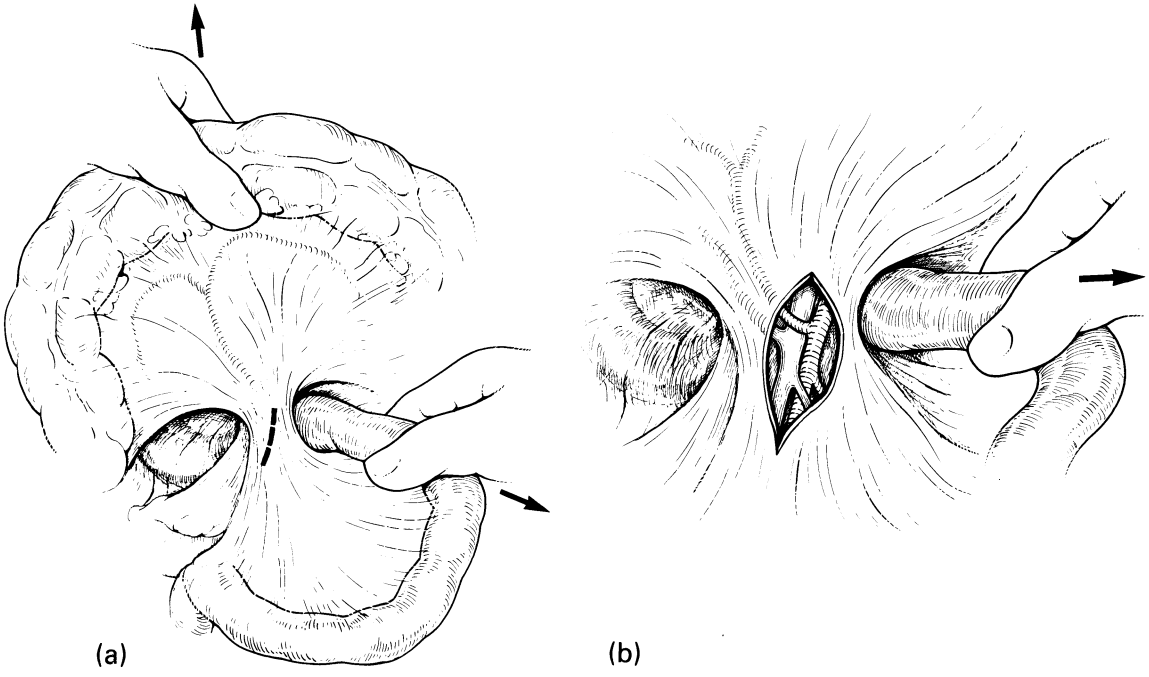


Figure 12.11(a–d) Superior mesenteric embolectomy: (a) the transverse mesocolon is elevated superiorly and the duodenojejunal flexure drawn to the left, putting the root of the mesentery under tension. (b) incision of the peritoneum then exposes the superior mesenteric artery and vein

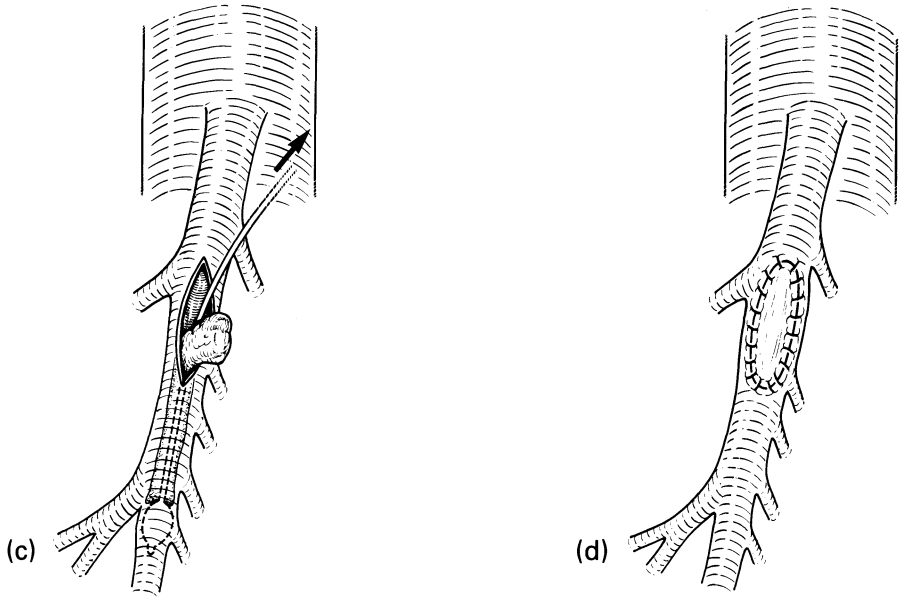


Figure 12.11 continued (c) superior mesenteric arteriotomy at the middle colic origin. A distal thrombectomy is then achieved using a Fogarty catheter. (d) arteriotomy closed with a vein patch

clot, heparin–saline is instilled distally and the arteriotomy closed with a vein patch.

If a good antegrade flow cannot be established by catheter thromboembolectomy and the distal tree is clear, an aortomesenteric or iliomesenteric bypass graft may be inserted, using the initial arteriotomy site for distal graft attachment.

After revascularization, it may be advisable to wait for approximately 20 minutes before assessing gut viability. Quite often, segments which seemed non-viable prior to revascularization are observed to regain their viability. Any obviously infarcted bowel should then be resected. Segments of marginal viability are best left for reassessment at a 'second look' procedure 12–24 hours later. Doppler ultrasound and fluorescein studies[27,28] have not yet proved sufficiently reliable to obviate the need for this 'second look' procedure in clinically borderline cases.

Following revascularization every effort should be made to maximize gut perfusion. This may necessitate continued use of cardiac support drugs, and measures to correct any hypovolaemia or acidosis. Mesenteric vasoconstriction may persist after an otherwise successful embolectomy and an intra-arterial vasodilator infusion has been utilized post-embolectomy in some centres[29]. Systemic antibiotics begun at the time of surgery should also be continued into the postoperative period. As with emboli elsewhere, postoperative anticoagulation should be instituted to minimize the risk of recurrent embolization (see Chapter 6).

Non-surgical treatment

Patients with peripheral emboli, or those with proximal emboli and critical cardiac disease, may be initially managed non-operatively providing peritoneal signs are absent. Several therapeutic options are available, including low-dose intra-arterial streptokinase[30,31], vasodilator infusion[29] or both. Repeat arteriography is essential to check that progress is being made. In addition, careful clinical observation should be maintained throughout. If peritoneal signs develop, immediate laparotomy should be undertaken irrespective

of the arteriographic status.

An alternative non-operative approach is percutaneous transfemoral catheter extraction; which may be particularly appropriate in poor risk cases with a major embolus (see renal embolectomy Chapter 6). The embolus may be either dislodged into a non-critical vessel[32] or retrieved via a femoral arteriotomy under local anaesthesia[33].

SUPERIOR MESENTERIC ARTERY THROMBOSIS

Whereas a survival rate of 50% or more may nowadays be expected in mesenteric embolization, acute superior mesenteric artery thrombosis is still almost invariably fatal[24,25]. This poor outlook may be due to delay in diagnosis, more extensive gut infarction and greater difficulty in revascularization compared with simple embolectomy.

Clinically the onset of mesenteric thrombosis is usually insidious, with central abdominal pain which persists and becomes more severe and which may be accompanied by diarrhoea or vomiting. As with superior mesenteric embolization, physical signs in the abdomen are conspicuous by their absence initially, though later on the features of infarction appear with distension, tenderness, sometimes bloody diarrhoea and progressive systemic effects. Abdominal plain films may show non-specific dilated intestinal loops and the leucocyte count is markedly elevated. A number of these patients give a history of previous intestinal angina and weight loss and most will show evidence of atherosclerotic occlusive disease elsewhere.

If the diagnosis is considered at a sufficiently early stage, an arteriogram should be obtained. This will distinguish thrombotic occlusion from embolization or non-occlusive ischaemia and will allow proper surgical planning. Mesenteric thrombosis may appear on arteriography as an arrest of contrast medium at, or immediately adjacent to, the vessel origin. Additional occlusive disease may be present in the other visceral arteries with a variable degree of collateral formation (see above). Unfortunately, in many cases the diagnosis is not considered until a late stage when arteriography is clearly inappropriate.

Once the condition is recognized immediate laparotomy should be undertaken. In the early case the bowel may appear acutely ischaemic but largely retrievable. This fortunate and uncommon state of affairs is most likely to be observed in patients requiring urgent laparotomy for an acute exacerbation of intestinal angina, or those who develop abdominal pain following mesenteric arteriography. If both the coeliac and superior mesenteric arteries are found to be occluded, an antegrade aortocoeliac or aortohepatic bypass may be inserted, as previously described. Coeliac revascularization alone will usually be sufficient to restore intestinal viability in these cases and poses less of a stress on these acutely ill patients than a double revascularization. Where coeliac bypass is not sufficient to restore gut perfusion or only the superior mesenteric artery is occluded, the latter vessel may be reconstructed by reimplantation or antegrade bypass. In either case a preliminary distal thrombectomy may be necessary before completing the reconstruction.

One advantage of grafts originating from the supracoeliac aorta is that they seem relatively protected from secondary infection, even when intestinal resection or enterostomy is necessary. Nevertheless, in the presence of established or incipient gangrene it is advisable to use autogenous vein rather than prosthetic materials for these procedures. In favourable cases, antegrade visceral artery bypass via a thoracoretroperitoneal approach may be considered and may permit the use of prosthetics. Once the reconstruction is completed, the peritoneum is opened to allow inspection of the gut.

Following revascularization, an interval of approximately 20 minutes should be allowed before assessing the viability of the bowel. Obviously infarcted bowel should then be resected with end-to-end anastomosis. As previously described, a 'second look' procedure may be considered where there are doubts regarding gut viability.

In the great majority of patients with superior mesenteric artery thrombosis, diagnosis is delayed and by the time operation is undertaken the bowel is extensively infarcted. Typically, gangrenous changes are found extending throughout the entire small bowel and proximal colon with a

foul serosanguineous exudate in the peritoneal cavity. Extensive gut resection may be attempted, with either primary anastomosis or exteriorization, but postoperative problems are severe and very few patients survive this form of therapy[34].

NON-OCCLUSIVE MESENTERIC ISCHAEMIA

Acute mesenteric ischaemia may occur as a result of severe splanchnic vasoconstriction. This in turn may be precipitated by factors such as cardiogenic shock, hypovolaemia, septicaemia and digitalis or vasopressor therapy. Occasionally, splanchnic vasoconstriction is superimposed on a major visceral artery stenosis[35].

The clinical presentation may be similar to other forms of acute intestinal ischaemia. Thus abdominal pain may occur, with or without gastrointestinal emptying, usually against a background of congestive cardiac failure or recent myocardial infarction. A profound leucocytosis may be present, with little or no physical signs in the abdomen in the initial stages.

Arteriography should be obtained urgently to distinguish occlusive from non-occlusive ischaemia and to allow intra-arterial vasodilator therapy. Arteriographic findings in non-occlusive ischaemia include diffuse tapering of the distal mesenteric branches, segmental spasm and reflux of contrast following a selective mesenteric injection[36].

Various vasodilators have been used for intra-arterial infusion including papaverine, phenoxybenzamine and prostaglandin E. It may be necessary to maintain the infusion for 24 hours or longer according to the arteriographic response and the clinical course. This approach should be coupled with measures to improve the underlying cardiac status and to correct other causative factors. In patients with congestive cardiac failure, digitalis should be discontinued because of its vasoconstrictor effect and, if necessary, dopamine or isoprenaline substituted along with diuretic therapy. Any volume deficit or electrolyte imbalance should also be corrected. Those patients in whom splanchnic vasoconstriction is superimposed upon a proximal visceral artery stenosis should undergo percutaneous transluminal angioplasty prior to commencing vasodilator infusion.

In many patients infarction can be averted by the above measures. However, if peritoneal signs develop during therapy, laparotomy should be undertaken and any necrotic segments of bowel resected as appropriate.

SUPERIOR MESENTERIC VEIN THROMBOSIS

Superior mesenteric vein thrombosis usually occurs as a complication of some other disorder such as abdominal sepsis or neoplasm, disseminated intravascular coagulopathy, the contraceptive pill, splenectomy or cirrhosis[37]. The presenting features may include abdominal pain, vomiting and sometimes diarrhoea. As with other forms of acute mesenteric ischaemia, physical signs in the abdomen are absent in the initial stages.

At operation the bowel is cyanosed and oedematous, usually with segmental infarction. The mid-small bowel tends to be maximally affected and the colon is usually spared. Further examination will reveal a normal pulse in the trunk of the superior mesenteric artery, though flow may be absent or diminished in the periphery. In thin subjects, thrombus may be visible in the veins of the mesentery.

The basis of treatment is gut resection with postoperative anticoagulation to limit clot propagation[38]. Venous thrombectomy is of little value because the thrombus usually extends right up to the bowel wall. A routine second or even third look procedure is advisable in this condition because of the frequency of thrombotic recurrence[37]. Overall prognosis is better than in acute arterial thrombosis, chiefly because the more limited extent of gut infarction[25,37].

VISCERAL ARTERY ANEURYSMS

The visceral arteries are an uncommon site for aneurysmal disease. However, as in the case of occlusive lesions, visceral aneurysmal disease is being recognized with increasing frequency nowadays as a result of the more widespread use of abdominal arteriography.

SPLENIC ARTERY ANEURYSMS

These are by far the commonest type of visceral artery aneurysm[39]. They are particularly associated with medial dysplasia, portal hypertension and repeated pregnancies[40]. Atherosclerosis is a common histological finding but is more often a secondary rather than a primary event. Splenic artery aneurysms usually involve the main trunk of the artery, particularly the distal portion, but may be present at multiple sites. Although these aneurysms are occasionally associated with vague upper abdominal pain, most are asymptomatic and are discovered by the incidental finding of curvilinear calcification in the left upper quadrant on plain abdominal X-ray. Similar calcification may occur with a tortuous splenic artery, a splenic cyst or a renal mass, and arteriography is therefore required to confirm the diagnosis. Rarely they may present with sudden abdominal pain and cardiovascular collapse due to rupture.

The incidence of rupture is low, probably amounting to less than 2% in the unscreened population[22,40]. Rupture may occur into the lesser sac initially, followed by free intraperitoneal rupture some time later ('double-rupture' phenomenon). Less commonly, splenic artery aneurysms may rupture into a viscus or into the splenic vein resulting in a large arteriovenous fistula. The risk of rupture may be increased in pregnancy particularly at the time of labour or delivery and the mortality rate may then be very high[41].

Splenic artery aneurysms can be effectively treated by surgery or embolization and most patients should be offered one or other of these modalities. Specific indications for treatment include aneurysms in women of child-bearing age, especially if discovered during pregnancy, symptomatic cases and those aneurysms which exceed 2 cm in diameter.

Surgery is the method of choice in young female patients because of proven effectiveness and avoidance of radiological exposure. The simplest method of correction is proximal and distal ligation with laying open of the sac. This is preferable to excision, which may necessitate dissection into the pancreas. The spleen may be left intact, as viability is assured by the short gastric arteries. Aneurysms located in the splenic hilum are best treated by splenectomy. If the aneurysm is large, an additional distal pancreatectomy may also be required.

Embolization may be considered in the remaining patients, particularly if there are operative risk factors. Steel coils or detachable balloons have proved effective for this purpose[42] (see Chapter 8). As with surgical exclusion, there should be ample collateral flow to ensure splenic viability.

HEPATIC ARTERY ANEURYSMS

Aetiological factors in hepatic artery aneurysms include atherosclerosis, medial dysplasia, trauma and infection. The majority of reported cases have been extrahepatic, occurring just proximal or distal to the gastroduodenal artery. Most intrahepatic aneurysms are of the false variety and occur as a result of previous penetrating trauma.

Hepatic artery aneurysms are frequently symptomatic, giving rise to right upper quadrant pain or biliary obstruction. They may also bleed into the biliary tree, peritoneal cavity or on occasions directly into the gut. Others may be diagnosed as a result of calcification on X-ray or following a hepatic ultrasound scan. Arteriography should be undertaken to confirm the diagnosis and to plan therapy.

Aneurysms of the common hepatic artery may be simply treated by surgical exclusion, or by embolization if there are operative risk factors. Although liver perfusion may be maintained via the gastroduodenal artery, this is sometimes deficient and a safer option in surgical cases is to combine exclusion with graft replacement (Figure **12.12a, b**).

Aneurysms of the proper hepatic artery may be managed by tangential excision and patching if the neck of the sac is narrow, or if not, by common–proper hepatic artery bypass[43]. Where the lesion is too distal for reconstruction, it may be embolized or ligated on the assumption that portal-venous and collateral flow will ensure hepatic viability.

Intrahepatic aneurysms may sometimes spontaneously regress. Those that persist can be effectively managed by embolization[44].

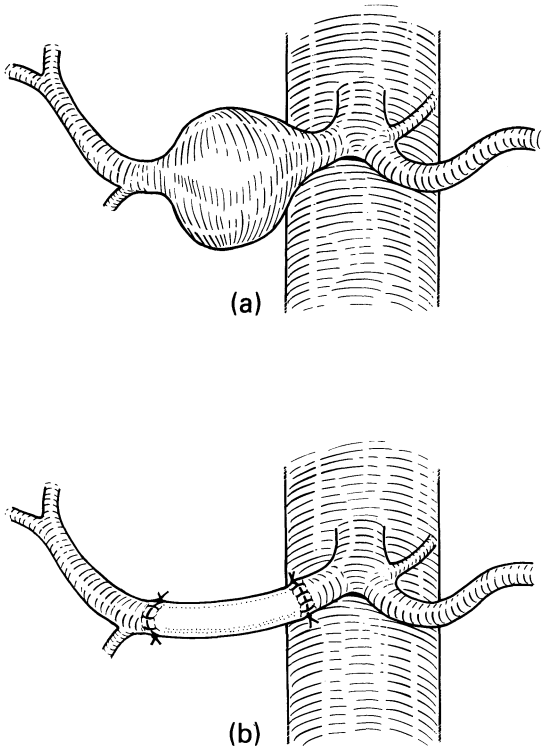


Figure 12.12(a, b) Common hepatic artery aneurysm treated by resection and PTFE graft replacement

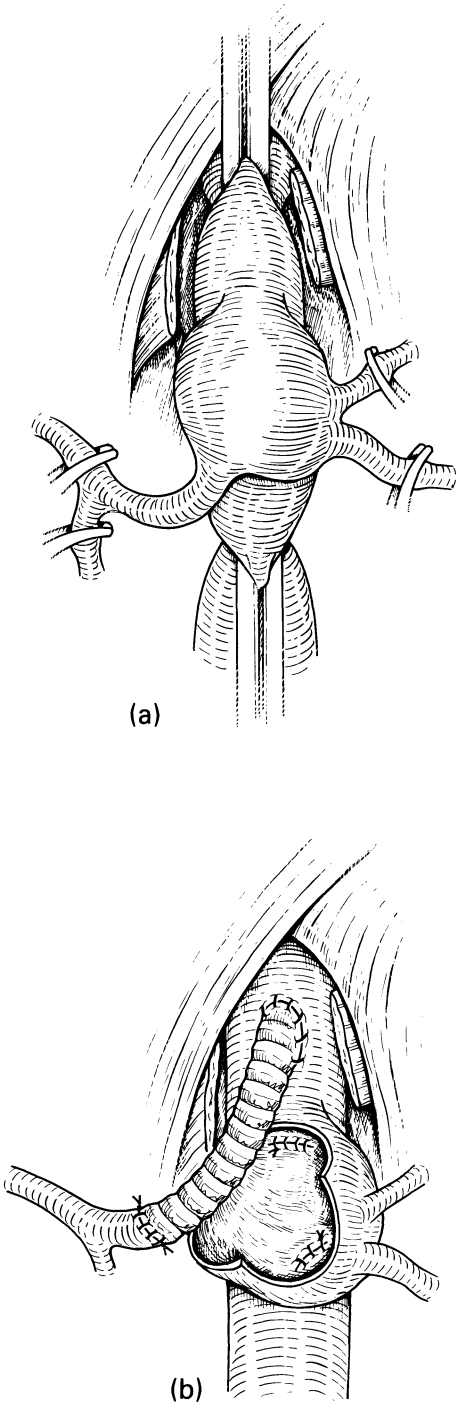


Figure 12.13(a, b) Coeliac artery aneurysm treated by exclusion and antegrade aortohepatic bypass

COELIAC ARTERY ANEURYSMS

Atherosclerosis or medial dysplasia are the pathological factors underlying most coeliac artery aneurysms. Occasionally these lesions may be associated with epigastric or back pain. Intestinal angina may also occur, either from intrasaccular thrombus or as a result of additional occlusive disease in the other visceral arteries. However, the great majority of cases are asymptomatic and

are discovered either during arteriography for coexistent aortoiliac aneurysmal or occlusive disease, or during an abdominal operation for an unrelated condition.

Operation is advisable in all cases because of the high mortality rate when rupture occurs[22]. The simplest method of management is to exclude the aneurysm by endosaccular suture. Distal perfusion may then be restored by an aortohepatic bypass graft (Figure **12.13a, b**). When there is additional infrarenal aortic disease, combined aortic and visceral artery reconstruction should be undertaken (technique see above).

SUPERIOR MESENTERIC ARTERY ANEURYSMS

These are most commonly mycotic in origin, and arise during the course of bacterial endocarditis or a non-cardiac septicaemia. Others are due to medial dysplasia, atherosclerosis or trauma.

Superior mesenteric artery aneurysms usually present with central or upper abdominal pain. A tender pulsatile abdominal mass may sometimes be detected. Pain may be due to expansion within the mesentery, though there may be an element of visceral ischaemia if collaterals are compromised or if superior mesenteric flow is reduced by intrasaccular thrombus. Rupture may occur at an early stage in mycotic lesions, while acute thrombosis is an additional possible hazard. Arteriography is helpful in establishing the diagnosis and will define the extent of collateral flow around the lesion.

Distally sited aneurysms may be managed by excision with an accompanying segmental bowel resection. Aneurysms of the proximal superior mesenteric artery are more of a problem. Occasionally, sufficient collaterals may have developed around the lesion to permit aneurysmal exclusion alone. Where this is not the case, the superior mesenteric artery should be reconstructed with an autogenous vein bypass graft[45]. In mycotic aneurysms, intensive systemic antibiotic therapy should be instituted to prevent graft colonization and the development of fresh lesions at other sites.

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13

Surgery of the Renal Arteries

Renal revascularization is primarily undertaken to cure or improve renovascular hypertension. A second major objective may be the preservation or restoration of renal function. Although the exact incidence of renovascular hypertension has not been clearly defined, renal artery occlusive disease is commonly regarded as the most important surgically correctable cause of hypertension. The underlying renal artery pathology is usually either atherosclerosis or fibromuscular dysplasia. Rarer causes include Takayasu's disease or other forms of arteritis, neurofibromatosis, abdominal aortic coarctation, chronic traumatic or embolic lesions and arteriovenous fistulae.

The diagnosis of renovascular hypertension should be particularly considered in young subjects, those with sudden acceleration of hypertension, older patients whose hypertension is refractory to medical treatment, hypertensive patients with early renal insufficiency and patients who have hypertension and an abdominal bruit[1].

The most widely used screening test for renovascular hypertension has been the rapid sequence intravenous pyelogram. This may be accurate in over 70% of patients with atherosclerotic renovascular disease, but is less helpful in young adults with fibromuscular dysplasia or in the paediatric age group[2]. Isotope renography[3] is associated with a high incidence of false positive and false negative results and is of limited value as a diagnostic screen. Peripheral plasma renin estimation has also been unreliable, though improved results have been claimed when combined with angiotensin blockade[4]. Intravenous digital subtraction angiography represents an improvement on these methods and may be particularly useful for follow-up after surgery, or other treatment[5]. However, resolution is less than that provided by conventional arteriography. In particular, subtraction artefacts may occur and in some cases there may be difficulty in visualizing the distal renal branch arteries.

Conventional arteriography is the most reliable method for delineating renal arterial disease. This may be feasible on an outpatient basis if sufficiently small (e.g. 5F) catheters are used. Resolution can be enhanced and the volume of contrast reduced by intra-arterial digital subtraction. A flush aortic injection is performed first, with AP and oblique views to show the general appearances of the renal arteries and to define any coexistent aortic or visceral artery disease. Selective catheterization with multiple views may then be employed if renal vascular anatomy remains difficult to define.

Atherosclerosis predominantly affects the

origin or proximal third of the main artery and gives rise to an eccentric or less commonly a concentric narrowing, often with post-stenotic dilatation. Bilateral lesions are present in 25% of cases. Fibromuscular dysplasia usually involves the middle or distal segments of the main artery and/or the branch arteries, with a 50–80% incidence of bilateral involvement. The main pathological types of dysplasia are intimal fibroplasia, medial fibroplasia, medial hyperplasia and perimedial fibroplasia[6]. By far the most frequent variant is medial fibroplasia and this gives rise to the classical 'string of beads' appearance on arteriography with multiple constrictions and intervening aneurysmal outpouchings. Other radiological findings may include focal stenosis, beaded narrowing and medial dissection with fusiform dilatation[7].

Accurate assessment of the distal tree beyond the obstructive lesions is as essential here as at other sites, and a thin clear cortex with attenuated vessels, intrarenal microstenoses or multiple areas of infarction are all relative contraindications to attempted revascularization.

Once an obstructive lesion has been identified on arteriography, an attempt should be made to assess its functional significance. Split renal function studies based on ureteric catheterization data have been discontinued in most centres due to unreliability and potential morbidity. Selective renal vein renin levels may be more helpful, and increased secretion on the ischaemic side with contralateral suppression is highly predictive of a favourable response either to surgery or to angioplasty[3,8]. Unfortunately, non-lateralization is misleading, since up to 50% of such patients may still be benefited by renal revascularization or nephrectomy[9,10]. Renal vein renin values should therefore be regarded as only a partial guide to therapy and if a significant arteriographic lesion is identified, definitive treatment by surgery or angioplasty should be undertaken, even if renin values are not diagnostic.

Stenotic lesions are usually regarded as significant when the lumen diameter is reduced by more than 50%. Collateral vessels are a certain sign that a lesion is haemodynamically significant, but are not commonly encountered other than in certain types of arterial dysplasia. Where there are bilateral renal artery stenoses both sides may be reconstructed at the same time if the lesions are severe. However, when there is obvious disparity between the two sides, the more severely stenosed vessel should be operated on first, in the expectation of a good blood pressure response.

Although renal artery surgery is principally undertaken to cure or improve renovascular hypertension, an additional or alternative objective may be the preservation of renal function. Renal artery disease may be both bilateral and progressive and, in atherosclerotic cases at least, may terminate in occlusion[11]. This disease progression is one of the factors responsible for the deterioration in renal function and loss of renal tissue which may occur in some patients with renovascular hypertension managed non-operatively[12]. Surgical correction of a severe renal artery lesion may not only protect the kidney from future deterioration but also restore or substantially improve renal function where impairment already exists. Severe azotaemia is a relative contraindication to revascularization since advanced parenchymal disease is nearly always then present. An exception to this rule is the patient with chronic bilateral renal artery occlusion and a viable kidney on one or both sides. These patients may be severely azotaemic yet respond in dramatic fashion to renal revascularization[14].

The success of reconstructive surgery in patients with total renal artery occlusion is dependent on preservation of renal viability by collateral flow. Favourable signs of salvageability include renal size greater than 9 cm, viable glomeruli on intraoperative biopsy, patency of the distal arterial tree on preoperative arteriography and a brisk back-bleed from the distal renal artery at surgery[15,16]. None of these criteria is absolute, and where doubt remains an intraoperative renal arteriogram may be decisive[17]. If this shows severe intrarenal disease or multiple areas of infarction, revascularization is unlikely to succeed and nephrectomy may be a more appropriate treatment option.

RENOVASCULAR SURGERY VS. TRANSLUMINAL ANGIOPLASTY

Renal artery surgery may be expected to cure or significantly improve hypertension in over 90% of patients with fibromuscular dysplasia and in over 80% of patients with focal atherosclerosis[18–20]. Operative mortality rates are now less than 1% in many series. Patients with generalized atherosclerosis were formerly regarded as an unfavourable surgical group[21], but good results are now being recorded in these cases as a consequence of better preoperative selection, intensive perioperative cardiovascular monitoring and improved surgical techniques[22,23].

The success rates following percutaneous transluminal angioplasty appear comparable to those achieved by surgical reconstruction, at least in the short-term. Thus, hypertension may be cured or improved in over 90% of patients with fibromuscular dysplasia and in approximately 80% of atherosclerotic patients in whom a technically successful dilatation has been possible[24–26]. Renal function may also be improved in a proportion of cases, though angioplasty may be less successful in this respect than surgery[27]. Initially a high recurrence rate was reported but with present techniques restenosis should not occur in more than 5% cases. Moreover, those patients who develop restenosis can usually be successfully redilated. Possible complications of angioplasty include intimal dissection, occlusion, distal embolization and perforation. However, in experienced hands morbidity should be minimal and mortality nil.

Although long-term follow-up is not yet available, transluminal angioplasty appears to be the treatment of choice in fibrous stenoses of the main renal artery or its proximal branches. Patients with non-ostial and non-occluded atheromatous lesions may also be treated by this method. Although angioplasty has obvious appeal in patients with operative risk factors some caution is required in this group. Many of these patients have severe aortic atherosclerosis which may not only limit the response but also increase the risk of embolization or other catheter-related accidents.

Where angioplasty is technically unsuccessful or results in a serious renal artery complication, surgical revascularization may be required. A surgical approach may also be specifically indicated in patients with atherosclerotic renal ostial stenosis or complete occlusion. In the former, the obstruction is due to aortic atheromatous encroachment on the renal artery origin. Dilatation merely deforms the aortic plaque for a short time and recurrence is inevitable[28]. Complete occlusions have been successfully recanalized by catheter techniques[29] but this is exceptional and these lesions should be regarded as primarily surgical. Paediatric patients with ostial hypoplasia should also be treated surgically in view of the recognised complications which may follow dilatation at this site[30]. Finally, surgery may be preferred for complex branch renal artery disease. Although proximal branch lesions are eminently treatable by angioplasty using a 'steerable' co-axial system (see Chapter 15), more distal lesions or combined aneurysmal and stenosing fibrous disease are best managed by microsurgical reconstruction.

PRE- AND INTRAOPERATIVE MANAGEMENT

Preoperative assessment in atherosclerotic cases should include careful screening for coronary artery and cerebrovascular disease, particularly in view of the reduction in perfusion pressure which may follow successful renal revascularization (see Chapters 2 and 9). If significant lesions are detected in these territories they should be corrected before undertaking renal artery surgery.

In the immediate period prior to surgery, antihypertensive drugs should be reduced to a minimum. If continued treatment is required, methyldopa with supplementary oral vasodilators should be substituted for other agents as this will provide greater stability during anaesthesia. More severe cases can be controlled with a carefully regulated nitroprusside infusion. Patients with renovascular hypertension often have a depleted blood volume, particularly if diuretic therapy has been utilized, and adequate fluid loading is important to prevent ischaemic tubular damage. An intravenous infusion should therefore be commenced preoperatively and in most cases 1 litre of Ringer's lactate with 500 ml colloid is given over a 12 hour period prior to anaesthetic induction.

Preoperative insertion of a Swan–Ganz catheter is advisable in patients with clinical evidence of ischaemic heart disease particularly if there is documented left ventricular dysfunction (see Chapter 2). Fluid therapy is then adjusted to maintain wedge pressure and cardiac output at normal levels throughout the perioperative pèriod. These measures will prevent myocardial ischaemia in most patients, though occasionally additional nitroprusside or nitroglycerin may be needed if echocardiographic abnormalities or other signs of left heart strain appear during supracoeliac occlusion[31].

Most renovascular procedures can be accomplished without incurring more than 20 minutes warm ischaemia. If the kidney is likely to be ischaemic for a longer period or if there is impaired renal function prior to surgery, intermittent cold perfusion with heparinized Ringer's lactate at 4 °C may be utilized, with or without inosine[32,33]. This may be particularly relevant when combined aortic and renal revascularization is being undertaken.

ANTEGRADE AORTORENAL BYPASS

Although the conventional method of aortorenal bypass utilizes the infrarenal aorta as source, this may have a number of disadvantages in atherosclerotic patients. This part of the aorta may already be diseased or subject to disease progression, while the risk of graft closure may be further increased by the turbulence created by the retrograde graft trajectory. Antegrade bypass from the supracoeliac aorta avoids these problems and is the currently preferred method of managing atherosclerotic renovascular disease.

Access for supracoeliac bypass may be best achieved by an anterior xiphopubic approach. A thoraco-retroperitoneal incision may also be used[34], but the addition of a thoracotomy may increase operative morbidity.

