Aortic Arch Replacement for Dissection

Marc R. Moon and D. Craig Miller

Aortic dissections that involve the aortic arch are usually type A, but can be retrograde extensions of type B dissections. Chronic arch dissections can remain asymptomatic for years, requiring only careful long-term surveillance and blood pressure control to minimize the likelihood of expansion; however, when aneurysmal dilatation occurs, surgical intervention becomes necessary. Aneurysmal dilatation of a dissected arch is not necessarily the result of neglect or medical treatment failure, as it can potentially complicate successful medical or surgical treatment of patients with either type A or type B dissections. Type A dissections include all of those that involve the ascending aorta, irrespective of the site of the primary tear or distal extension of the dissection. In the acute setting, operative intervention is carried out to prevent the expected sequelae of rupture with cardiac tamponade, acute aortic regurgitation caused by loss of commissural suspension, or myocardial infarction caused by coronary artery involvement. In these patients, the operative mortality risk is 10% to 25% compared with a 90% mortality rate at 3 months for nonoperative management (approximately 1% per hour for the first 48 hours).[1-10] Type B dissections do not involve the ascending aorta, and can be treated successfully with either medical or surgical therapy.[11,12] Both types, however, can involve the aortic arch, either as the location of the primary intimal tear or after distal or proximal extension. Controversy exists as to the best treatment for acute dissections that originate or involve the transverse arch.

Acute Dissection: Should the Arch be Replaced?

Acute type A dissection. In patients with acute type A dissections, surgical treatment of the arch, whether or not it is the site of the primary tear, can include "simple" ascending aortic replacement, "hemi-arch" replacement with a beveled, open distal aortic anastomosis under profound hypothermic circulatory arrest (PHCA) and ascending replacement, or complete transverse arch replacement with reimplantation of the arch vessels. We have moved away from simple ascending aortic replacement on the basis that circulatory arrest allows a more sound distal anastomosis, enables direct inspection of the arch and proximal descending thoracic aorta (with repair or resection of distal fenestrations, when appropriate), allows antegrade true lumen arterial reperfusion and rewarming after recannulation of the proximal arch graft, and avoids potential clamp injuries to the distal ascending aorta. The latter two options are distinctly different in that hemi-arch replacement requires only 15 to 30 minutes of circulatory arrest at 20°C to 22°C, whereas total arch replacement requires a substantially longer period of circulatory arrest time and lower core temperatures, both of which prolong cardiopulmonary bypass (CPB) and impair coagulation homeostasis. The Mount Sinai group reported circulatory arrest times ranging from 45 to 67 minutes for patients with acute and subacute arch dissections in which an aggressive approach to resecting arch tears was employed (total arch replacement was necessary in 11 of 19 patients); this appears to have been associated with increased bleeding, as evidenced by the need for a Cabrol shunt between the periprosthetic space and the venous circulation (the older wrap inclusion techniques were used then) in 40% of patients.[4] In their hands, however, this approach was associated with an acceptable early mortality rate of 21% for these complicated, high risk patients. Series from Japan[13,14] and France[15] also advocated total arch replacement for such patients with dissections that involved the arch, reporting mortality rates of 11% to 23%.

On the other hand, Crawford et al[16] and Borst et al[17] were more cautious, reporting that the probability of operative death more than doubled in certain individuals if arch replacement was carried out for patients with acute type A aortic dissections. Borst et al reported the Hannover series of partial (open-distal) versus total arch replacement for patients with aneurysms (n = 58) or dissections (n = 92, acute; n = 54, chronic). Their reported circulatory arrest times were remarkably low for both partial and total arch replacements (17 ± 6 minutes and 34 ± 13 minutes, respectively), but the mortality rate increased substantially with total arch replacement for patients with aneurysms (3% partial arch *v* 19% total arch), acute dissections (12% partial arch *v* 36% total arch), or chronic dissections (6% partial arch *v* 20% total arch). In Crawford's series at Baylor, among 82 patients with acute type A dissections, one third of patients underwent combined ascending and arch replacement, whereas the procedure was limited to the ascending aorta in two thirds.[16] The operative death rate increased from 17% for ascending aortic replacement alone to 31% for patients in whom the procedure included at least the proximal arch (hemi-arch replacement). Arch repair was an independent predictor of operative mortality, although it seemed to reduce the need for late reoperations on the distal aorta. Additionally, advanced New York Heart Association functional class, diabetes, and the need for concomitant coronary artery bypass grafting appreciably increased the risk if the procedure was extended into the arch, whereas their impact after isolated ascending aortic replacement was minimal. In the combined Stanford-Duke series of patients with aortic dissections due to arch tears (n = 47 patients), advanced age, number and severity of dissection related complications, and coexistent medical illnesses adversely influenced the already high operative risk.[18]

We currently prefer the hemi-arch, open distal technique for patients with acute type A dissections to allow for assessment of the extent of intimal disruption and the condition of the arch vessels and to ensure a more sound distal anastomosis (this technique has been previously published in this journal).[19] We resect the tear if practical, but do not perform complete arch replacement in this setting unless there is rupture or impending rupture of the arch itself, especially if other risk factors are present. Inherent in this approach is the known potential for dilatation of the distal false channel requiring subsequent reoperation. Close follow-up is essential to monitor the progression of false lumen dilatation and to decide when the appropriate time for surgical intervention is necessary. Follow-up of patients with aortic dissection, whether treated medically or surgically, should include a computed tomographic or magnetic resonance imaging scan before hospital discharge and at 3 months. Scans can then be obtained at 6 month intervals for 1 to 2 years, then annually throughout the patient's life if there are no major pathoanatomical changes detected over time. Strict blood pressure control and negative inotropic therapy are also essential; DeBakey et al[2] found that aneurysms subsequently developed in 46% of patients with uncontrolled hypertension, but only in 17% with controlled blood pressure.

Acute type B dissection. In patients with acute type B dissections, we currently treat most individuals with medical therapy; in general, surgical intervention is undertaken for those with complications, eg, acute rupture or leak, the presence of a large false aneurysm, ischemia of distal end-organs, or progression of the dissection during medical therapy manifested by persistent or recurrent pain. We take a more aggressive surgical approach, however, in younger, healthy patients and those with the Marfan syndrome. The advent of endovascular stent-grafting for patients with acute type B dissections has also changed the decision-making process, but this experimental treatment modality is confined to just a few centers around the world. In the Stanford-Duke series of patients with type B dissections, the 30-day mortality rate for patients with no compelling indication for emergency operation was 10% with medical therapy and 19% with surgical therapy.[11] Patients who develop ischemic complications due to distal aortic branch compromise undergo angiographic investigation and stenting of branch vessels or the aorta itself, with or without fenestration of the dissection flap.[20-22] We have always considered early operation (proximal descending aorta replacement, or, currently, a stent-graft to cover the primary intimal tear) in younger, low-risk patients in an attempt to prevent the adverse late sequelae of the disease; although this aggressive approach is intuitively logical and today is endorsed by the Mount Sinai and Yale groups, its use and effectiveness compared with medical therapy alone remains unknown at this time.[12,22] We do, however, avoid manipulation and clamping of the arch during operations in patients with acute type B dissections by using circulatory arrest and constructing an "open proximal" aortic anastomosis. In the early Stanford surgical series, the operative mortality rate for patients with an acute type B dissection caused by an arch tear was 75%, compared with 32% if the tear was in the descending aorta.[3] In the Stanford-Duke series of patients with primary intimal tears located in the arch, none of the four patients who underwent arch repair at the time of descending thoracic replacement survived, and only one of three patients without concomitant arch repair survived.[18] After 2 years, 75% ± 22% of medically-treated patients were alive compared with only 14% ± 13% of the surgically-treated group; however, by 3 years, all the medical patients had died. Clearly, these historical data from the 1970s indicate that substantial room for improvement existed in dealing with patients with type B dissections originating from an arch tear, including newer surgical techniques that rely on profound hypothermic arrest.[7,23-26]

Fate of the Distal False Lumen

Radiographic studies have shown that the distal false lumen remains patent in the majority of patients (85%) after proximal thoracic aortic repair, but the persistence of the false channel does not portend a catastrophic late outcome; in fact, it may be the only source of blood flow to major organs in some patients.[27] In the Mount Sinai series, persistence of the false lumen was associated with the development of late thoracic aortic false aneurysms, but the incidence was relatively low and long-term survival was not substantially different between patients with a closed versus patent distal false lumen.[28] Two of 18 (11%) patients with a persistently patent distal false lumen required late reoperations. Event-free survival at 5 years was 84% for patients with a thrombosed false lumen, versus 63% for those with a patent false lumen (P = not significant [NS]). Resection of a primary arch intimal tear did not necessarily obliterate the distal false lumen; 23% of patients who underwent partial or total arch replacement for an arch tear had a patent false lumen. Persistence of the false channel, therefore, does not appear to correlate with successful exclusion of the primary intimal tear, but depends more on the presence of distal fenestrations between the true and false lumens.[27]

More intensive long-term medical follow-up is necessary to decrease the death rate from late aortic rupture by prompting earlier reoperation in patients whose chronic dissections become aneurysmal. Of all late deaths in the early Stanford series, at least 15% were caused by late aortic rupture, and in DeBakey et al's 20-year series, 29% of late deaths were caused by aortic rupture.[2,4] Subsequent aneurysmal formation occurred more often with dissections that involved the entire descending and abdominal aorta (35%) compared with dissections limited to either the ascending (14%) or proximal descending (16%) aorta.[2] Overall, late aneurysms developed in 29% of patients after successful initial surgical treatment of patients with acute dissections. Reoperation in this setting should not be considered a treatment failure, but rather a timely and prudent treatment of the late aortic sequelae of aortic dissections. In the Stanford series, the linearized rate of reoperation was 3.4% per patient year; or, in actuarial terms, 13% ± 4% of patients at 5 years and 23% ± 6% at 10 years had required an aortic reoperation, postoperatively.[4] The reoperative rate was similar in patients with type A or type B dissections, but higher in younger patients and in those with primary tears in the arch (27% ± 13% at 1 year); however, even in patients with primary arch tears, the site of reoperation rarely included the arch itself. Among the 21 reoperations required in 135 hospital survivors, 8 were on the proximal aorta and 13 were for distal aortic replacement. Arch replacement was performed in three patients with type A dissections and in two patients with type B dissections. Aneurysmal dilatation of chronic dissections involving the aortic arch is uncommon, but its surgical treatment can be quite challenging; this is the subject of this current review.

Indications For Operation on the Aortic Arch

Consideration of surgical intervention should probably be more aggressive in patients with arch false aneurysms caused by chronic dissection than in those caused by degenerative aneurysms. In Pressler and McNamara's[29] series, aortic rupture was the cause of death in 77% of patients with thoracic aortic aneurysms due to dissection compared with only 44% of degenerative aneurysms; however, this disparity is due in part to the fact that the diagnosis of aortic dissection was made during the acute phase shortly before fatal rupture in many patients with dissections in this historical retrospective study. Hirose et al[30] reported that the growth rate of aneurysms was greater in the arch (0.56 cm/year) than in the descending thoracic (0.42 cm/year) or abdominal aorta (0.28 cm/year). The Mount Sinai group found that once an aneurysm reached 5 cm, its growth rate increased substantially; aneurysm expansion rate was also higher in smokers.[31] Juvonen et al[32] also developed a model to predict the risk of rupture in patients with thoracic aneurysms based on aneurysm size, age, and the presence of pain or chronic obstructive pulmonary disease; unfortunately, these data did not include aortic dissections. The Yale group found that the growth rate increased with aneurysm size and was higher in the presence of a dissection; furthermore, descending aneurysms expanded faster than more proximal thoracic aortic aneurysms (Fig I).[33] Indications for resection of a chronic dissection involving the aortic arch include: (1) Arch diameter (true and false lumens combined) greater than 6 to 7 cm or greater than twice the size of the normal thoracic aorta for that individual; (2) enlargement of more than 7 to 10 mm in 1 year; (3) symptoms attributable to the dissection such as pain, hoarseness, swallowing or respiratory difficulties, caval obstruction; or (4) localized saccular protrusion of the false lumen, which might put the patient at a higher risk of rupture. Patients with the Marfan syndrome are generally younger and are more likely to experience rapid progressive aortic dilatation[33-35]; therefore, arch replacement should generally be considered when expansion exceeds 3 to 5 mm per year or when the aortic diameter exceeds 5 cm in healthy individuals.

Surgical Technique: General Principles

Aortic pathology that is superior to the left mainstem bronchus on computed tomographic scan can be addressed effectively via a median sternotomy. If surgical access is required beyond the left mainstem bronchus, a left thoracotomy or bilateral anterior thoracotomy is required. In this review, we only describe our techniques for arch replacement in patients with chronic dissections via the anterior approach. For arch replacement in chronic dissections, the "elephant trunk" methods, in which a free segment of graft is left dangling distally in the descending aorta, is ideal to simplify future aortic operations.[36,37] The dissection most often extends far distally, and although the descending aorta

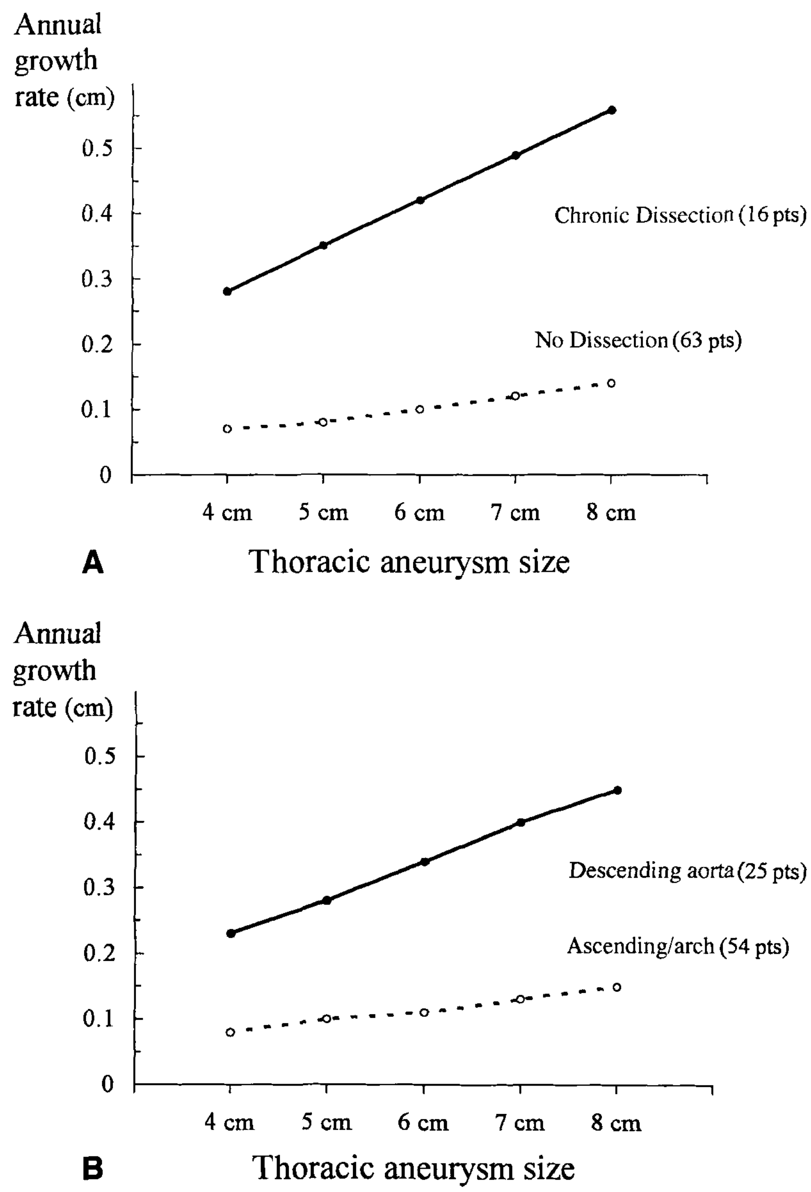


Fig I. Annual growth rate of thoracic aortic aneurysms based on (A) dissection status and (B) location of the aneurysm. (Data from Coady MA, Rizzo JA, Hammond GL, et al: What is the appropriate size criterion for resection of thoracic aortic aneurysms? J Thorac Cardiovasc Surg 113:476-491, 1997.[33])

may not yet be aneurysmal at the time of arch replacement, the distal graft is available for either a later planned procedure or when dilatation of the descending thoracic aorta becomes sufficient to warrant further replacement.

CPB. If previous ascending aortic replacement has been performed and redo sternotomy can be accomplished safely, the arterial perfusion cannula may be inserted into the previous graft to provide antegrade flow during the period of cooling. Otherwise, CPB (Fig II) is commenced initially using either a subclavian or femoral arterial cannula, and the arterial line is "Y-ed" to allow subsequent recannulation of the arch graft for antegrade CPB flow after arch reconstruction. Over time, we have been using the subclavian artery more often, following the lead of Sabik et al[38] from the Cleveland Clinic, hoping that this will minimize the incidence of CPB malperfusion of vital end-organs. When femoral arterial cannulation is performed in a patient with a chronic dissection, bilateral tympanic membrane temperature (and/or bilateral radial artery pressure) monitoring is used to ensure equal cerebral cooling rates, myocardial temperature is measured, and the descending aorta is interrogated with transesophageal echocardiography to make sure that neither the false nor true lumen becomes obliterated at the onset of

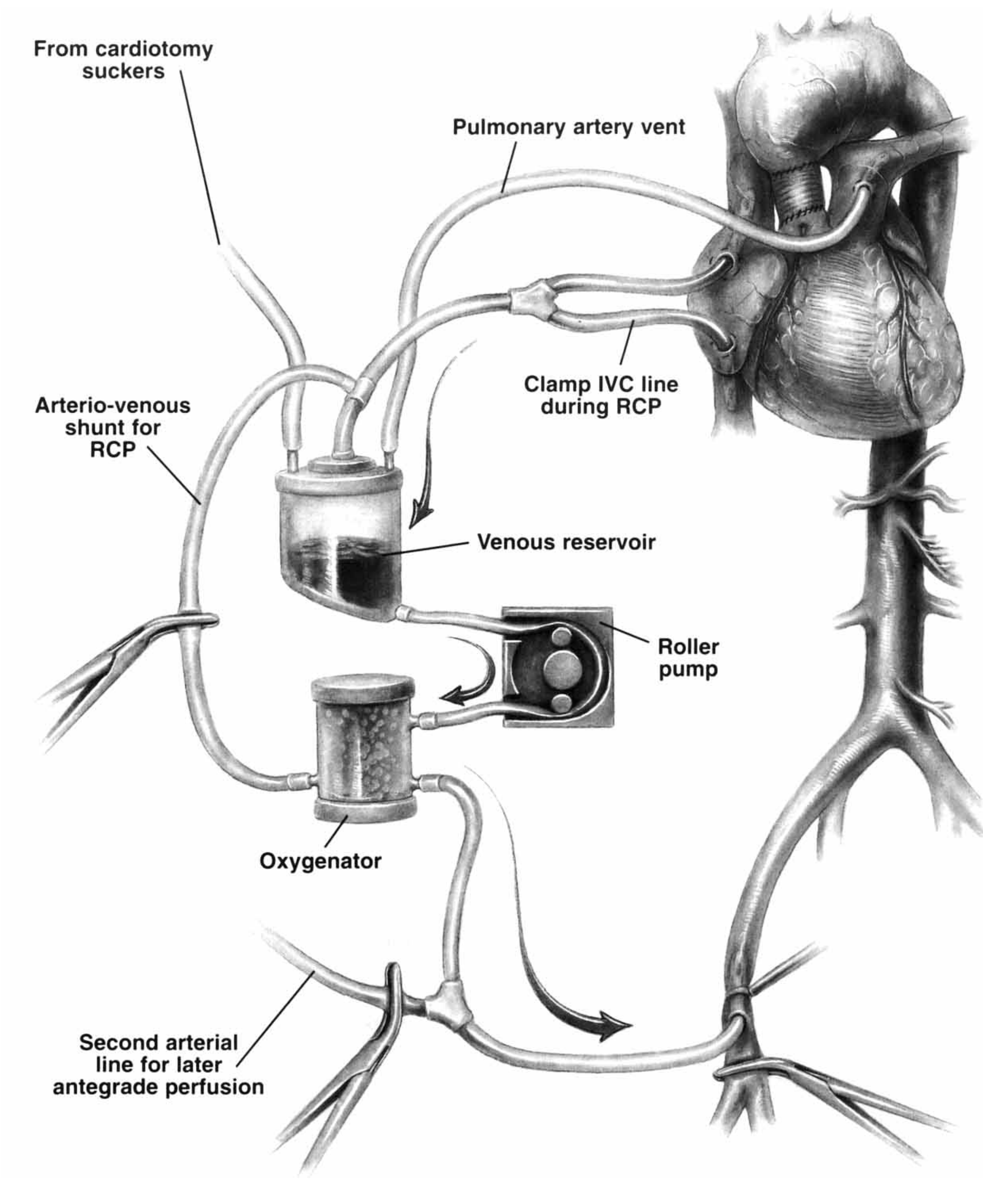


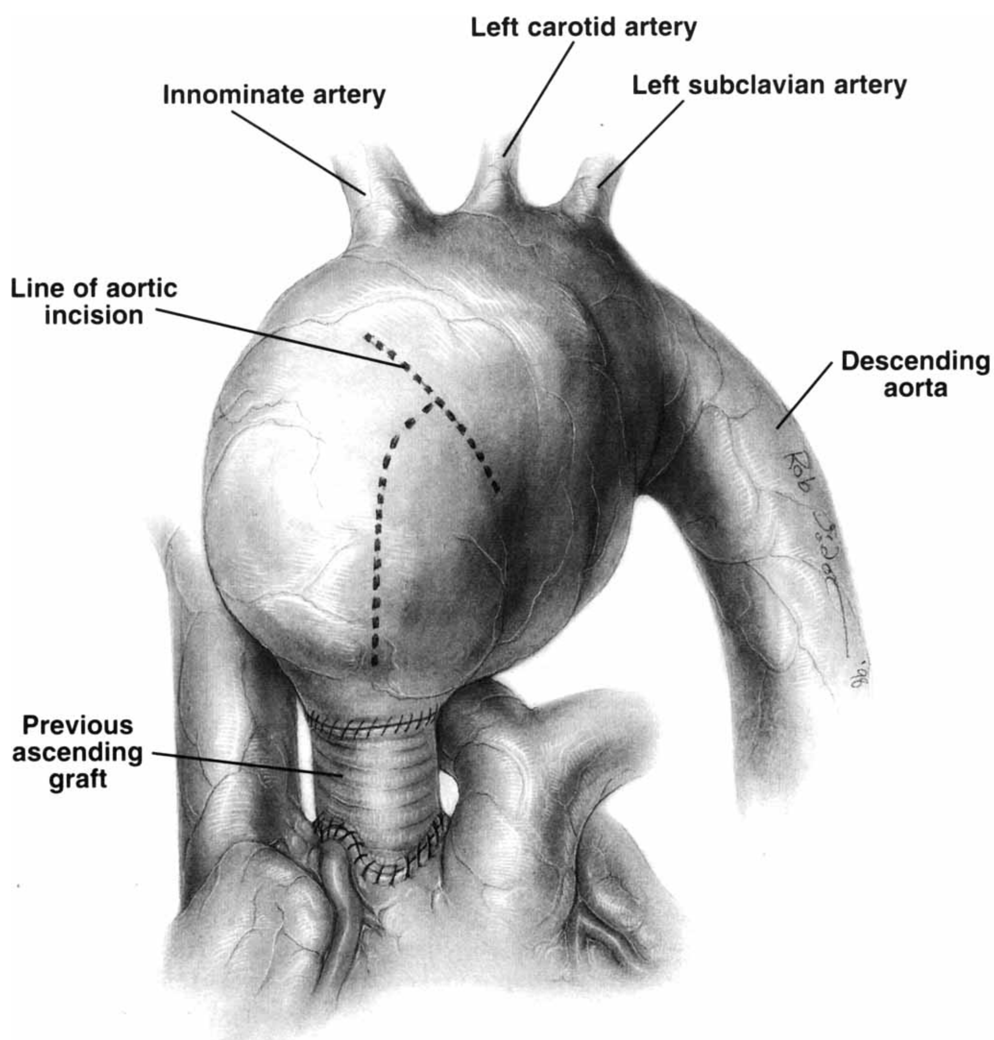
Fig II. Schematic of the CPB circuit for aortic arch replacement using profound hypothermic circulatory arrest and retrograde cerebral perfusion (RCP). IVC, inferior vena cava.

retrograde CPB perfusion. Malperfusion of important aortic arch branches or abdominal aortic tributaries at this point can be a catastrophic complication. We cannulate the femoral or subclavian artery with the stronger pulse (contrary to our policy in patients with acute dissections), which usually communicates directly with the aortic true lumen above, but prefer the right side if femoral vein cannulation should become necessary. If a venous cannula needs to be passed into the right atrium, using the right femoral vein affords the most reliable direct access route to the inferior vena cava (IVC) by minimizing problems navigating the guidewire or cannula across the IVC bifrucation. In complicated cases where ascending aortic false aneurysms have eroded into the posterior table of the sternum, CPB can be instituted and the patient cooled to 22°C using femoral-femoral cannulation before the sternotomy is completed under PHCA; a 28F multifenestrated venous cannula (DLP Medical Products, Medtronic, Inc, Grand Rapids, MI) is placed at the right atrial-superior vena cava (SVC) junction under echocardiographic guidance and a centrifugal (suction) pump system is used to augment venous drainage. Otherwise, standard bicaval venous cannulation is employed with tourniquets around both the SVC and the IVC. Once the chest is open and CPB is initiated, a 14F sump vent can be placed into the pulmonary artery to reduce bronchial return to the left heart. Alternatively, if severe aortic valvular regurgitation is present or a false aneurysm of the ascending aorta has eroded into the left atrium or left ventricle creating a fistula or the ventricle cannot be kept adequately decompressed by manual massage, we prefer using a large (20F) apical left ventricular (LV) sump vent. The CPB circuit also includes an arteriovenous shunt for retrograde cerebral perfusion (RCP) during periods of circulatory arrest.