The left renal artery is exposed by opening the peritoneum over the proximal infrarenal aorta and extending this incision to the left along the inferior border of the pancreas. The inferior mesenteric vein will usually need to be divided during this process (Figure **13.1a**). An alternative

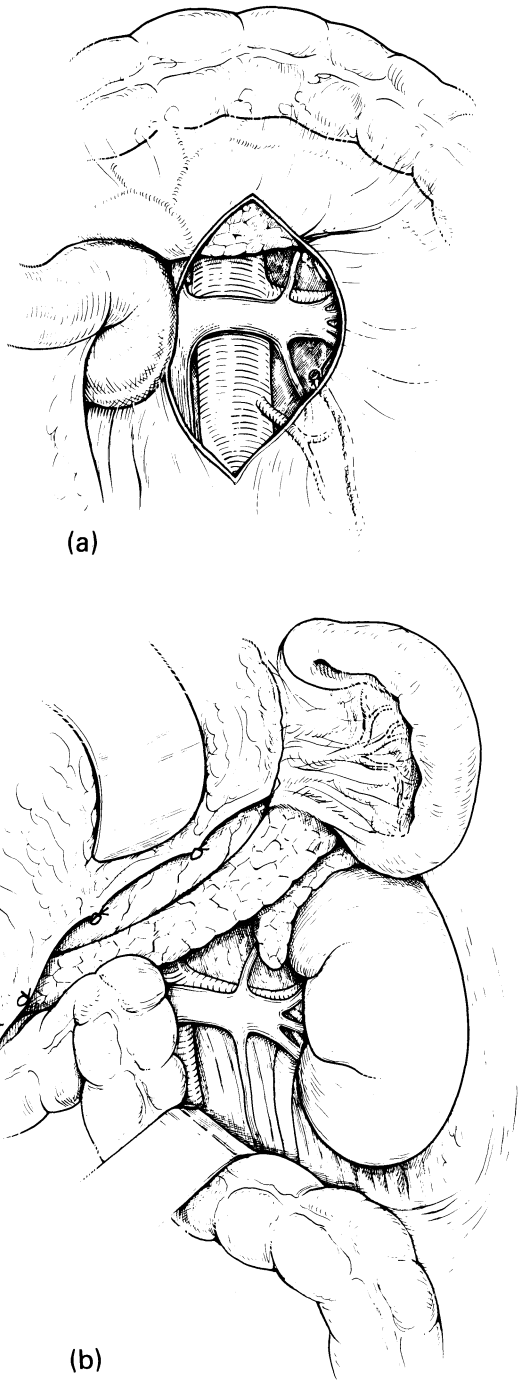
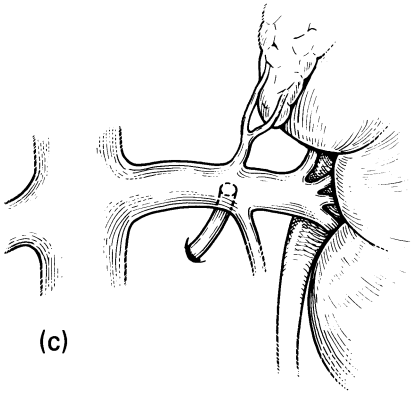
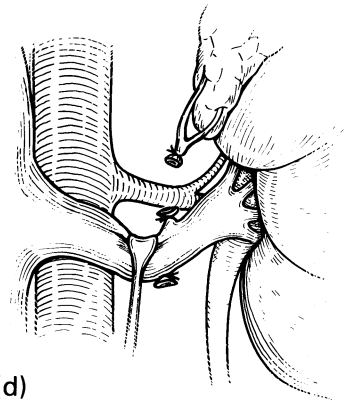


Figure 13.1(a–d) Exposure of the left renal artery: (a) the infrarenal aorta and left renal vessels are exposed from in front. (b) alternative retroperitoneal exposure



(c)



(d)

Figure 13.1 continued (c) anatomy of the left renal vein. A common pattern is shown with a lumbar branch joining the posterior aspect of the main vein. (d) the branches of the left renal vein have been divided and the vein retracted to expose the distal renal artery

method of exposing the left renal artery is to mobilize the left colon medially without disturbing the spleen (Figure **13.1b**).

The left renal vein overlies the artery and is fixed in position by its adrenal, gonadal and lumbar tributaries (Figure **13.1c**). These should be ligated and divided so that the vein can be retracted sufficiently to expose the artery (Figure **13.1d**). The distal portion of the latter and its primary branches are then controlled with Silastic loops.

The right renal artery is best exposed by displacing the hepatic flexure inferiorly and sweeping the duodenum and head of the pancreas medially off the front of the inferior vena cava (Figure **13.2a**). Again, the renal vein will require mobilization and retraction before the artery can be displayed (Figure **13.2b**).

The supracoeliac aorta is now approached via the gastrohepatic ligament. With the oesophagus held to the left and the left lobe of the liver to the right, the diaphragmatic hiatus is split and a 6–10 cm segment of supracoeliac aorta mobilized for clamp placement.

An appropriate graft is then selected. Autogenous vein has had limited durability in antegrade bypass to the visceral arteries[35] and prosthetics, either PTFE or knitted Dacron, are generally preferred for both visceral and renal artery revascularization in atherosclerotic patients. If Dacron is used, a segment may be cut from a standard bifurcation graft so as to produce a flange for aortic attachment (see Figure **12.3**). PTFE has the advantage that it is softer and sews in more easily to the distal renal artery, though its long-term behaviour in this position is unknown. If both kidneys are to be revascularized, a branched or bifurcation prosthesis may be utilized.

Systemic heparin is given and the supracoeliac aorta cross-clamped. An elliptical aortotomy is made on the anterolateral surface and the graft attached in end-to-side fashion (Figure **13.3a**). The aorta is then flushed via the open end of the graft and after placing a rubber-shod clamp across the graft origin, aortic flow is restored.

Bypass grafts to the left renal artery may be routed in front of or behind the pancreas (Figure **13.3b**). Although a retropancreatic route is said to predispose to graft compression this is more

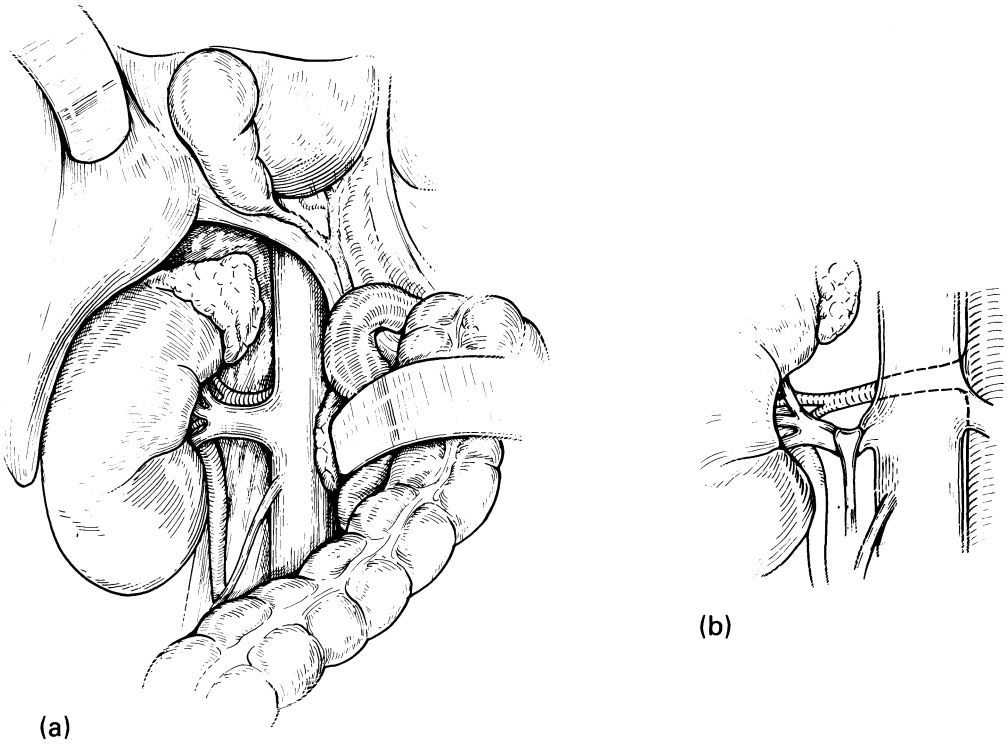


Figure 13.2(a, b) Exposure of the right renal artery: (a) medial reflection of the colon and duodenum. (b) right renal vein retracted to expose the distal portion of the artery

of a concern with vein grafts than prosthetics. If a prepancreatic route is utilized, the prosthesis should be wrapped in omentum to isolate it from the gastrointestinal tract. Bypass grafts to the right renal artery may be routed through the epiploic foramen (Figure **13.3c**).

Distal graft attachment may be by end-to-end or end-to-side anastomosis. The former is preferred as flow rates are better and there is less risk of kinking. After estimating the graft length under tension, the renal artery is transected beyond the macroscopic disease and the distal stump spatulated. The graft is tailored to match and anastomosed with a running prolene suture. Interrupted sutures may be used if the artery is small but are not necessary as a routine. The usual precautions of flushing and irrigation are observed before the suture line is completed and flow is restored to the kidney.

In some patients the intima of the renal artery is abnormal well beyond the main atherosclerotic lesion. End-to-end anastomosis in these cases may risk intimal splitting as the graft and artery are drawn together. This difficulty may be avoided by attaching the graft initially in end-to-side fashion. A ligature is then placed adjacent to the heel of the anastomosis converting the latter into an effective end-to-end configuration (see Figure **13.3b**).

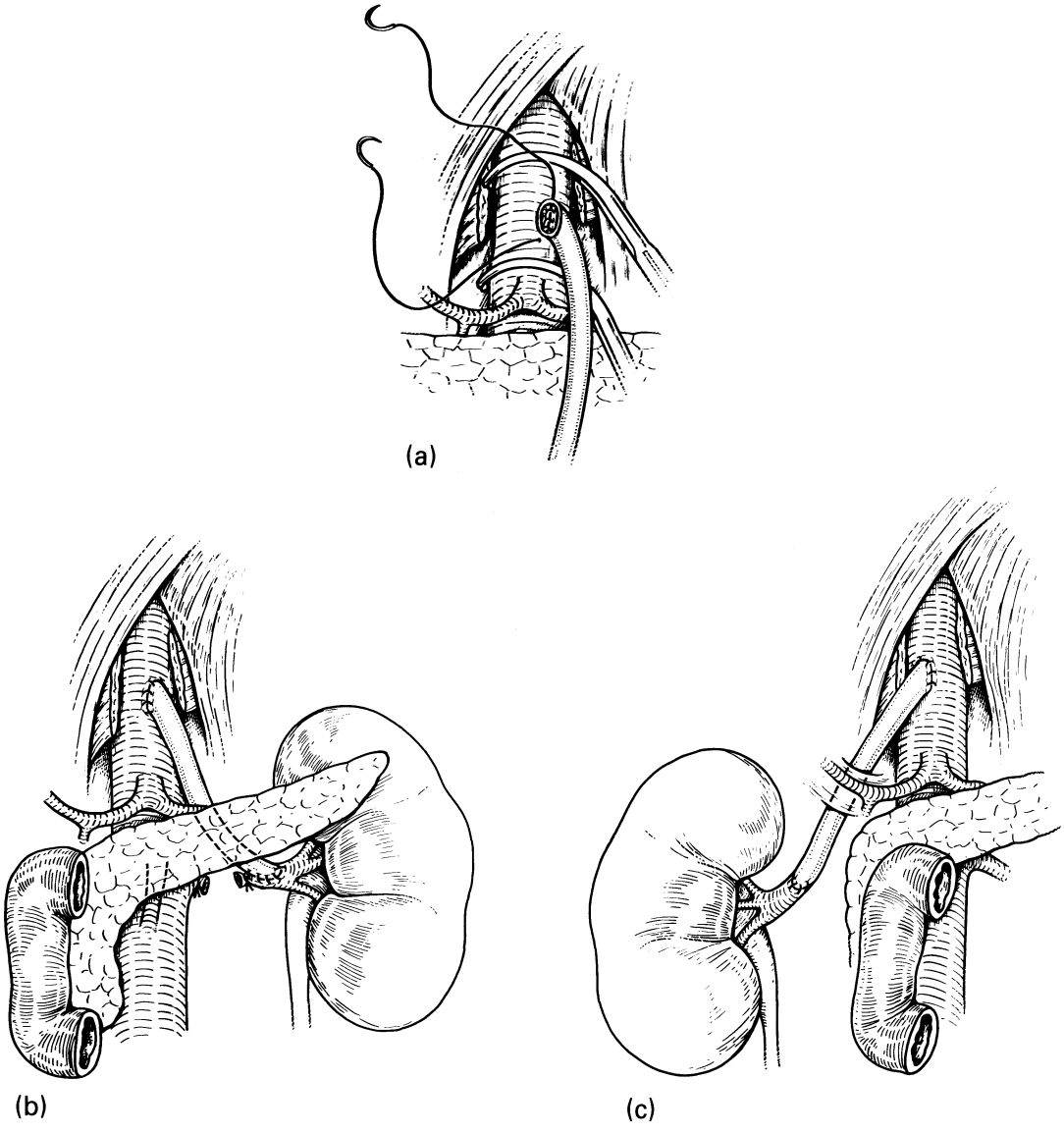


Figure 13.3(a–c) Antegrade aortorenal bypass: (a) PTFE graft attached to the anterolateral aspect of the supracoeliac aorta. (b) bypass to the left renal artery, in this case passing behind the pancreas. (c) bypass to the right renal artery via the epiploic foramen

RETROGRADE AORTORENAL BYPASS

In most patients with fibromuscular dysplasia the infrarenal aortic wall is reasonably well preserved and may be utilized for graft attachment. Access is easier than at supracoeliac level and infrarenal bypass may therefore be preferred, despite the theoretical haemodynamic disadvantages of the retrograde route.

Autogenous saphenous vein has been the most widely used bypass material in these younger patients. This material lends itself well to anastomosis with the distal renal artery or its branches, while there are particular advantages in an autogenous reconstruction when durability may be required for several decades. The long-term results of aortorenal vein grafts have generally been satisfactory, though stenosis and occlusion continue to occur in a small percentage of cases[36]. Dilatation is a specific risk of vein grafts in this position, particularly in children, though this is seldom sufficiently severe to require reoperation.

Autogenous internal iliac artery grafts are relatively free from late dilatation and stenosis and are regarded as the optimum material for renal revascularization in paediatric subjects[37]. Factors which may limit the use of internal iliac artery autografts on a wider scale include inadequate length and possible involvement by atherosclerosis or fibromuscular dysplasia.

Access for retrograde aortorenal bypass may be achieved via a subcostal incision extending across the opposite rectus muscle (Figure **13.4a**). On the right side the renal artery and infrarenal aorta may be exposed by reflecting the colon and duodenum medially as previously described. This exposure has the advantage that both ends of the bypass remain in the same operative field. The left renal artery may also be exposed by a similar retroperitoneal dissection or alternatively by the anterior transperitoneal route (Figure **13.1a, b**).

A segment of saphenous vein is then removed from the distal thigh. If possible, this should include a branch with which to enlarge the heel of the aortic anastomosis. A segment without valves may also be an advantage (see Chapter 5).

After systemic heparinization, the infrarenal aorta is cross-clamped and a longitudinal incision made on the anterolateral wall. Excision of an ellipse of aorta should be avoided in the case of a vein graft implantation, because this may lead to undue splaying of the vein. The graft is anastomosed end-to-side at this site with the tip directed inferiorly using a 5/0 prolene suture (Figure **13.4b**).

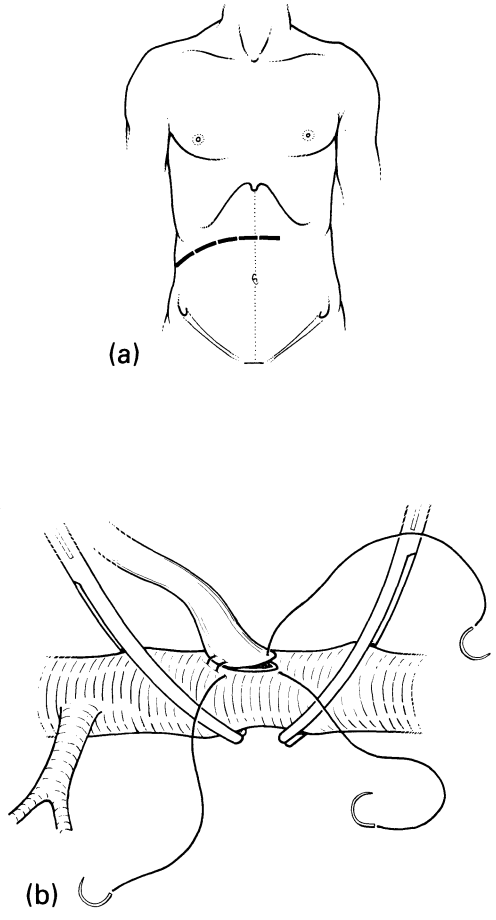
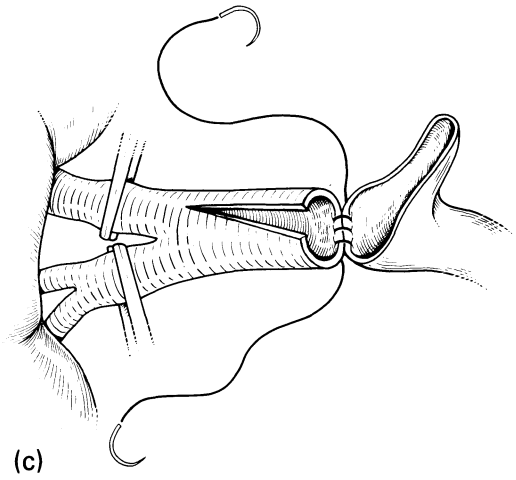
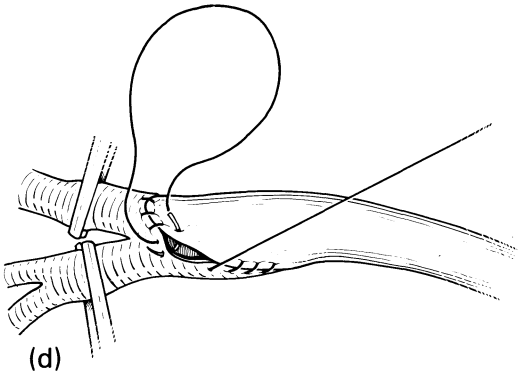


Figure 13.4(a–e) Retrograde aortorenal bypass: (a) the renal artery and aorta are exposed via a retroperitoneal subcostal incision. (b) a saphenous vein graft is attached end-to-side to the infrarenal aorta

The graft is then exposed to arterial tension and the length and lie estimated. On both sides the graft should ultimately adopt a gentle curve as it aligns itself to the distal renal artery. On the right, a course anterior to the inferior vena cava is preferred, since vein grafts in the retrocaval position may be liable to compression as a result of organizing perigraft haematoma. Once these decisions have been taken, the renal artery is transected beyond the macroscopic disease and a spatulated end-to-end anastomosis completed using 6/0 prolene (Figure **13.4c–e**). Interrupted sutures may be useful if the renal artery is small, and are routinely employed for distal autograft attachment in children in order to allow for subsequent growth.





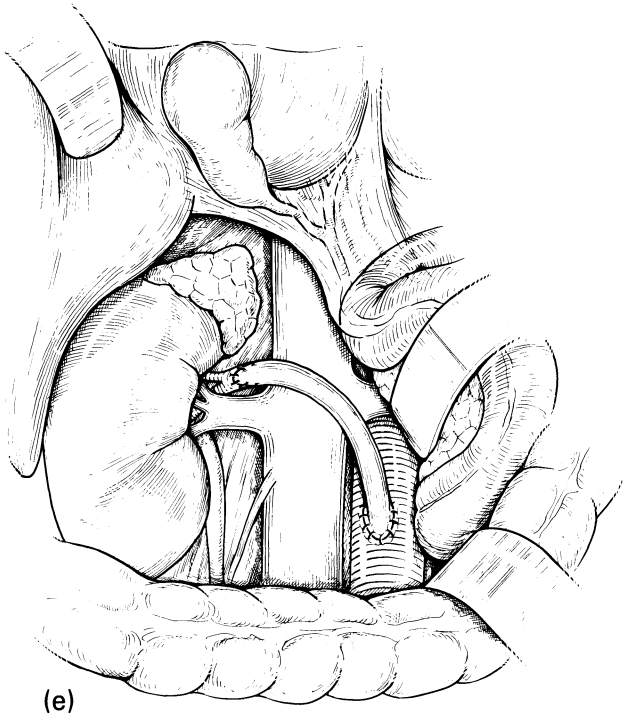


Figure 13.4 continued (c, d) distal spatulated anastomosis. (e) completed reconstruction

MANAGEMENT OF BRANCH ARTERY LESIONS

Isolated branch lesions can be managed by percutaneous transluminal angioplasty or by intraoperative dilatation at the time of aortorenal bypass (Figure **13.5**). However, patients with extensive branch involvement secondary to fibromuscular dysplasia are more satisfactorily managed by direct branch reconstruction.

In situ repair is usually possible providing there are disease-free branches outside the renal hilum[38]. These reconstructions are best undertaken with optical magnification and microsurgical instruments. The most versatile graft is autogenous saphenous vein and two or more segments may be sewn together to provide a branched arrangement (Figure **13.6a**). The graft is first attached to the anterolateral surface of the aorta. End-to-end anastomosis of the graft branches to each of the renal artery branches is then carried out using interrupted 7/0 sutures. These anastomoses should be performed sequentially, so that the kidney remains perfused throughout (Figure **13.6b, c**).

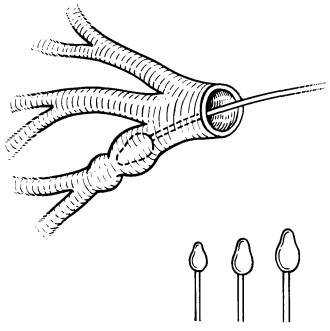


Figure 13.5 Method of managing branch artery stenoses by intraoperative graduated dilatation prior to distal attachment of an aortorenal bypass

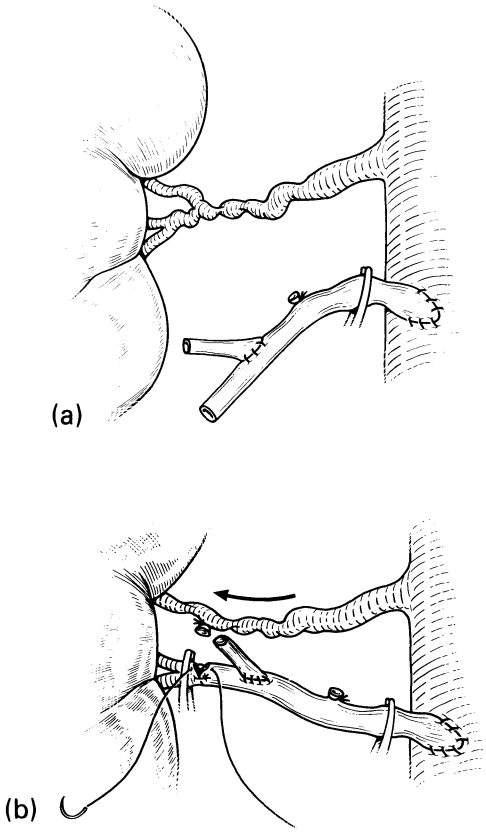


Figure 13.6(a–c) Repair of branch artery lesions using a branched saphenous vein graft: (a) the graft is attached to the infrarenal aorta. (b) end-to-end anastomosis of one limb of the graft to a branch artery is undertaken while the remainder of the kidney is perfused. (c) second branch anastomosis completed

Other methods of reconstruction may include side-to-side branch union to form a larger communal opening for graft attachment (Figure **13.7a–c**) or distal end-to-end graft–artery anastomosis with end-to-side branch reimplantation (Figure **13.8**). Single bypass to an isolated branch artery or branch resection with direct reanastomosis may also occasionally be utilized.

Ex vivo reconstruction is indicated for intrarenal branch involvement or following a failed *in situ* repair. The kidney may be completely removed or alternatively the ureter may be left intact and the kidney brought out onto the abdominal wall for repair. In the latter instance the ureter should be temporarily occluded to prevent retrograde blood flow into the kidney. Cooling may be achieved by simple flushing with chilled Collins' solution followed by immersion in ice slush saline[39].

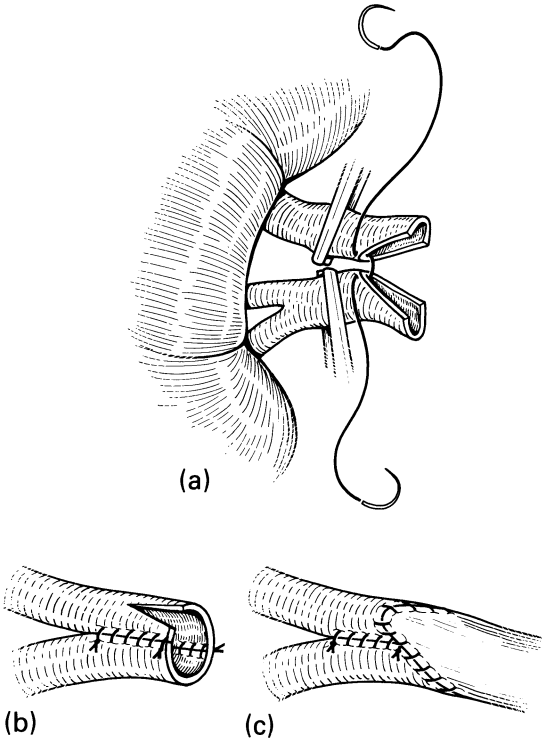


Figure 13.7(a–c) Side-to-side anastomosis of adjacent branch arteries creating a larger common opening for distal aortorenal graft attachment

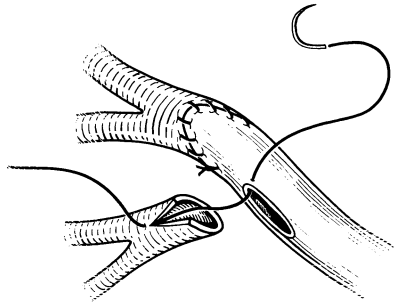


Figure 13.8 Branch artery reimplantation into the side of a vein graft

Internal iliac artery autograft is the preferred material for these reconstructions since it can be obtained with several of its branches. If the internal iliac artery is unsuitable, a multibranched saphenous vein graft may be substituted. In most cases optical loupes will provide sufficient magnification, though occasionally the operating microscope may be needed if very small branches are involved. Vascular repair may be performed with interrupted 7/0–9/0 sutures, using similar methods to those already described.

The repaired kidney should then be perfused via a pulsatile perfusion unit to check the integrity of the anastomoses. Addition of indigo carmine to the perfusate will confirm that distribution is even throughout the kidney. Thereafter the kidney is either replaced in the renal fossa, or else autotransplanted to the iliac fossa using the same techniques as for renal allotransplantation[40,41].

AUTOTRANSPLANTATION

Renal autotransplantation in association with ex-vivo repair has been used in a number of renovascular disorders including distal branch stenotic or aneurysmal disease, previously unsuccessful *in situ* repair and injuries to the renal vasculature[39]. More controversially, autotransplantation alone has been advocated as an alternative to aortorenal

bypass in children with ostial stenosis[42] and in adults with main artery atheromatous or fibrous disease[43]. Certainly autotransplantation provides good anastomotic conditions and is virtually failproof in the hands of those experienced in renal allotransplantation. However, it represents much more of an undertaking than aortorenal bypass or other forms of *in situ* repair, and inevitably leads to loss of the collateral supply to the kidney. As a result, more conventional methods of revascularization continue to be preferred for the great majority of patients with renovascular hypertension.

Remaining indications for autotransplantation may include certain cases of abdominal aortic coarctation[44], aortic aneurysms involving the main or accessory renal arteries[45], patients with a surgically difficult aorta but disease-free iliac vessels[46], and aortic dissection with renal artery compromise[47].

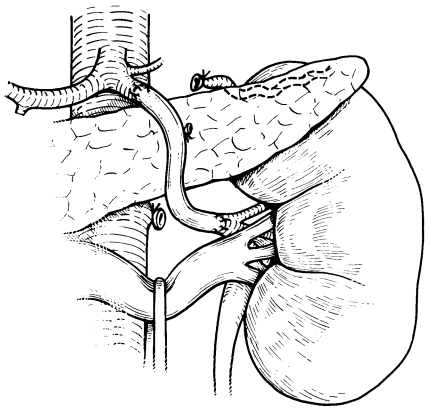


Figure 13.9 Splenorenal bypass. In this example a saphenous vein graft has been placed between the splenic artery and the left renal artery. This is a more satisfactory method than turning down the distal splenic artery for direct anastomosis.

OTHER TYPES OF BYPASS

In the presence of coexistent infrarenal aortic atherosclerosis, renal revascularization may be achieved either by supracoeliac aortorenal bypass as previously described or, if the aortic lesion is more severe, by combined aortic and renal artery replacement (see below). However, if there are significant operative risk factors or an aortic prosthesis is already in place, other methods of renal revascularization may be considered. These include various bypass procedures using the splenic, hepatic, superior mesenteric or iliac arteries as source[46,48] (Figures **13.9, 10**). The latter vessels are themselves liable to atherosclerotic involvement and careful arteriographic and intraoperative assessment is essential before proceeding. Subsequent disease progression in the donor vessels is an obvious concern with these methods and durability may be less than with in line aortic bypass.

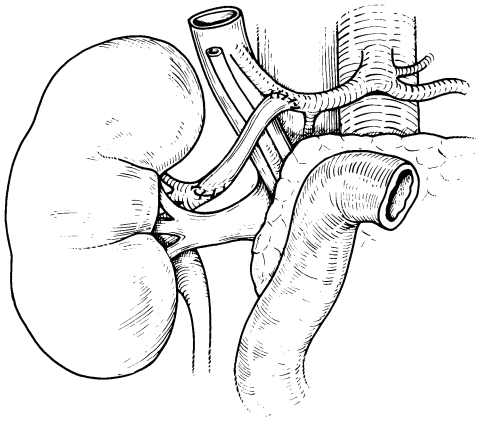


Figure 13.10 Hepatorenal bypass revascularizing the right kidney

TRANSAORTIC RENAL ENDARTERECTOMY

This is an alternative to bypass grafting which may be considered in patients with atherosclerotic renovascular disease. Its advantages are that it is an autogenous technique and that both renal arteries can be reopened under one short period of aortic occlusion. It may be particularly useful where there are multiple accessory arteries with ostial narrowing due to aortic atheroma (see Figure **14.15**). If necessary, transaortic endarterectomy may be combined with clearance of similar lesions at the visceral artery origins or with an aortoiliac reconstruction (see Chapters 3 and 12).

As with all transluminal eversion procedures, a major concern is the distal end point in the branch vessel and this method should not be attempted if the disease extends significantly beyond the immediate proximal segment of the renal arteries. In addition, endarterectomy should be avoided in the presence of aortic aneurysmal or pre-aneurysmal degeneration. In these cases an overdeep cleavage plane is likely to be developed within the aortic wall, leading to a thick loosely adherent distal edge in the branch vessel.

Access for transaortic renal endarterectomy may be achieved by a midline abdominal incision. A flank approach with medial reflection of the left kidney has also been advocated[49], but access to the right renal artery is then limited and exposure less than optimal if additional infrarenal aortic reconstruction is required.

The left renal vein is first mobilized by dividing its branches, and the left renal artery isolated as previously described. The suprarenal aorta is then

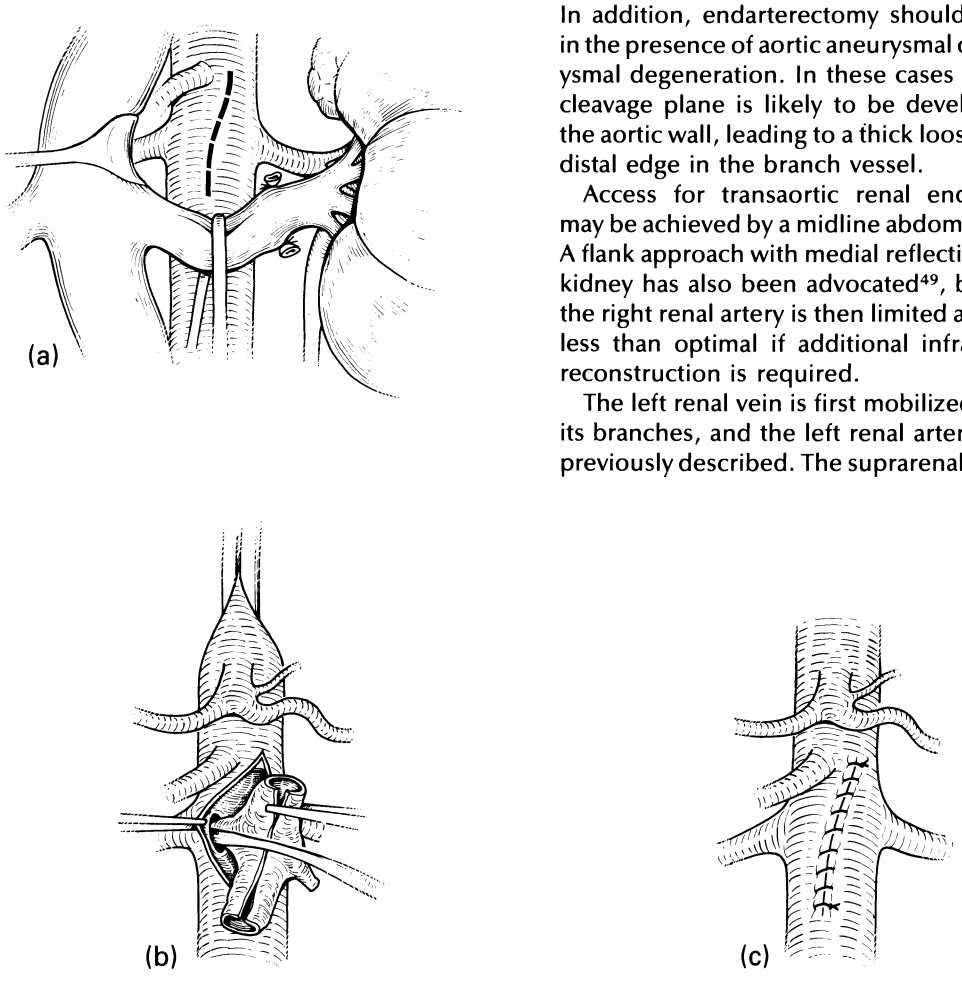


Figure 13.11(a–c) Transaortic renal endarterectomy: (a) anterior approach to the aorta and renal artery origins. (b) the aorta is opened longitudinally and a sleeve of intima removed in continuity with the renal orifice lesions. (c) aortotomy closed and renal perfusion restored

progressively cleared up to and including the superior mesenteric origin by cephalic retraction of the pancreas. Access to the proximal right renal artery may be gained by retracting the vena cava laterally following division of one or two pairs of lumbar veins as appropriate (Figure **13.11a**). In nearly all cases transaortic renal endarterectomy can be achieved without the need to divide the left renal vein. However, if the vein does require section, it should be re-anastomosed using two hemicircumferential sutures at the end of the procedure.

After heparinization, a soft jaw clamp is applied to each renal artery beyond the macroscopic disease and the supracoeliac and infrarenal segments of the aorta then similarly controlled. The aorta is then opened longitudinally at the level of the renal arteries and the incision extended to the left of the superior mesenteric artery origin (Figure **13.11a**).

The aortic intima is transected at the distal limit of the aortotomy at a point of minimal thickening, and a cleavage plane developed proximally in the aortic wall. At the proximal limit of the aortotomy the intima is similarly cut across just short of the superior mesenteric origin. The renal orifices are then cleared in turn. Gentle traction is applied to the atheromatous core and the cleavage plane extended into each renal artery, as the latter is everted into the aortic lumen. With use of the spatula in a progressively more superficial plane the lesion should separate as a thinly feathered edge, leaving the distal intima adherent to the underlying media.

Once clearance is complete, the aorta and renal arteries are flushed in turn and the lumen rinsed with heparin–saline. If the distal aortic intima is loose, tack-down stitches may be required, though this is not frequently necessary. The aortotomy is then closed with a running suture and flow released into the distal aorta and the renal arteries (Figure **13.11c**). The renal arteries should then be carefully palpated and assessed with ultrasound. In doubtful cases an intraoperative aortogram should be obtained. If this shows any suggestion of an intimal flap, a separate arteriotomy should be made in the affected renal artery and the loose intima extracted. Continuing concern regarding the distal intima may necessitate conversion to a bypass technique.

COMBINED AORTIC AND RENAL ARTERY SURGERY

Simultaneous aortic and renal artery reconstruction may be required when a main or accessory renal artery is involved in an aortic aneurysm, or arises from a diseased aortic segment which is to be replaced (see Figures **2.18, 19; 14.15, 16**). A variety of methods are available for achieving renal revascularization in these circumstances, including reimplantation, endosaccular anastomosis, bypass grafting, or endarterectomy (see Chapters 2 and 14).

Combined aortic and renal artery surgery may also be needed when a significant renal artery atherosclerotic lesion is associated with infrarenal aortic disease (Figure **13.12a**). In the majority of these patients renovascular hypertension or impaired renal function will be present, but aortic cases with severe asymptomatic renal artery lesions may also be considered for dual reconstruction. Failure to correct the latter at the time of aortic surgery may risk renal artery occlusion either in the perioperative period or subsequently as a result of disease progression. Although secondary renal artery reconstruction may still be possible at a later date, this may be more difficult and the patient's general condition less favourable than at original presentation.

Usually the aortic lesion will be either an obvious aneurysm or symptomatic occlusive disease. However, patients with renal artery stenosis and clinically mild or asymptomatic aortoiliac occlusive disease may also be considered for dual replacement if the aortographic appearances are severe (e.g. patulous segments or gross ulceration which carry a threat of embolization) providing there are no significant operative risk factors.

Combined aortic and renal artery reconstruction may carry a higher operative mortality than either procedure alone[50,51]. Particular risk factors include azotaemia, the need for bilateral renal or additional visceral artery reconstruction, aneurysmal rather than occlusive aortic disease, ECG evidence of myocardial ischaemia and diffuse peripheral vascular disease[52].

When several of these factors are present it may be advisable to modify the therapeutic approach. Thus the renal (and visceral) artery stenosis may be managed by initial percutaneous angioplasty

and the aortic lesion by graft replacement or extra-anatomic bypass. If angioplasty is not possible or is inappropriate, e.g. renal artery obstruction due primarily to aortic atheroma, a staged surgical approach may be considered involving an initial splenorenal or hepatorenal bypass followed by aortic replacement at a later date[48].

Operative technique

Combined aortoiliac and renal endarterectomy is a possible method of managing selected patients with aortorenal occlusive disease[51]. However, most patients requiring dual reconstruction are best managed by graft replacement.

A xiphopubic incision is made and the infrarenal aorta and renal arteries displayed as previously described. The juxtarenal aorta is often degenerate in these cases (Figure **13.12a**) and conventional infrarenal clamping may risk dislocating atheromatous sludge into the renal arteries. In such instances, proximal aortic control may be more safely achieved at supracoeliac level.

An appropriate sized aortic prosthesis is selected and an elliptical opening cut in the stem. A 6 mm PTFE graft or flanged Dacron side arm is then attached. A PTFE aortic prosthesis with a venous sidearm is an alternative combination which is particularly useful when the distal renal artery is small or fragile. A second extension limb may be attached on the opposite side of the prosthetic stem if both kidneys are to be revascularized.

Heparin is given and the aorta transected immediately below the renal arteries. The aortic prosthesis is then inlaid or attached by direct end-to-end anastomosis according to the state of the aortic wall (Figure **13.12b**). After completing the anastomosis, a rubber-shod clamp is placed across the prosthesis and the supracoeliac clamp is removed (Figure **13.12c**).

The renal sidearm is then exposed to arterial tension so that a decision can be taken on optimum graft length (Figure **13.12d**). On the right side, prosthetic grafts may be routed behind the vena cava to avoid the possible risk of subsequent duodenal erosion. A more direct route is utilized on the left. The distal renal artery is then transected and spatulated and after tailoring the graft, an end-to-end anastomosis is completed as previously described (Figure **13.12e**). A similar procedure may be carried out on the opposite side if both kidneys require revascularization. Finally, the distal limbs of the aortic graft are attached to the iliac or femoral arteries as appropriate.

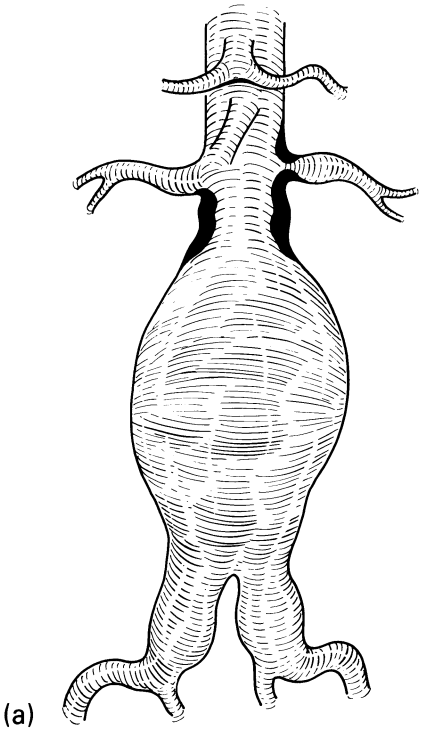


Figure 13.12(a–e) Combined aortic and renal artery bypass: (a) infrarenal aneurysm with left renal artery stenosis. The juxtarenal aorta is degenerate in this example, precluding clamp application at this site

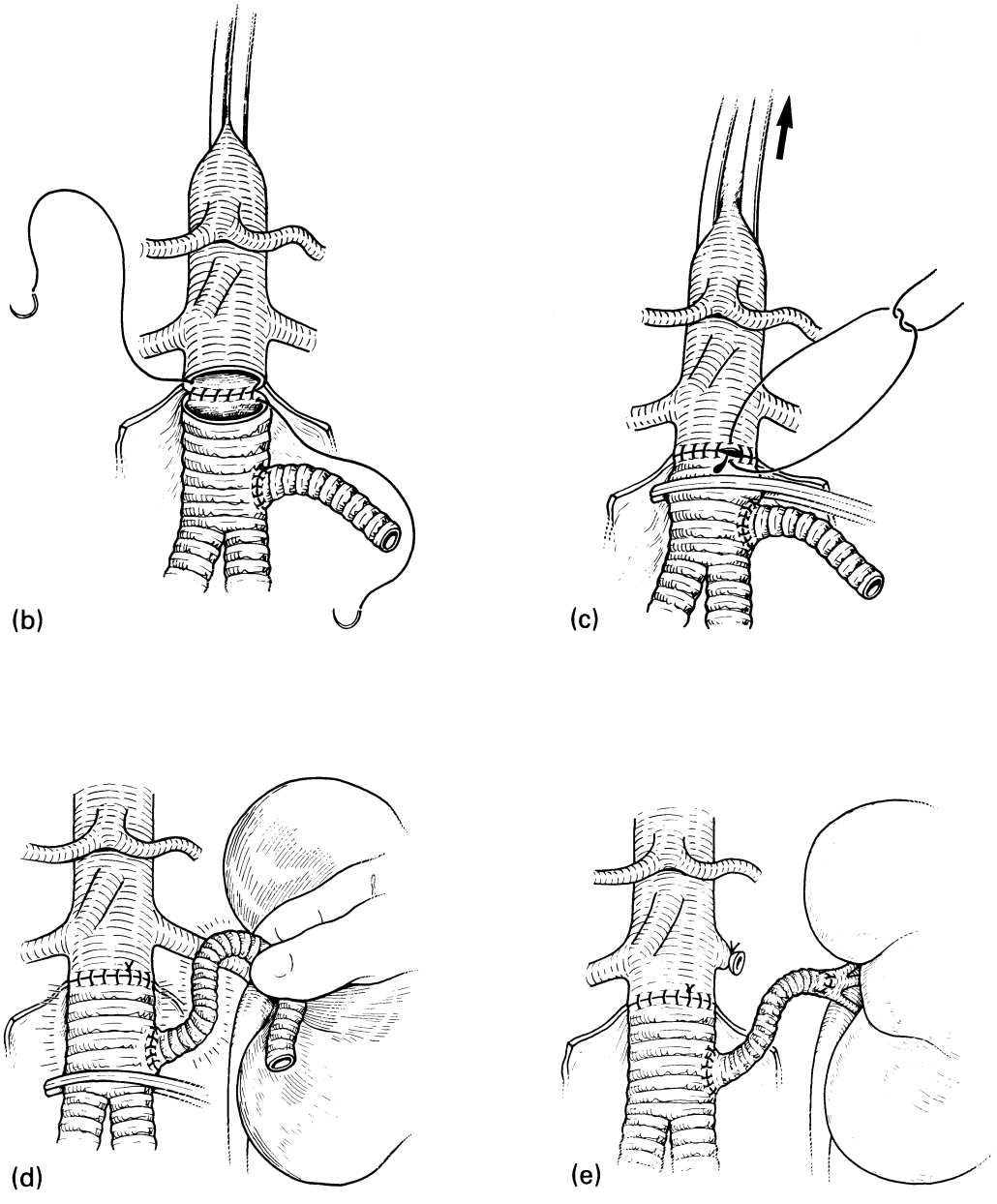


Figure 13.12 continued (b) the aortic graft with a renal sidelimb is inserted under supracoeliac control. (c) the aortic anastomosis is completed and the supracoeliac clamp removed. (d) renal graft length is estimated by exposure to arterial tension. (e) completed bypass to the renal artery

COARCTATION OF THE ABDOMINAL AORTA

Abdominal aortic coarctation is rare, accounting for less than 2% of all coarctations of the aorta[53]. Some lesions represent a congenital hypoplasia of the aorta and its visceral branches[54]. Others are due to Takayasu's arteritis or rarely to other disorders such as neurofibromatosis or fibromuscular dysplasia[55]. The aortic involvement may take the form of suprarenal, inter-renal or infrarenal narrowing. Less commonly there may be a diffuse involvement of the entire abdominal aorta. Distal thoracic or arch lesions may coexist, particularly in patients with Takayasu's disease. The most frequent site of involvement is the inter-renal aorta and additional narrowing of the renal artery ostia is usually then present. Multiple accessory renal arteries are frequently found in this type of coarctation and may complicate revascularization.

Most patients with coarctation of the abdominal aorta present with severe hypertension in childhood or early adult life. The hypertension is usually secondary to renal artery stenosis though suprarenal coarctations may reduce renal perfusion directly, with or without additional renal artery stenosis. Disabling lower limb ischaemia is uncommon, though peripheral pulses are nearly always reduced. Similarly, although the coeliac and superior mesenteric artery origins are often also narrowed[56], intestinal angina is rare due to effective collateralization chiefly from the inferior mesenteric artery[53].

Hypertension is often difficult to control by medical means and some form of intervention is required at an early stage to prevent congestive cardiac failure or cerebral haemorrhage. In some patients with suprarenal coarctation, hypertension may be improved by transluminal dilatation of the narrowed aortic segment. However, the main factor responsible for hypertension in most coarctation patients is renal artery ostial stenosis, and this may not be amenable to dilatation[30].

One surgical solution in young subjects is to patch the narrowed aortic segment with autogenous material. The renal arteries may then be reimplanted into the patch or revascularized by an autogenous aortorenal bypass (Figure **13.13a, b**). This approach offers maximum flexibility

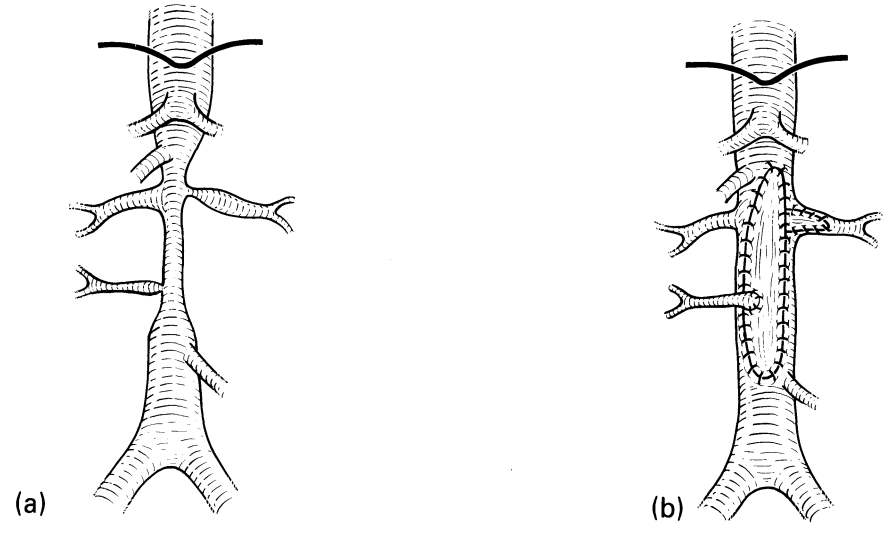


Figure 13.13(a, b) (a) Inter-renal coarctation in childhood. The left main and right lower pole accessory renal arteries are stenosed. (b) Reconstruction by autogenous aortoplasty with extension into left renal artery. The accessory right renal artery has been reimplanted into the patch

should further surgery be required later in life, and avoids the difficulty of gauging the length and size of an aorto–aortic bypass graft in a growing child.

In older subjects the definitive treatment is a thoracoabdominal aortic bypass. If the descending thoracic aorta is also involved, the ascending aorta may be used as source. The renal arteries may then be revascularized by reimplantation or by means of a bypass graft originating from either the native aorta or the aorto–aortic bypass (Figure **13.14a, b**).

Additional visceral artery reconstruction is necessary if there are overt intestinal ischaemic symptoms or if critical collateral routes have been jeopardized during aortic bypass insertion or renal revascularization. Prophylactic gut revascularization may also be considered in asymptomatic patients with severe multivessel involvement (see Chapter 12). Reconstruction methods include reimplantation of the inferior mesenteric artery and coeliac/superior mesenteric bypass from the aortic prosthesis or native aorta.

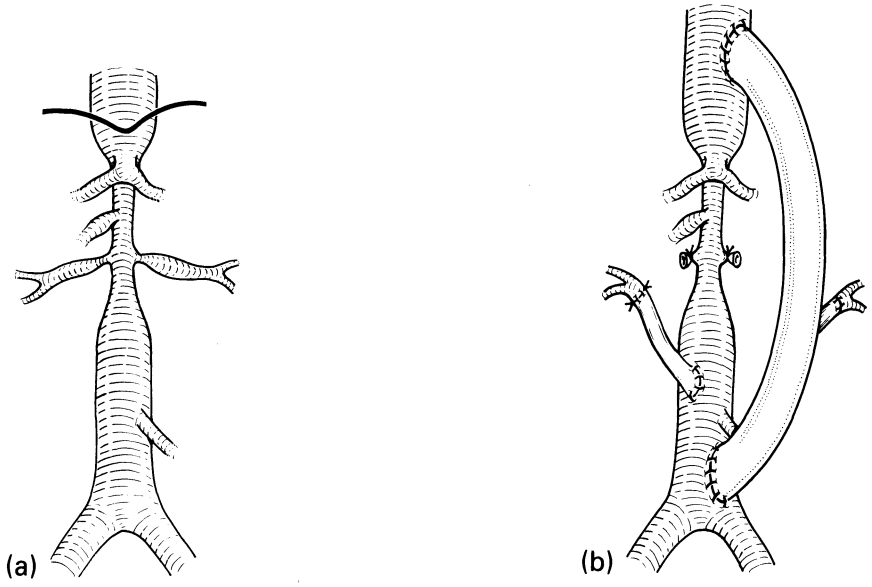


Figure 13.14(a, b) Inter-renal coarctation with bilateral renal artery stenosis. In this case a thoracoabdominal PTFE bypass has been inserted. The right kidney is revascularized by means of a saphenous vein graft from the distal aorta. The left aortorenal graft originates from the aortic bypass

RENAL ARTERY ANEURYSMS

Renal artery aneurysms are uncommon, but are being recognized with greater frequency nowadays as a result of the increased use of arteriography in evaluating hypertension. The main categories are aneurysmal dilatations associated with fibromuscular dysplasia, aneurysmal dissections, microaneurysms which result from arteritis associated with collagen and other disorders, aneurysmal arteriovenous malformations, and macroaneurysms of undetermined origin[57,58].

Macroaneurysms are the most important type of aneurysm and two groups may be recognized: saccular and fusiform. The great majority of aneurysms are saccular and located chiefly at the primary or secondary branch bifurcations. Fusiform aneurysms are usually secondary to a proximal stenosis and are equally distributed throughout the renal artery. The pathology underlying renal artery macroaneurysms appears to be a congenital or acquired degeneration of the media. Atherosclerosis with calcium deposition may be present in some cases but these changes are usually regarded as a secondary event[57].

Although renal artery aneurysms may be associated with loin pain or haematuria, most are asymptomatic and are discovered during arteriographic evaluation of hypertension. Approximately 20% of renal artery aneurysms are calcified and are discovered by the chance finding of signet ring calcification on a plain abdominal film. Others present with calyceal distortion on a pyelogram carried out for other purposes.

The main clinical importance of renal artery aneurysms lies in their relationship to hypertension and their potential for rupture. The association with hypertension is more than coincidental and may result from compression of adjacent arteries, thrombus propagation within the aneurysm or distal embolization. In other cases the hypertension may be due to coexistent branch artery stenotic disease, though this may not necessarily be apparent on preoperative arteriography[59].

Renal artery aneurysms may rupture into the retroperitoneum, peritoneal cavity, renal pelvis or into an adjacent vein creating a large arteriovenous fistula. The risk of rupture is difficult to assess but in the unscreened population is probably very low indeed[60]. An increased incidence of rupture is said to be associated with aneurysms over 2 cm in diameter and those which are non- or part-calcified[61]. As with splenic artery aneurysms, rupture may occur in late pregnancy or postpartum and may then be associated with particularly dire consequences[62].

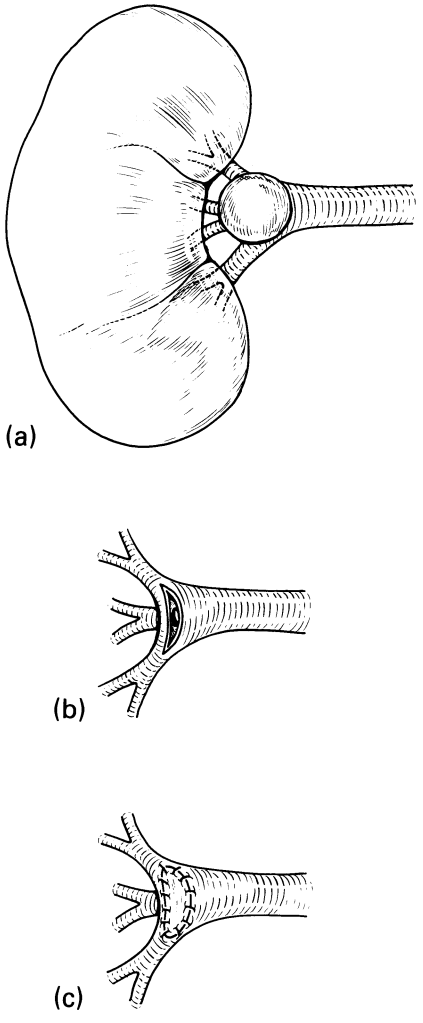


Figure 13.15(a–c) (a) Saccular aneurysm at the renal artery bifurcation. (b) Tangential excision of aneurysm. (c) Repair using an autogenous vein patch

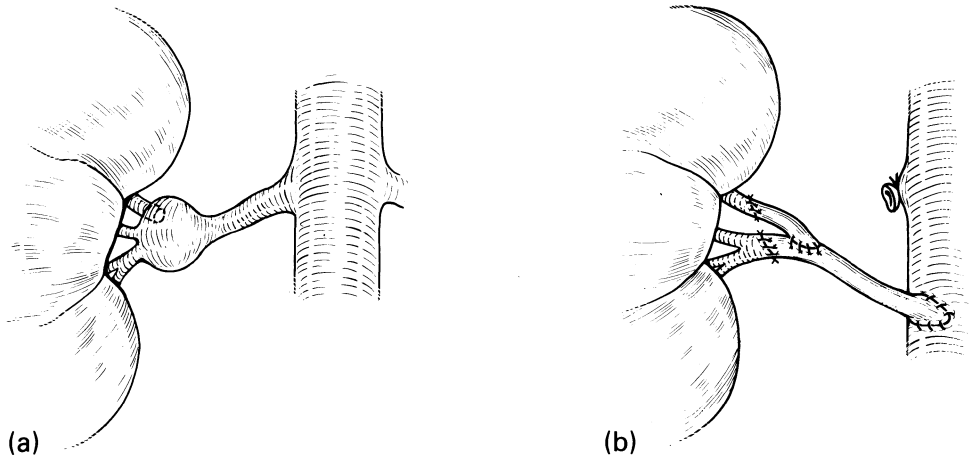


Figure 13.16(a, b) (a) Renal artery aneurysm involving the primary branches. (b) *In situ* resection of the aneurysm and replacement with a branched autograft

Indications for operation include the presence of symptoms, large or expanding aneurysms, documented renovascular hypertension, and evidence of intrasaccular thrombus on arteriography, with or without prior distal embolization. In addition, operation should be advised as a routine in women of child-bearing age because of the catastrophic results of rupture in pregnancy. Those patients not subjected to surgery should be carefully followed up with serial arteriography.

The type of arterial reconstruction utilized depends on the site of the aneurysm, the number and size of the branch vessels involved and the presence or absence of occlusive disease. Most cases can be managed by *in situ* repair with or without regional hypothermia.

Proximal fusiform or saccular aneurysms involving the main artery can be managed by excision and aortorenal bypass on standard lines. Saccular aneurysms at the primary bifurcation may sometimes be amenable to tangential excision, providing that the rest of the artery wall is disease-free. The resulting defect should then be closed with a vein patch (Figure **13.15b, c**). However in most cases the branch arteries cannot be preserved and reconstruction using a branched autogenous graft is necessary (Figure **13.16a, b**). Options for distal graft attachment are as previously described.

More complex lesions involving multiple branches, or aneurysmal disease at secondary or tertiary branch level, may be optimally managed by *ex vivo* repair.

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14

Complications of Aortic Surgery

Surgery for aortoiliac occlusive or aneurysmal disease is nowadays highly successful. Nevertheless, complications may still occur which may threaten the patient's limbs or life. As with arterial procedures at other sites, early complications (within 30 days of surgery) are principally due to technical or judgemental errors. Late complications may arise from degenerative changes within the reconstruction or in the adjacent host vessels. A number of complications may also occur at sites outside the immediate reconstruction zone. These include general complications, such as myocardial infarction or respiratory insufficiency, as well as complications more directly related to the aortic procedure, e.g. visceral ischaemia, renal and ureteric problems, sexual dysfunction and spinal cord ischaemia.

EARLY COMPLICATIONS IN THE RECONSTRUCTION ZONE

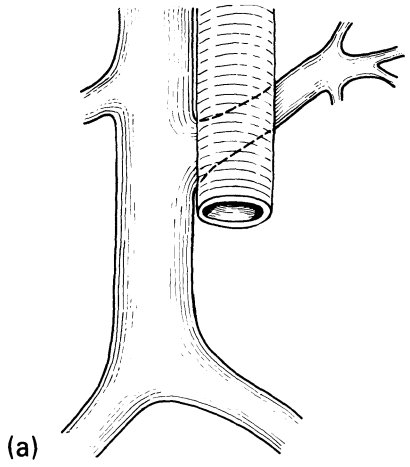
HAEMORRHAGE

Venous haemorrhage is the most troublesome type of intraoperative blood loss. One of the commonest sources is injury to the iliac veins or vena cava during attempts to mobilize the distal aorta or iliac arteries. This risk is greatest in patients with large adherent aneurysms, in re-do cases, in inflammatory aortitis and in patients with a periadventitial reaction to a recent occlusion.

In order to avoid venous injury certain adjustments may need to be made in surgical technique. Thus, if there is marked adherence between the arterial and venous axes in patients with aortoiliac occlusive disease, a bypass technique may be a safer option than endarterectomy. Similarly in re-do cases, endoluminal balloon catheters may be used for vascular control with inlay graft attachment, or alternatively the diseased zone may be excluded and revascularization achieved by bypass at a distance.

In aneurysm surgery, encirclement of the common or internal iliac arteries can and should be largely avoided. Thus, if a straight tube graft is being inserted, it is sufficient to clear only the front and sides of the common iliac arteries and then to clamp these from in front. Where a bifurcated graft is required, end-to-side graft attachment to the external iliac artery with distal common iliac ligation or oversewing of the common iliac origin will avoid the need to mobilize the common iliac arteries (see Figure **2.7b, c**). In the case of large common iliac aneurysms involving the iliac bifurcation, the graft can be attached to the external iliac artery and the internal iliac origin sewn off from within the aneurysmal sac. A similar endosaccular approach may be used for dealing with additional internal iliac aneurysmal disease.

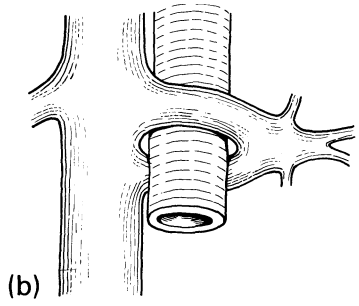
Venous haemorrhage may also occur from injury to the lumbar veins during attempts to obtain proximal aortic control. Again, a minimum dissection technique restricting mobilization to the front and sides of the aorta will avoid this problem.



Damage to the left renal vein and left renal vein–vena caval junction is a particular risk in ruptured aneurysms due to overhasty attempts at proximal aortic control. This hazard may be avoided by digital dissection within the periaortic haematoma keeping well to the left of the midline and directly on the surface of the aneurysm.

Venous anomalies increase the likelihood of intraoperative venous haemorrhage. The most common anomaly is a retroaortic left renal vein (Figure **14.1a**). Failure to identify the left renal vein in its normal position should immediately initiate a search behind the aorta to avoid damaging a posteriorly located vessel during aortic clamp placement. Other venous anomalies include a left-sided vena cava and duplication of the cava (Figure **14.1c**). The former may not interfere with the aortic reconstruction unduly but the latter may require transection of the left-sided component to allow access to the proximal aorta[1].

If venous haemorrhage occurs despite the above precautions, it should be controlled by digital compression rather than by blind attempts at clamp application. Where the injury is in the form of a linear tear adjacent to the arterial axis,



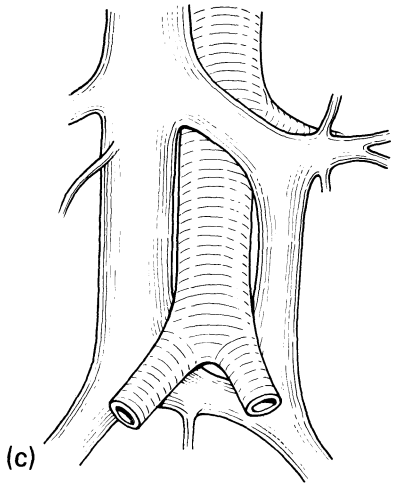


Figure 14.1(a–c) Venous anomalies which may complicate aortic surgery: (a) retroaortic left renal vein. (b) circumaortic renal venous collar. (c) duplicated inferior vena cava

the simplest method of repair is to sew the edge of the venous defect to the adventitia of the artery. The arterial reconstruction is then appropriately modified, e.g. bypass at a distance instead of endarterectomy. Occasionally, if the artery and vein have been partially separated at the time of injury, it may be possible to directly repair the laceration over a partial occlusion clamp. Tears at the iliac vein–vena caval confluence which extend behind the arterial axis are more difficult to manage. Often the quickest solution is to transect the overlying artery (both common iliac arteries if necessary). Reflection or rotation of the arterial axis will then give unimpeded access for venous repair.

Intraoperative arterial haemorrhage is usually less of a problem. It may occur from the lumbar or inferior mesenteric arteries, but most commonly it is the result of leakage through the interstices of the graft or from one of the suture lines. Graft leakage may be avoided by thorough preclotting of all knitted Dacron grafts or alternatively by using a woven or impregnated Dacron prosthesis or PTFE. Suture line bleeding may be avoided by a careful suturing technique with the finest possible needles and by test-irrigating for deficiencies before declamping. If leakage still occurs despite these precautions, extra stitches will be required. These should be inserted under clamp control since attempts to suture a pulsating vessel will merely enlarge the hole. However, if the proximal and distal graft attachments have been completed, reclamping may carry at least a theoretical risk of thrombus deposition within the graft. An alternative is to secure the suture line with pledget-backed sutures inserted into the unclamped vessel (Figure **14.2**).

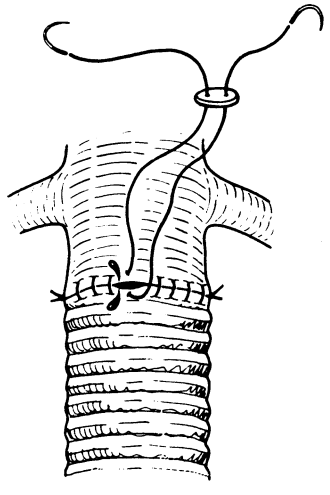


Figure 14.2 A defect in the aortic suture line is closed with a Teflon or Dacron pledget

Postoperative bleeding may occur from an unligated artery or vein or from one of the suture lines. More commonly a coagulation disorder is at fault e.g. hypofibrinogenaemia, thrombocytopenia, disseminated intravascular coagulation or heparin overdose. Signs of postoperative bleeding include an unstable systemic arterial blood pressure, decreased urinary output and a low central venous or left atrial pressure. After transfusion these parameters improve, only to fall again. Where clinical signs are less certain, an emergency aortogram may be helpful in showing suture line leakage or displacement of arterialized structures by haematoma[2].

Immediate reoperation is necessary in all cases of suspected postoperative bleeding once any coagulation defect has been corrected with fresh frozen plasma, platelet concentrate etc. An overlooked vessel in the mesentery, retroperitoneum or abdominal wall may then be found and ligated. Rarely the cause is a splenic injury following retractor trauma or separation of adhesions. Anastomotic bleeding may be due to the suture cutting out from the aortic wall or less commonly to rupture of the suture material itself. The latter may result from instrumental trauma, perforation by a supplementary stitch or the sawing effect of calcific plaque. Treatment requires partial dismantling of the anastomosis and insertion of a fresh suture. Smaller defects due to intimal cut-out may be managed by pledget-backed sutures as previously described. In either case an external Dacron wrap may be used to reinforce the suture line.

THROMBOSIS

Acute thrombosis after endarterectomy or bypass in the aortoilofemoral system may be due to judgemental or technical errors (cf. femoro–distal bypass, Chapter 5).