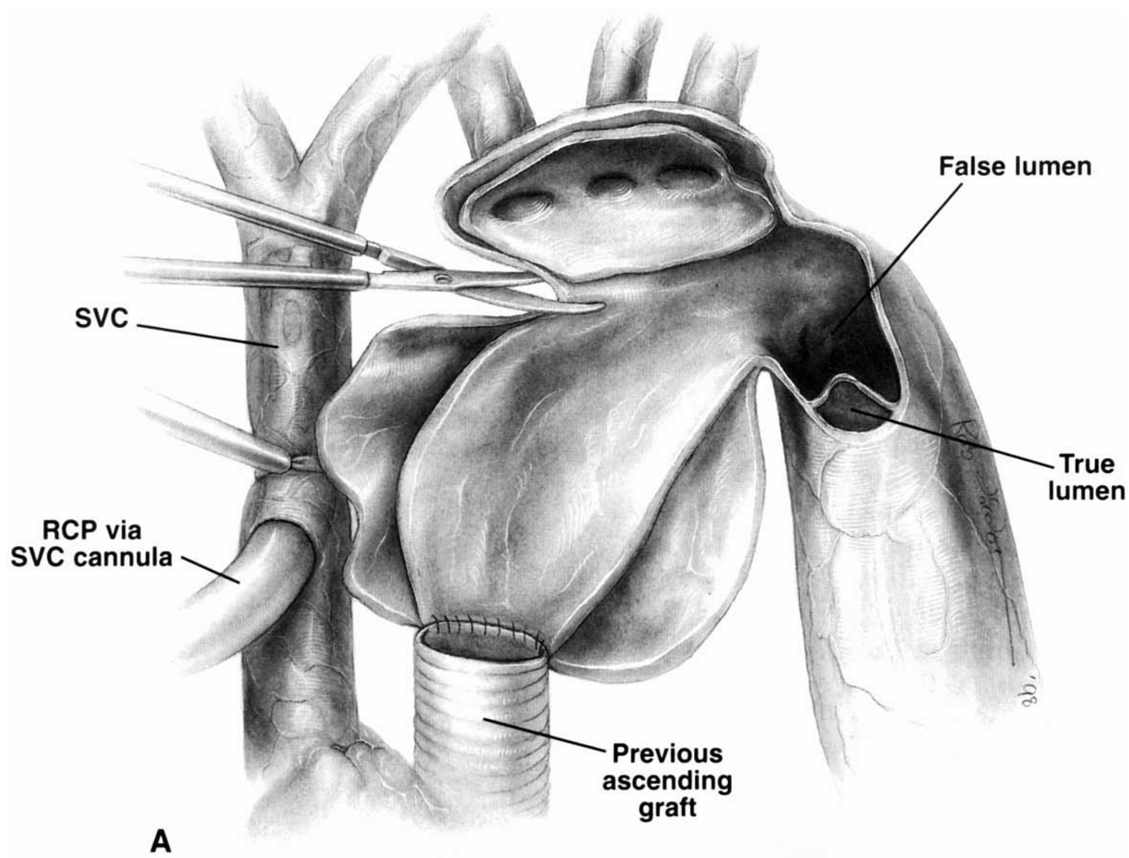
Preparation for circulatory arrest. Because of previous ascending aortic operations or extensive aneurysms, it commonly is not possible to clamp the ascending aorta safely before turning off the pump. For total arch replacement, the patient is cooled to 15°C (bladder temperature), which theoretically allows 30 to 45 minutes of safe PHCA time. At 18°C, cerebral metabolic rate is 40% of baseline, at 15°C, 25% to 30% of baseline, and at 8°C to 10°C, less than 10% to 12% of baseline.[39-41] Systemic core cooling should progress at a rate of 1°C per minute, and usually takes 30 to 45 minutes. It is important to remember that during cooling, the heart will fibrillate, usually at around 20°C to 25°C; thereafter, transesophageal echocardiography must be used continuously to avoid LV distention. If the heart fibrillates at a much warmer temperature, then coronary malperfusion should be suspected immediately, and appropriate alternative arterial cannulation performed quickly, eg, subclavian artery or LV apical cannulation across the (native) aortic valve into the ascending aorta or switching to the other femoral artery. Just prior to circulatory arrest, mineralocorticoids (dexamethasone, 10 mg intravenously [IV]) and phenobarbital (1 to 2 mg/kg IV) are given, and the head is kept packed continuously in ice. Myocardial protection using intermittent retrograde blood cardioplegia is preferred, in conjunction with a Daily myocardial cooling jacket (Daily Medical Products, San Diego, CA). At these low systemic temperatures, however, special attention needs to be given to insulating and protecting the left phrenic nerve. After turning off the pump, 1,000 mL of cold blood cardioplegia is infused retrograde, the caval tourniquets are cinched down, the patient is minimally exsanguinated (only 500-1,000 mL are drained into the venous reservoir), and the cooling jacket is positioned over both the right and the left ventricles. Myocardial temperature is kept below 10°C, and cardioplegia is reinfused retrograde every 20 to 30 minutes for washout purposes; the amount of blood cardioplegia given is small (300 mL), and these intermittent infusions are stopped when the effluent blood coming out of the coronary ostia turns pink or red.

RCP. During circulatory arrest, the patient is placed in the Trendelenberg position, and we commence intermittent, low pressure, low flow RCP via the SVC with the IVC cannula clamped to flush air and particulate matter from the brain. RCP is initiated at 100 mL per minute to 500 mL per minute to keep the innominate vein pressure less than 20 mm Hg; the cardiotomy reservoir volume is maintained using blood collected from the field by the cardiotomy suckers. Retrograde cerebral perfusion supplies only approximately 20% to 25% of the metabolic requirements of the brain, but when combined with topical cooling, it helps maintain cerebral hypothermia and does have a flushing effect.[42-46] By keeping the proximal venous pressure (innominate vein) less than 20 mm Hg and limiting the time of RCP we attempt to minimize the risk of cerebral edema. Usui et al[43] found that SVC flow rates did not correlate with the stroke rate (18% if less than 300 mL/min versus 19% if greater; $P > .78$), but there was a significantly higher stroke rate in patients with increased SVC pressure (greater than 25 mm Hg, 28% stroke rate; less than 25 mm Hg, 16% stroke rate; $P < .03$). Others have found that the stroke incidence increased progressively as the circulatory arrest times became longer, especially when the period of arrest exceeded 45 to 60 minutes.[47,48]

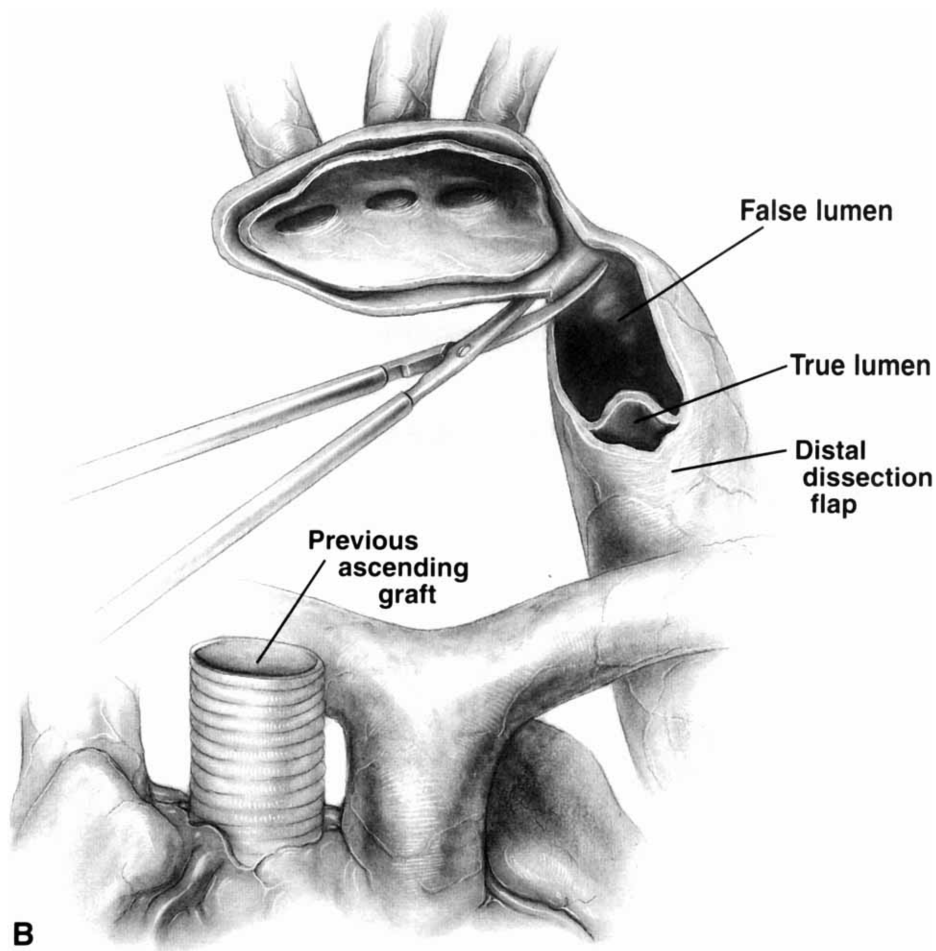
SURGICAL TECHNIQUE



1 The mid-ascending aorta is opened, and after identification of the true and false lumens, the aorta is transected just proximal to the innominate artery. Careful delineation of the aortic arch pathological anatomy, ie, relationship of the true and false lumens with respect to the arch vessels, is paramount at this stage of the operation.

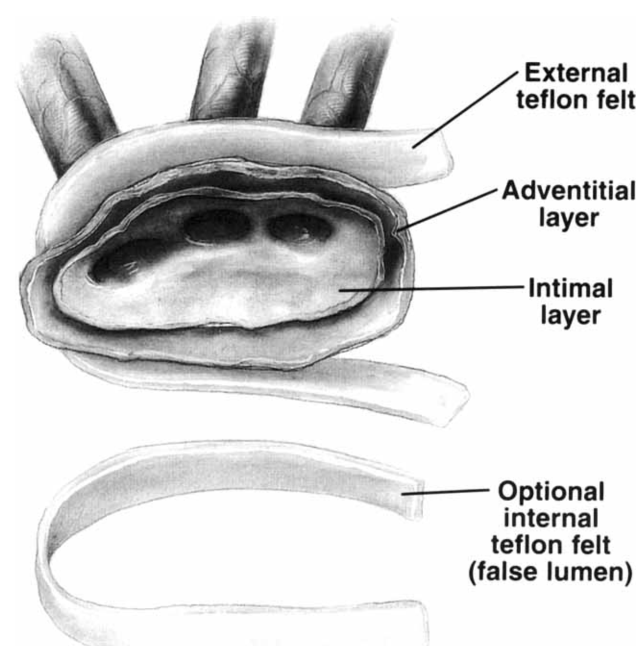


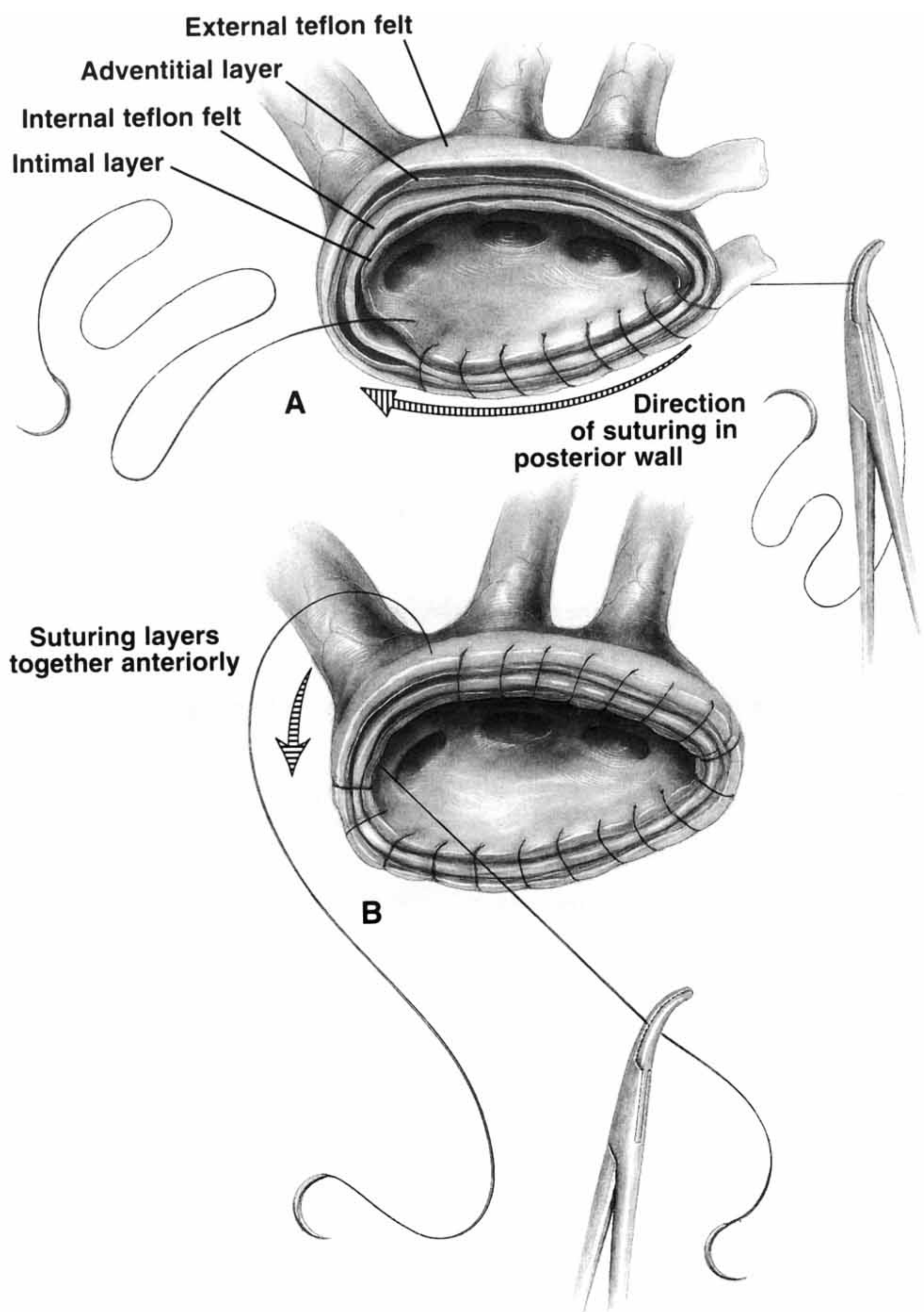
2 (A) If the origin of the arch vessels is close to the dissection flap but the arch vessels are not dissected per se, the intima and adventitia are divided together to leave a 5 to 10 mm rim around their ostia. If the arch vessels are dissected, it is generally not possible nor wise to direct blood flow exclusively into the true lumen; the false lumen may be responsible for some or all of the distal arterial blood flow. A wedge of the dissection flap is excised from each vessel, allowing subsequent perfusion of both (true and false) channels. The flap within each dissected branch is incised for a distance of approximately 10 mm, and amputated sharply without leaving any freely mobile piece.