Judgemental errors include underestimating stenotic or embologenic disease proximal to the reconstruction zone, commencing or terminating a bypass or endarterectomy at a site where the artery wall is too pathological, and mis-assessing the quality of the distal run-off. These errors may be minimised by adequate preoperative and intraoperative assessment and by appropriate adjustments in surgical technique, e.g. extending the bypass or endarterectomy to a site where the artery is less diseased, or carrying out an additional profunda reconstruction or femoro–distal bypass to secure an adequate run-off (Figure **14.3**).

Technical errors which may lead to bypass graft occlusion include intimal splitting at a suture line or distal clamp site, a narrowed anastomosis, unevacuated thrombus within the graft, and twisting or compression of the graft particularly in relation to the sigmoid mesentery or inguinal ligament. Technical errors in an aortoiliac or iliofemoral endarterectomy may involve incomplete removal of the endarterectomy specimen, an overdeep cleavage plane, a false passage, a distal intimal flap, an overthick proximal edge and stenosis of an arteriotomy closure.

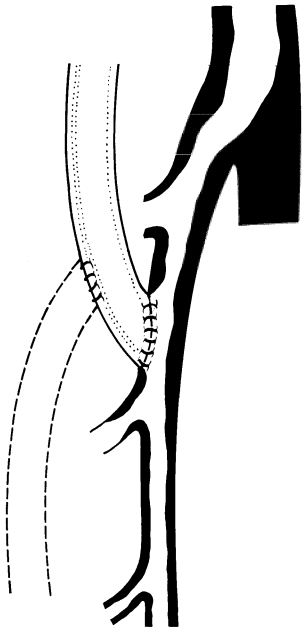


Figure 14.3 Early aortofemoral graft thrombosis due to deficient profunda run-off. Correction requires thrombectomy and insertion of a femoro–distal extension

Most of these technical errors may be apparent at their inception, while routine intraoperative arteriography should ensure that subclinical lesions such as anastomotic narrowing or a small intimal dissection are detected before the patient leaves the operating room. If, despite these precautions, a graft limb or endarterectomy occludes in the immediate postoperative period, the patient should be returned to the operating room and the lower end of the reconstruction reopened. A retrograde catheter thrombectomy is then carried out, compressing the opposite groin to avoid contralateral embolization. Once patency has been restored any obvious underlying error at the distal anastomosis or endarterectomy site is then corrected. This may variously involve patch insertion, anastomotic relocation or extension of the distal arteriotomy to allow extraction of loose intima and fixation of the distal intimal edge. A completion arteriogram is then obtained as a routine to confirm the adequacy of the reconstruction and the patency of the distal run-off.

Occasionally inspection of the distal anastomosis or the distal endarterectomy end point fails to reveal an obvious error. Providing a good down-bleed has been restored, the operative procedure may be confined to retrograde thrombectomy on the assumption that the occlusion was due to propagation of unevacuated thrombus within the reconstruction zone or in the distal run-off vessels. However, a retrograde intraoperative arteriogram should be additionally obtained to exclude a proximal technical error. This is particularly important in endarterectomy cases, and if a proximal lesion is identified the safest option is to replace the endarterectomized segment with a bypass graft. Failure to restore a good down-bleed in a prosthetic reconstruction is an indication for graft exchange, if necessary using a crossover route.

Although postoperative occlusion of one limb of an aortoiliac bypass graft or one side of an aortoiliac endarterectomy may be similarly managed by reopening the distal limit of the reconstruction zone, a simpler solution is a femorofemoral crossover graft assuming that the opposite side is normal. This can be inserted through undissected tissues, if necessary under local anaesthesia, and avoids the need for iliac exposure.

ARTERIAL OCCLUSION DISTAL TO THE RECONSTRUCTION

Acute postoperative limb ischaemia may be due to causes other than failure of the aortic reconstruction. If a strong femoral pulse is palpable, infrainguinal thrombosis or embolization should be suspected.

Thrombosis may be superimposed on a previously stenotic or aneurysmal infra-inguinal vessel or there may be thrombotic extension distal to a chronic superficial femoral occlusion. Both events are uncommon nowadays as a result of intraoperative systemic heparinization. However, it is essential that heparinization is adequate throughout the clamp period and that hypercoagulation problems are recognized and corrected[3].

A more common cause of acute limb ischaemia after aortic surgery is embolization[4]. Measures which should be taken to reduce this problem include early control of the iliac vessels before the aorta is approached, careful selection of clamp sites, once only aortic clamp application, external flushing of all clamps and thorough graft aspiration and irrigation before the distal suture lines are completed. Although some authors have advocated routine distal passage of a Fogarty catheter prior to completing the distal anastomosis, this is largely unnecessary and is indeed potentially dangerous.

Pre-exploratory arteriography is helpful in patients who develop limb ischaemia following aortic surgery and who have a retained femoral pulse. This may be obtained by a single film hand injection technique in the operating room. If an embolus is demonstrated, this may be removed by standard embolectomy from the groin, popliteal fossa or ankle, as appropriate (see Chapter 6). Acute thrombosis in the superficial femoral artery may be managed by femoro–distal bypass, providing there is a patent run-off. Consecutive thrombosis below a chronic superficial femoral occlusion may sometimes be amenable to distal thrombectomy and bypass grafting. However, in many of these cases the below-knee vessels are also atherosclerotic and it may be difficult to re-establish distal patency by surgical means. Local low-dose streptokinase may then be considered, but if ischaemia is severe, amputation may become inevitable.

In all patients who develop acute arterial occlusion following aortic surgery, muscle ischaemia may be severe and revascularization may need to be accompanied by wide fasciotomy. Additional measures may be necessary to counteract the systemic effects of such ischaemia (see Chapters 6 and 7).

LATE COMPLICATIONS IN THE RECONSTRUCTION ZONE

The principal late *in situ* complications of aortoiliac reconstruction are occlusion and false aneurysm formation. Arterial infection may be an early or late complication and is considered in a separate section.

LATE GRAFT OCCLUSION

The most common causes of late aortoiliac or aortofemoral graft failure are atherosclerotic progression just beyond the distal limit of the reconstruction or in the inflow tract, failure to bypass significant disease at the initial operation, anastomotic intimal hyperplasia, and graft-related problems such as kinking, or fragmentation of mural thrombus. In some cases late reconstruction failure may be precipitated by a hypotensive event. Usually there is an underlying anatomical abnormality, but occasionally severe hypotension alone may be responsible.

Symptomatic patients with late graft occlusion should be reassessed by arteriography, if necessary using the translumbar or transaxillary route. Views should be obtained of the profunda, the

contralateral iliofemoral axis and where possible the below-knee vessels. An attempt may then be made to lyse the thrombus column using a local low-dose streptokinase infusion. Although this approach on its own is not a long-term solution[5] it may be usefully combined with angioplasty or local surgical revision if an underlying stenosis is identified. Restoration of patency is most likely with recently occluded grafts though occasionally quite late cases may respond. Distal embolization is a particular hazard of lytic therapy when applied to prosthetic grafts[6,7] but most cases will respond to continued infusion (see Chapter 15).

Clot lysis takes time, and where the limb is in jeopardy an immediate surgical approach should be made. Surgery is also indicated when thrombolysis is unsuccessful or results in only partial improvement, or when the rest of the graft is defective and requires replacement. The surgical options available are retrograde thrombectomy[8], replacement of part[9] or all of the graft[10], and extra-anatomic bypass[11]. In nearly all cases these measures must be supplemented by revision of the outflow tract. As the superficial femoral artery is usually occluded, this centres on a reconstruction of the profunda. Where the latter is poorly developed or extensively diseased, a supplementary femoro–distal bypass will be necessary in order to secure an adequate run-off (see Chapter 3).

Retrograde transfemoral thrombectomy is perhaps the simplest surgical option for managing aortofemoral graft limb occlusion, providing aortic inflow is good and the rest of the graft is free from gross abnormalities. This approach is quite suitable for occluded PTFE grafts which have a thin pseudointima and are easy to thrombectomize (Figure **14.4a, b**). However, in the case of Dacron prostheses, retrograde thrombectomy may lead to scuffing of the thick pseudointima and there may be some risk of early rethrombosis. This is particularly likely if it is necessary to use a ring stripper to free adherent clot[12]. After achieving thrombectomy, an arteriogram of the proximal graft should be obtained to confirm the completeness of clot removal. A small catheter inserted from the distal end is a convenient method of opacifying the graft. If appearances are satisfac-

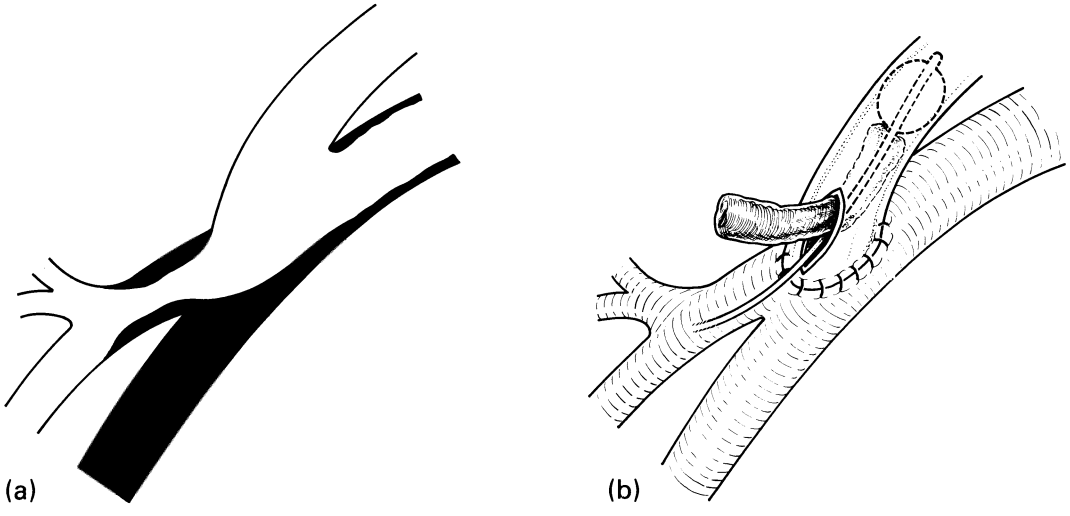


Figure 14.4(a–d) Late aortofemoral PTFE graft failure due to profunda stenosis (the superficial femoral artery is occluded): (a) pathology. (b) retrograde catheter thrombectomy

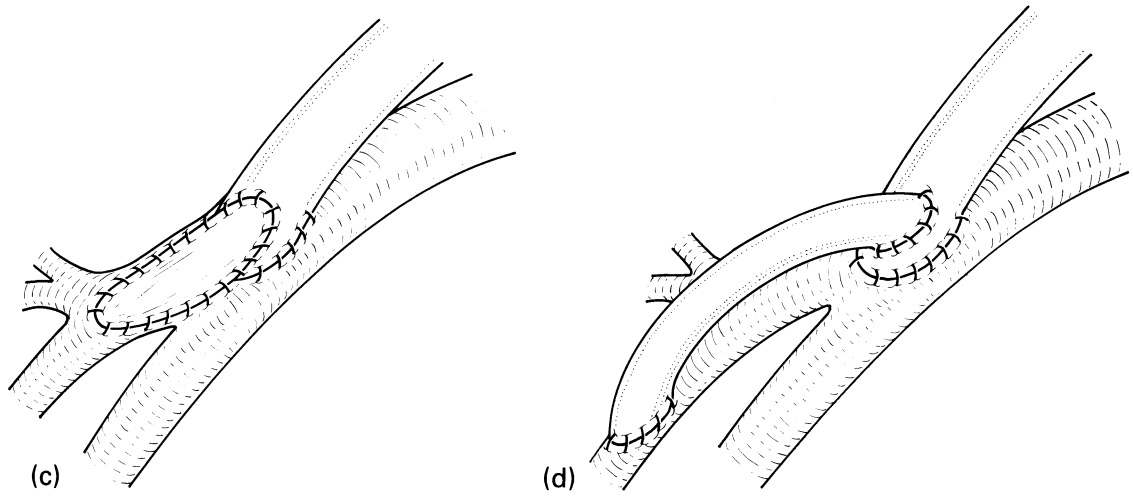


Figure 14.4 continued (c) patch angioplasty of the profunda. (d) PTFE graft bridging the stenotic zone

tory, attention should be turned to revising the graft outflow. This may involve insertion of a patch or a bridging graft from the thrombectomized limb to the profunda distal to the stenotic zone (Figure **14.4c, d**). Although most occluded aortofemoral grafts can be reopened by retrograde thrombectomy, long-term patency may be less than that achieved by graft replacement[10,13,14] or extra-anatomic bypass[11] and in consequence the latter methods are usually preferred.

Crossover grafting is a particularly good method of revascularization providing the contralateral graft limb is sound and aortic inflow is unimpeded. A suprapubic femorofemoral route or a route through the space of Retzius may be used in cases of unilateral aortoiliac graft occlusion. In the case of aortofemoral graft thrombosis, a donor site on the contralateral graft limb proximal to the inguinal ligament may be preferred as this will avoid the need to reopen the groin (Figure **14.5a**). A retroperitoneal exposure is employed and the graft is passed through the space of Retzius to the recipient groin. Here it may be attached end-to-side to the profunda beyond the occlusive zone or end-to-end to the spatulated profunda stump, with or without endarterectomy (Figure **14.5b, c**).

In a significant number of patients the entire reconstruction must be replaced because of additional proximal aortic disease, stenosis or false aneurysmal change at other anastomoses, or because of defective graft geometry. The latter includes a perpendicular implantation, kinking of the graft limbs (Figure **14.6a, b**), mismatch in graft–artery size and an overlarge end-to-side distal anastomosis with stasis. Anatomic regrafting may be possible (technique see false aneurysm section). However, where access to the infrarenal aorta is particularly difficult, the new graft may be attached to the ascending, descending or supracoeliac aorta[15,16]. Graft replacement is, of course, a major undertaking and poor risk patients may be alternatively managed by axillofemoral bypass, if ischaemia is sufficiently disabling to warrant further surgery, and if the distal run-off is adequate.

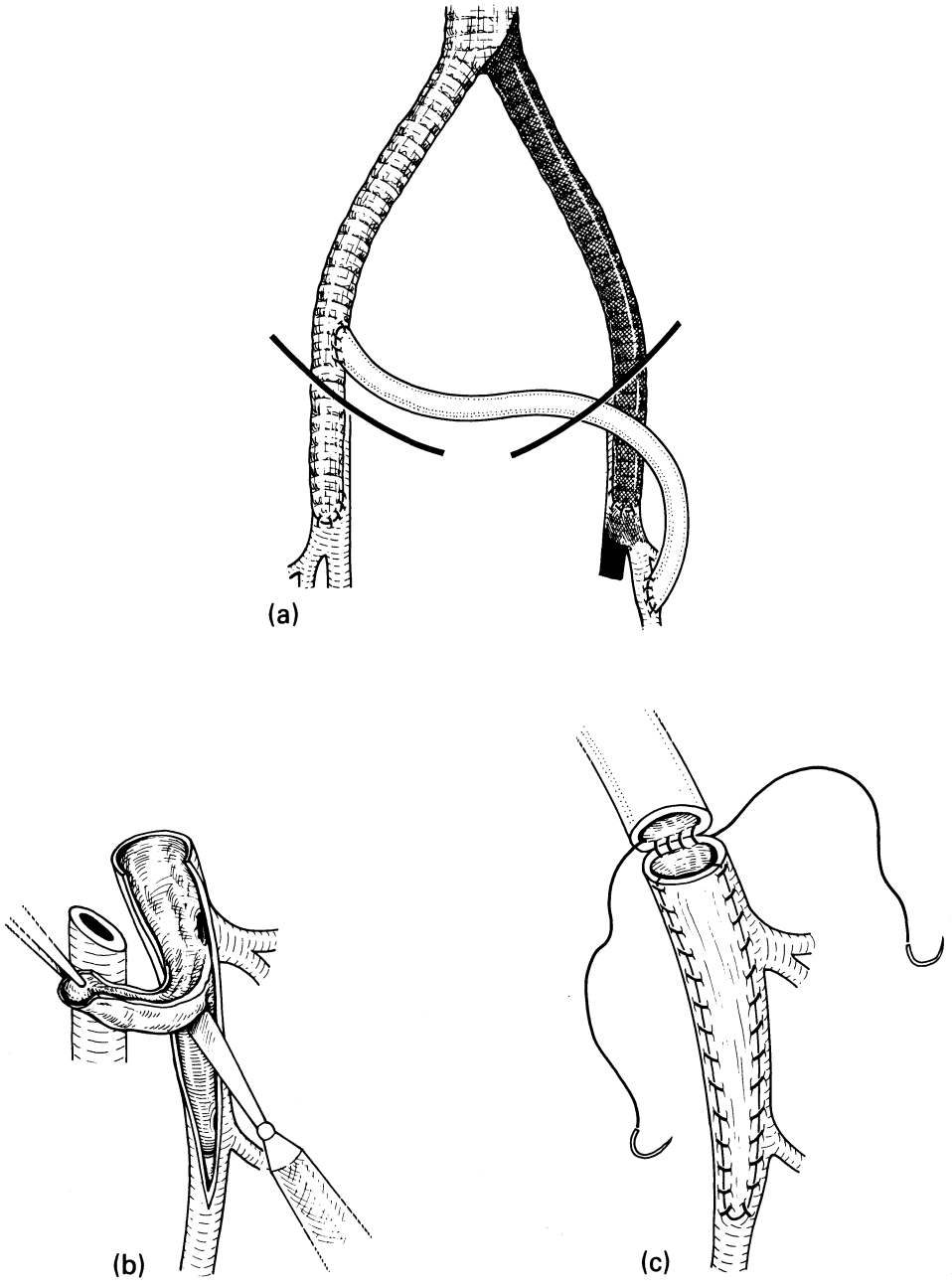


Figure 14.5(a–c) (a) Late occlusion of the left limb of an aortofemoral graft treated by crossover ilioprofunda (PTFE) bypass. (b, c) method of developing profunda outflow by endarterectomy and vein patch prior to graft attachment

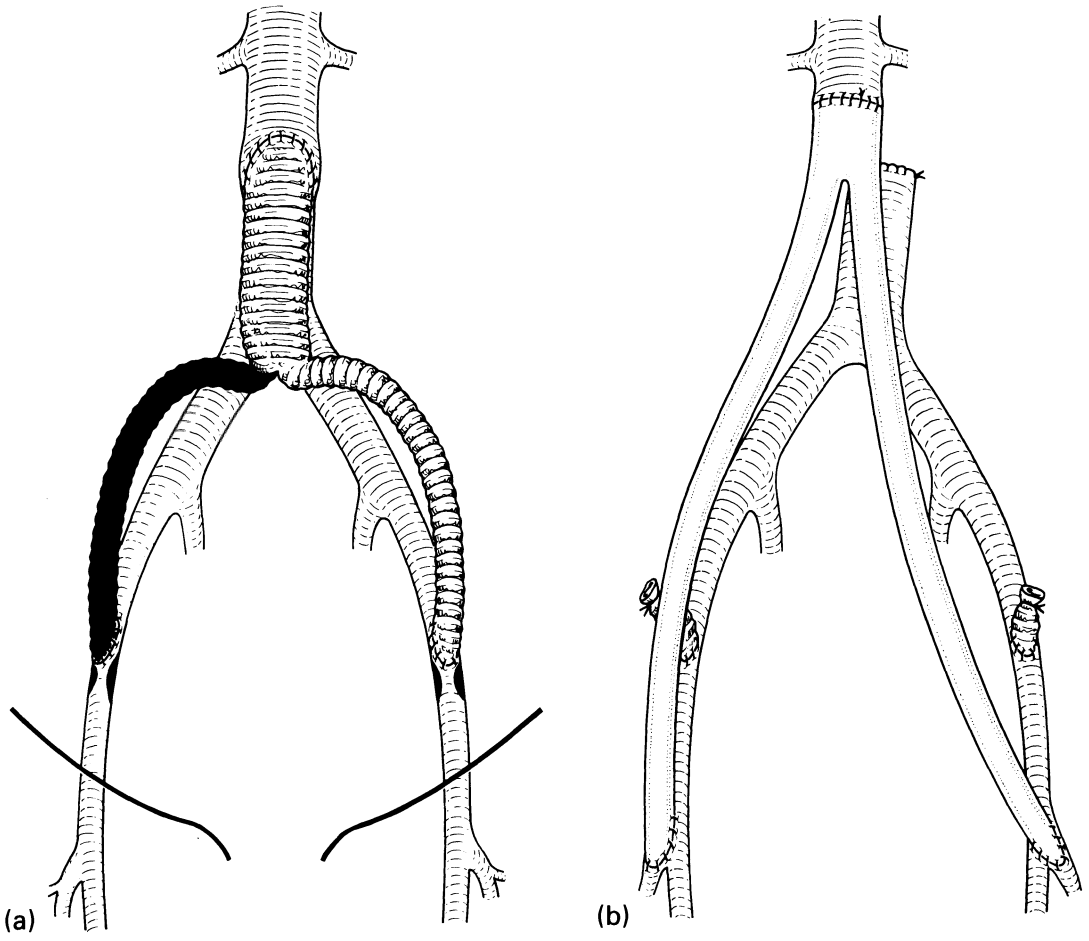


Figure 14.6(a, b) Late aortoiliac graft failure requiring graft replacement: (a) overlong prosthetic stem with kinking of the iliac limbs. Note additional intimal hyperplasia at the distal anastomoses. (b) a new aortofemoral prosthesis (PTFE) has been inserted

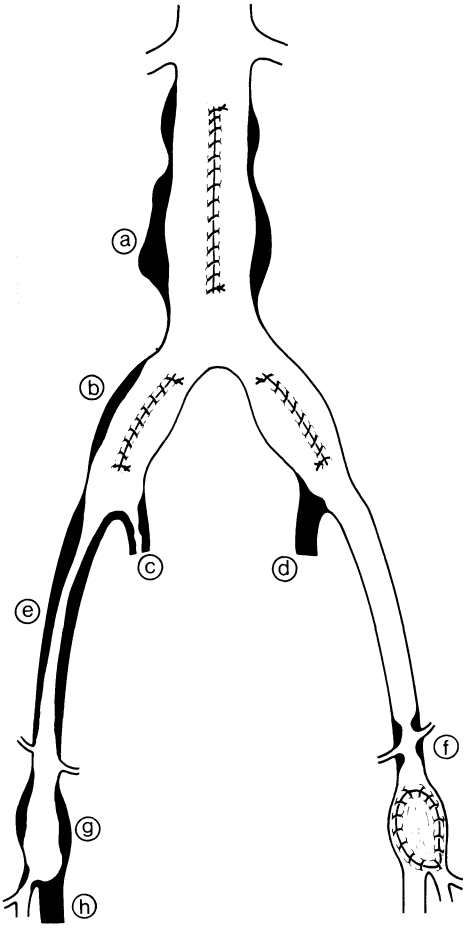


Figure 14.7 Late degenerative changes after aortoiliofemoral endarterectomy. *Key*: (a) Segmental dilatation of the aorta with mural thrombus; (b) fusiform dilatation of the common iliac arteries with mural thrombus; (c) atheromatous internal iliac stenosis; (d) internal iliac occlusion; (e) diffuse stenosis of the external iliac artery; (f) intimal hyperplasia at the iliofemoral junction (re-anastomosis site); (g) dilatation of the common femoral artery after patch angioplasty, again with mural thrombus; (h) atherosclerotic progression in the distal run-off: occlusion of the superficial femoral artery and profunda stenosis

Although the majority of patients with late aortoiliac or aortofemoral graft failure can be successfully managed by re-do surgery, a small number continue to lose their limbs from this event[17]. In addition, long-term patency after reoperation is not as good as that achieved by the primary procedure, irrespective of the mode of treatment[8,14]. In contrast, intervention prior to occlusion will prolong the functional life of the reconstruction and moreover correction at this stage may be relatively easy to achieve, either by percutaneous dilatation or local operative revision. As at femoropopliteal level, regular graft surveillance using vascular laboratory testing or digital subtraction angiography is hence advisable so that the failing reconstruction can be detected prior to occlusion.

LATE ENDARTERECTOMY FAILURE

As in the case of bypass graft occlusion, late failure after aortoiliac or iliofemoral endarterectomy is commonly due to atherosclerotic progression in the inflow or outflow tracts, particularly the latter. In addition, degenerative changes leading to occlusion may occur in the endarterectomy zone itself (Figure **14.7**). These include intimal hyperplasia, particularly in the external iliac segment or at suture lines, recurrent atheroma and late dilatation. Aneurysmal changes may occur in the aorta–common iliac segment after 10–15 years, or at an earlier date in the common femoral arteries if patch graft angioplasty has been utilized. These dilated zones may harbour mural thrombus, which may embolize the run-off bed or progress to complete occlusion.

Late occlusion after aortoiliac or iliofemoral endarterectomy may be a relatively innocuous event since the internal iliac or lumbar arteries may remain patent and reperfuse the profunda system. However, in some patients recurrent ischaemic symptoms may be sufficiently severe to merit further surgery. The procedure of choice in most cases is an aorta–biprofunda bypass. A bilateral reconstruction is usually necessary since even with an initial unilateral endarterectomy, atherosclerotic involvement of both iliac axes is the rule by the time occlusion has occurred.

FALSE ANEURYSMS

Although false aneurysms may complicate any arterial suture line, the most common site is in the groin after prosthetic bypass. Three basic mechanisms may be involved in their production: failure of the suture material, detachment of the suture from the prosthesis, or cutting out of the intact suture from the wall of the artery. Now that silk is no longer used for graft anastomoses, suture failure is not an important cause. Although the use of monofilament sutures is open to criticism on theoretical grounds[18], in practice prolene has proved much safer than its earlier counterparts[19] and is widely used for vascular work. Detachment of the suture from the prosthetic edge should also be rarely implicated, providing the graft has been cut properly and the sutures placed at a sufficient depth.

In most cases false aneurysms are due to the suture cutting out from the artery wall. Multiple factors have been implicated in this process, including progressive degeneration of the host vessel, inadequate depth of suture placement, excessive endarterectomy, wound complications such as infection, perigraft haematoma or lymph accumulation, and reoperation[20–22]. In fact mechanical factors are of prime importance in most cases: a graft which has been sutured under too much tension, turbulence in an end-to-side anastomosis, graft dilatation, compliance mismatch and flexion–extension movements of the hip which 'work' the suture line by pulling on the graft.

The usual time interval between operation and appearance of a false aneurysm is several years, apart from cases due to technically inadequate suture placement or infection which may present within the first few postoperative weeks. Femoral false aneurysms may be recognized by the appearance of a pulsatile mass in the groin. Less commonly embolization, graft limb occlusion or rupture may occur. Aortic false aneurysms may produce abdominal pain and a mass, but most are impalpable and are discovered either during investigation of a concomitant femoral false aneurysm, or because of a complication such as embolization, graft limb compression or rupture. The latter may take place into the gut, retroperitoneum or vena cava. Iliac false aneurysms are prone to similar complications. In addition, they are particularly likely to produce hydronephrosis, and the presence of unilateral or bilateral ureteric obstruction in a patient with previous aortic surgery should always suggest this possibility.

Although some authors have advocated a conservative approach to small femoral aneurysms, in our view surgical intervention should be considered in virtually all anastomotic aneurysms because of the seriousness of their complications.

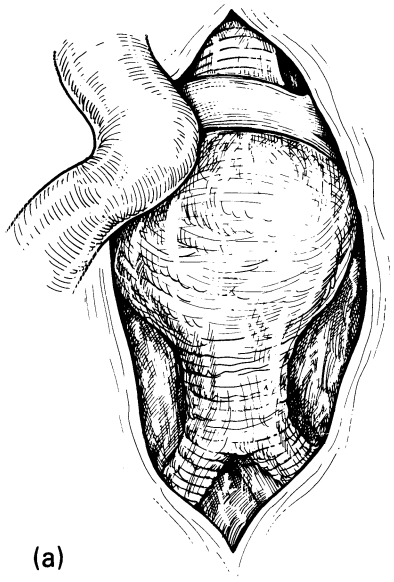
Preoperative assessment should include arteriography and a CT or ultrasound scan. Arteriography may not fully define the extent of a false sac, because of intraluminal thrombus, but is helpful in showing additional abnormalities in the prosthesis and in the inflow or outflow tracts. The most accurate method of assessment is a CT scan and this will detect lesions not apparent on either arteriography or ultrasound scan as well as providing information on the state of the adjacent aorta[23].

It is important to exclude an infective aetiology, particularly when a false aneurysm appears within the first few months of surgery. Suggestive features include fever, leucocytosis, and CT evidence of fluid or oedema around the graft. Operative appearances supplemented by Gram staining should confirm this supposition (see below).

Management of aortic false aneurysm

Aortic false aneurysms may be associated with similar lesions at either or both of the distal anastomoses, so that in most cases the entire graft must be replaced.

The abdomen is reopened by the customary xiphopubic incision. A sternal retractor may be placed at the upper end of the wound in case access to the supracoeliac aorta is required. The posterior peritoneum is opened to expose the graft area and false sac (Figure **14.8a**). Often the duodenum is firmly adherent to the aneurysm and should be left undisturbed. If the graft has been attached end-to-side to the aorta, proximal control can usually be obtained immediately below the renal arteries and this segment should be cleared for clamp placement. The stem of the prosthesis is then similarly mobilized by opening the perigraft sheath.



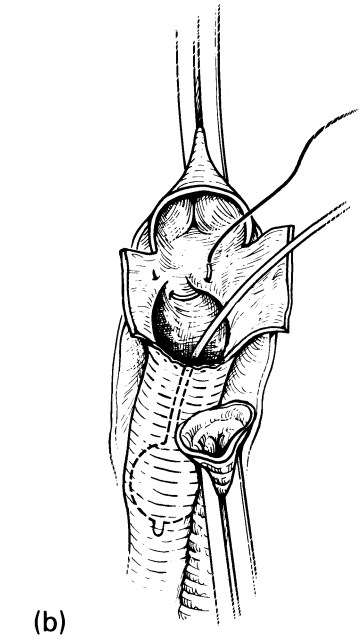


Figure 14.8(a, b) (a) A false aneurysm complicating a previous end-to-side aortic bypass. (b) The proximal infrarenal aorta and the prosthetic stem have been clamped and the graft detached. Back-bleeding from the distal aorta is controlled by balloon catheter while the aorta is oversewn

After heparinization, clamps are applied and the false aneurysm is entered. Back-bleeding from the distal aorta may be controlled by manual compression or by balloon catheter while the prosthesis is detached and the distal aorta oversewn (Figure **14.8b**). The proximal aorta is then sectioned and a new graft attached in end-to-end fashion. This anastomosis should be reinforced with an external Dacron cuff to prevent recurrence. If dissection is difficult the posterior wall of the aorta may be left intact and the graft inlaid (see Chapter 2).

Where the false aneurysm complicates a previous end-to-end aortic anastomosis, proximal control below the renal arteries may be more difficult to achieve. In these cases the aorta should be either clamped at supracoeliac level, or controlled by an endoluminal balloon passed into the proximal aorta via a puncture site in the body of the prosthesis. After obtaining distal control, the aneurysm is opened and the graft replaced as above.

If there is insufficient usable aorta below the renal arteries for graft attachment, the aorta may be closed sagittally, taking care to avoid the renal artery origins. The new graft is then attached end-to-side at supracoeliac level (Figure **14.9**) Alternatively, the aorta may be transected above the renal arteries and the new prosthesis inserted here with reattachment of the renal arteries to the sides of the prosthetic stem. Prior renal cooling, with or without inosine, is advisable if this type of manoeuvre is envisaged, as the ischaemic time for the second kidney may exceed 20 minutes (see Chapter 13).

Once the aortic anastomosis has been completed, the distal ends of the old graft are detached from the iliac or femoral arteries and the prosthesis completely removed. The limbs of the new graft are then passed distally to the groins, either via a new lateral route created by digital dissection from above and below, or via the old graft tunnels if the new prosthesis is of smaller size. Occasionally the previous distal anastomosis sites are reusable, in which case a cuff of graft is preserved for attachment to the new limb. However, in most cases there is either a coexistent false aneurysm or local atherosclerotic degeneration. In these circumstances the old anastomosis site should be closed and the new limb reattached more distally (see below).

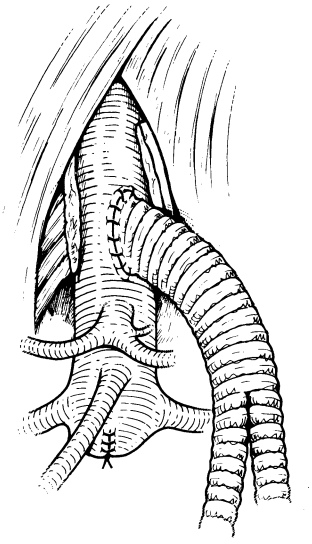


Figure 14.9 Technique for inserting a new aortofemoral bypass if the infrarenal aorta is unsuitable. The infrarenal aorta is closed (this is done sagittally to avoid compromising the renal artery origins) and the new graft is attached end-to-side to the supracoeliac aorta

False aneurysm in the groin

If there are additional abnormalities such as kinking of the graft limbs, stenosis or false aneurysm at the aortic anastomosis, or if an excessively thick or partly detached graft lining is encountered at operation, the entire graft should be replaced. In the absence of such changes a false aneurysm in the groin may be managed by local reconstruction.

The old groin incision is reopened and the prosthesis identified proximal to the aneurysm. Depending on the site of the previous anastomosis, the inguinal ligament may or may not require division for proximal control. The perigraft sheath is then opened just proximal to the sac and the prosthesis mobilized for clamping. If possible the superficial and deep femoral vessels should be displayed beyond the aneurysm to allow optimum selection of a new anastomosis site. However, conventional dissection may be limited by postoperative fibrosis and endoluminal control is an easier option in some cases.

Heparin is given systemically and the graft inflow clamped. The aneurysm is then entered and back-bleeding controlled digitally while graft separation is completed. Balloon catheters are then inserted into the afferent and efferent orifices (Figure **14.10a**). Often the superficial femoral artery is occluded and only the profunda and proximal common femoral arteries need to be controlled. Thrombus within the sac is evacuated and sent for culture along with the distal portion of the graft. The state of the efferent orifices is then assessed and the medial circumflex femoral artery either clamped, if it is to be included in the reconstruction, or oversewn. Unless the original arteriotomy is reusable, which is exceptional, the proximal common femoral artery should then be similarly oversewn from within the aneurysmal sac.

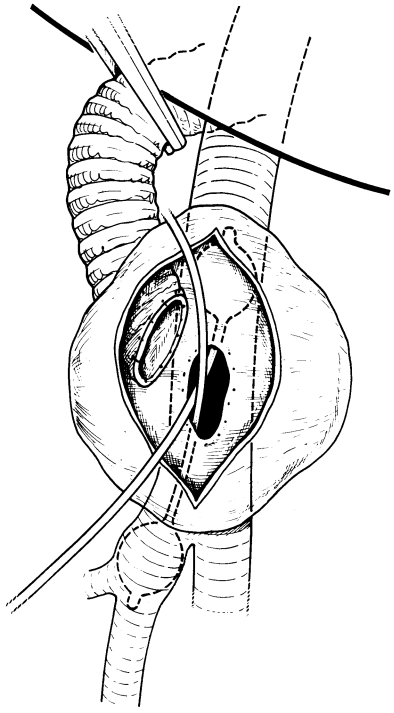


Figure 14.10a–d False aneurysm in the groin: (a) vascular control achieved by conventional clamping supplemented by endoluminal occlusion catheters

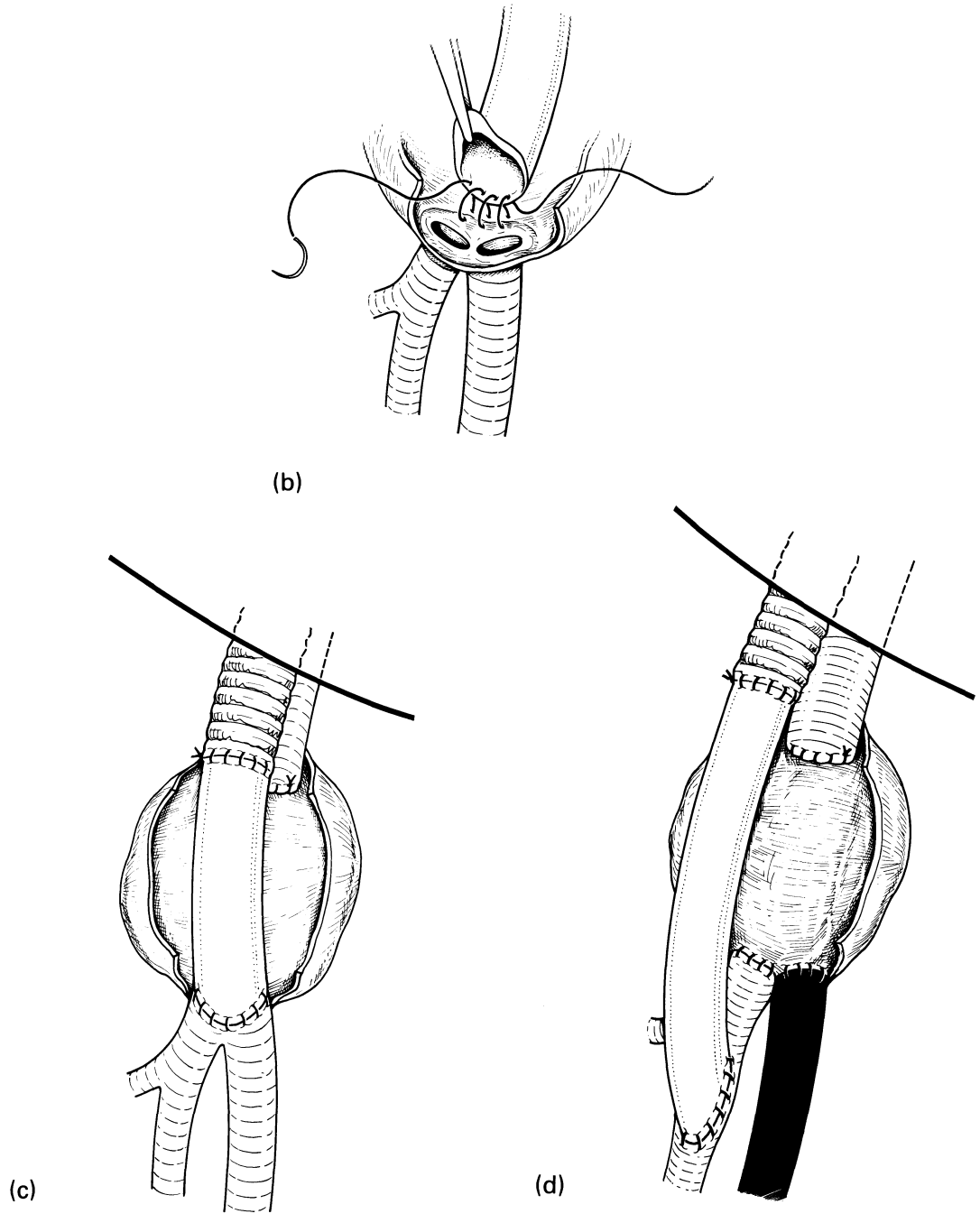


Figure 14.10 continued (b) in this example both branches of the common femoral artery are patent and the distal common femoral artery usable. A PTFE segment is inlaid at the bifurcation. (c) the PTFE segment is then attached to the old graft. (d) preferred method of reconstruction when the superficial femoral artery is occluded, with or without additional profunda stenosis

In nearly all cases a new anastomosis site should now be sought at a more distal level. If the common femoral bifurcation is sufficiently preserved, a PTFE segment may be attached here using an inlay technique around both branch orifices (Figure **14.10b**). Where the superficial femoral artery is occluded the graft is inlaid around the profunda origin. The new prosthetic segment is then cut to length and anastomosed in spatulate fashion to the old graft limb (Figure **14.10c**).

In many patients the profunda origin is rather rigid or stenosed and a more satisfactory method of reconstruction is to sew off the profunda or common femoral bifurcation from within the aneurysmal sac. The new prosthetic segment is then attached end-to-side to the profunda or perforator trunk beyond the sclerotic zone (Figure **14.10d**). This technique provides optimum conditions for anastomosis as well as avoiding any difficulty due to mismatch in graft–artery size. Indeed it may be applicable even when the superficial femoral artery is patent, an additional branch being inserted to the latter vessel using the profunda limb as source (see Figure **4.18**).

Whatever type of reconstruction is utilized, the new prosthetic segment should be wrapped in the false sac before closure. In addition, the distal anastomosis may be reinforced with an external Dacron sleeve to minimize recurrence.

Iliac false aneurysm

An isolated false aneurysm at the distal anastomosis of an aortoiliac prosthesis may be managed on similar lines. An abdominal incision is made and the prosthetic limb controlled at a convenient site proximal to the aneurysm. Distal control is usually possible by clamping rather than by endoluminal catheter. The false sac is then entered and the graft detached from the arteriotomy. The latter is then closed and a PTFE or Dacron segment attached to the old graft limb and extended down to a new anastomosis site in the groin.

COMPLICATIONS OUTSIDE THE RECONSTRUCTION ZONE

Complications which may occur at sites other than the reconstruction zone include visceral ischaemia, renal and ureteric complications, sexual dysfunction and spinal cord ischaemia.

VISCERAL ISCHAEMIA

Visceral ischaemia is an uncommon but particularly serious complication of aortic surgery. The left colon is the site most frequently affected, though small bowel ischaemia may also occur[24]. The basis for postoperative visceral ischaemia is usually operative interruption or perioperative thrombosis of a major visceral vessel in the presence of limited collateral supply. Systemic factors are often contributory and indeed on their own may be sufficient to cause visceral infarction (see Chapter 12). These factors include myocardial infarction, arrhythmias and cardiac failure, as well as features more specific to the postoperative period such as hypovolaemia, acidosis or bacteraemic shock.

Ischaemia in the superior mesenteric territory

Failure to recognize significant visceral artery disease preoperatively may lead to lethal postoperative small bowel infarction[25]. Good quality arteriography is essential before embarking on aortic surgery and should include lateral views of the visceral artery origins. These should be obtained preferably as a routine and certainly whenever there is mesenteric underfilling ('desert' sign) or a prominent meandering mesenteric collateral or pancreaticoduodenal arcade on the AP film (see Chapter 12). If significant visceral artery disease is identified, the surgical tactics should be adjusted to ensure that the superior mesenteric and coeliac arteries are revascularized, and that collateral routes from the inferior mesenteric or internal iliac arteries are preserved.

In some cases the small bowel may be observed to become acutely ischaemic during the course of an aortic procedure. Signs include pallor or cyanosis of the bowel, lack of peristalsis and ab-

sent pulsation in the mesentery. One explanation for these events is that significant visceral artery occlusive disease may have been overlooked preoperatively and ligation of the inferior mesenteric artery or its meandering mesenteric branch during aortic resection has interrupted a critical supply route (Figure **14.11**).

A second explanation is an intraoperative reduction in cardiac output which has localized in the splanchnic territory as a result of an underlying superior mesenteric artery stenosis. Even in the absence of the latter, intraoperative hypotension, if severe enough, may produce quite marked changes in the small bowel. This may be observed sometimes in operations for ruptured aneurysm.

A third possible mechanism for intraoperative small bowel ischaemia is superior mesenteric embolization. Mural thrombus on a suprarenal ulcerative plaque or ectatic segment may detach during mobilization, clamping or declamping of the supracoeliac aorta. Less commonly, emboli may be detached retrogradely from the infrarenal aorta, e.g. during correction of a total infrarenal aortic occlusion.

Management of intraoperative small bowel ischaemia requires preliminary correction of any hypotension followed by exploration of the proximal segment of the superior mesenteric artery, to establish whether or not a significant occlusive lesion is present. The coeliac axis may also require similar assessment. Lesions at either site may then be corrected, usually by means of bypass grafts from the aortic prosthesis (see Chapter 12). If palpation of the proximal superior mesenteric artery reveals a bounding pulse at the origin with absent pulsation distally, an embolus is the likely cause and treatment is immediate embolectomy.

Small bowel ischaemia may also become manifest postoperatively if the patient is dependent on a precarious collateral supply and a low flow state supervenes, e.g. hypovolaemia or cardiac insufficiency. During the first few days after aortic surgery diagnosis may be extremely difficult because of the usual postoperative abdominal dis-

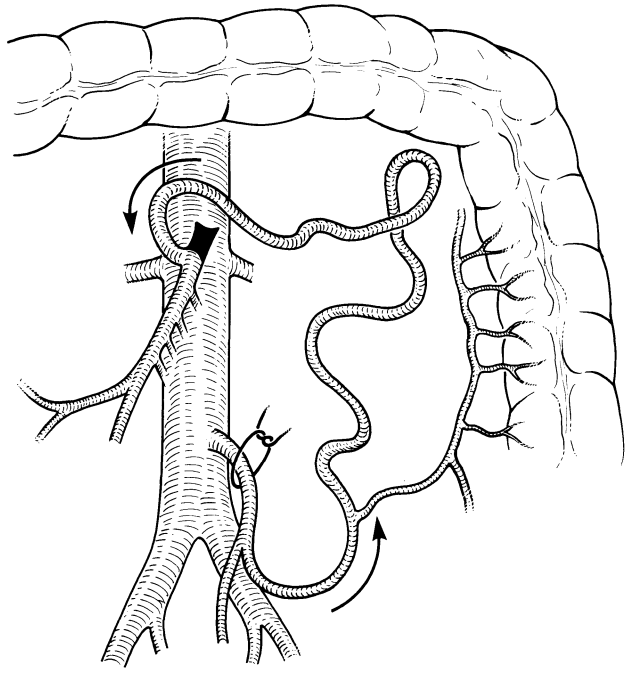


Figure 14.11 Superior mesenteric occlusion with a meandering mesenteric collateral. Ligation of the inferior mesenteric artery during aortic replacement may precipitate small bowel ischaemia

comfort. Early features include persistent abdominal pain and hyperperistalsis. At this stage the ischaemia is usually reversible but with the passage of time infarction supervenes, the abdomen becoming progressively more distended, tender and silent. The patient's mental state may become altered and there may be hypotension, acidosis and oligo-anuria. The white blood cell count is markedly raised without any other obvious cause and abdominal screening may show dilated, inert, thick-walled loops of small bowel with intraperitoneal fluid. Gas bubbles in the gut wall or portal vein are a late but pathognomonic sign of visceral gangrene.

Immediate reoperation should be undertaken on the slightest suspicion of visceral ischaemia. The risk of a negative laparotomy is preferable to overlooking a remediable situation and allowing progression to infarction. Ideally, the status of the visceral circulation should be assessed by arteriography prior to surgery but often clinical circumstances make this impossible.

At laparotomy in the early case the gut may still be largely viable and vascular reconstruction may be undertaken with or without bowel resection (see Chapter 12). Unfortunately most cases are reopened late and the situation is then irretrievable.

Ischaemia of the left colon

The incidence of symptomatic ischaemic colitis following aortic surgery is of the order of 1–2%. However, routine employment of postoperative colonoscopy suggests that the true incidence is much higher[26]. Most cases have been reported after reconstruction for aneurysmal rather than occlusive disease, with the highest incidence following operation for ruptured aneurysm[24].

The most important aetiological factor in postoperative colonic ischaemia is probably operative interruption of inferior mesenteric artery perfusion. This may occur when a large patent vessel is ligated or excluded during aortic replacement, or when the vessel origin is compromised during an aortoiliac endarterectomy. In most instances interruption of the inferior mesenteric artery during aortic resection is well tolerated since there is usually an adequate collateral supply. However, the latter may be limited if there is additional superior mesenteric or internal iliac artery occlusive disease or if the marginal artery is deficient either from natural causes or following previous colectomy. In these circumstances inferior mesenteric artery ligation may result in colonic ischaemia.

Other intraoperative events which may predispose to colonic ischaemia include ligation of the left colic and sigmoid arteries instead of the inferior mesenteric origin (Figure **14.12**), or inclusion of these branches in the retroperitoneal closure. Either of these events will reduce superior mesenteric inflow. Operative trauma to the vessels in the sigmoid mesocolon and acute loss of internal iliac perfusion may also predispose to colonic ischaemia. Loss of internal iliac perfusion may result from operative exclusion, a post-endarterectomy intimal flap or intraoperative embolization. Systemic hypotension may also be a major factor in left colon ischaemia, either by directly reducing flow or by closing down collateral pathways when the inferior mesenteric artery

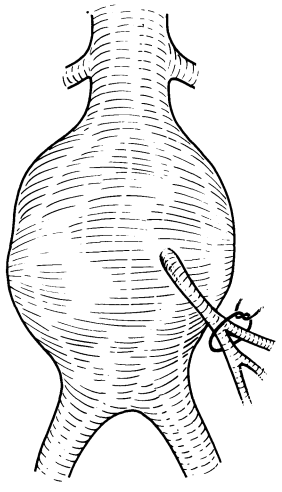


Figure 14.12 Incorrect ligation of the inferior mesenteric artery: the left colic branch has been included in the ligature. This may compromise collateral inflow to the colon from the superior mesenteric territory

has been ligated or both internal iliac arteries have been excluded. The important contributory role played by hypotension may account at least in part for the prevalence of ischaemic colitis in patients with ruptured aneurysm[27].

In some patients colonic ischaemia may not develop until the postoperative period. One example where this may occur is following operative exclusion of the distal aorta, the inferior mesenteric and the internal iliac arteries. The colon may be initially viable but if the cardiac output decreases postoperatively there may be extensive thrombosis in the excluded segment resulting in colonic infarction.

The clinical features of postoperative colonic ischaemia are governed by the depth and severity of colonic wall involvement. In reversible cases there may be premature postoperative diarrhoea which may or may not be bloody[28], left-sided abdominal discomfort, a rather prolonged ileus and a leucocytosis. These features usually persist for several days and then slowly improve.

If full thickness infarction occurs the clinical course is more serious, with pyrexia, abdominal tenderness and distension, diarrhoea which may be bloody, a leucocytosis and signs of increasing toxicity. Other presentations include isolated rectal haemorrhage, a pneumoperitoneum and late abscess or fistula formation secondary to localized perforation.

Plain films of the abdomen should be obtained at an early stage and may show distension of the colon and sometimes the small bowel, or possibly a pneumoperitoneum from perforation. If a gastrografin enema is performed, this may show 'thumbprinting' or spiculation particularly in the sigmoid region or occasionally thoughout the left colon. However, the most useful investigation is proctocolonoscopy. This may reveal a pale oedematous mucosa with haemorrhage in the milder cases, necrotic ulcers with pseudomembranes in more severe cases, and finally a greenish-black slough denoting gangrene.

Patients with potentially reversible ischaemia may be managed conservatively with intravenous fluids, nasogastric suction and antibiotics. Repeat colonoscopic studies should be made to confirm that the ischaemic process is resolving. Where the submucosa as well as the mucosa has been ischaemic, healing may result in stricture formation. Mild strictures may sometimes be managed by dilatation though if obstructive symptoms are marked a colonic resection may eventually be required.

Patients suspected of having colonic infarction require immediate reoperation. The necrotic segment should be excised and a proximal colostomy performed with drainage. If the rectum has to be removed it is essential to reperitonealize the pelvic floor to isolate the aortic prosthesis from the open perineum. In addition, an omental pedicle should be brought down into the pelvis to cover the reconstruction. Local irrigation with antibiotic or antiseptic solutions and high dose systemic antibiotics should be commenced intraoperatively and continued into the postoperative period.

The overall mortality rate among patients who develop colonic ischaemia following aortic surgery is approximately 50% and approaches 90% in transmural infarction[26]. These figures underline the necessity for reimplanting the inferior mesenteric artery during aortic resection whenever a large patent vessel is encountered (Figure **14.13**). In addition, efforts should always be made to preserve at least one internal iliac artery during the aortic procedure (see Chapter 2).

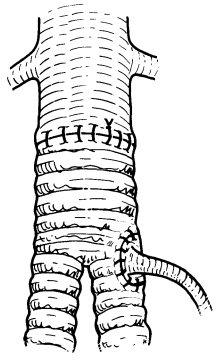


Figure 14.13 Reimplantation of the inferior mesenteric artery during aortic replacement

RENAL AND URETERIC COMPLICATIONS

Renal artery complications

Preoperative recognition of abnormalities of the renal arteries or kidneys will reduce the incidence of intraoperative and postoperative complications. Thus the discovery of an accessory lower pole artery or a duplicated renal artery on arteriography will prevent inadvertent ligature or damage during the aortic procedure (these accidents may be responsible for postoperative hypertension). In addition the presence of these anomalies may lead to a change in the surgical plan, e.g. end-to-side aortic bypass attachment instead of end-to-end (Figure **14.14a, b**); employment of an aortic endarterectomy to clear a stenosed accessory renal artery origin (Figure **14.15a, b**); and reimplantation of an accessory artery during aortic aneurysmectomy (Figure **14,16a, b**).

Anomalies of the kidney itself may also be evident during preoperative work-up and include horseshoe kidney and ectopic kidney. The former may or may not complicate aortic management according to its blood supply and the state of the renal isthmus (see Chapter 2). An ectopic kidney with a renal artery arising from the aortic bifurcation or iliac axis usually does not present a problem, though if the orifice is involved in the atheromatous process it may be endarterectomized and reimplanted into the reconstruction.

Coexistent renal artery occlusive disease should also be fully evaluated prior to aortic surgery. In most instances severely obstructive lesions are corrected at the time of the aortic procedure. Preoperative percutaneous angioplasty or preliminary hepatorenal or splenorenal bypass may be alternative methods of managing the renal artery lesion in poor risk cases (see Chapter 13).

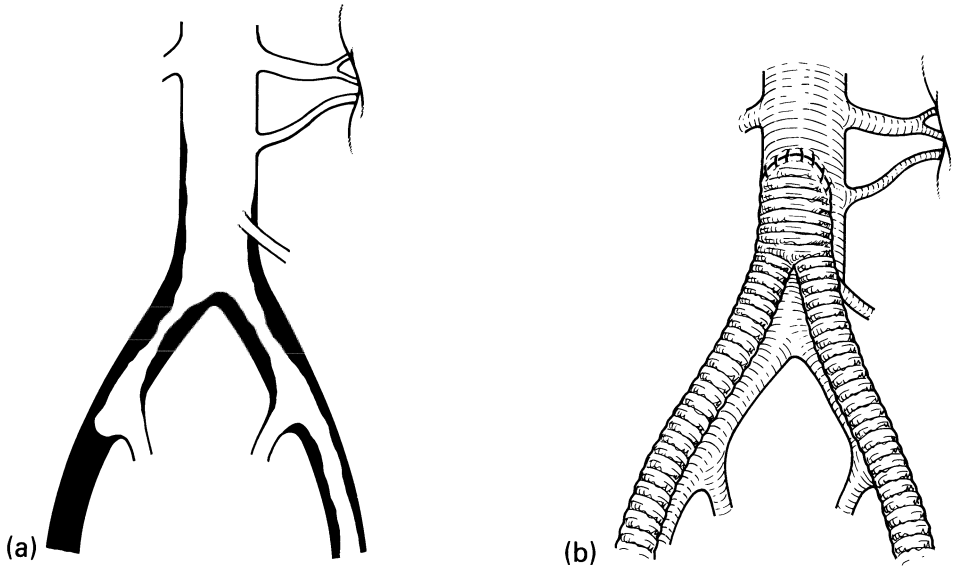


Figure 14.14(a, b) Accessory lower pole renal artery in a patient with extensive aortoiliac occlusive disease: (a) pathology. (b) end-to-side aortofemoral bypass attachment preserving antegrade flow in the accessory renal artery (and also in the inferior mesenteric artery)

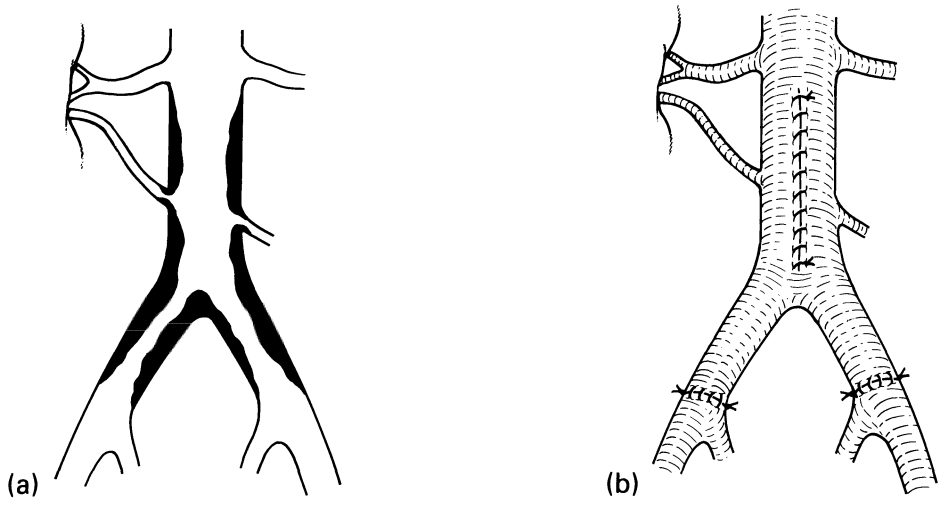


Figure 14.15(a, b) (a) Accessory right lower pole renal artery in a patient with Leriche syndrome. (b) Endarterectomy of the aortoiliac segment including the origin of the accessory renal artery

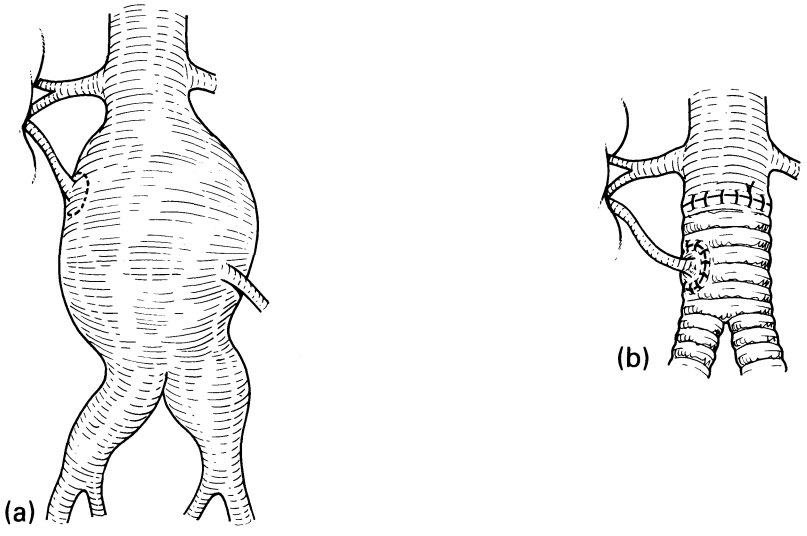


Figure 14.16(a, b) Reimplantation of an accessory renal artery in a patient with an abdominal aortic aneurysm

Intraoperative renal artery accidents are uncommon. Acute thrombosis may supervene in a severely stenosed vessel during supracoeliac clamping. Thrombosis may also occur if an infrarenal aortic clamp is placed adjacent to the renal artery origin and fractures or elevates intimal plaque. A similar intimal dissection may occur if an aortic thromboendarterectomy is taken too close to the renal artery origins (Figure **14.17a, b**).

Renal artery embolization is an alternative cause of acute intraoperative occlusion. Antegrade embolization may occur from an unrecognized ulcerative or ectatic lesion in the suprarenal aorta during mobilization of this segment or on clamp application or removal (Figure **14.18**).

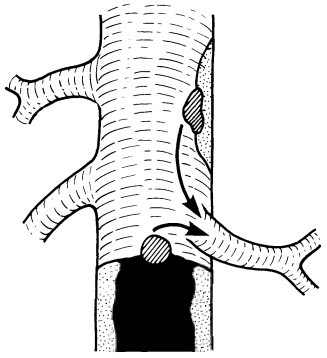


Figure 14.18 Possible sources of intraoperative renal artery embolization: suprarenal mural plaque or infrarenal aortic thrombosis

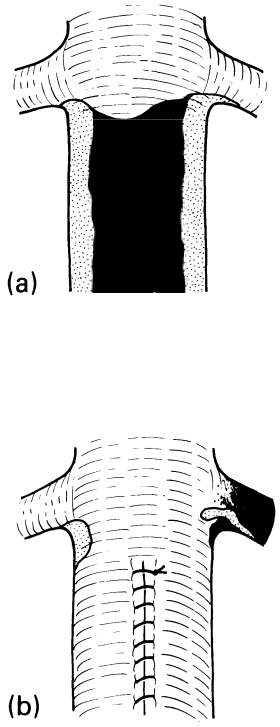


Figure 14.17(a, b) (a) Infrarenal aortic occlusion. In this case the atheroma extends up to the renal artery origins. (b) Aortic endarterectomy has encroached on the left renal artery origin resulting in an intimal flap and thrombosis

Thrombus may also be dislodged retrogradely from the infrarenal aorta, particularly during correction of a total aortic occlusion.

Intraoperative renal artery occlusion may be suspected if there is haematuria, a reduction in urinary output or a change in colour of the kidney (this may be partial or total, depending whether a branch artery or the main vessel is involved). Operative inspection will then reveal a pulseless vessel. Revascularization should be immediately undertaken either by bypass grafting or embolectomy (see Chapters 6 and 13).

Renal artery complications may occasionally become manifest in the postoperative period. Thus, sudden severe hypertension may be due to an uncorrected renal artery stenosis or an intraoperative renal artery accident. Bilateral renal artery occlusion or a unilateral arterial complication in a solitary kidney may result in oligo-anuria. The definitive investigation is arteriography following which the patient should be returned to the operating room for revascularization, usually by means of a bypass graft. However, if the patient's condition is unfavourable for re-laparotomy, an attempt may be made to restore renal artery patency by transfemoral catheter thromboembolectomy[29,30] or by percutaneous transluminal angioplasty[31].

Acute renal failure

Acute renal failure is a major concern after aortic surgery, particularly in patients with ruptured aneurysm[32]. Factors responsible for postoperative renal dysfunction may be grouped into central hypotensive causes, local ischaemic causes and a group of miscellaneous factors. The first group includes preoperative and intraoperative haemorrhage, declamping shock, myocardial insufficiency and inadequate fluid replacement. Local ischaemic causes may comprise intraoperative renal artery accidents (see above), prolonged supracoeliac or renal artery clamping, and atheromatous microembolization. Miscellaneous causes include disseminated intravascular coagulation, gut or muscle necrosis, and nephrotoxins such as anaesthetic agents, contrast media or aminoglycoside antibiotics. In all groups acute renal failure is more likely when there is pre-existing renal impairment.

Although some of the above factors are difficult to avoid completely, careful attention to surgical technique and anaesthetic support measures may minimize the risk of postoperative renal impairment. Obviously intraoperative haemorrhage must be kept to a minimum and all losses promptly replaced. Thereafter fluid replacement should be adjusted according to left heart pressures[33,34]. Additional volume loading is advisable immediately prior to clamp release and this together with staged declamping should avoid any fall in systemic arterial pressure.

In patients with a ruptured aneurysm, rapid aortic control and expeditious graft insertion are crucial. In particular additional blood loss, whether from venous injury, through the graft or from technical errors at the suture lines, should be avoided at all costs. Where it has been necessary to divide the left renal vein for access, this should be re-anastomosed on completion in order to maximally safeguard renal function[35].

Renal microembolization is a relatively unrecognized but important cause of postoperative dysfunction[36]. This may arise as a result of applying an infrarenal clamp to a markedly degenerate aorta, loose atheromatous material being squeezed directly into the renal artery origins. Alternatively, debris may be stripped from the aortic wall by blood jetting against the jaws of the clamp. In view of this risk, it is advisable to use supracoeliac control whenever the aorta is markedly degenerate at the usual clamp site. In addition the proximal aortic clamp should be applied once only, since repeated cross-clamping may fracture otherwise intact aortic intima and predispose to renal as well as distal embolization.

Additional protection for the kidneys is not usually necessary during supracoeliac occlusion since the period of circulatory arrest is normally only a matter of minutes. However, when renal ischaemia is likely to exceed 20 minutes, e.g. during combined aortic and renal replacement, or in thoracoabdominal aneurysm surgery, it may be advisable to cool the kidneys via the open aorta and to supplement this with systemic or regional inosine (see Chapters 2 and 13).

Ureteric complications

These often lead to loss of the kidney. They may be categorized under the headings of intraoperative injury, postoperative fistula and stricture formation.

The risk of dividing or damaging the ureter is greatest in re-do surgery, when the ureter may be embedded in a fibrotic reaction around a graft, endarterectomized segment or aneurysmal sac. Immediate repair may not necessarily risk arterial infection providing the repaired ureter is wrapped in omentum to separate it from the arterial reconstruction.

A postoperative ureteric fistula is most commonly due to ischaemic necrosis, though occasionally an unrecognized operative injury may be the cause. It usually presents as a retroperitoneal collection during the second postoperative week. This may be distinguished from a lymphocele by its urea content on aspiration and by the demonstration of a contrast leak on intravenous pyelography. Other features may include septicaemia or pyonephrosis. In view of the risk of secondary arterial infection, these cases are best treated by nephrectomy, with an omental wrap to cover the arterial reconstruction.

A postoperative stricture is the commonest ureteric complication[37]. This may be intrinsic due to devascularization, but most are secondary to extrinsic compression. Aetiological factors include graft misplacement (Figure **14.19a**), a retroperitoneal reaction associated with perigraft haematoma or lymphocele, and false aneurysm formation[38]. Patients with an inflammatory type of aortic aneurysm may also have continuing ureteric hold-up postoperatively though most cases settle (see Chapter 2). In a significant number of patients postoperative hydronephrosis is the first sign of graft infection and may antedate more overt septic manifestations by a considerable period of time.

Patients with ureteric obstruction following aortic surgery may present with loin pain, pyonephrosis, septicaemia or renal impairment. However, the majority of cases are asymptomatic and are detected only during routine follow-up ultrasound examination[38]. Not all patients require treatment since in many cases the degree of obstruction is mild and non-progressive while others spontaneously regress as the post-implant retroperitoneal reaction settles. Specific indications for treatment include progressive obstruction, renal impairment or secondary sepsis.