2 (continued) (B) The "island" of arch surrounding the great vessels is dissected free just beyond the left subclavian artery.

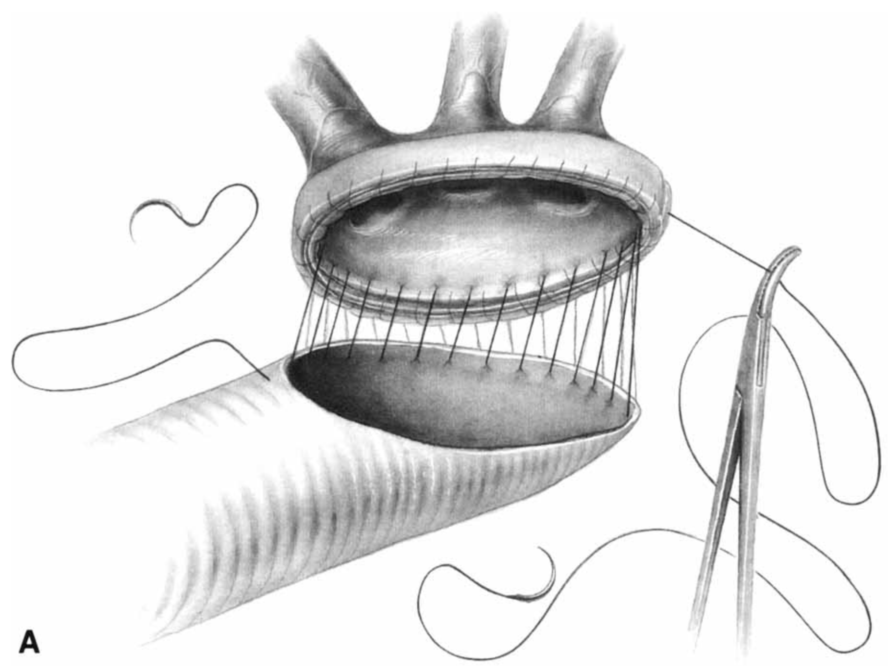
3 The arch cuff is constructed with a strip of Teflon felt or knitted, double velour Dacron graft material (Meadox Medicals/Boston Scientific Corp, Oakland, NJ) between the intimal and adventitial layers. When the tissues are extremely friable (eg, in patients with the Marfan syndrome), if necessary, the arch cuff can also be reinforced externally with another strip of fabric. In the acute setting, this Teflon felt "sandwich" technique has been associated with reduced distal false lumen patency, suggesting a more secure anastomosis with less intimal tearing.[28]





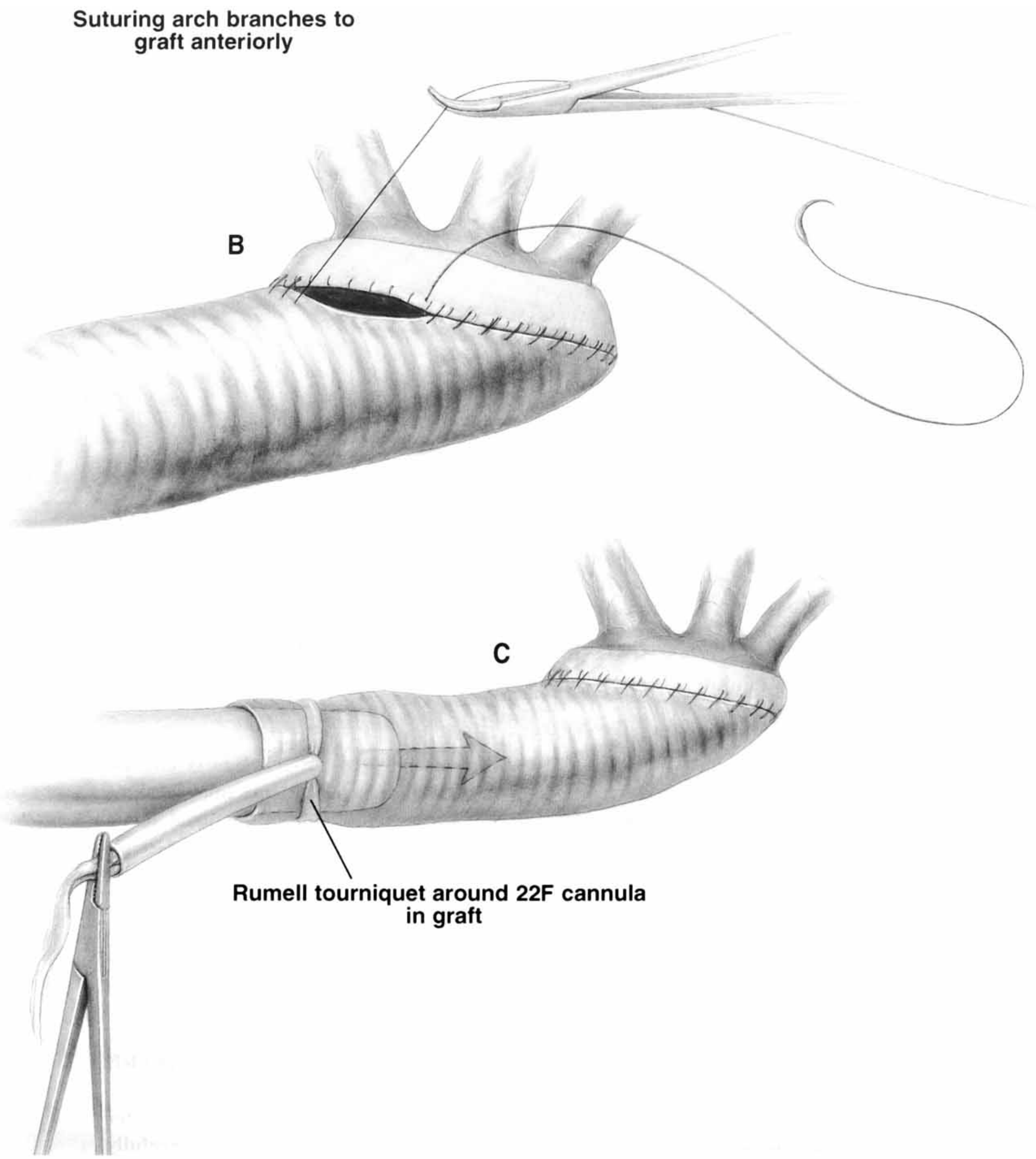
4 A "whip-stitch" of long (137 cm) 4-0 SH-1 Prolene suture (Ethicon, Inc, Somerville, NJ), is started distal to the left subclavian, (A) progressing first along the posterior wall, then (B) anteriorly to complete the buttressed arch island.

Suturing graft to arch branches posteriorly



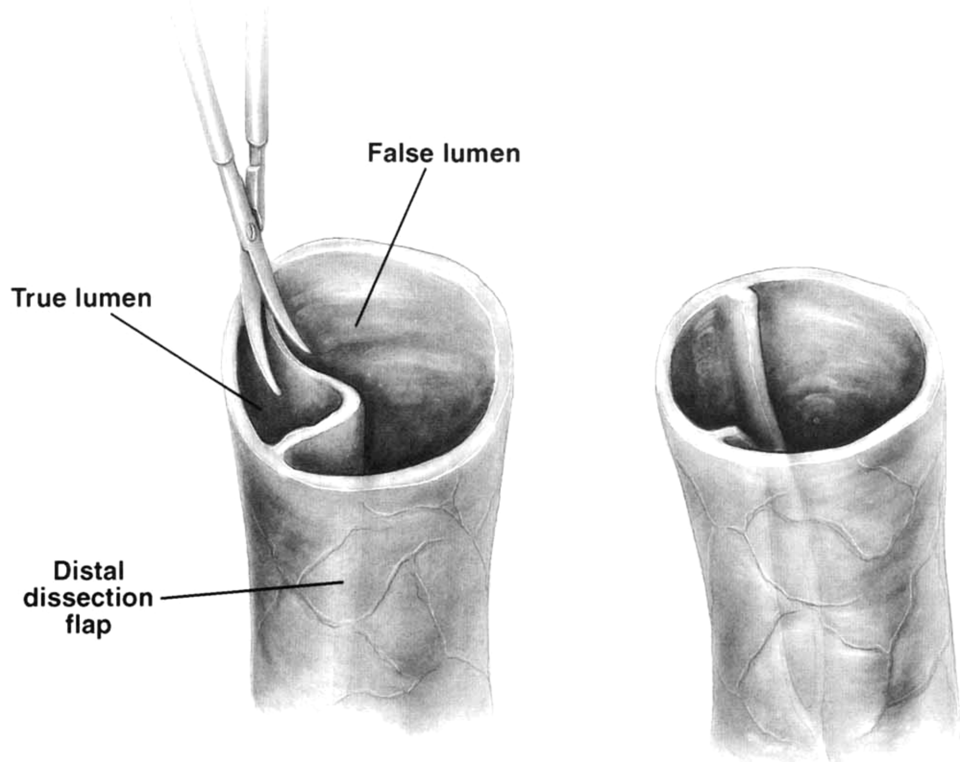
A

5 (A) During complex arch procedures or when the expected PHCA time is long, we frequently prefer to sew a separate graft to the arch vessels at this time to allow selective antegrade cerebral perfusion (SACP) during completion of the distal aortic anastomosis. Our technique is a modification of that originally developed by Griepp and associates,[41,49] which offers optimal exposure during each anastomosis and allows the surgeon time to perform the distal aortic anastomosis in a methodical manner; the technical soundness of this distal anastomosis is key, and less than a perfect anastomosis can lead to potentially fatal bleeding or the late development of pseudoaneurysms. The reconstructed arch cuff is sewn to the beveled end of a small (14 to 18 mm) collagen-impregnated (Hemashield) knitted or woven double velour vascular graft (Meadox Medicals/Boston Scientific Corp, Oakland, NJ). Using a 137 cm 3-0 SH or 4-0 SH-1 Prolene suture, the anastomosis is started distal to the left subclavian artery orifice, running first along the posterior wall. The initial suture bites are placed loosely and then the graft is parachuted down to allow better visualization of this most critical portion of the anastomosis. A nerve hook is carefully used to ensure that the sutures are symmetrically positioned and tight. Extreme gentleness is required when arcing the needle on its complete one-half curve through the aorta to prevent intimal needle hole tears, especially along the posterior wall when the surgeon is placing the sutures from outside-to-inside the aorta. The first assistant must also be very delicate with the "following" traction to avoid tearing the fragile aortic tissue. Although not preferred, if the distal arch is densely adherent to the posterior mediastinal tissues and circumferential dissection of a full-thickness cuff is not possible, the distal-most part of the arch island can be left intact. In this case, the bites taken beyond the left subclavian employ an inclusion technique and are very deep to ensure incorporation of large bites of the adventitia.

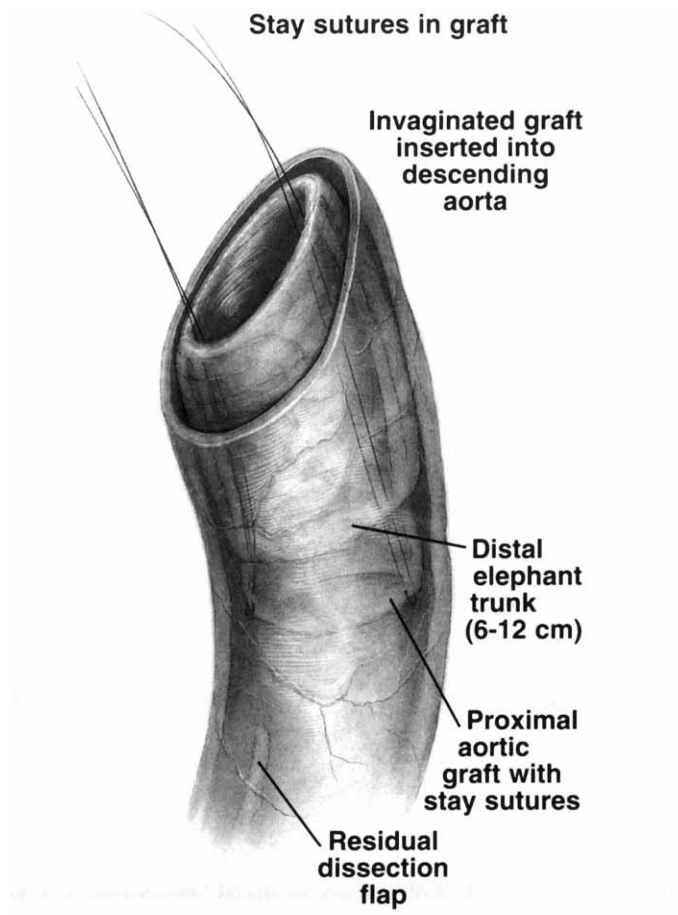


5 (continued) (B) The posterior suture is continued just anterior to the innominate artery, and the anastomosis is completed by running the other arm of the suture anteriorly along the patch. It is occasionally necessary to temporarily suspend retrograde cerebral perfusion to improve visualization during critical portions of the anastomosis.

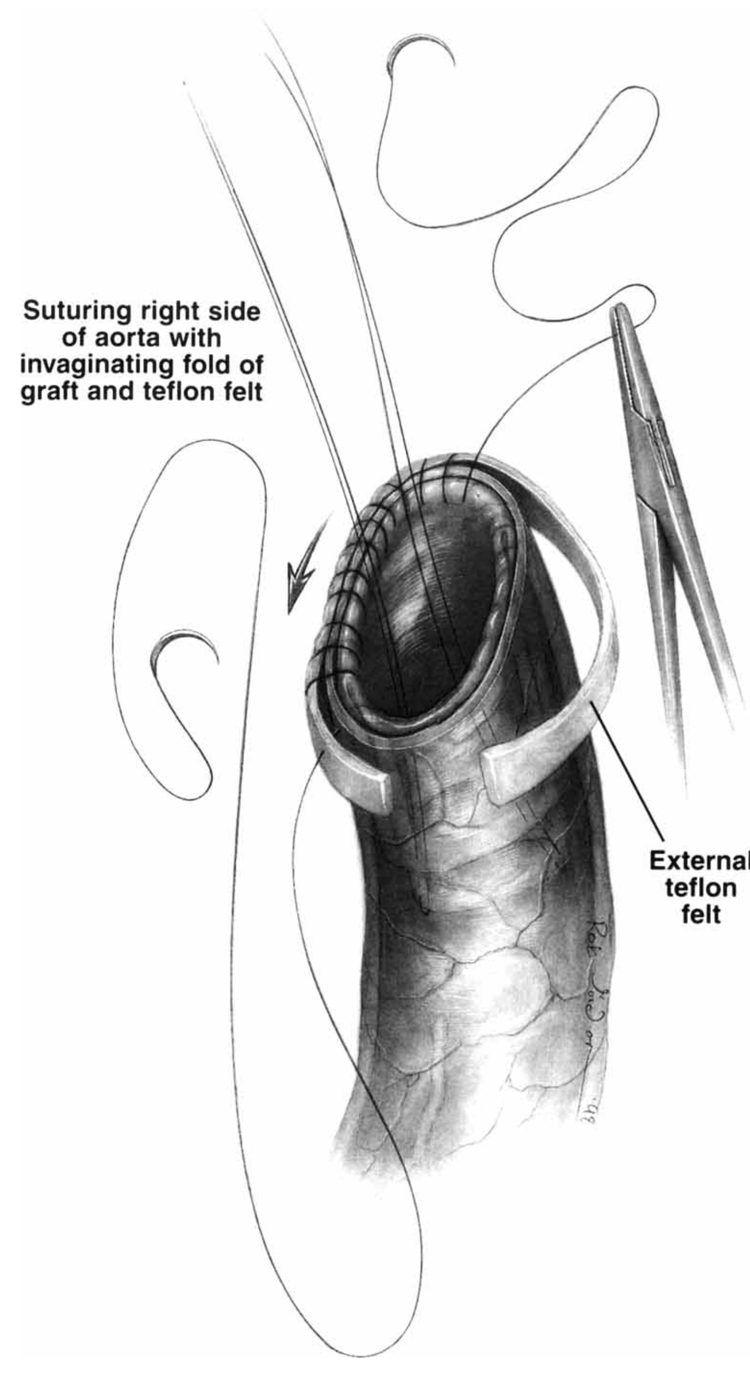
(C) The arch vessels are de-aired using low flow RCP, steep Trendelenberg position, and finger manipulation of the carotid and subclavian arteries. An arterial cannula is then inserted into the proximal end of the arch graft, retrograde RCP is discontinued, the IVC cannula is unclamped, the femoral artery *cannula is clamped, and SACP is started to the arch vessels at 15°C to 20°C to keep the radial artery pressure* approximately 50 mm Hg; usually, flow rates of 500 to 900 mL per minute are sufficient. This arterial cannula can remain in this position throughout the remainder of the procedure, if desired. Once antegrade cerebral perfusion is restored, the difficult distal anastomosis can be performed carefully and without excessive time constraints.



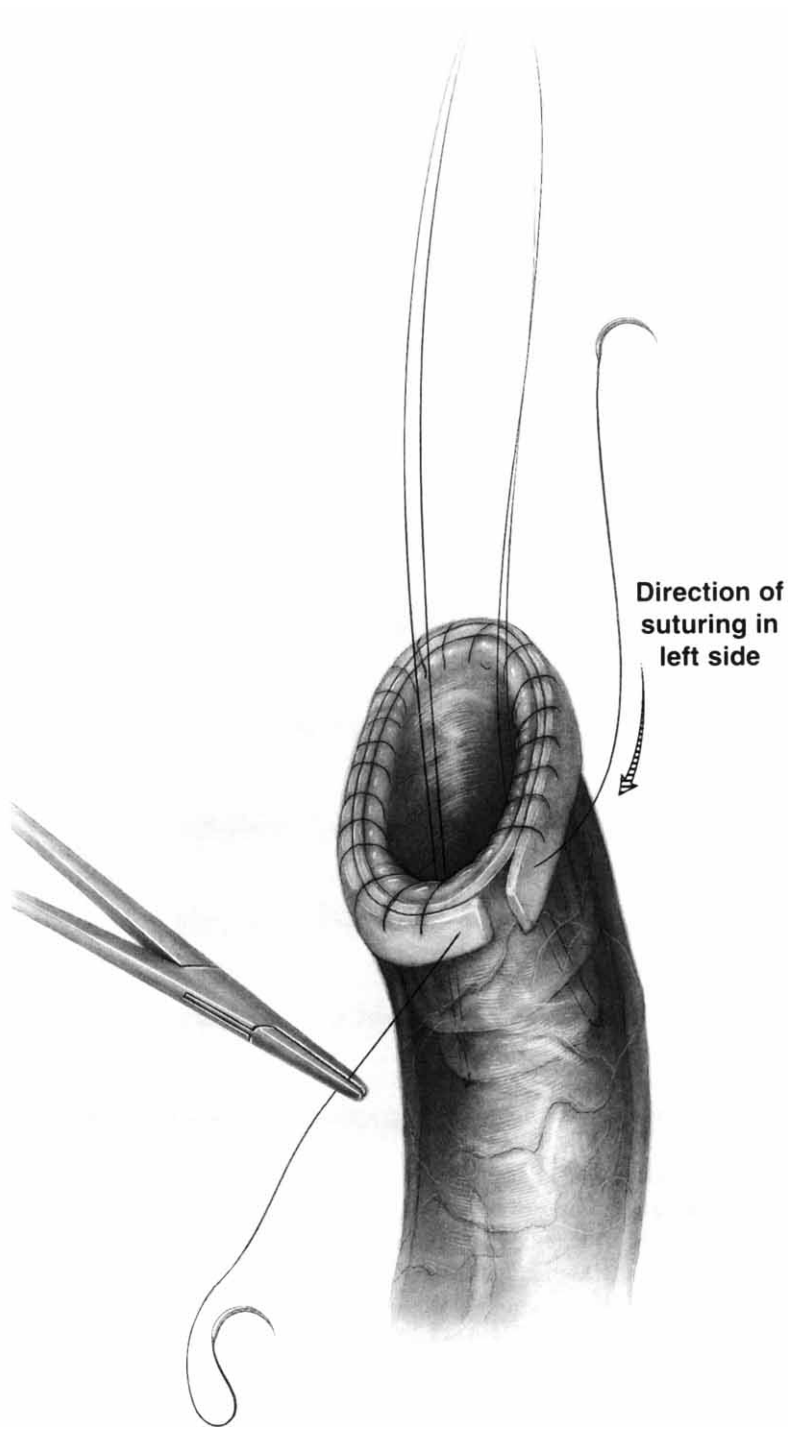
6 The distal arch or proximal descending thoracic aorta is preferentially prepared for an end-to-end, full thickness distal anastomosis. Care should be taken to avoid injury to the phrenic or vagus nerves, which often are adherent to the dissected aorta at this level. Tissue planes are often obliterated, and circumferential dissection around the aorta can be hazardous. In this case, the greater curve is dissected free, but the lesser curve (near the ligamentum) can be left intact, if necessary. We feel this provides a less secure distal anastomosis, but it may be the only option in some patients. On the other hand, when the dissected aorta does not naturally narrow into a "waist" just beyond the left subclavian artery, we feel it is imperative to do a full-thickness, circumferential anastomosis at the level of the proximal descending aorta; only this technique will enable the surgeon to "neck down" the inordinately large aorta safely and perform a sound distal graft anastomosis. No attempt is made to redirect distal aortic flow into the true lumen because both the true and/or the false channels may be responsible for critical aortic branch flow downstream. The dissection flap (or septum between the true and false lumens) is incised longitudinally along its borders as far distally as can be visualized; the excision of the flap must extend for a distance that is longer than the length of the elephant trunk graft to avoid compromise of distal aortic flow due to the end of the elephant trunk graft ending up inadvertently in one lumen or the other.



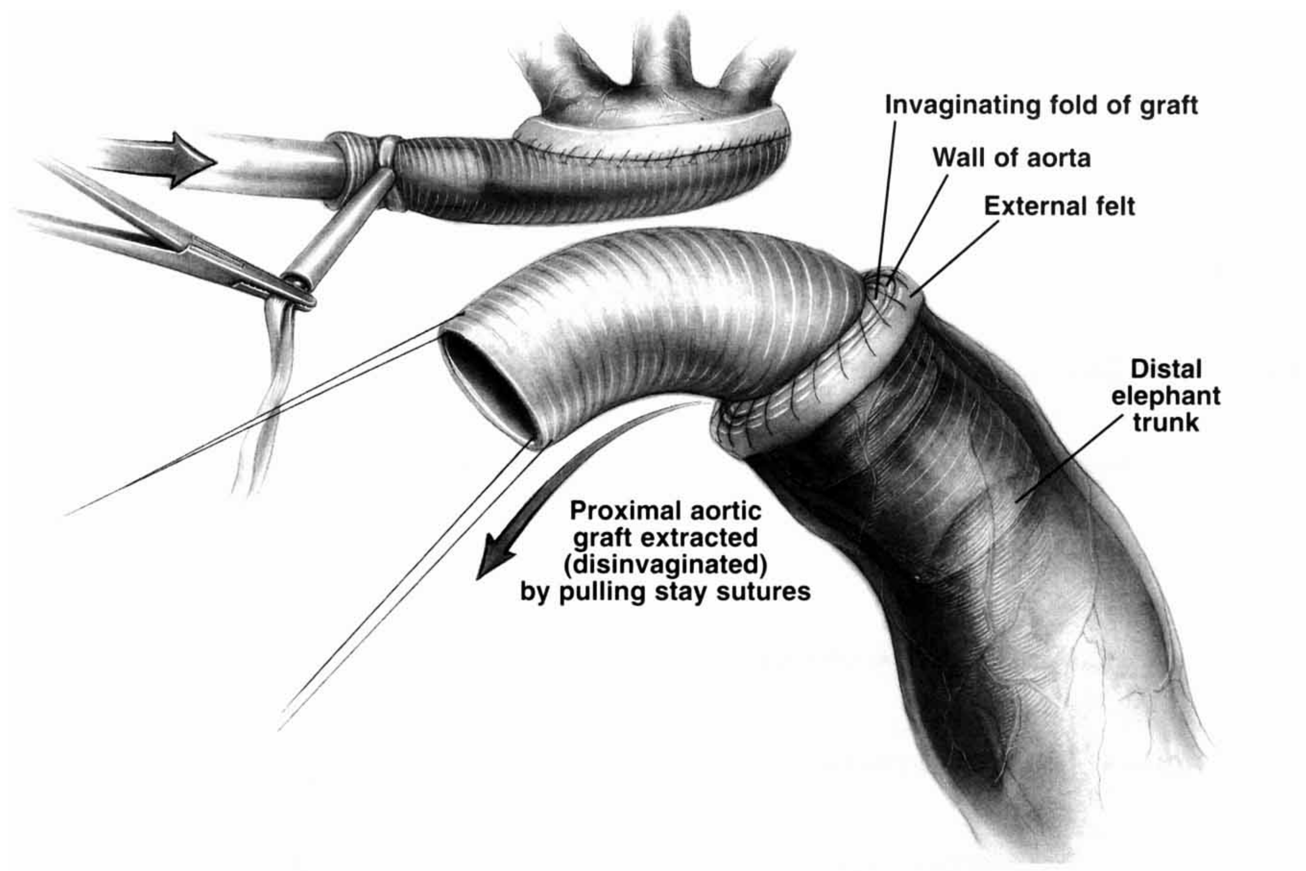
7 Two long stay stitches are sutured to the proximal end of a Hemashield woven double velour Dacron graft; graft size is selected on the basis of the preoperative computed tomographic scan (considering that the true and false lumens in the descending aorta will form one common aortic chamber) or by passing valve sizers into the descending aorta after the dissection flap is excised. The future arch segment of the graft is invaginated into the elephant trunk portion, which should be only 5 to 10 cm long to avoid kinking or occlusion of one of the distal aortic channels. The doubled-up graft is inserted into the descending aorta such that the distal aortic anastomosis between the invaginating fold of the graft and the buttressed cuff of the descending thoracic aorta can be accomplished precisely under direct vision in an end-to-end, full-thickness manner.