Most extrinsic ureteric strictures can be managed by careful ureterolysis and intraperitonealization. If the ureter is compressed as a result of misplacement posterior to the limb of a prosthesis, the easiest solution is to temporarily transect the latter and transpose the ureter anteriorly (Figure **14.19b**). The alternative option of ureteric division and re-anastomosis may risk graft sepsis should urinary leakage occur. An intrinsic ureteric stricture may be managed by balloon dilatation or by resection-anastomosis with an accompanying omentopexy (Figure **14.19c**). Complex ureteric reconstructions are best avoided because of the risk of infecting the arterial prosthesis and if the above options are not feasible, nephrectomy is the safest course. This is also the treatment of choice if there is a secondary pyonephrosis.

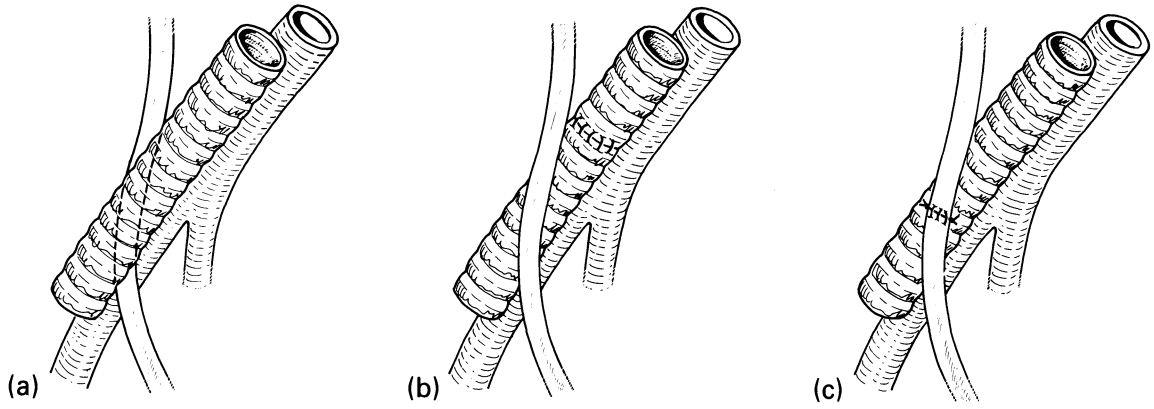


Figure **14.19(a–c)** Ureteric stricture following aortofemoral bypass: (a) extrinsic compression due to a technical error in which the graft has been placed anterior to the ureter. (b) treatment requires anterior transposition of the ureter after temporary graft division. (c) intrinsic ureteric stricture, treated by resection–anastomosis. Omentum is then interposed between the repaired ureter and the arterial prosthesis

SEXUAL DYSFUNCTION

Postoperative difficulties with erection or ejaculation may occur as a result of interruption of the autonomic nerve fibres investing the lower aorta and proximal common iliac arteries[39] (Figure **14.20**). Impotence may also follow operative impairment of pelvic blood supply. With appropriate adjustments in surgical technique, errors in both areas may be avoided and the incidence of postoperative sexual dysfunction reduced[40].

In aneurysm cases, nerve damage may be minimized by restricting the dissection for vascular control to the aorta immediately below the renal arteries, and to the common iliac bifurcation or external iliac artery on either side. The aorta is opened along the right anterolateral wall to avoid the predominantly left-sided pre-aortic plexus. For the same reason the inferior mesenteric artery should be sewn off from within the sac rather than externally ligated, if it is not to be preserved for reimplantation. Tunnelling the graft limbs through the unopened common iliac arteries may be an additional means of avoiding nerve damage where appropriate (see Figure **2.7d**). Finally, when the reconstruction has been completed the redundant aortic sac should be double-breasted over the prosthesis rather than excised, taking care to avoid catching the nerve plexus with the securing stitches (see Figures **2.8, 10**).

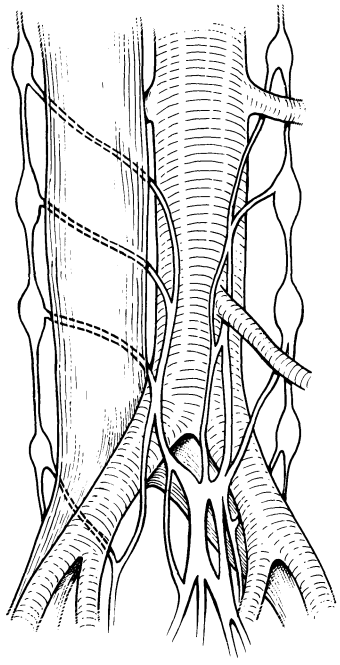


Figure 14.20 Diagrammatic representation of the pre-aortic and hypogastric autonomic plexi

In ruptured aneurysms it is obviously much more difficult to carry out a nerve-sparing dissection since rapid control is essential. In addition, haematoma from the rupture may itself disrupt autonomic nerve supply and for both these reasons the incidence of postoperative sexual dysfunction is greater than in elective cases.

Aortoiliac endarterectomy is more likely than a bypass technique to result in damage to the autonomic nerve fibres, since it requires more extensive arterial dissection. This damage may be kept to a minimum by incising the periaortic tissue longitudinally and mobilizing the aorta within this plane. Transverse incisions inevitably transect nerve filaments in the periaortic layer and should therefore be avoided. Preservation of the nerve fibres crossing the front of the left common iliac artery is particularly important and one solution is to transpose the distally divided common iliac artery to an anterior position before undertaking eversion endarterectomy (see Figure **3.27a**). Alternatively the common iliac artery may be left largely undisturbed and clearance achieved using ring strippers from the distal end (see Figure **3.29**). Because of the greater difficulties with nerve preservation during endarterectomy, bypass grafts may be preferred in younger male subjects, providing adequate internal iliac perfusion can still be assured (see below). Lumbar sympathectomy may add to the denervation already sustained and is therefore also best avoided in sexually active male patients.

Impotence may occur after aortic surgery as a result of interrupting the pelvic blood supply. Although potency may still be retained when both internal iliac arteries are occluded[41], this is unusual and efforts should always be made to preserve internal iliac perfusion on at least one side during aortic surgery. In the case of an aortofemoral bypass, this may necessitate using an end-

to-side aortic anastomosis instead of end-to-end attachment, if there is significant external iliac occlusive disease. Similarly, during aortoiliac endarterectomy, great care must be taken to avoid creating an intimal flap in the internal iliac artery. Although significant ostial plaque may be easily removed via a distal common iliac arteriotomy, blind extraction of more distal intima may lead to dissection and acute occlusion (see Figure **3.37**).

Intraoperative embolization of the internal iliac arteries is another mechanism which may contribute to postoperative impotence. Intimal damage from clamping a degenerate aorta is one source of atheromatous emboli and, as previously indicated, once only aortic clamp application is routinely advisable. Any clots or debris should be carefully flushed externally and the graft thoroughly aspirated and irrigated before restoring distal flow.

In some cases arterial reconstructive surgery may improve pre-existing impotence. This particularly applies to patients with occlusive disease in the distal aorta and common iliac arteries (Leriche syndrome). Aortoiliac endarterectomy with a nerve-sparing technique may be highly successful in restoring potency in these cases. A similar result may also be achieved by aortofemoral bypass if retrograde internal iliac perfusion can be assured.

Other patients are impotent as a result of a localized occlusion of the common iliac artery or a high grade internal iliac stenosis. These patients may respond favourably to unilateral endarterectomy or to percutaneous transluminal angioplasty. However, when the internal iliac arteries are more diffusely atherosclerotic, improvement in erectile function is unlikely.

A third site where vascular occlusive disease may cause impotence is in the pudendal artery or its branches. Lesions at these sites will not be evident on standard arteriography and selective or superselective catheter studies may be needed for full delineation. In some of these cases direct corporeal revascularization procedures may be possible using microsurgical techniques[42,43].

Where vascular reconstruction is not feasible or is unsuccessful, a penile prosthetic device may be considered. This may also be helpful in postoperative patients who are impotent as a result of nerve damage.

SPINAL CORD ISCHAEMIA

Ischaemia of the spinal cord manifest by partial or complete paraplegia is a very rare complication of abdominal aortic surgery. Most of the reported cases have followed aneurysm surgery, particularly ruptured aneurysm[44].

The distal spinal cord receives its main blood supply from the artery of Adamkiewicz which is the largest radicular artery. This usually arises from an intercostal artery at the approximate level of T9 or T10, but may occasionally originate from an infrarenal lumbar artery. The latter may be interrupted during abdominal aortic replacement. Even so, collateral flow from adjacent intercostal or lumbar arteries into the anterior spinal artery is usually sufficient to maintain spinal cord function. However, if the anterior spinal artery is deficient or if collateral flow is impaired by atherosclerotic involvement of the radicular vessels or by operative hypotension, then interruption of the great radicular artery may result in paraplegia. Hypotension is a particularly important factor and explains the predilection for ruptured aneurysm cases.

Little can be done to prevent this complication, apart from avoiding perioperative hypotension. Reimplantation of large lumbar arteries into the graft has been advocated, but size alone is no indication of their relative contribution to spinal cord supply. However, the risk of paraplegia is increased when the aorta requires extensive replacement (e.g. thoracoabdominal aneurysm) and reimplantation of prominent upper lumbar or intercostal arteries may then be advisable (see Chapter 2).

INFECTION IN AORTIC SURGERY

Infection is perhaps the most feared complication of aortic surgery. It is essentially a problem of prosthetic grafts and these only will be considered, though similar management principles can be applied to the rare infections which complicate other aortic procedures.

Aortic graft sepsis may occur with an estimated average incidence of 1–2%. Grafts in the femoral position are more prone to this complication than those performed solely through an abdominal incision. Although improved results have recently been reported in managing these infections[45,46], most series continue to report a high morbidity and mortality rate, particularly when the aortic portion of the graft is involved[47,48].

The most common organism in aortofemoral prosthetic infection is staphylococcus, though recent reports have indicated an increasing incidence of Gram-negative or mixed infections[48]. *S. aureus* predominates in early infections, whereas *S. epidermidis* is more frequently cultured in late aortofemoral sepsis[49]. Aortoiliac graft sepsis is predominantly due to Gram-negative organisms, reflecting the frequency of underlying enteric fistulization.

In approximately half of the cases clinical signs and symptoms of infection appear within the first 30 days of surgery. In the remainder, the clinical features are delayed, in some cases for up to several years after the initial event. This may mean that organisms have been lying dormant since their introduction at operation. Other explanations for late onset of infection include secondary colonization during an incidental bacteraemia, late enteric fistulization, and unrelated intra-abdominal sepsis.

The most common presentation of aortofemoral graft sepsis is with infection in the groin. This may take the form of a fluctuant mass, a frank abscess, a draining sinus or wound dehiscence. Once the graft or suture line is involved there may be haemorrhage, a false aneurysm, graft occlusion or septic embolization. In some low grade chronic infections, graft limb thrombosis or false aneurysm may occur without any other signs of sepsis, and unless this possibility is always kept in mind the true diagnosis may be overlooked with disastrous consequences.

Aortoiliac graft infection or those uncommon infections which are restricted to the aortic end of an aortofemoral graft may present more insidiously and are more difficult to diagnose. Clinical features may include vague abdominal or back pain, otherwise unexplained fever or leucocytosis, ureteric obstruction and metastatic abscesses. Untreated, the infection may eventually disrupt the aortic suture line with formation of a false aneurysm, retroperitoneal or intraperitoneal haemorrhage, or enteric fistulization.

The diagnosis of an infected graft may be self-evident when there are signs in the groin. Cultures may be obtained from a sinus or from aspiration of a mass. Blood cultures may also be taken, though these are often negative unless there is an underlying aortoenteric fistula. Sinography may be helpful in establishing the diagnosis and in delineating the proximal extent of the infection. However, skip lesions may occur and a negative sinogram does not necessarily rule out proximal graft involvement.

The most accurate method of assessing intra-abdominal graft status is a CT scan[50]. This may show fluid around the graft, or a false aneurysm at the aortic anastomosis. Alternatively there may be air in the periprosthetic tissues or in the aorta, signifying an enteric fistula[51]. Tomographically guided needle aspiration of perigraft fluid may provide bacteriological confirmation of the diagnosis[52] and will allow specific antibiotic therapy to be commenced before operative manipulation.

In some patients with chronic low grade aortoiliac graft sepsis the diagnosis may remain elusive. A gallium-67 or indium-111 scan may then be helpful in localizing the infective process to the graft area[53,54]. Endoscopy may be additionally considered where there is a possibility of underlying enteric fistulization (see below). Arteriography is of more help in planning revascularization than in establishing the diagnosis of graft sepsis. However, in some cases the possibility of infection may be suggested by the presence of a false aneurysm or prominent crimping due to loss of graft incorporation.

MANAGEMENT OF INFECTION IN THE GROIN

Not all infections in the groin require graft removal. If the graft is patent and there is neither haemorrhage from the suture line nor a false aneurysm, and if blood cultures are negative, a conservative approach may be justified. High-dose systemic antibiotics are commenced and subsequently adjusted according to sensitivity reports. The patient is returned to the operating room and the groin incision reopened and thoroughly debrided. The wound may then be either packed with povidone–iodine soaks or continuously irrigated with the same solution[55]. Spontaneous healing by secondary intention may occur, or alternatively the wound may be closed by secondary suture or with a rotation flap[56] once all the infection has completely settled.

Graft-artery involvement as evidenced by suture line bleeding, false aneurysm, or graft occlusion is an indication for removal of the graft either completely or in part, usually with alternative revascularization. If the infection appears to be localized to one groin it may be possible to preserve most of the graft, thereby avoiding a major abdominal procedure. However this approach should only be considered when the graft limb is patent, septicaemia is absent and there is no suggestion of proximal involvement on intravenous pyelogram, sinogram, CT scan or aortogram. A final decision whether or not local graft removal should be attempted will depend on the appearances of the graft on iliac exploration.

If a local procedure seems a possibility, the patient is prepped and draped for iliac exposure and for distal limb bypass. Particular care must be taken to isolate the infected groin from the operative field. A short retroperitoneal approach is then made to the iliac portion of the graft. If the exterior of the graft looks abnormal and oedema or periprosthetic fluid is present, the proposed plan for limited graft removal must be abandoned and steps taken to remove the entire prosthesis (see below). This course should be followed even if Gram staining is negative[49]. However, if the iliac portion of the graft appears soundly incorporated, a 3–4 cm segment should be resected and the distal graft tunnel closed over with retroperitoneal tissue in order to isolate the remainder of the reconstruction from the infected groin.

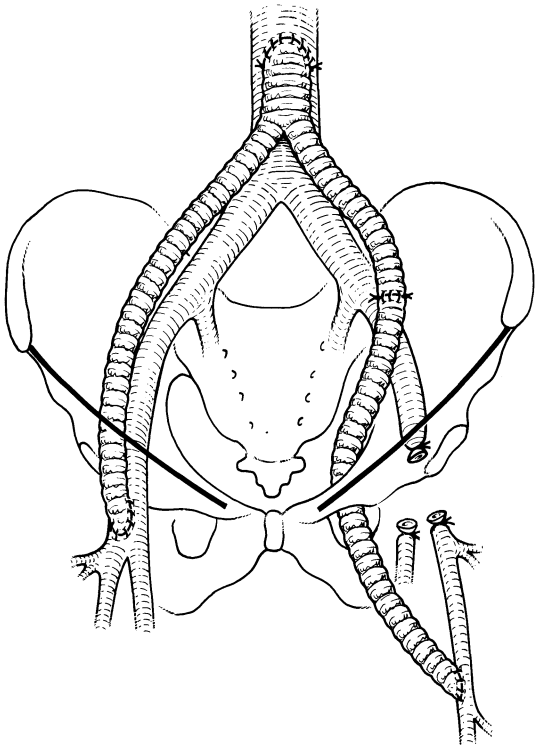


Figure 14.21 Obturator bypass for aortofemoral graft sepsis confined to one groin. The infected femoral end of the aortofemoral bypass has been removed and the femoral vessels have been ligated

The proximal graft stump may then act as source for obturator bypass[57] (Figure **14.21**). However, durability of obturator grafts has been rather limited in our experience and a preferable option is to route the new graft laterally around the groin. The simplest lateral course is a subcutaneous one passing just medial or lateral to the anterior superior iliac spine and then diagonally down the thigh to the distal femoropopliteal segment or below-knee vessels. Alternatively the graft may be routed deep to sartorius and rectus femoris for distal attachment to the mid-profunda femoris or the superficial femoral artery in the adductor canal (Figure **14.22**).

Once the new bypass is functioning, the incisions are closed and sealed. The infected groin

is then opened, and the distal segment of the aortofemoral prosthesis is removed. Often the infection will have destroyed the common femoral artery and after graft removal the safest course is to separately ligate the common, superficial and deep femoral vessels. Occasionally it may be possible to preserve the communication between the superficial and deep femoral vessels by oversewing the bifurcation with prolene. The groin incision is then lightly packed with povidone–iodine soaks and left open. Healing is prompt once the infected foreign material has been removed.

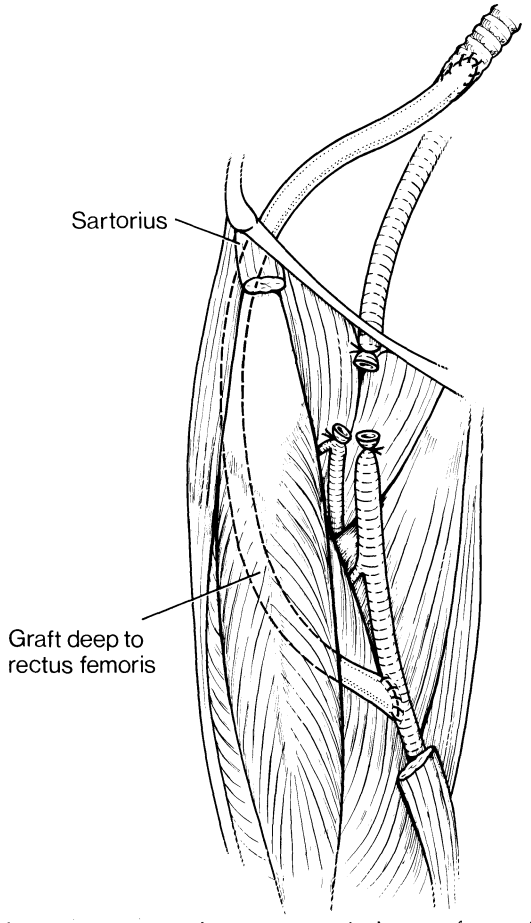


Figure 14.22 Lateral extra-anatomic bypass for graft sepsis in the groin. The new bypass is attached to the iliac limb of the original prosthesis and tunnelled deep to rectus femoris, rejoining the superficial femoral artery in mid-Hunter's canal. (sartorius not shown completely)

The above order of events applies to patients presenting with groin sepsis but without serious haemorrhage. If severe bleeding occurs, the infected zone should be opened first and the arterial axis interrupted with disconnection of the graft. The patient should then be reprepped and draped and extra-anatomic bypass carried out as an entirely separate procedure using fresh instruments, gloves, etc. The distal infected segment of graft is then finally removed via the groin.

MANAGEMENT OF INTRA-ABDOMINAL GRAFT INFECTION

Patients with aortofemoral graft infection which is not confined to the groin and those who develop infection after aortoiliac or aortic graft placement require complete graft removal. Occasionally graft excision may be the only measure required, if the prosthesis is occluded and the collateral supply sufficient to maintain limb viability. Residual or recurrent ischaemic symptoms in these cases may merit *in situ* regrafting at a later date, using the supracoeliac or thoracic aorta as source[46,58]. In most patients with aortoiliac occlusive disease and virtually all those with aneurysmal disease, aortic graft removal must be accompanied by immediate revascularization. A selective policy of revascularization according to clinical and Doppler criteria has been employed by some authors in an attempt to limit the magnitude of the surgical undertaking[59]. However these criteria may not be completely reliable and irreversible leg or colonic ischaemia may result if revascularization is delayed[60].

Graft removal and extra-anatomic bypass

Those patients who present with haemorrhage from the groin wound require local graft disconnection and ligation of the femoral vessels, as previously described. Thereafter the patient is reprepped and draped and an axillofemoral (or popliteal) bypass inserted. In the absence of serious groin haemorrhage extra-anatomic bypass is undertaken as the opening move. Once this is in place the infected aortic prosthesis should be

removed, either under the same or a subsequent anaesthetic, according to the patient's condition. High-dose systemic antibiotic cover is essential to minimize the risk of the new graft becoming infected as well as to prevent continuing aortic sepsis. If the infection is complicated by aortic haemorrhage, or an expanding false aneurysm, abdominal graft removal should precede extra-anatomic bypass since aortic control is obviously the priority. However, the lower limbs may then become markedly ischaemic before revascularization is achieved and the accompanying acidosis may add to the risk of renal failure. This approach should therefore only be used in urgent circumstances.

Extra-anatomic revascularization is usually straightforward in cases of aortoiliac graft infection since the groins have not been disturbed. Insertion of an axillofemoral bypass with a suprapubic femorofemoral crossover follows conventional lines (see Chapter 3). Revascularization in patients with aortofemoral graft sepsis is more difficult and additional measures are necessary to circumvent the femoral areas. The following account is concerned solely with this problem.

The choice of which side to use for the axillofemoral bypass is governed by the relative state of the subclavian–axillary arteries and the ease of graft tunnelling around the groins. In the rare event that neither axillary artery is suitable the ascending aorta may be used. The groins must be carefully excluded during prepping and draping, and provision made for crossover grafting and for possible bypass extension to the popliteal–tibial vessels.

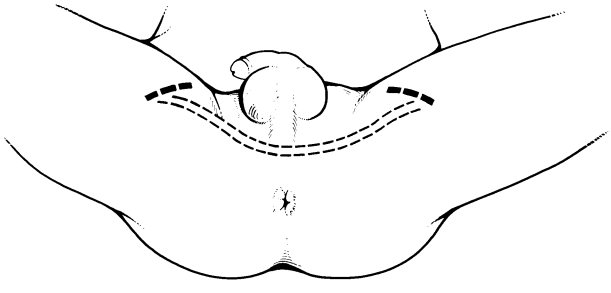


Figure 14.23 Operative position for transperineal femorofemoral bypass

Although bilateral axillofemoral grafts routed laterally round each groin may be utilized, a single axillofemoral graft with a femorofemoral extension will provide better graft flow and late patency (see Chapter 3). The crossover may have a high take-off passing across the lower abdomen and skirting laterally around the opposite groin. However, a transperineal route may be preferable as this will not only avoid contamination from the groins but will allow the crossover graft to originate close to the distal end of the axillofemoral limb. This arrangement ensures maximum flow throughout the full length of the graft[61]. In order to route the graft via the perineum, the patient should be positioned with the legs slightly flexed and abducted and with the scrotum fixed to the anterior abdominal wall (Figure **14.23**).

The donor axillary artery and the distal graft attachment sites are then exposed and the axillo–distal tunnelling completed. The tunnel for the transperineal crossover graft is developed by digital dissection posterior to the scrotum from an upper medial thigh incision on either side. The midline raphe will require to be breached to put the two sides in communication. In women a comparable crossover tunnel can be developed just below the pubic symphysis. Tapes may be left in the graft tunnels to facilitate subsequent graft passage. In order to reduce the risk of contamination, antibiotic–soaked sponges may be placed in the incisions which are not in immediate use.

Systemic heparin is then given and an 8mm PTFE graft is anastomosed end-to-side to the proximal axillary artery. This is then led down the trunk in the preformed tunnel to enter the leg lateral to the groin. The iliac crest may sometimes require notching during this manoeuvre to prevent graft compression. Distally the graft may be attached to the mid-profunda, superficial femoral or popliteal–tibial vessels, according to availability. In the case of the longer types of bypass, a sequential attachment to more than one vessel may augment graft flow and improve patency[62]. A second PTFE segment, or alternatively a length of autogenous saphenous vein from the distal thigh, is then attached close to the distal end of the axillofemoral limb. This is then led through the perineal tunnel to the opposite side where it is anastomosed to the mid-profunda and/or superficial femoral artery, or to the more distal vessels (Figure **14.24**).

Once the grafts are in place and functioning, all the incisions are closed and sealed. If possible the aortic prosthesis should now be removed under the same anaesthetic. However, if the procedure has already been rather lengthy and the aortic suture line does not appear at immediate risk, graft removal may be undertaken at a second operation a few days later. In this event the original graft limbs should be disconnected in the groins to prevent secondary seeding of the extra-anatomic bypass and to reduce competitive flow.

Removal of the aortic graft is achieved using a xiphopubic approach. The duodenum and small bowel are first displaced away from the graft area, taking care to exclude the possibility of an enteric fistula. The proximal infrarenal aorta is prepared for clamping as is the prosthesis below the anastomosis. If access is difficult, proximal aortic control may be obtained at supracoeliac level. Clamps are applied above and below the anastomosis and the graft is detached from the aorta. If the femoral limbs have already been disconnected the entire prosthesis can now be removed. If not, this step is postponed until the aorta has been closed.

Where there has been a previous end-to-side aortic graft attachment it may sometimes be pos-

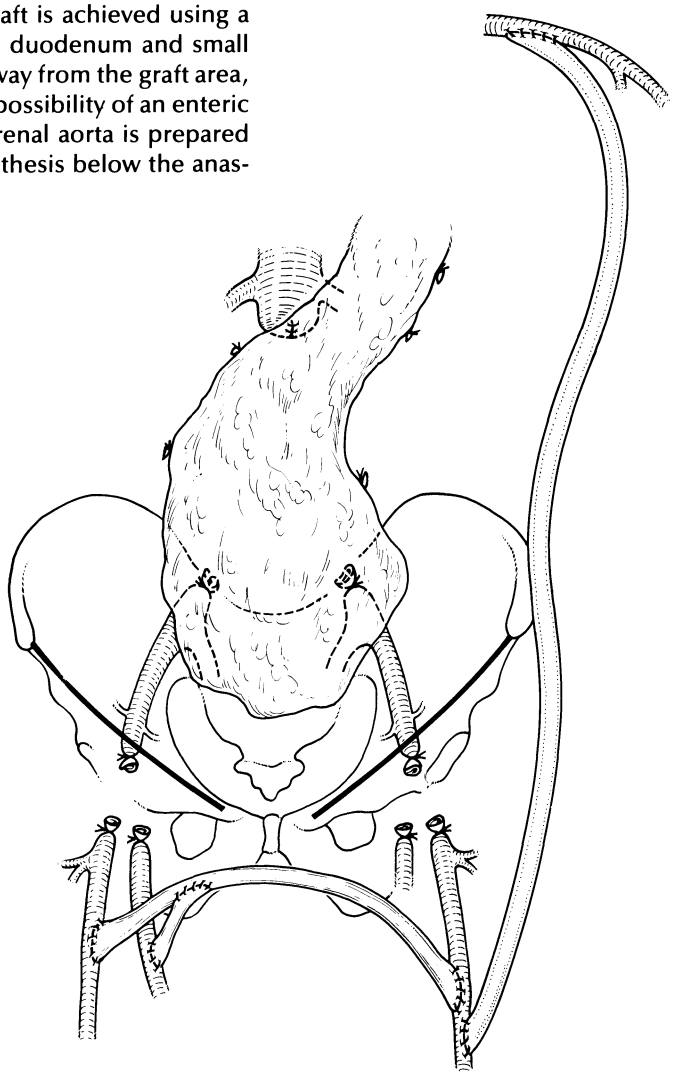


Figure 14.24 Infection of an entire aortofemoral graft. A PTFE axillofemoral bypass has been inserted with a femorofemoral (perineal) crossover using autogenous vein (the right profunda and superficial femoral arteries are both patent). The infected aortic prosthesis is then removed and the graft bed covered with omentum

sible to close the aortotomy directly, thereby preserving aortic flow. However, this is uncommon and in most cases it is necessary to transect the aorta completely with ligation of the distal stump or iliac arteries.

Management of the proximal aortic stump is critical. A thorough débridement should be carried out and the aorta resected back to healthy tissue. The stump is then closed with a running prolene suture and a proximal ligature. If there is insufficient aorta below the renal arteries to permit ligation, the stump may be closed with a series of interrupted mattress sutures followed by a second running prolene suture across the distal end. In particularly difficult cases the aortic stump closure may be reinforced with a flap of prevertebral fascia[63], a seromuscular jejunal patch[64] or with autogenous vein pledgets[65]. Alternatively the aorta may be transected above the renal arteries to obtain better tissue for closure. Renal revascularization may then be achieved using autogenous grafts from the supracoeliac aorta or from the hepatic and splenic arteries (see Chapter 13).

The groins are then opened and the distal anastomoses dismantled, allowing complete removal of the infected prosthesis. As previously described, the femoral vessels may need to be separately ligated if there is extensive destruction. However, where it is essential to maintain retrograde internal iliac perfusion, the common femoral arteriotomy on the best preserved side may be closed directly or with an autogenous patch. This manoeuvre increases the risk of secondary haemorrhage compared with femoral ligation and should not be used as a routine.

Finally, the retroperitoneum is thoroughly irrigated with antibiotic saline or dilute povidone–iodine solution and a viable pedicle of omentum brought down over the aortic stump. Sump drains are placed to the graft bed exiting on the side opposite to the axillofemoral bypass. These may be used for drainage and postoperative irrigation with antibiotic or antiseptic solution. The abdomen is then closed in layers and the groin wounds lightly packed with povidone-iodine soaks.

In situ autogenous reconstruction

In situ autogenous reconstruction of the aortoiliofemoral segment is an alternative to remote subcutaneous bypass following aortic graft removal[45]. Where the infected aortic prosthesis has been attached end-to-side to the aorta, distal perfusion may sometimes be maintained by a combination of autogenous patching and endarterectomy of the native aortoiliofemoral vessels. If an end-to-end aortic graft has been previously placed, *in situ* repair is more difficult but not impossible. Appropriate conduits may be constructed from segments of saphenous vein or endarterectomized superficial femoral artery.

These *in situ* techniques require a rather prolonged operating time even with two surgical teams. However, by maintaining aortic flow the hazards of stump blowout and ascending thrombosis are avoided. As a result, these methods have led to an improved survival rate among patients with aortofemoral graft sepsis[45,66].

AORTOENTERIC FISTULAE

Aortoenteric fistulae may be of two types: primary or secondary. Primary fistulae arise as a result of rupture of an abdominal aortic aneurysm, usually into the third part of the duodenum. Their management is discussed in Chapter 2. Most reported aortoenteric fistulae are of the secondary type which are nearly always associated with previous aortic graft insertion. These lesions have had a particularly poor prognosis in the past. This has been partly due to delayed recognition, surgery being undertaken only when exsanguinating haemorrhage has supervened. A second unfavourable factor has been continuing aortic sepsis with stump blowout, refistulization or multi-system failure[67–69].

The most frequent site of involvement is the distal duodenum, though cases involving the small bowel, colon, or stomach have also been reported. Three main macroscopic varieties of fistula may be recognized. In the first type there is a direct communication between the aortic suture line and the gut (Figure **14.25a**). In the second type the gut communicates with a false aneurysm at the graft–aorta suture line (Figure **14.25b**). In the third variety, the so-called paraprosthetic fistula or graft–enteric erosion, the bowel opens onto the graft surface and the suture line is not initially involved (Figure **14.25c**). Although the exact sequence of events leading to fistulization

is often unclear, mechanical factors and infection may each play a role and are often combined[53].

The majority of patients with aortoenteric fistula present with gastrointestinal haemorrhage. This may resemble any of the other major causes of bleeding but when this occurs in a patient who has had previous aortic surgery, it should be attributed to an aortoenteric fistula until proved otherwise. The time interval between aortic surgery and the onset of gastrointestinal bleeding is very variable. Cases have been reported as early as the first postoperative week while in other patients the interval may be as long as 15 years. The most common pattern of bleeding is intermittent low volume melaena which may occur for several weeks or months prior to admission. Haematemesis or rectal bleeding are alternative presentations. A number of patients exhibit 'herald bleeding' which is characterised by an initial brisk episode usually with haematemesis and hypotension. This ceases spontaneously but is then followed hours or days later by more massive haemorrhage. The fact that exsanguinating haemorrhage is rarely the initial presenting feature means that in virtually all cases it should be possible to intervene surgically while the patient is still stable.

Many patients with aortoenteric fistula have additional abdominal or back pain and fever, and these features may be present for some time before the onset of gastrointestinal haemorrhage. Indeed, in patients with paraprosthetic fistulae septic features predominate with recurrent chills, metastatic abscesses, anaemia and guiac-positive stools but no overt gastrointestinal bleeding[70].