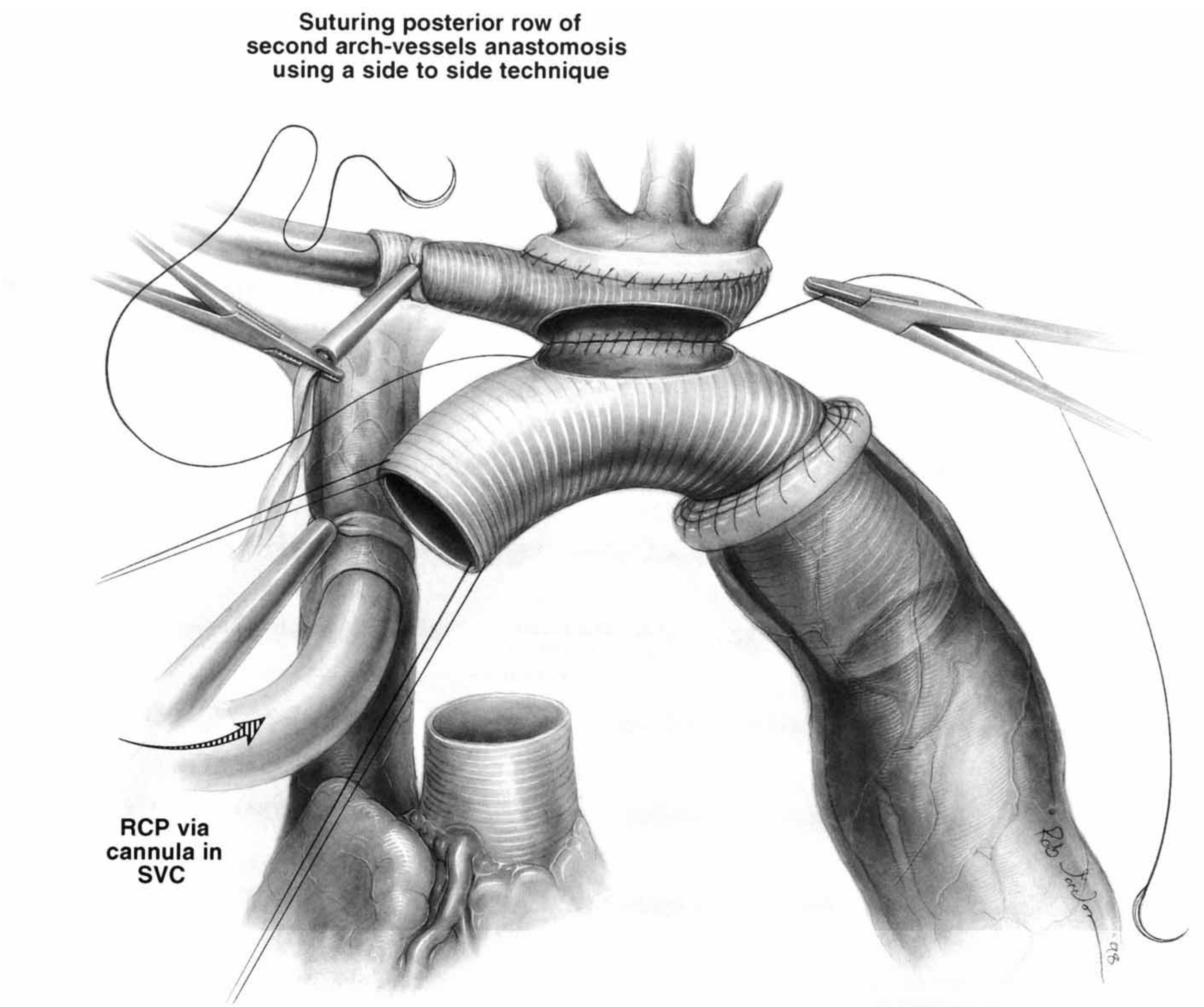
8 A Teflon strip is occasionally used to reinforce the adventitial layer of the dissected native aorta; this step is important if the aortic diameter at this point is much larger than the graft. Similarly, it is essential in the rare patients with acute aortic dissections who require complete transverse arch replacement. Starting in the posterior corner, the posteromedial aspect of the anastomosis is completed with a 137 cm 3-0 SH Prolene suture through the invaginated fold of the graft, inside-to-outside the aorta, and then through the Teflon felt strip. Tightening the posterior suture line with a nerve hook at this stage is important.



9 The distal aortic anastomosis is then completed anteriorly by running the other arm of the Prolene suture upward. This difficult anastomosis is best completed with a combination of forehand and backhand suture placement by both the surgeon and the first assistant. Meticulous attention to detail during this distal anastomosis is paramount to prevent troublesome bleeding or late pseudoaneurysm development, because most of this anastomosis is here-after inaccessible.

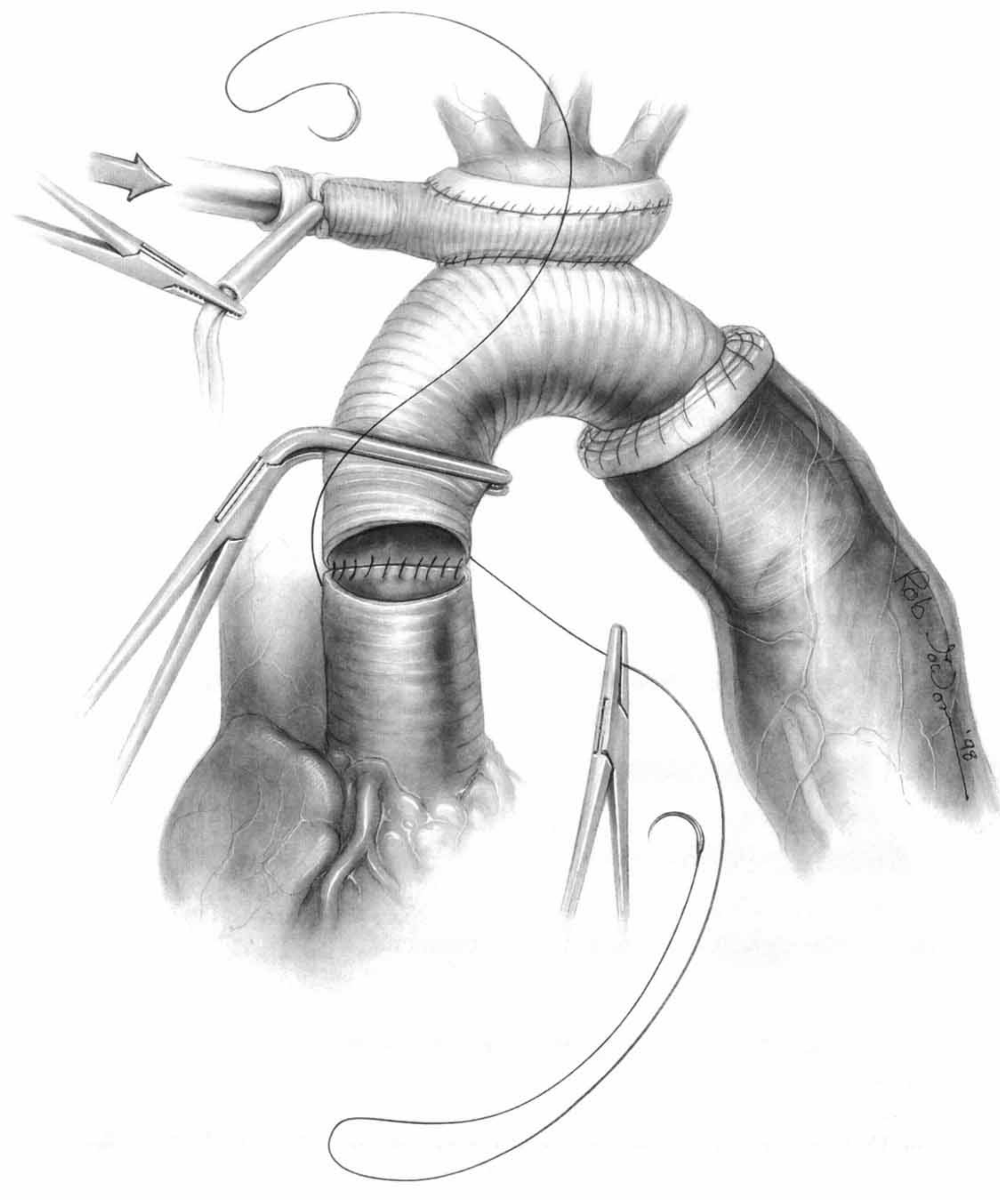


10 The stay stitches on the (proximal) end of the graft are then pulled out to extract the invaginated portion of the graft, and the distal elephant trunk graft is inspected for kinks and length. One should be able to see directly into both the true and the false lumens distally.

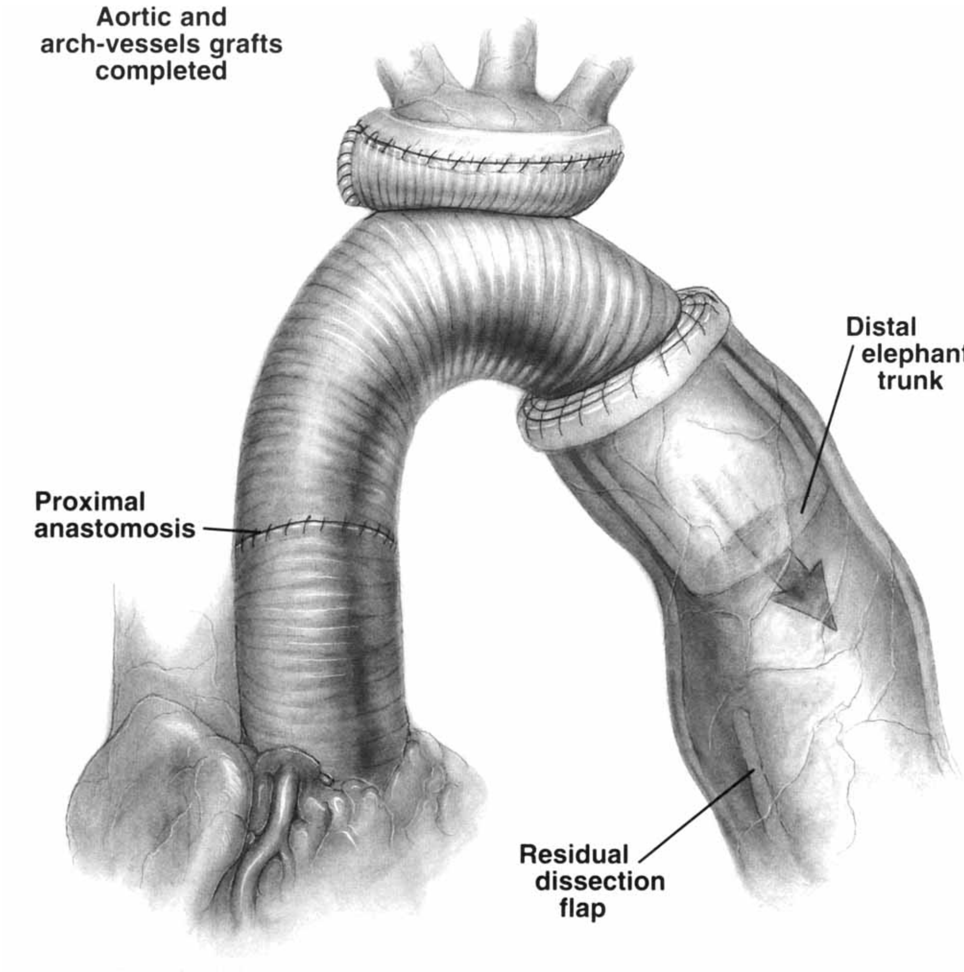


11 SACP is discontinued, the IVC cannula is clamped, and RCP is started again. Equal-sized ellipses (approximately 4 cm × 1 cm) are excised from the inferior portion of the great vessel graft and the superior portion of the main aortic arch graft, and a side-to-side anastomosis between the two grafts is completed using a 4-0 SH-1 Prolene suture. The distal aorta is allow to fill passively during this anastomosis to remove air, which is aided by return of dark blood backward down the carotid arteries.

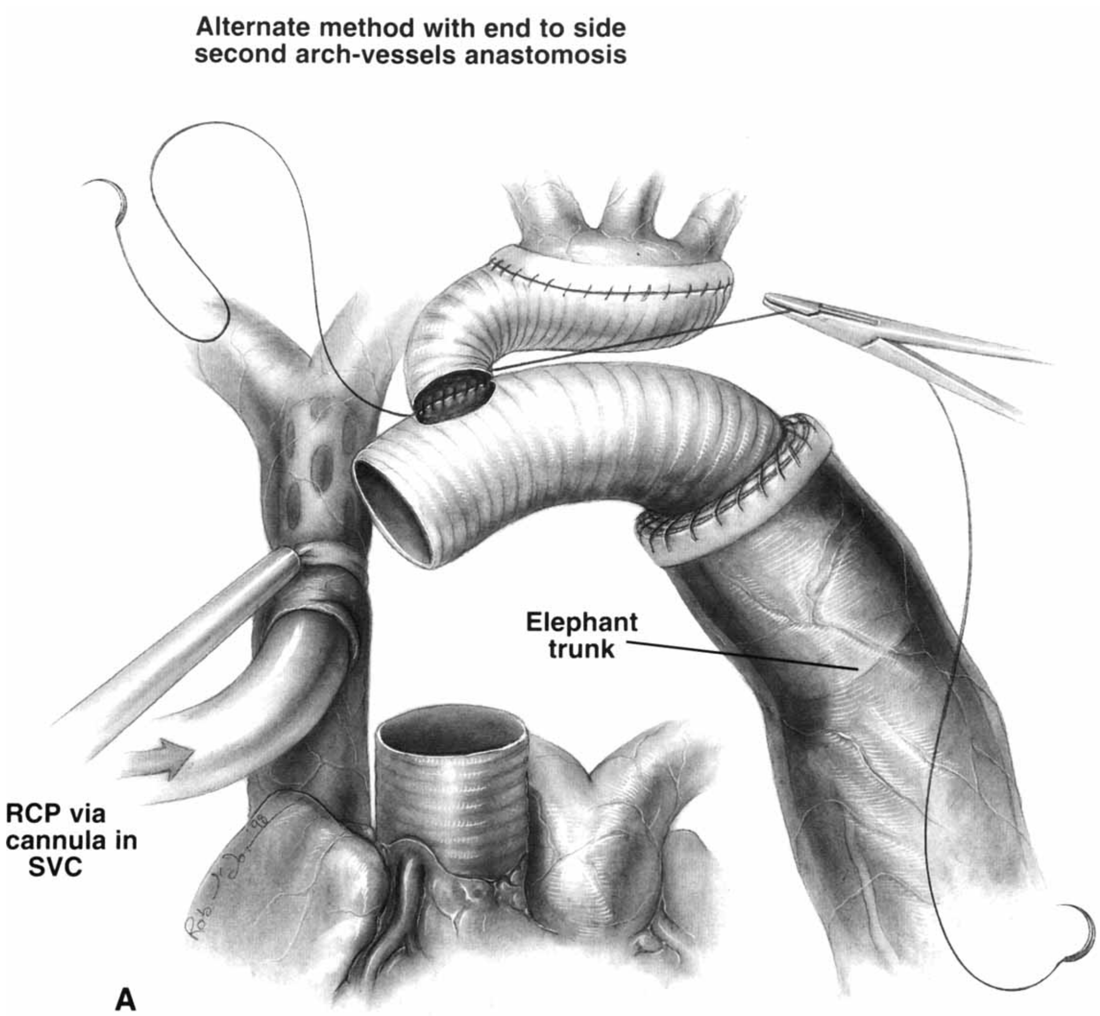
Suturing proximal anastomosis between new and previous aortic grafts



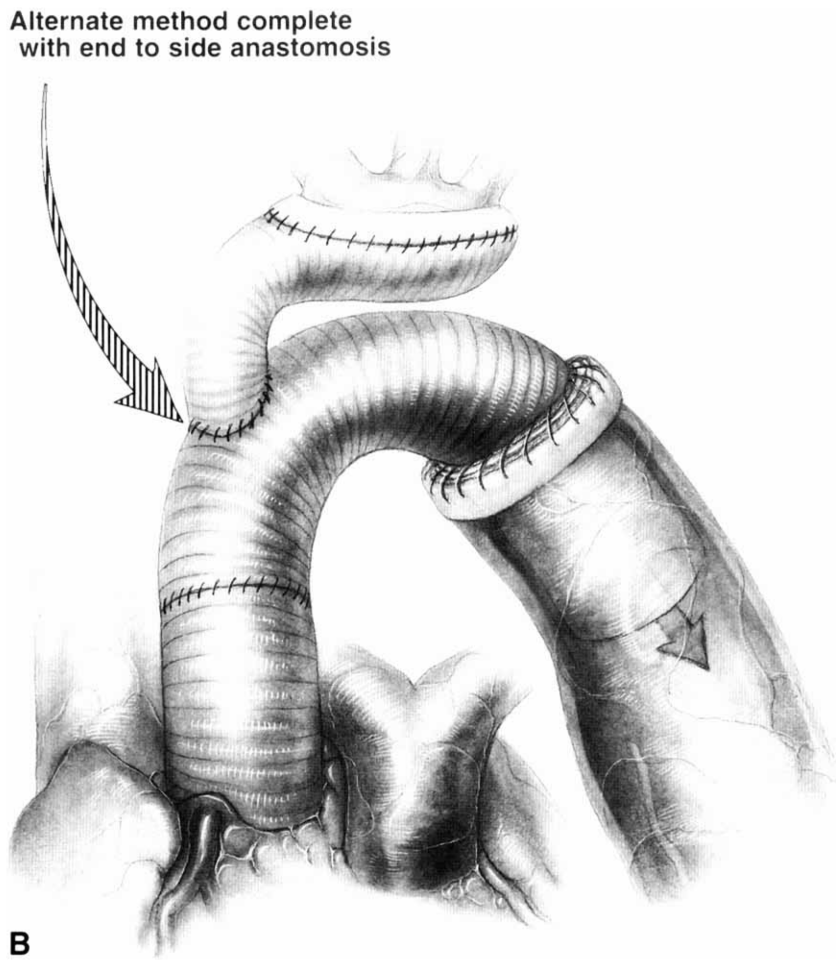
12 The head vessels are again de-aired, a cross-clamp is placed on the proximal aortic arch graft, and antegrade CPB perfusion resumed throughout the body using the cannula in the end of the great vessel graft. Alternatively, angled arterial cannula is inserted via a stab in the arch graft. Systemic rewarming is also started. Depending on the particular anatomical situation, the proximal anastomosis is then completed to an existing ascending aortic graft or to the native proximal aorta, with or without replacement of the aortic valve, as previously described.[19,50] A few of these patients have not had an ascending aortic procedure done previously, in which case the most appropriate aortic root reconstruction is done now. In this example, a graft-to-graft anastomosis is completed with a 4-0 SH-1 Prolene suture. A common error is to make the arch graft segment too long, predisposing to kinking of the graft or aortic valve regurgitation if the native valve is preserved. The ascending graft should be beveled, but, as described by Griepp,[49] such that the "toe" actually goes to the greater curve of the arch near the innominate artery; this maneuver foreshortens the graft and helps prevent kinking or bowing of the graft to the patient's right.



13 Prior to removal of the cross-clamp, 1,000 to 1,500 mL of warm blood cardioplegia (20 mEq/L potassium) is infused retrograde, the pulmonary artery or LV vent is clamped, the vena caval tourniquets are released, the lungs are ventilated, and the heart is filled with blood to expel air. An aspirating vent is placed into the ascending aortic graft, and the heart is meticulously de-aired prior to removal of the cross-clamp. Systemic rewarming generally takes 2 to 3 times longer than cooling (approximately 90 minutes), and should progress at 1°C every 3 to 5 minutes. The temperature gradient between arterial infusion and venous return blood should be less 10°C during rewarming to prevent bubble formation, and we agree with Griepp's group that the arterial blood temperature should never exceed 37°C in order not to exacerbate neurological injury. After resuscitation of the heart and rewarming of the patient to 36°C to 37°C, CPB is discontinued in the usual fashion. The arterial cannula is removed and either the end of the great vessel graft or the stab wound in the arch graft are oversewn.



14 An alternative method for reconstituting flow to the arch vessels (in lieu of that depicted in 11) is with an end-to-side anastomosis between the great vessel graft (after removing the cannula used for SACP) and the main aortic arch graft. 4-0 SH-1 Prolene suture is used for this anastomosis, which was originally described by Griepp and associates.[41,49] In this case, antegrade CPB perfusion is resumed throughout the body using an angled arterial cannula, which is inserted into the arch graft (not shown).



14 (continued)

Postoperative Hemostasis

We routinely administer high dose (30gm) ϵ-aminocaproic acid during almost all CPB cases, including arch replacement and redo arch replacement, and try to avoid the use of aprotinin in circulatory arrest cases because of the uncertainty about diffuse thrombosis after periods of no or low blood flow. If aprotinin is used, the circulating whole blood heparin concentration is always kept above 3.5 U/mL, and the infusion is stopped during the circulatory arrest interval. The most important factor concerning postoperative bleeding is precise, meticulous surgical technique and assiduous attention to the aortic tissue, which can be very fragile; however, circulatory arrest times of greater than 30 to 45 minutes and CPB times exceeding 4 hours can be expected in some patients undergoing primary or redo transverse arch replacement for chronic aortic dissection, and rapid coagulation factor replacement (including 4 to 5 units of fresh frozen plasma, 6 to 12 units of platelets, and 10 units of cryoprecipitate) can help combat the coagulopathy that can accompany these procedures. Patience and time dedicated to careful inspection of all visible anastomoses, coupled with multiple infusions of coagulation factors when necessary, usually allows one to achieve satisfactory hemostasis before leaving the operating suite. In Europe, "French glue" (gluteraldehyde resorcine formol; GFR) and other glues and adhesives have been advocated to reapproximate the intimal and adventitial aortic layers that aid in controlling hemorrhage, but this is most applicable to patients with acute dissection; furthermore, these substances are not available in the United States.[15,51]

Surgical Results

Reported series limited to patients with chronic dissections involving the aortic arch do not contain large numbers of patients, but the morbidity and mortality risks today are generally similar to those reported for other operations involving the arch. In eight major reports, the reported average operative mortality rate for aortic arch replacement was 15% (range, 6% to 23%) (Table 1).[14,17,43,48,52-55] The operative mortality rate was similar for patients with degenerative aneurysms (6% to 22%) and dissections (6% to 19%). The reported mortality rates of 8% to 16% for patients with acute aortic dissections undergoing arch replacement are remarkably low, as they are for patients with chronic dissections (2% to 11%); however, most of these reports are from centers that specialize in this field and have extensive experience with thoracic aortic surgery.[14,17,48,54] The expected outcome in all hospitals around the world is probably closer to the somewhat higher rates reported in the Japanese cooperative study that surveyed 49 centers.[43] In summary, in experienced hands the mortality risk should be less than 10% to 15% for arch replacement in patients with chronic dissections. Postoperative hemorrhage requiring reoperation occurred in 3% to 19% of patients, and the risk of permanent neurological injury averaged 6% (range, 3% to 12%) (Table 2). The risks of developing renal dysfunction (5% to 14%), pulmonary insufficiency (5% to 39%), and LV failure or low cardiac output syndrome (7% to 34%) were similar to that seen in patients undergoing other types of thoracic aortic aneurysm repair.

Replacement of the aortic arch for chronic aortic dissection remains one of the most challenging operations in cardiovascular surgery, but with improved perfusion methods, more thoughtful preoperative and intraoperative planning, and expedient, but precise, surgical technique, the success rates have improved markedly over the last decade.