Investigation of patients who present with an acute bleed should be completed within hours of admission because of the possibility of imminent severe haemorrhage. Blood should be drawn for cross-matching and intravenous lines inserted in readiness for immediate surgery. Blood cultures are frequently positive in patients with aortoenteric fistulae[69] and once blood has been obtained for this purpose, high-dose systemic antibiotics should be commenced. The most useful investigation is endoscopy and this should be undertaken in the operating room in case serious bleeding

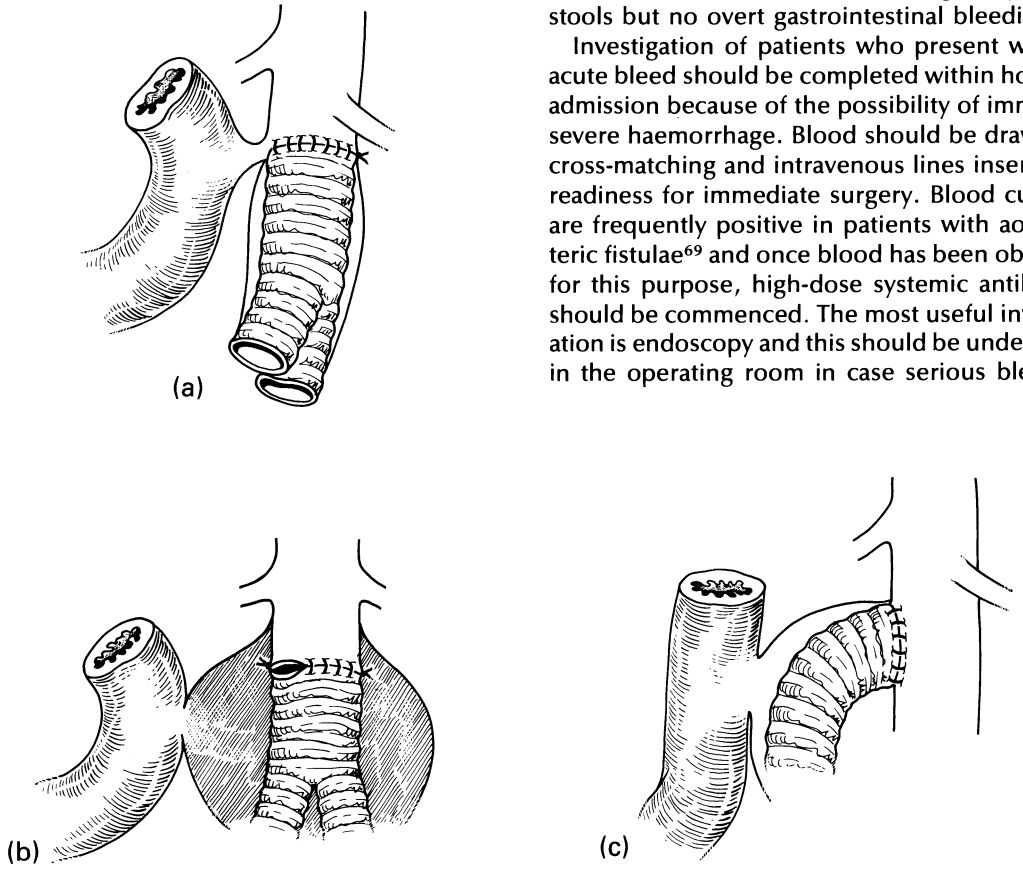


Figure 14.25(a–c) Aortoduodenal fistula: (a) direct type. (b) false aneurysm with secondary rupture into the duodenum. (c) paraprosthetic fistula

ensues. Although it is rare to visualize the graft through the bowel wall, bleeding or punctate ulceration in the distal duodenum is good evidence of an aortoenteric fistula, particularly if the rest of the examination is normal. Peptic ulcer, varices etc. of course may coexist but unless these lesions are actively bleeding they should be discounted. Colonoscopy may be useful in the small group of patients who present with rectal haemorrhage.

If time permits, a CT scan should be obtained and may show air in the aorta or periprosthetic tissues or other features denoting graft sepsis (see above). Arteriography may aid surgical planning particularly where there is distal occlusive disease and may occasionally contribute to the diagnosis by demonstrating a contrast leak or anterior bowing of the prosthetic stem[70]. Other studies such as barium roentgenography have had a low diagnostic yield in most reports and are not generally worth pursuing. In some cases it may be impossible to establish the existence of a fistula preoperatively. Rather than embark on another round of possibly fruitless investigations it is safer to explore the patient forthwith. Although the diagnosis may occasionally prove to be something other than aortoenteric fistula, this policy is preferable to overlooking a fistula and allowing massive haemorrhage to ensue.

Operative management

Where the diagnosis has been established preoperatively and the patient is not actively bleeding, an axillofemoral (popliteal) bypass should be inserted as the first stage. If the fistula complicates an aortofemoral prosthesis, it is safer to assume that the groins are infected and route the extra-anatomic bypass accordingly.

After closing and sealing all the incisions the abdomen is opened and the region of the fistula exposed. Proximal aortic control is obtained immediately below the renal arteries, though if there is considerable scarring or a false aneurysm, supracoeliac clamping may be required. The prosthesis is then similarly clamped and the duodenum detached by sharp dissection. Any backbleeding from the fistula site can usually be controlled by digital compression. In some cases the fistula opening is quite posterior and the diagnosis is only established after fully mobilizing the duodenal loop off the prosthesis.

Enteric repair is undertaken first in order to avoid further contamination. A direct transverse closure may be possible but in paraprosthetic fistulae in particular there may be more extensive damage necessitating bowel resection. The bowel may then be reanastomosed in end-to-end fashion, though if the defect is too great the ends should be closed, and continuity restored by end-to-side attachment of the proximal jejunum to the second part of the duodenum (Figure **14.26a, b**).

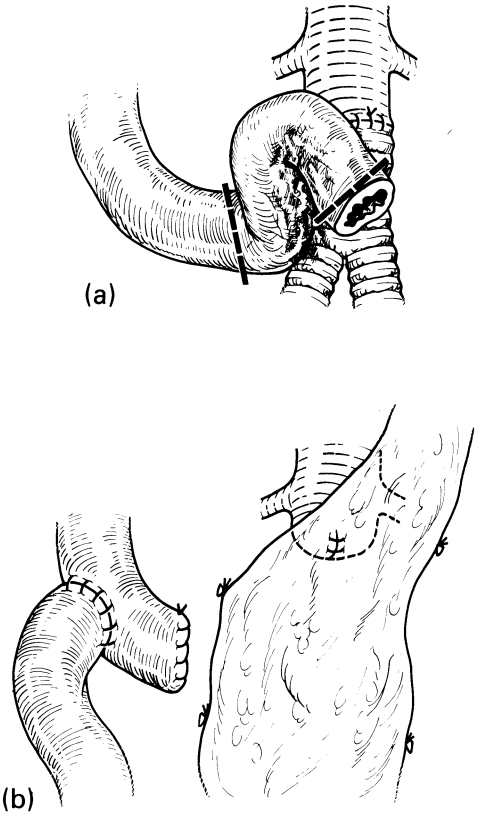


Figure 14.26(a, b) Paraprosthetic fistula with extensive duodenal destruction: (a) pathology and limits of bowel resection. (b) the aortic prosthesis has been removed and gastrointestinal continuity restored by end-to-side jejunoduodenal anastomosis

The prosthesis should now be removed and the proximal aorta oversewn as previously described. An omental pedicle is brought down over the graft bed and the abdomen is closed with drainage.

In the presence of active haemorrhage or when the diagnosis is uncertain, the above order of events should be reversed, with laparotomy as the opening move. If the patient is hypotensive, immediate aortic control should be obtained either by supracoeliac clamping or manual compression at the hiatus while blood volume is rapidly restored. Once the patient is stable the infrarenal dissection may be pursued and the clamp position readjusted as appropriate. Fistula closure and graft excision may then follow the lines already indicated. The abdomen is closed and an extra-anatomic bypass then inserted after reprepping, draping and a change of gown, gloves and instruments.

Two other therapeutic options are available when laparotomy is undertaken as the first stage in management. The first involves *in situ* reconstruction of the aortoiliofemoral segment by autogenous methods once the bowel has been repaired and the aortic graft has been removed (see above). The other possibility which may be considered is enteric repair followed by anatomic regraft (Figure **14.27**) or, in the case of paraprosthetic fistulae, bowel repair alone leaving the aortic graft in place[71]. These methods are suitable only in the absence of gross sepsis, and providing the colon is not involved. On completion an omental pedicle should be brought down over the aorta and graft. Local graft irrigation may be used postoperatively along with high dose systemic antibiotics. Although a small number of successes have been reported with this approach, there is a significant incidence of continuing aortic sepsis and refistulization and complete removal of the aortic graft is a safer option[60,67]. However, in patients severely obtunded by haemorrhage or poor risk cases with limited distal run-off, these compromise solutions may be worth considering.

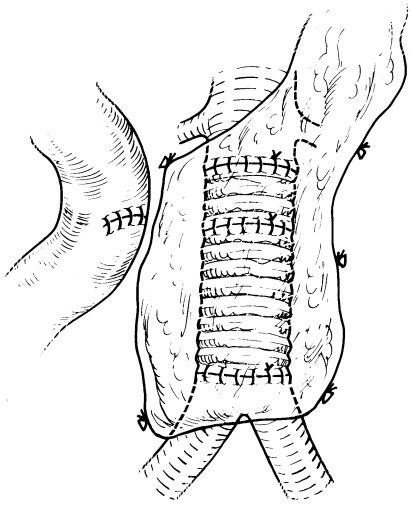


Figure 14.27 Aortoduodenal fistula in a poor risk case. The proximal portion of the original aortic graft has been replaced *in situ* with a new prosthetic segment. The bowel has been repaired and omentum interposed

PREVENTION OF INFECTION IN AORTIC SURGERY

Prevention of infection is obviously of prime importance in view of the difficulties in treating the established case.

Preoperatively, specific attention should be given to treating infected lesions on the feet or intercurrent sepsis elsewhere and to improving the patient's general condition. Transfemoral arteriography may predispose to groin wound complications and it may be advisable to defer elective surgery for at least 10–14 days after this procedure[72]. Antibiotics active against staphylococci and Gram-negative organisms including anaerobes are given as a routine. These should be commenced premedication and continued until all lines and catheters have been removed.

In the operating room strict asepsis must be observed throughout. Thorough skin prepping and careful draping are essential. In addition, drapes should always be kept dry to avoid contamination by capillary action. It is particularly important to prevent the graft from coming into contact with exposed skin, and plastic wound drapes are helpful in this respect. However, the single most important measure in preventing graft infection is a meticulous surgical technique. Groin exposures should be made using a lateral incision to avoid cutting into lymphoadipose tissue. If it is necessary to divide any lymphatic

tissue, this should be sutured or ligated. Similar care should be taken with haemostasis, particularly when re-operating on a fresh wound since a haematoma around the graft invites infection. Antibiotic– or antiseptic–soaked sponges should be placed in the groin wounds when the latter are not in use. On completion the femoral wound should be carefully closed in layers, with suction drainage if necessary. If soft tissue cover is deficient or wound complications are anticipated, a sartorius flap may be used to cover the graft.

Attention to postoperative groin wound complications is also important. An obvious haematoma should be evacuated in the operating room and the incision reapproximated in layers. A postoperative lymphocele may sometimes be managed by repeat aspiration. If this is unsuccessful, or if there is persistent lymphorrhoea, the safest course is to reopen the wound and suture–ligate any obvious lymphatics. A suction drain is then inserted at a distance and the incision reclosed in layers. Minor skin necrosis may be treated by débridement and local wound care. More extensive dehiscence may sometimes be amenable to direct resuture, though there is often a soft tissue deficiency which may necessitate a rotation or advancement flap[56].

At the aortic end great care should be taken to avoid even minor trauma to the duodenum. If the duodenum is adherent to the wall of an aneurysm it is best left undisturbed and the graft inlaid via a more lateral aortotomy. Large lymphatics are often encountered in the retroperitoneum, particularly in patients requiring emergency laparotomy, and these should be ligated to avoid a postoperative accumulation of chyle. Where an end-to-end aortic anastomosis has been performed, the suture line may be reinforced with an external Dacron cuff. This in itself will not prevent infection but will aid haemostasis and may prevent subsequent false aneurysm formation. Most importantly, an adequate amount of soft tissue should always be interposed between the duodenum and the aortic suture line. In aneurysm cases a covering flap may be created from redundant sac. In addition, the duodenum should be distanced from the suture line by sewing the left peritoneal edge to the periaortic tissue on the right side of the aorta and graft[73]. Where the prosthesis and suture line cannot be effectively covered, liberal use should be made of an omental wrap. This is particularly important in re-do cases.

All patients with arterial prostheses are at risk of graft colonization during incidental bacteraemia. This risk may be particularly great in the early postoperative months when graft incorporation may be deficient. Antibiotic cover should therefore be provided as a routine for events such as dental extraction, urinary catheterization, barium enema, proctocolonoscopy and repeat arteriography.

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15

Percutaneous Angioplasty

Percutaneous transluminal angioplasty, first described by Dotter and Judkins in 1964[1] using coaxial catheters, has gained more widespread acceptance since the development by Gruntzig of a double-lumen catheter with a cylindrical polyvinyl balloon[2] (Figure **15.1**). This non-elastic material allows high pressures to be exerted on the lesion without risk of overdistension of the balloon above a predetermined diameter. The balloon catheter considerably improved the results and lowered the complication rate of peripheral angioplasty, and resulted in extension of the method to other areas, notably the coronary and renal arteries. Many other vessels are technically amenable, using a combination of selective catheterization and angioplasty techniques.

Though angioplasty is only useful in selected cases, usually those with relatively localized disease, it has several obvious advantages: morbidity and cost are relatively low, and the length of inpatient stay and rehabilitation time are markedly reduced compared with surgery. In the event of failure, the chances of successful surgery are very seldom compromised. Long-term patency figures are as yet unknown, but initial good patency rates are being maintained up to 3 years[3,4] and there are similar 5-year figures available from early workers[5]; in any event recurrences are usually amenable to repeat angioplasty.

The mechanism of angioplasty has been extensively studied in animals and cadavers. There is some impaction and longitudinal extrusion of soft atheromatous material, but the main events in balloon dilatation appear to be endothelial desquamation and the formation of one or more intimal clefts extending into the plaque material. Stretching of the media with consequent increase in diameter of the artery as a whole is another factor in successful dilatation[6]. The intimal clefts are often visible angiographically as an extraluminal track simulating a dissection. Various degrees of lumen distortion and irregularity are also often present. Over the ensuing weeks healing occurs with the formation of a fibrous neo-intima; retraction of the atheroma between the clefts may result in further lumen enlargement.

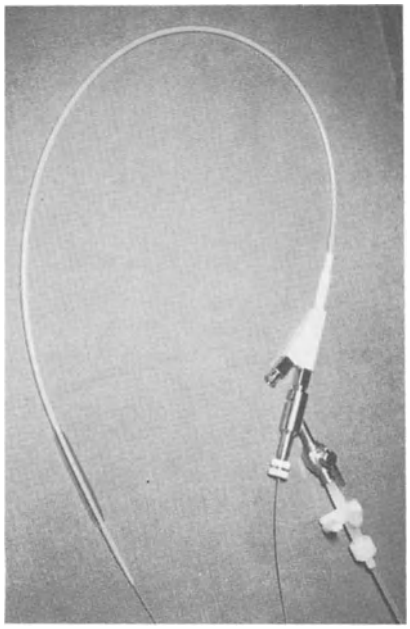


Figure 15.1 Double-lumen balloon catheter for peripheral angioplasty, showing Y-connector which allows pressure measurement, etc, through the catheter tip with the guide wire in place. The oblique (open) port is for the balloon lumen

PERIPHERAL ANGIOPLASTY

INDICATIONS

Angioplasty sometimes gives rise to complications requiring surgery (see below), therefore in general it should be applied only in patients whose symptoms warrant surgery. However, with careful technique the risks associated with dilating a stenosis are very small; patients with claudication may then be justifiably regarded as candidates for angioplasty even though surgery might not be contemplated.

The possible prophylactic role of angioplasty when a stenosis is found in a patient with little or no relevant symptoms is debatable: dilatation of a vein graft or anastomotic stenosis would certainly be indicated to prevent progression to occlusion, but the advisability of such intervention in native vessel disease is not yet established.

Angioplasty can be used in conjunction with surgery, in addition to its use in the treatment of stenoses associated with bypass grafts. For example, dilatation of an iliac stenosis can be performed prior to femorofemoral grafting for an iliac occlusion on the opposite side, or ipsilateral femoropopliteal bypass. Run-off can be improved perioperatively by dilatation of femoral stenoses in an iliac reconstructive procedure, or tibial stenoses during femoropopliteal bypass.

If there is threatened limb loss in a patient with surgical risk factors, angioplasty may be attempted even if the disease distribution is not ideal.

The ideal lesion is a short isolated stenosis of the iliac, femoral or popliteal artery. Multiple discrete stenoses are associated with equivalent results, but recurrence appears more likely in very long lesions, i.e. when the vessel is diffusely diseased.

With careful technique, complete occlusions can be recanalized, but angioplasty should generally be limited to relatively short lesions: iliac occlusions greater than 6 cm are associated with a significant risk of embolization[7], and femoral occlusions greater than 10 cm long tend to recur[2]. Occlusions less than 2 months old should not be attempted because of the higher risk of embolization before organization has taken place: local fibrinolytic therapy can be offered for these lesions (see below).

Atheromatous stenosis of the abdominal aorta is readily amenable to angioplasty: to obtain sufficient luminal diameter, two balloons are usually inflated alongside each other. Relatively localised lesions of the tibial arteries can be dilated, usually in conjunction with angioplasty of proximal disease, but careful technique is necessary to avoid damage.

COMPLICATIONS

As angioplasty is performed in arteriopaths and significant stenoses or occlusions are intentionally traversed, inevitably complications sometimes occur. However, with rigid adherence to the techniques to be described these can be minimized. Haematomas at the puncture site are usually self-limiting, but occasionally need surgical intervention. Other complications of surgical significance include acute thrombosis at the puncture site or at the lesion, though this may be amenable to local fibrinolytic therapy. Clinically significant embolism is, perhaps surprisingly, an infrequent occurrence, being more likely in the recanalization of occlusions rather than stenosis dilatation. Pseudo-aneurysm formation and vessel perforation are very rare. Overall, the incidence of complications requiring surgery, in experienced centres, is 1-2%[8]. Though this author firmly believes that angioplasty should only be performed by those with considerable experience in percutaneous catheterization, clearly close surgical cooperation is essential for complication management, as well as patient selection.

TECHNIQUE

One effect of balloon dilatation on the arterial wall is to produce a roughened inner surface, which is liable to platelet aggregation and thrombosis. Platelet inhibitors or anticoagulants should therefore be used. Most workers use antiplatelet drugs, either aspirin alone or in combination with dipyridamole, for 2 days before and for 5–6 months after angioplasty. Also, 'conventional' ionic radiographic contrast media are hyperosmolar by a factor of five to eight: they have been

shown to cause endothelial damage *in vitro*, and they have been incriminated in accelerating the atheromatous process and in inducing thrombosis which is sometimes seen after angiography[9]. The denuded endothelial surface of the vessel wall immediately after angioplasty is probably particularly vulnerable to such osmotic damage. The use of one of the new lower osmolality contrast media (for example iohexol, ioxaglate or iopamidol) is therefore recommended during angioplasty.

Catheter introduction

Iliac dilatation (Figures **15.2** and **15.3**) is best performed by a retrograde ipsilateral route, even if the femoral pulse is absent or severely diminished, as there is less departure from standard angiographic technique and more direct control of the guide wire and catheter course is possible. Aids to femoral artery puncture include a needle with ultrasound flow probe, visualization of the

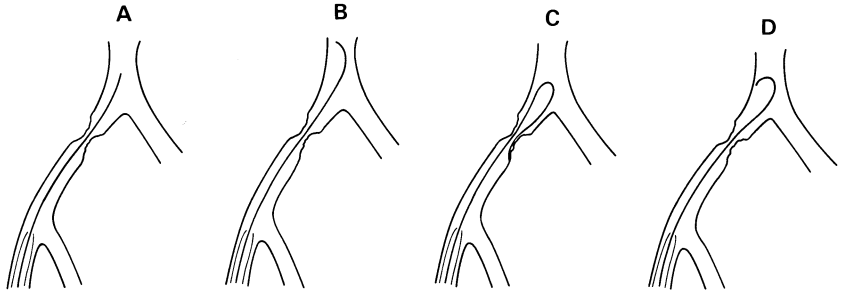


Figure 15.2(a–d) Various guide wire configurations used to traverse an iliac stenosis: (a) straight tip. (b) large (e.g. 15 mm radius) J curve acting as eccentric tip. (c) loop formed when tip abuts on artery wall. (d) small (e.g. 3 mm radius) J curve. (From *Clinical Radiology*[8] with permission)

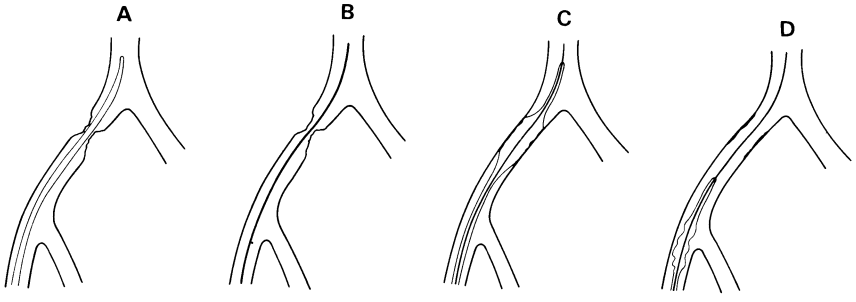


Figure 15.3(a–d) Stages in iliac dilatation: (a) 'pre-dilatation' with tapered catheter. (b) exchange guide wire for insertion of balloon catheter. (c) balloon inflation. (d) balloon collapsed, catheter withdrawn to check distal pressure with fine wire kept beyond lesion. (From *Clinical Radiology*[8], with permission)

artery on screening by injection through a catheter introduced from the opposite side, or knowledge of the relationship of the vessel to bony landmarks from previous angiograms. In practice the artery can almost always be found by trial and error, as its position in the groin is always approximately known. Abolition of the trans-stenotic pressure gradient is the main criterion of success in iliac dilatation, the angiographic appearance being of secondary importance, therefore the initial post-stenotic arterial pressure should be measured before the lesion is crossed.

For femoral/popliteal angioplasty, a high antegrade common femoral puncture is often used, though with practice selective puncture of the superficial femoral artery is usually possible, and this lessens the risk of haematoma. The puncture is aided in obese patients by a pillow placed beneath the buttocks. If the common femoral artery is punctured, the guide wire tends to enter the profunda. Passage into the superficial femoral artery is aided by altering the needle angulation in a horizontal or lateral/medial direction, or using a guide wire with a medium curve J-tip (see below). A curved catheter can be used as a last resort. When the superficial femoral artery has been entered with a guide wire and a catheter is introduced over it, there may be buckling in the groin as the catheter is introduced or advanced, particularly in obese patients. Placing a pillow beneath the buttocks and having an assistant hold the wire straight and taut usually help. Alternatively a special rigid 'Lundqvist' wire can be used to stiffen the catheter, but great caution is required, and its use is not recommended for traversing the lesion.

As pressure measurement is unreliable in femoral angioplasty, often the only criterion of technical success at the time of the procedure is angiographically assessed lumen improvement or flow improvement on screening.

In both iliac and femoral/popliteal angioplasty, 7 French catheters and 0.035 inch diameter guide wires are generally used.

Traversing the lesion and dilatation

The critical part of the angioplasty procedure is to cross the lesion intraluminally; once this is achieved, a successful result is almost assured.

The lesion is first crossed with a guide wire to lessen the risk of subintimal passage. The wires are available with either a straight tip or with a terminal 'J' curve, ranging in radius from 1·5 to 15 mm. The last few centimetres of the wire are made without an inner core to give more flexibility, thus aiding atraumatic passage and allowing bends to be negotiated; moveable-core wires can be used to alter the length of the flexible segment if desired.

The tip configuration used to negotiate a lesion varies: in stenoses a straight wire usually succeeds, but eccentric stenoses may require a large J curve, for example 15 mm radius acting as an eccentric tip, the direction of which can be altered by rotation (Figure **15.2**). Rarely a small J curve is required. The use of force is neither necessary nor desirable. If much resistance is encountered, the wire is passing intramurally. If a suitably floppy tip is used, sometimes the tip of the wire engages the arterial wall but does not pass subintimally when the wire is advanced further; instead a loop forms, which crosses the lesion successfully and eventually the tip of the wire disengages and follows the loop through (Figure **15.2**). When the wire has crossed the stenosis and free advancement beyond is possible, indicating an intraluminal course, a catheter is introduced over the guide wire to 'pre-dilate' the lesion (Figure **15.3**). A smooth, straight catheter with a long tapered tip is recommended to minimize the risk of embolization.

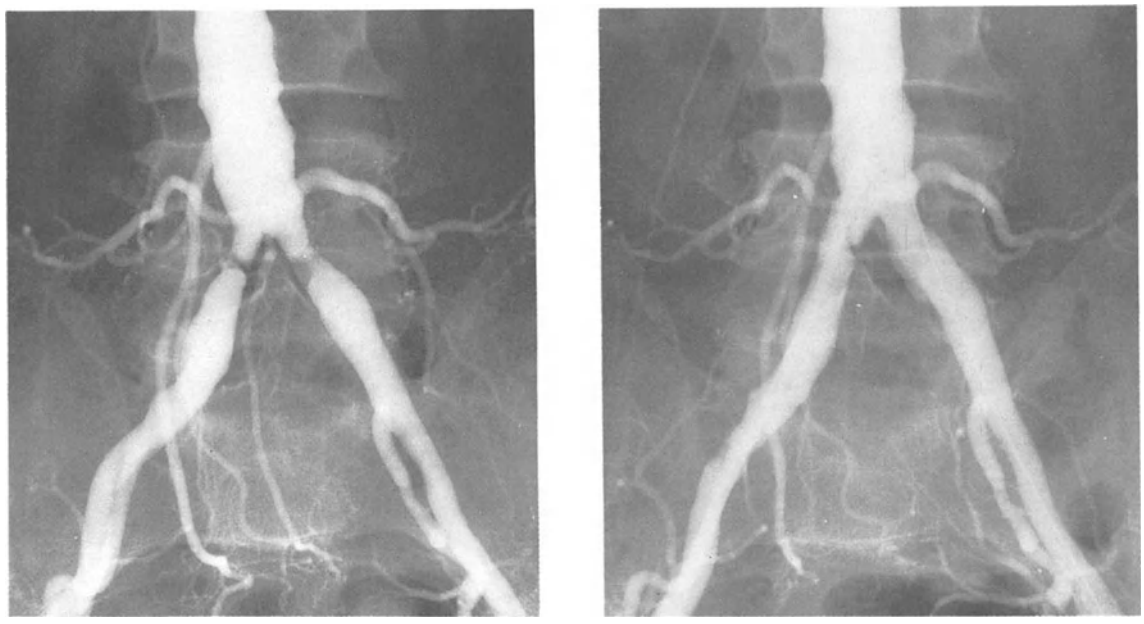
The tip configuration needed to traverse an occlusion is more difficult to predict, and personal preferences abound. This author uses a straight guide wire first, as for stenoses. Slight resistance is acceptable in occlusions therefore gentle forward pressure may be needed, but if any greater resistance is encountered the wire is withdrawn a few millimetres and held in place while the catheter is advanced over it until it is near the resistance point; the guide wire is then gently re-advanced. Most occlusions can be traversed in this step-wise manner. Sometimes a small J wire is needed to initially engage the occlusion, and occasionally different configurations, for example eccentric tips, are needed within the lesion. Towards the distal end of the occluded segment, withdrawal of the wire and

contrast injection via the catheter will often reveal a narrow irregular channel communicating with the patent lumen beyond. This is then easy to negotiate with a guide wire.

If the vessel wall is inadvertently entered, failure is likely as any other guide wire will probably follow the false track. In stenoses, this may cause acute occlusion, but in already established occlusions this is most unlikely to result in a complication unless the false track is continued distally by the use of force, in which case patency of the distal vessels may be compromised.

When the pre-dilating catheter has traversed the lesion, the guide wire is removed and an intraluminal position confirmed by observing backbleeding and injecting contrast medium. At this stage, 5,000 units of heparin are usually given to inhibit thrombosis. In femoral/popliteal dilatation the tibial arteries tend to go into spasm if they are instrumented; this tendency can be minimised by the intra-arterial injection of an anti-spasmodic, for example 10 mg tolazoline. The guide wire is repassed and the pre-dilating catheter is exchanged over this for the chosen balloon catheter; the guide wire must be kept beyond the lesion throughout. The balloon diameter is chosen by reference to the angiogram, predicting the desired lumen size from adjacent normal vessels wherever possible. In practice, 6 to 8 mm diameters are usually used for iliac lesions, 4 or 5 mm for the femoral/popliteal arteries, and 3 mm for tibial dilatation. The optimum balloon length is 4 cm, even for short lesions, to allow a stable position during inflation. In long lesions sequential overlapping balloon positions are used; longer balloons, for example 8 to 10 cm, are useful for these.

The fully deflated balloon will traverse the puncture site without the need for a sheath; passage is aided by folding it in a spiral fashion and rotating the catheter as it is introduced. Suction (with a syringe) should be applied whenever the catheter is moved, but especially when traversing the puncture site and the lesion itself. When the balloon catheter has crossed the lesion, the guide wire is exchanged for one of 0.018 inch diameter. This fine wire is kept beyond the lesion throughout the procedure, so that when the catheter is



(a) (b)

Figure 15.4(a, b) (a) Bilateral common iliac stenoses in 62 year old female with 3 year history of claudication at 50 yards. (b) After angioplasty. Intra-arterial gradients completely abolished. Complete symptom relief

withdrawn from the lesion to assess the result of the dilatation, an intraluminal route is assured if the lesion has to be recrossed for further dilatation (Figure **15.3**); otherwise there is a tendency to enter the arterial wall at the dilatation site, thus potentially ruining the procedure. The guide wire has to be of very small diameter to allow pressure measurement and contrast injection through the catheter tip. At the operator end of the catheter a Y-connector, with adjustable seal to prevent back-bleeding, is used (Figure **15.1**).

The balloon is inflated with a 1:1 contrast medium/saline mixture as a compromise between radiographic visibility and viscosity. Pressures of about 6–8 atmospheres are used and these are relatively easily achieved by hand with a 10 cc syringe. A simple in-line monitor is advisable to monitor the inflation pressure: pressures above 8 atmospheres are rarely necessary in peripheral angioplasty even if the balloon can withstand them. The optimum inflation time is not known – about 30-60 seconds is common practice. Often several inflations are given. The stenosis may initially indent the balloon: this is useful to confirm that the balloon is correctly positioned, but disappearance of the indentation on further inflation is of limited value in assessing progress. In occlusions, the site of an underlying stenosis may be detected by this initial indentation. Particular attention should be paid to dilating this area to reduce the chance of reocclusion.

Two balloons inflated alongside each other, inserted via each groin, are often needed in dilatation of an aortic stenosis because of the relatively large vessel diameter. A double balloon technique is also needed where a lesion involves the orifice of an adjacent large vessel, for example where an iliac lesion encroaches on the aortic bifurcation or a proximal superficial femoral lesion involves the profunda origin. The first balloon is then used to dilate the lesion and the second is placed in the lumen of the adjacent vessel to prevent it becoming occluded during the dilatation.

If balloon rupture occurs it is usually without consequence as the balloons are filled with incompressible fluid and an explosive effect does not occur. There is usually little difficulty removing them percutaneously as they are designed to rupture longitudinally; if difficulty is experienced an arteriotomy may still be avoided by the use of a large sheath.

If the balloon catheter has to be exchanged, the fine wire must be changed for a standard gauge wire (advancing the catheter beyond the lesion first) to guide the new catheter through the puncture site.

Contralateral (crossover) angioplasty

If angioplasty from an ipsilateral puncture fails or is precluded by proximity of the lesion to the groin or because of postoperative scarring, an approach from the opposite groin can be used. This method is also useful if there is a catheter already in place on one side, for example for initial diagnostic angiography; angioplasty of a vessel on the opposite side can then be performed without the need for a second arterial puncture. However, the crossover technique does not give the same direct control of the wires and catheters afforded by the ipsilateral route: transmitted sensation and manipulation are both diminished. It is therefore generally best reserved for stenosis dilatation only; occlusions are unlikely to be successfully recanalized. Advancement of the catheter through the stenosis and subsequent exchange to the balloon catheter can present some difficulties especially if the aortic bifurcation has an acute angle, but these should be surmountable by the following technique:

The contralateral iliac artery is first catheterized with a 'side-winder' catheter, the loop being formed by advancing the catheter to the aortic arch and rotating in a clockwise direction. Withdrawal to the abdominal aorta and appropriate direction of the tip will result in the opposite iliac artery being engaged; further withdrawal causes the tip to advance distally. The lesion is then crossed with a guide wire, 'pre-dilated' with the catheter and exchange made for the balloon catheter as previously described. As the initial catheter is advanced through the stenosis, and as the balloon catheter is being introduced, an assistant should hold the guide wire completely still and taut; this reduces any tendency for the catheter or guide wire to form a loop in the aorta.

Lower limb angioplasty from the axillary route

This alternative can be tried if a groin approach fails or is not feasible. Local complications are more common than with femoral catheterization, and manipulation of the wires and catheters may be more difficult due to the arterial angulations and distance from the puncture site; these factors should be borne in mind before undertaking an axillary approach.