TABLE 1. Operative Mortality for Partial or Total Replacement of the Aortic Arch

Institution	Author	Year of Publication	Years of Study	Inclusion Criteria	Overall Mortality Rate No. (%)	Routine Aneurysms No. (%)	Dissections No. (%)	Acute Dissections No. (%)	Chronic Dissections No. (%)
Texas Heart Institute	Livesay et al[52]	1983	1976-1982	Arch	14/60 (23)	—	—	—	—
New York University	Galloway et al[53]	1989	1978-1988	Ascd & Arch	25/165 (15)	—	—	—	—
Mount Sinai	Ergin et al[48]	1994	1985-1992	Circulatory arrest	30/200 (15)	8/44 (18)	—	9/55 (16)	—
Hannover, Germany	Borst et al[17]	1994	1980-1994	Arch	27/204 (13)	6/58 (10)	21/146 (14)	15/92 (16)	6/54 (11)
Baylor	Coselli et al[54]	1995	1987-1993	Arch	14/227 (6)	7/110 (6)	7/117 (6)	4/48 (8)	3/69 (4)
Japan-cooperative	Usui et al[43]	1996	1990-1992	Circulatory arrest	45/228 (20)	13/58 (22)	32/170 (19)	—	—
Suresnes, France	Bachet et al[55]	1997	1984-1995	Arch	23/138 (17)	—	—	—	—
Osaka, Japan	*Okita et al[14]*	*1998*	*1993-1996*	*Arch*	*15/148 (10)*	*11/78 (14)*	*4/70 (6)*	*3/28 (11)*	*1/42 (2)*

TABLE 2. Postoperative Complications for Partial or Total Replacement of the Aortic Arch

Institution	Author	Stroke No. (%)	Hemorrhage-Reexploration No. (%)	Renal Dysfunction No. (%)	Respiratory Insufficiency No. (%)	Cardiac Dysfunction No. (%)
Texas Heart Institute	Livesay et al[52]	6/60 (10)	11/60 (19)	6/60 (10)	8/60 (13)	6/60 (10)
New York University	Galloway et al[53]	4/165 (2)	—	—	—	—
Mount Sinai	Ergin et al[48]	22/200 (11)	19/200 (10)	19/200 (10)	54/200 (27)	68/200 (34)
Hannover, Germany	Borst et al[17]	10/204 (5)	—	—	—	—
Baylor	Coselli et al[54]	7/227 (3)	7/227 (3)	11/227 (5)	88/227 (39)	16/227 (7)
Japan-cooperative	Usui et al[43]	27/228 (12)	—	32/228 (14)	84/228 (37)	17/228 (7)
Suresnes, France	Bachet et al[55]	5/138 (4)	—	8/138 (6)	7/138 (5)	—
Osaka, Japan	Okita et al[14]	6/148 (4)	18/148 (12)	8/148 (5)	22/148 (15)	10/148 (7)

REFERENCES

1. Hirst AE, Johns VJ, Kime SW: Dissecting aneurysm of the aorta: A review of 505 cases. Medicine 37:217-279, 1958
2. DeBakey ME, McCollum CH, Crawford ES, et al: Dissection and dissecting aneurysms of the aorta: Twenty-year follow-up of five hundred twenty-seven patients treated surgically. Surgery 92:1118-1134, 1982
3. Miller DC, Mitchell RS, Oyer PE, et al: Independent determinants of operative mortality for patients with aortic dissections. Circulation 70:153-164, 1984 (suppl 1)
4. Haverich A, Miller DC, Scott WC, et al: Acute and chronic aortic dissections: Determinants of long-term outcome for operative survivors. Circulation 72:22-34, 1985 (suppl 2)
5. Lansman SL, Raissi S, Ergin MA, et al: Urgent operation for acute transverse aortic arch dissection. J Thorac Cardiovasc Surg 97:334-341, 1989
6. Crawford ES, Svensson LG, Coselli JS, et al: Surgical treatment of aneurysm and/or dissection of the ascending aorta, transverse aortic arch, and ascending aorta and transverse aortic arch: Factors influencing survival in 717 patients. J Thorac Cardiovasc Surg 98:659-674, 1989
7. Svensson LG, Crawford ES, Hess KR, et al: Dissection of the aorta and dissecting aortic aneurysms: Improving early and long-term surgical results. Circulation 82:24-38, 1990 (suppl 4)
8. Kouchoukos NT, Wareing TH, Murphy SF, et al: Sixteen-year experience with aortic root replacement: Results of 172 operations. Ann Surg 214:308-320, 1991
9. Galloway AC, Colvin SB, Grossi EA, et al: Surgical repair of type A aortic dissection by the circulatory arrest-graft inclusion technique in sixty-six patients. J Thorac Cardiovasc Surg 105:781-790, 1993
10. Fann JI, Smith JA, Miller DC, et al: Surgical management of aortic dissection during a 30-year period. Circulation 92:113-121, 1995 (suppl 2)
11. Glower DD, Fann JI, Speier RH, et al: Comparison of medical and surgical therapy for uncomplicated descending aortic dissection. Circulation 82:39-46, 1990 (suppl 4)
12. Miller DC: The continuing dilemma concerning medical versus surgical management of patients with acute type B dissections. Semin Thorac Cardiovasc Surg 5:33-46, 1993
13. Kazui T, Kimura N, Yamada O, et al: Total arch graft replacement in patients with acute type A aortic dissection. Ann Thorac Surg 58:1462-1468, 1994
14. Okita Y, Takamoto S, Ando M, et al: Mortality and cerebral outcome in patients who underwent aortic arch operations using deep hypothermic circulatory arrest with retrograde cerebral perfusion: No relation of early death, stroke, and delirium to the duration of circulatory arrest. J Thorac Cardiovasc Surg 115:129-138, 1998
15. Bachet J, Goudot B, Dreyfus G, et al: The proper use of glue: A 20-year experience with the GFR glue in acute aortic dissection. J Card Surg 12:243-255, 1997
16. Crawford ES, Kirklin JW, Naftel DC, et al: Surgery for acute dissection of ascending aorta: Should the arch be included? J Thorac Cardiovasc Surg 104:46-59, 1992
17. Borst HG, Buhner B, Jurmann M: Tactics and techniques of aortic arch replacement. J Card Surg 9:538-547, 1994
18. Yun KL, Glower DD, Miller DC, et al: Aortic dissection resulting from tear of transverse arch: Is concomitant arch repair warranted? J Thorac Cardiovasc Surg 102:355-370, 1991
19. Yun KL, Miller DC: Technique of aortic valve preservation in acute type A aortic dissection. Op Tech Card Thorac Surg 1:68-81, 1996
20. Moon MR, Dake MD, Pelc LR, et al: Intravascular stenting of acute experimental type B dissections. J Surg Res 54:381-388, 1993
21. Slonim SM, Nyman U, Semba CP, et al: Aortic dissection: Percutaneous management of ischemic complications with endovascular stents and balloon fenestration. J Vasc Surg 23:241-253, 1996
22. Slonim SM, Nyman UR, Semba CP, et al: True lumen obliteration in complicated aortic dissection: Endovascular treatment. Radiology 201: 161-166, 1996
23. Crawford ES, Coselli JS, Safi HJ: Partial cardiopulmonary bypass, hypothermic circulatory arrest, and posterolateral exposure for thoracic aortic aneurysm operation. J Thorac Cardiovasc Surg 94:824-827, 1987
24. Caramutti VM, Dantur JR, Favaloro MR, et al: Deep hypothermia and circulatory arrest as an elective technique in the treatment of type B dissecting aneurysm of the aorta. J Card Surg 4:206-215, 1989
25. Stone CD, Greene PS, Gott VL, et al: Single-stage repair of distal aortic arch and thoracoabdominal dissecting aneurysms using aortic tailoring and circulatory arrest. Ann Thorac Surg 57:580-587, 1994
26. Miller DC: Surgical management of acute aortic dissection: New data. Semin Thorac Cardiovasc Surg 3:225-237, 1991
27. Guthaner DF, Miller DC, Silverman JF, et al: Fate of the false lumen following surgical repair of aortic dissections: An angiographic study. Radiology 133:1-8, 1979
28. Ergin MA, Phillips RA, Galla JD, et al: Significance of distal false lumen after type A dissection repair. Ann Thorac Surg 57:820-825, 1994
29. Pressler V, McNamara JJ: Thoracic aortic aneurysm: Natural history and treatment. J Thorac Cardiovasc Surg 79:489-498, 1980
30. Hirose Y, Hamada S, Takamiya M, et al: Aortic aneurysms: Growth rates measured with CT. Radiology 185:249-252, 1992
31. Dapunt OE, Galla JD, Sadeghi AM, et al: The natural history of thoracic aortic aneurysms. J Thorac Cardiovasc Surg 107:1323-1333, 1994
32. Juvonen T, Ergin MA, Galla JD, et al: Prospective study of the natural history of thoracic aortic aneurysms. Ann Thorac Surg 63:1533-1545, 1997
33. Coady MA, Rizzo JA, Hammond GL, et al: What is the appropriate size criterion for resection of thoracic aortic aneurysms? J Thorac Cardiovasc Surg 113:476-491, 1997
34. Pyeritz RE: Marfan syndrome: Current and future clinical and genetic management of cardiovascular manifestations. Semin Thorac Cardiovasc Surg 5:11-16, 1993
35. Hwa J, Richards JG, Huang H, et al: The natural history of aortic dilatation in Marfan syndrome. Med J Aust 158:558-562, 1993
36. Borst HG, Frank G, Schaps D: Treatment of extensive aortic aneurysms by a new multiple-stage approach. J Thorac Cardiovasc Surg 95:11-13, 1988
37. Crawford ES, Coselli JS, Svensson LG, et al: Diffuse aneurysmal disease (chronic aortic dissection, Marfan, and mega aorta syndromes) and multiple aneurysm: Treatment by subtotal and total aortic replacement emphasizing the elephant trunk operation. Ann Surg 211:521-537, 1990
38. Sabik JF, Lytle BW, McCarthy PM, et al: Axillary artery: An alternative site of arterial cannulation for patients with extensive aortic and peripheral vascular disease. J Thorac Cardiovasc Surg 109:885-891, 1995
39. Mezrow CK, Midulla PS, Sadeghi AM, et al: Evaluation of cerebral metabolism and quantitative electroencephalography after hypothermic circulatory arrest and low-flow cardiopulmonary bypass at different temperatures. J Thorac Cardiovasc Surg 107:1006-1019, 1994
40. Mezrow CK, Gandsas A, Sadeghi AM, et al: Metabolic correlates of neurologic and behavioral injury after prolonged hypothermic circulatory arrest. J Thorac Cardiovasc Surg 109:959-975, 1995
41. Griepp RB, Ergin MA, McCullough JN, et al: Use of hypothermic circulatory arrest for cerebral protection during aortic surgery. J Card Surg 12:312-321, 1997
42. Midulla PS, Gandsas A, Sadeghi AM, et al: Comparison of retrograde cerebral perfusion to antegrade cerebral perfusion and hypothermic circulatory arrest in a chronic porcine model. J Card Surg 9:560-575, 1994
43. Usui A, Abe T, Murase M: Early clinical results of retrograde cerebral perfusion for aortic arch operations in Japan. Ann Thorac Surg 62:94-104, 1996
44. Griepp RB, Juvonen T, Griepp EB, et al: Is retrograde cerebral perfusion an effective means of neural support during deep hypothermic circulatory arrest? Ann Thorac Surg 64:913-916, 1997
45. Coselli JS, LeMaire SA: Experience with retrograde cerebral perfusion during proximal aortic surgery in 290 patients. J Card Surg 12:322-325, 1997
46. Juvonen T, Weisz DJ, Wolfe D, et al: Can retrograde perfusion mitigate cerebral injury after particulate embolization? A study in a chronic porcine model. J Thorac Cardiovasc Surg 115:1142-1159, 1998

47. Svensson LG, Crawford ES, Hess KR, et al: Deep hypothermia with circulatory arrest: Determinants of stroke and early mortality in 656 patients. J Thorac Cardiovasc Surg 106:19-31, 1993
48. Ergin MA, Galla JD, Lansman SL, et al: Hypothermic circulatory arrest in operations on the thoracic aorta: Determinants of operative mortality and neurologic outcome. J Thorac Cardiovasc Surg 107:788-799, 1994
49. Griepp RB, Ergin MA: Aneurysms of the aortic arch, in Edmunds LH (ed): Cardiac Surgery in the Adult. San Francisco, CA, McGraw-Hill Companies, Inc, 1997, pp 1197-1226
50. Miller DC, Mitchell RS: Composite aortic valve replacement and graft replacement of the ascending aorta plus coronary ostial reimplantation: How I do it. Semin Thorac Cardiovasc Surg 5:74-83, 1993
51. Laas J, Jurmann MJ, Heinemann M, et al: Advances in aortic arch surgery. Ann Thorac Surg 53:227-232, 1992
52. Livesay JJ, Cooley DA, Reul GJ, et al: Resection of aortic arch aneurysms: A comparison of hypothermic techniques in 60 patients. Ann Thorac Surg 36:19-28, 1983
53. Galloway AC, Colvin SB, LaMendola CL, et al: Ten-year operative experience with 165 aneurysms of the ascending aorta and aortic arch. Circulation 80:249-256, 1989 (suppl 1)
54. Coselli JS, Buket S, Djukanovic B: Aortic arch operation: Current treatment and results. Ann Thorac Surg 59:19-27, 1995
55. Bachet J, Goudot B, Dreyfus G, et al: How do we protect the brain? Antegrade selective cerebral perfusion with cold blood during aortic arch surgery. J Card Surg 12:193-200, 1997 (suppl)

From the Department of Cardiovascular and Thoracic Surgery, Stanford University School of Medicine, Stanford, CA.

Address reprint requests to D. Craig Miller, MD, Department of Cardiovascular and Thoracic Surgery, Falk Cardiovascular Research Center, Stanford University School of Medicine, Stanford, CA 94305-5247.

1522-2942/99/0401-0003$10.00/0