Examples of peripheral angioplasty are shown in Figures **15.4–15.7**

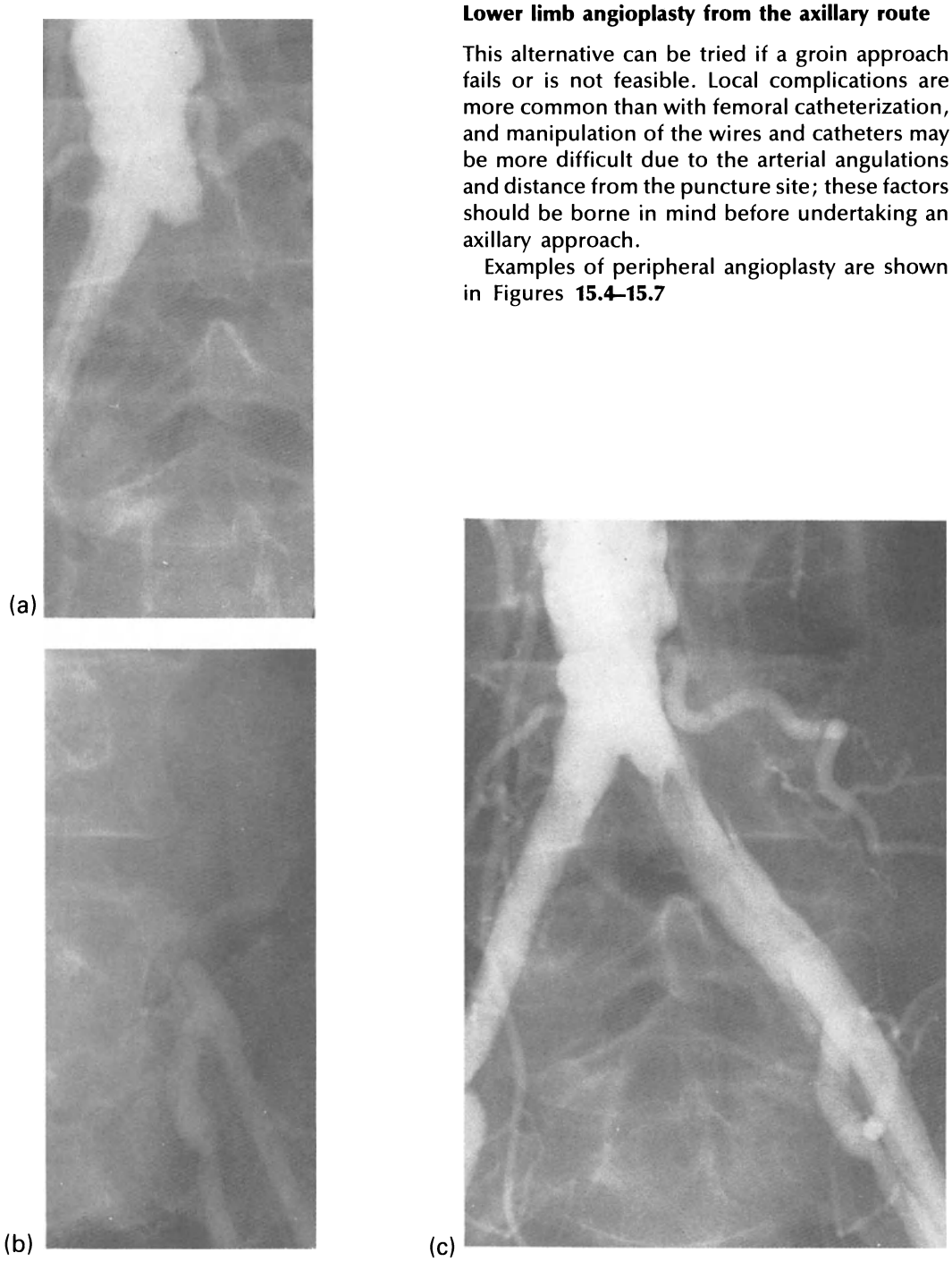
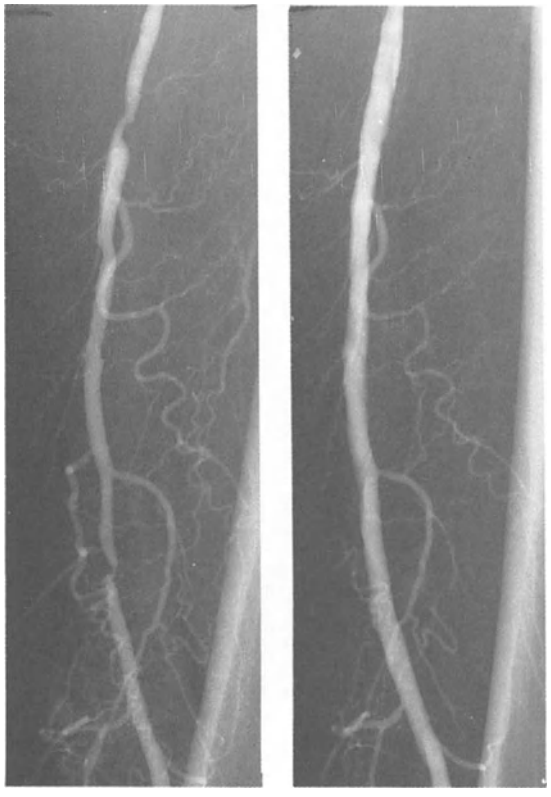


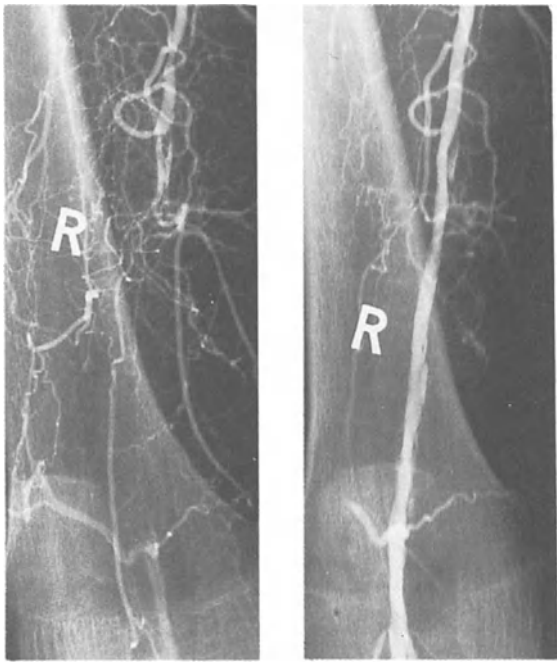
Figure 15.5(a–c) (a, b) Left common iliac occlusion in 58 year old female with severe claudication and night pain. (c) After angioplasty. Complete abolition of 70 mmHg gradient. Complete symptom relief



(a) (b)

Figure 15.6(a, b) (a) Superficial femoral stenoses in 78 year old diabetic with ischaemic calf and foot. (b) After angioplasty. Complete reversal of ischaemia

Figure 15.7(a, b) (a) Short femoral occlusion in 53 year old male with 3 year history of severe claudication. (b) After angioplasty. Complete symptom relief



(a) (b)

ANGIOPLASTY OF VESSELS REQUIRING SELECTIVE CATHETERIZATION

The two commonest areas in this category are the coronary and renal arteries. The former is outside the scope of this chapter.

RENAL ANGIOPLASTY

The usual indication for renal angioplasty is renovascular hypertension. A renovascular cause is present in only a small proportion of hypertensive patients and hitherto there has been no reliable, relatively non-invasive way of detecting it. Excretion urography, isotope renography and systemic venous renin estimation have reported false negative rates of 33%, 30% and 44% respectively[10]. Digital subtraction angiography, in which good visualization of the abdominal aorta and its branches is now possible by intravenously administered contrast medium, should prove to be a reliable way of demonstrating renal arterial lesions (see Chapter 13).

Once a stenosis is found it may be regarded as haemodynamically significant if the diameter reduction is greater than 50%, corresponding to a cross-sectional area reduction of 75%. The presence of collateral vessels would also indicate a significant lesion, but these are seldom seen except in complete occlusions. An isotope renogram, if positive, would be useful confirmatory evidence, but as previously mentioned a normal result does not exclude significant flow restriction.

The relevance of a lesion to the hypertension may be determined by selective renal vein renin estimation, split renal function studies or the detection of angiotension II dependency by saralasin infusion[10], but these tests are not uniformly reliable and such exhaustive investigation may not be justified. There is a case for carrying out angioplasty whenever a suitable, significant lesion is found with the appropriate clinical indication, usually poorly controlled hypertension on multiple drug therapy or hypertension in a young patient or child.

Angioplasty is also being increasingly used to preserve renal function, for example in bilateral disease or in stenosis of the artery to the only functioning kidney.

From a technical point of view, most renal artery stenoses are amenable to angioplasty. This applies to atheroma or fibromuscular dysplasia, though the clinical results are significantly better with fibromuscular dysplasia, and the recurrence rate is less[11]. Branch stenosis due to fibromuscular dysplasia is also usually accessible. One relatively unfavourable situation is when the lesion is primary aortic atheroma encroaching on the renal artery orifice rather than juxta-ostial stenosis: in these cases dilatation tends to displace the atheroma for a short time only, though the use of a large balloon may overcome this problem. Successful recanalization of complete renal artery occlusions has occasionally been reported[12], as has local streptokinase infusion in renal artery thromboembolism[13].

Technique

Successful angioplasty for hypertension may result in rapid reduction in blood pressure; there may be hypotension in the hours following the procedure, so that short acting anti-hypertensive drugs only should be used beforehand.

There are two main methods of performing renal angioplasty: the first, the *exchange wire method*, is similar in principle and practice to peripheral angioplasty. The vessel is first catheterized with a 7F pre-shaped selective catheter. The stenosis is crossed with a guide wire and then 'pre-dilated' with the catheter. Using a long (e.g. 200 cm) guide wire with its tip in a distal renal artery branch, exchange is made for the chosen balloon catheter. If the main vessel is very caudally directed, initial stenosis passage and/or catheter exchange may be difficult; an assistant holding the guide wire taut may help. If not, the second method of renal angioplasty, the *co-axial method*, may be used. This method may also be useful for very severe stenoses which cannot be crossed by the catheters used in the exchange method.

The co-axial method (Figure **15.8**) uses a 8F preshaped non-tapered catheter, introduced via a percutaneous sheath. This 'guiding catheter' is used to catheterize the renal artery. The factors governing choice of catheter shape are similar

to those in diagnostic catheterization, bearing in mind that the catheter tip should remain proximal to the stenosis when the vessel is entered. A 4F balloon catheter, with a very fine guide wire within it, is then introduced through the guiding catheter. Once the guide wire has been manoeuvred across the stenosis and into a distal renal artery branch, the balloon catheter is advanced through the stenosis. The arterial pressure is monitored simultaneously at the tips of both catheters so that continuous measurement of pre- and post-stenotic pressures is possible. The arrangement of the catheters and pressure connections is shown in Figure **15.8**.

As in peripheral angioplasty, several balloon inflations up to about 8 atmospheres may be needed to achieve the desired result. Pressure gradient abolition is a reliable criterion of success and is usually achieved with the co-axial system (Figures **15.8, 9**). In the exchange wire method pressure measurement may be unreliable because of the larger 7F catheter and its collapsed balloon profile partially obstructing the lumen.

Post-dilatation angiography should generally be performed by a mid-stream aortic injection to avoid the risk of re-crossing the dilated area with a selective catheter, unless the lesion is a safe distance from the renal ostium (Figure **15.9**).

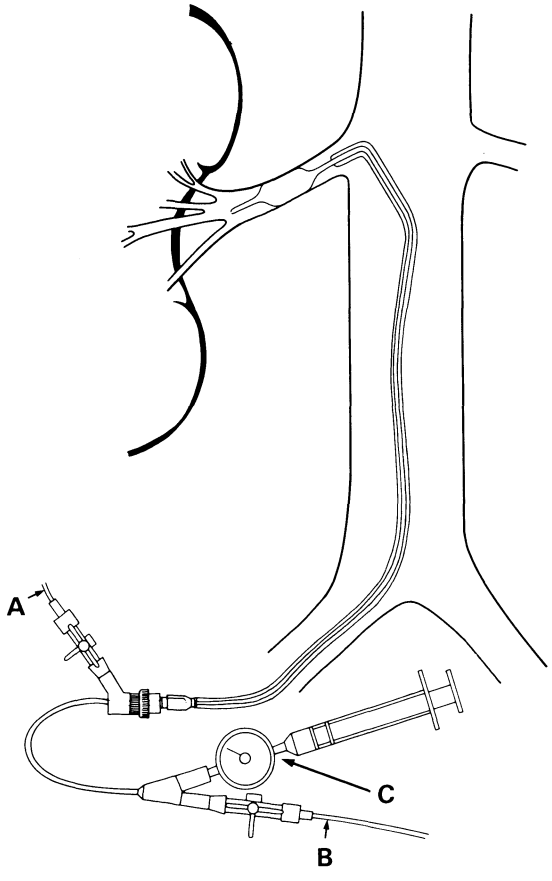


Figure 15.8 Co-axial method for renal angioplasty. Measurement of the pressure at the guiding catheter tip (pre-stenotic pressure) is by connection A, and that at the balloon catheter tip (post-stenotic pressure) is via connection B. The balloon is inflated by a hand-held syringe with an in-line pressure gauge (C). (From *Clinical Radiology*[8], with permission)

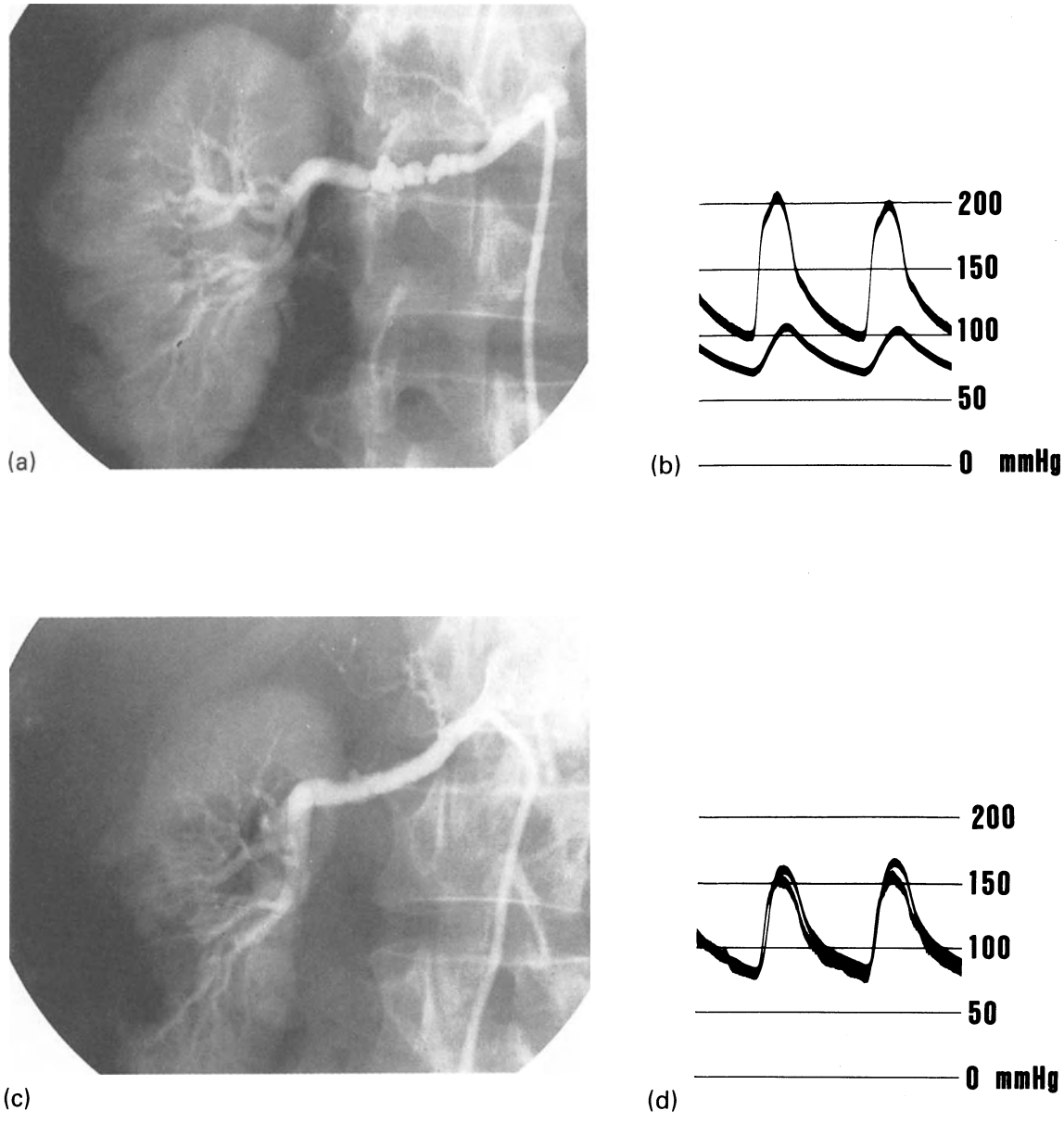


Figure 15.9(a–d) (a) Right renal artery stenosis in 38 year old female with 2 year history of severe, poorly controlled hypertension. (b) Pre- and post-stenotic pressure traces immediately after crossing the stenosis. (c) After angioplasty. Normotensive without any treatment from 2 hours after angioplasty. (d) Final pressure after dilatation

ANGIOPLASTY OF OTHER AREAS AND OF NON-ATHEROMATOUS LESIONS

Any vessel which can be selectively catheterized is technically amenable to angioplasty. Dilatation of the coeliac and superior mesenteric arteries is well described[8]; the caudal direction of these vessels may necessitate an axillary approach or co-axial method.

Angioplasty of the brachiocephalic vessels is being increasingly practised; this has been mainly concerned with proximal subclavian stenosis, but it is now evident that vertebral stenoses can also be safely dilated[14] (Figure **15.10**). The exchange wire method from a femoral approach, similar to renal angioplasty, is the method of choice. Percutaneous dilatation of internal carotid atheroma is only practised on a very limited basis because of the embologenic nature of the lesion.

Angioplasty of a wide variety of non-atheromatous lesions has been described[8]. As has been mentioned, renal fibromuscular dysplasia lends itself particularly well. Renal transplant artery stenosis is usually technically amenable, though the arterial angulations in internal iliac transplants may present some challenges: a contralateral femoral approach ('crossover' method) may be needed. Other suitable lesions include stenoses associated with venous bypass grafts and dialysis fistulae, and various arteriopathies such as Takayasu's disease or radiation-induced arteritis. Fibrous stenoses can be quite resistant to dilatation, and access to some of them may be more difficult because of postoperative scarring or distance from a suitable puncture site. However, if primary success is achieved, the results obtained appear to be equivalent to angioplasty of atherosclerotic disease.

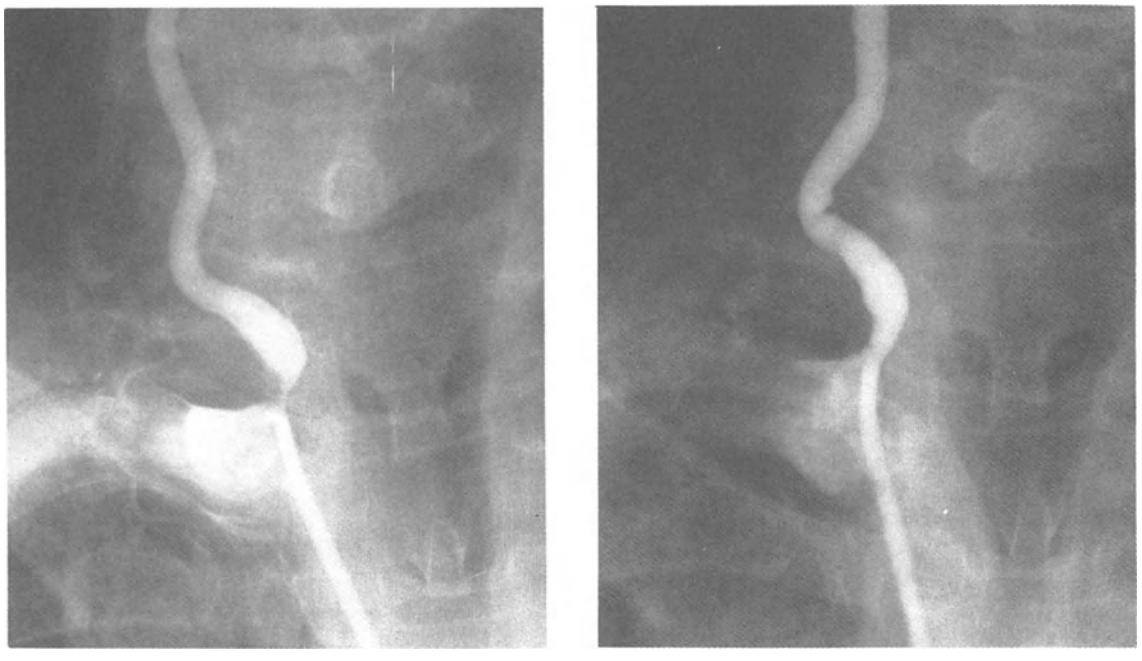


Figure 15.10(a, b) (a) Severe stenosis at origin of right vertebral artery in 61 year old male with disabling vertebrobasilar attacks for 1 year. (b) After angioplasty (femoral puncture, exchange wire method). Complete symptom relief

FIBRINOLYTIC THERAPY

Local low-dose administration of fibrinolytic agents is probably associated with less risk than systemic treatment, and increases the technical capability of angioplasty. Quoted success rates overall are about 75%[15–18], varying considerably with the type of case attempted. Lysis can almost always be acheived in acute thrombosis and, in this author's experience, in occlusions less than 3 weeks old. Some occlusions several months old can be treated, especially in the iliac arteries, but the technique does become more speculative with increasing age and length of lesion. Angioplasty complications such as acute post-procedure thrombosis or embolus after recanalisation of relatively fresh occlusions are amenable (Figure **15.11**), and some patients with occlusions too long and/or too recent for balloon angioplasty can be offered fibrionolytic therapy, especially if they are poor surgical candidates. The technique has a definite role in graft thrombosis[16], but there is increased risk of haemorrhagic complication if this is done within a few days of surgery[19] and extravasation is a well recognised problem with Dacron grafts, even when mature[18].

The literature so far has been mainly concerned with primary results, techniques and complications rather than patency, and it is difficult to glean meaningful figures from heterogeneous patient groups. If complete lysis can be achieved with correction of the underlying lesion, usually by balloon dilatation, patency rates appear to be reasonable and support the use of the technique in patients with little or no other therapeutic option.

The duration of infusion required to restore patency is unpredictable, therefore the method is not appropriate if there is critical ischaemia requiring immediate revascularization.

There is as yet no clear consensus of opinion on optimum dose schedules. As little as 5000 units of streptokinase per hour, sometimes for 24 to 48 hours, can be given, but there is now a general tendency to use a larger dose, for example 20,000 units per hour, to allow a shorter infusion time. There is also an 'accelerated' infusion time[15], in which 5000 to 10,000 units are given every few minutes. Though streptokinase is more widely used because of its greater availability and lower price than urokinase, the latter is favoured by some workers because it is non-antigenic and response to it may be more predictable than to streptokinase.

A catheter is advanced antegradely into the proximal part of the occlusion: contralateral catheterization is therefore needed for iliac or proximal femoral blocks. The angiographic status is checked periodically (every few hours in the lower dose schedules) and the catheter advanced if necessary. If some progress is not seen after 12 hours, eventual success is unlikely[17,18].

Contraindications to systemic fibrinolytic therapy include active bleeding, a pre-existing coagulation disorder, recent surgery or trauma, and a recent cerebrovascular accident. These probably also apply to local fibrinolytic therapy because a systemic lytic effect is often observed. Serious haemorrhagic complications are relatively unusual, provided the patient has not had surgery within the last few days[19], and provided the patient is carefully monitored, particularly if heparin is also given. Monitoring of the thrombin time and/or fibrinogen level has been recommended to detect a systemic effect, but the validity of these tests in predicting haemorrhagic complications has recently been questioned[19].

If significant puncture site bleeding occurs, the infusion should be stopped. Control should eventually be achieved by manual compression, though this may need to be quite prolonged. If this is not effective, fresh frozen plasma or even tranexamic acid may be needed.

Thrombosis may occur, paradoxically, on the catheter surface, especially if there is low flow due to persistent distal occlusion. This may cause retrograde thrombosis of the vessel or re-thrombosis of the recently recanalized segment. The risk may be lessened by a small catheter diameter (for example 3 or 5 French), a guide wire with a central lumen allowing infusion, by using an 'accelerated' regime of higher dose for shorter period[15], or by concomitant heparin infusion.

Embolism due to fragmentation of the disintegrating thrombus has been reported in up to 20% of cases[15]. This is usually of minor effect and responds to continuation of the infusion[15,19].

Massive myoglobinuria has been reported following restoration of blood flow to ischaemic and necrotic tissues. In five patients in one series[20],

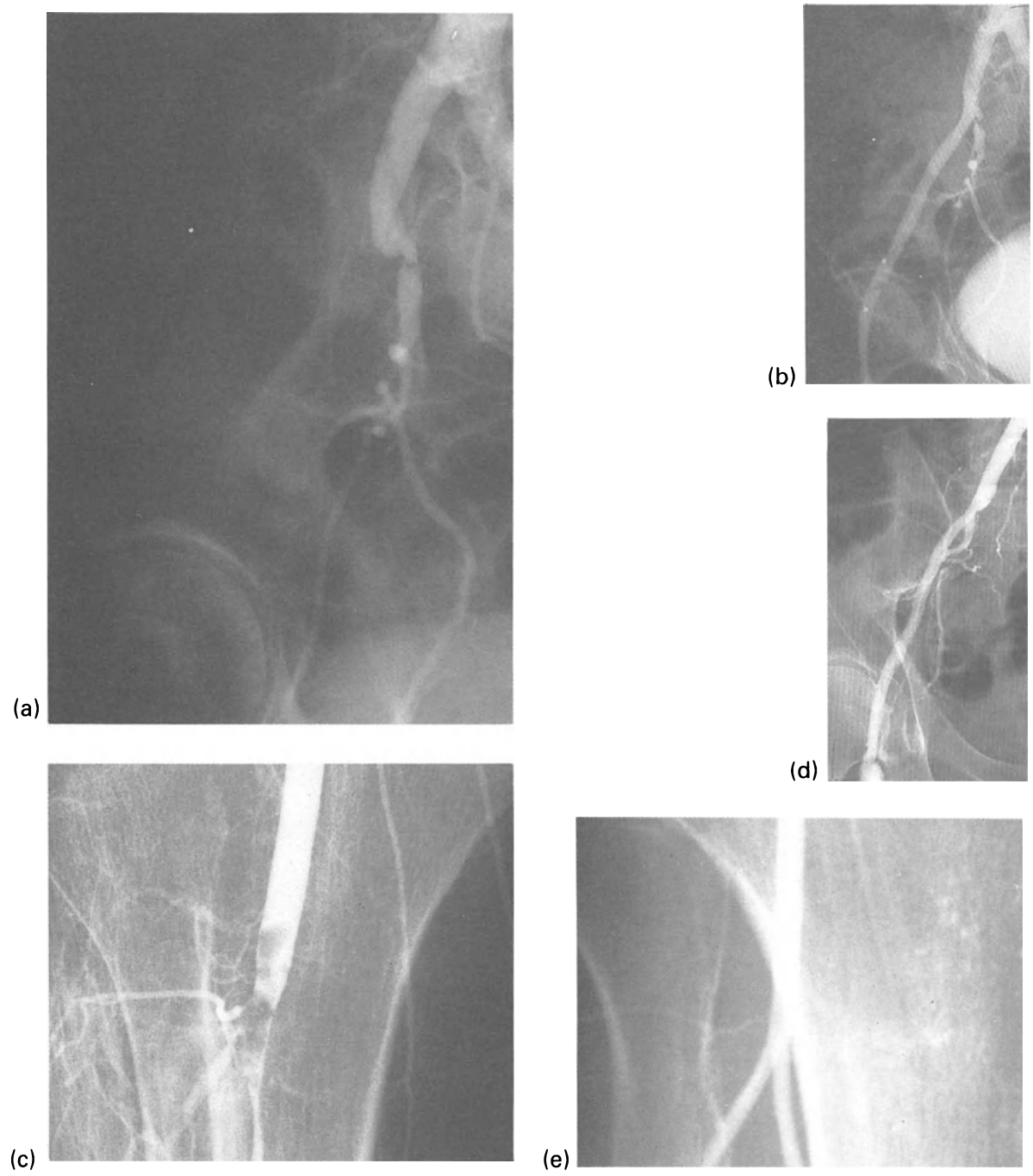


Figure 15.11(a–e) (a) A 64 year old male with acute thrombosis of right external iliac artery causing severe rest pain of 2 months' duration. Proximal part of occlusion could not be entered for fibrinolytic therapy, therefore angioplasty performed. (b) After angioplasty. Note loose thrombus adjacent to balloon. Popliteal embolus occurred. (c) Treated by local streptokinase infusion for 4 hours with reversal of ischaemia. (d, e) Appearances of right iliac and popliteal arteries at 6 months

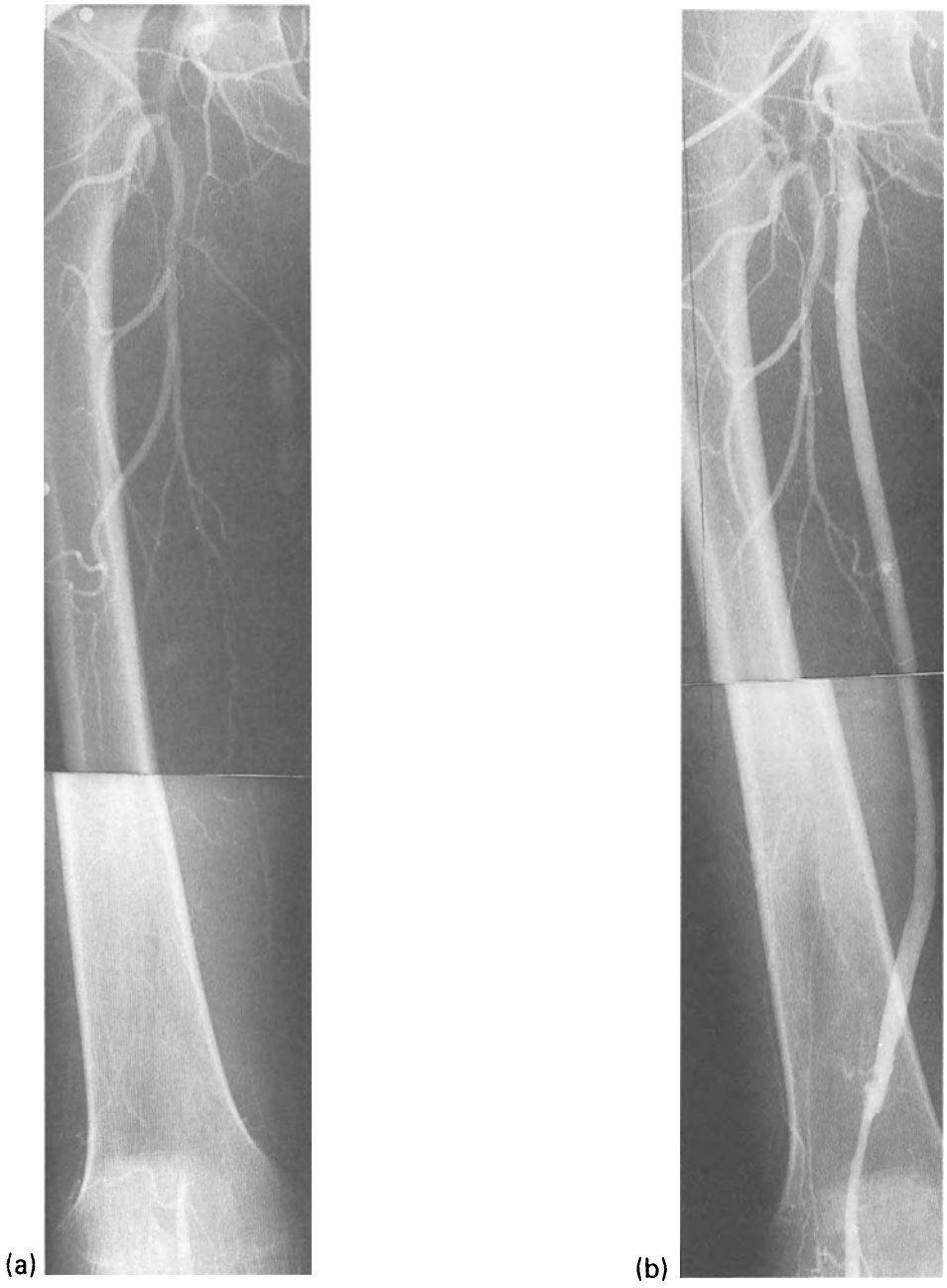


Figure 15.12(a, b) Male aged 64 years. Right femoropopliteal saphenous vein graft 3 years previously for intermittent claudication. Four week history of rest pain in right calf and foot. (a) complete occlusion of graft from its origin, normal profunda femoris, patent distal vessels. (b) after 36 hours local streptokinase infusion and balloon dilatation of stenosis at the distal anastomosis site, complete patency has been achieved.

acute tubular necrosis occurred in two and one died. This should be borne in mind if the method is being considered in patients with compartment syndrome due to ischaemia or those with much necrosis.

It is tempting, and may be correct, to dilate an underlying stenosis immediately after successful lysis: in this author's experience with streptokinase, immediate dilatation often gives the patient severe pain, probably due to a local toxic effect with endothelial damage. It may well be preferable therefore to wait for a few days, if possible, before dilating the underlying lesion.

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