# Surgical Management of Aortic Dissection

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Aortic dissection, the most common acute catastrophe involving the thoracic aorta, occurs 2 to 3 times as often as rupture of an aneurysm of the abdominal aorta. Each year in the United States more than 60,000 aortic dissections are reported.

The 1st attempt to repair a dissecting aneurysm took place in 1935,[1] when Gurin and colleagues performed a local "fenestration" procedure on the iliac artery. In 1955, DeBakey, Cooley, and Creech[2] reported excellent results after transthoracic treatment of 6 patients with use of the fenestration technique. Later reports stressed the importance of resecting the aneurysm, closing the false channel, and interposing a tube graft for definitive treatment.[3,4]

Because of high mortality rates, some investigators began to question the efficacy of surgical treatment.[5,6] Consequently, Wheat and Palmer and their associates[7,8] proposed medical treatment as an alternative to surgical correction. In their hands, a medical regimen consisting of antihypertensive and negative inotropic drugs resulted in a patient mortality of 10% to 20%, which appeared favorable when compared to other published mortality rates (21% to 90%) associated with the direct surgical approach at that time.[3,5,6,9-17] Because of the decreases in surgical mortality during the past decade, however, treatment has shifted again toward immediate surgical intervention for dissection in the ascending aorta. In acute dissections of the descending aorta, the trend is toward medical management to stabilize the patient and subsequent surgery, unless signs of acute or impending rupture develop. This review describes acute dissecting aneurysms and provides operative techniques for treating these difficult lesions.

## Anatomy and Pathophysiology

Acute aortic dissections are characterized by a separation within the wall of the aorta, usually in the medial layer, although the separation may be subadventitial in some areas. The origin of the term "dissecting" aneurysm is attributed to Laennec (1819). Although not the 1st physician to describe this lesion, Maunoir[18] published a vivid account of it in 1802: "Here the internal coat breaks at a given point, and the external coat is pouched out; and it alone opposes the effusion of blood which passes through the tear of the internal tunic . . . the pocket enlarges, and the blood sometimes dissects the artery in its entire circumference, and it (the aorta) is found in the center of the aneurysm entirely bathed in the aneurysmal blood."

With respect to cause and pathologic features, dissecting aneurysms of the descending aorta differ from those of the ascending aorta. In approximately two-thirds of the cases, dissecting descending aortic aneurysms are caused by atherosclerosis;[19] in an additional 20%, Erdheim's cystic medial necrosis is responsible. In contrast, ascending aortic dissection is usually caused by cystic medial necrosis. Both types of dissecting aneurysms are also associated with coarctation of the aorta and trauma; the proximal portion of the descending aorta is a common site of traumatic lesions.

The typical aortic dissection patient is a man, between 45 and 70 years of age, with a history of essential hypertension, although normotensive persons may also experience aortic dissection. Men tend to be affected 3 to 4 times as often as women. Most lesions begin as an intimal tear or split that is usually transverse and may be multiple. Such tears permit blood to enter the medial layer, forming a false channel. Alternatively, an intramural hemorrhage, particularly one that results from stretching of the aorta, may cause a spreading hematoma that perforates secondarily

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into the lumen. The most common area of predilection to dissection is in the ascending aorta (Type A), about 2 to 3 centimeters above the origin of the right and left coronary arteries. Another frequent site is the aortic isthmus, just distal to the left subclavian artery (Type B). The dissection tends to spread both proximally and distally, until the intramural blood finally reenters the true aortic lumen. In a large percentage of patients, the lesion extends progressively to involve all of the thoracic and abdominal aorta.

Clinical manifestations arise not only from the dissection itself, but also from the occlusion of aortic tributaries, which may also be sheared off by the dissecting process. Proximally, damage may extend to the coronary arteries and the aortic valve cusps, as well as the subclavian and intercostal arteries. Carotid artery involvement may result in neurologic deficits. The spinal arteries may be occluded, thereby causing temporary or even permanent paraplegia. If dissection and occlusion of the visceral vessels occur, the celiac, superior mesenteric, and renal arteries can be affected. Involvement and occlusion of a renal artery may produce additional hypertension and oliguria, thus aggravating the basic condition. Dissection extending to the iliac arteries, especially the left, will cause the corresponding lower extremity to become pulseless. No matter which portion of the aorta is affected, the adjoining tissues are usually subjected to compression, erosion, and infarction. In the absence of timely surgical intervention, the patient eventually succumbs to fatal aortic rupture, commonly into the pericardial or the left pleural cavity.

## Diagnosis

Acute dissecting aneurysms of the aorta are usually heralded by a sudden tearing, excruciating pain in the intrascapular or precordial area that occasionally radiates into the back or legs (in the case of a descending aortic aneurysm) or into the neck or arms (in the presence of an ascending aortic lesion). The differential diagnosis at the onset of pain may be dissection of the thoracic aorta, myocardial infarction, pulmonary embolus, rupture of a sinus of Valsalva aneurysm, aortoiliac occlusion, stroke, or perforated peptic ulcer. The pain tends to follow the path of dissection; it is rarely relieved by narcotics, and it persists in untreated individuals. Although most patients appear to be in a state of low perfusion, their systolic and diastolic blood pressures are far above their usual levels.

A number of methods can be used to diagnose acute dissecting aneurysms, including standard chest radiography, computed tomography, digital angiography, echocardiography, magnetic resonance imaging, and aortography.[20] The chest roentgenogram is abnormal in more than 90% of these patients, generally showing a dilated aorta, wide mediastinum, and effusion. Aortography remains the definitive diagnosis. Acute dissections are classified as those in which dissection, as evidenced by characteristic chest pain, has occurred within 2 weeks before admission. About half of our patients have had acute dissections, with acute inflammation of the aorta and relatively fresh blood clot in the false channel at the time of surgery.

## Treatment

When a patient's dissecting aneurysm is diagnosed, the physician must determine whether the lesion arises from the ascending or descending aorta, since there is a considerable difference in prognosis, depending upon the point of origin. Ascending aortic lesions are seriously life-threatening, due to the risk of intrapericardial hemorrhage and fatal cardiac tamponade. Therefore, acute ascending dissections should be regarded as emergencies and subjected to immediate surgical treatment.

In contrast, acute dissections of the descending aorta do not tend to be immediately life-threatening;[12] once this lesion has been diagnosed, the patient's condition can be stabilized before surgery, since imminent rupture is unlikely. Hypotensive agents such as nitroprusside, trimethaphan camsylate (Arfonad, Roche Laboratories; Nutley, New Jersey, USA), or so-called beta-blocking drugs may be used during the initial phase to reduce the tendency of the dissection to spread. We believe that most acute lesions should be operated on during the original period of hospitalization and observation; some may require urgent operation. Early surgery reduces the possibility of subsequent rupture and expansion, the latter being an almost inevitable consequence. A history of paraplegia, or paraparesis, and the absence of pulsation in the femoral arteries, particularly the left femoral artery, are indications for early surgery. Occasionally, a chronic, descending dissection may be discovered incidentally. We do not operate on patients with this condition if they are normotensive and their aneurysms are asymptomatic and not expanding.

## Anesthetic Management

Sudden increases in blood pressure should be avoided in the presence of an acute dissection. During the period of aortic cross-clamping for Type-B lesions, the anesthetist must be alert to changes in blood pressure. With proximal aortic occlusion, we believe that it is normal for the blood pressure to rise by 20 to 40 mmHg. When systolic pressure reaches 180 to 200 mmHg, however, cautious use of vasodilators is indicated. We prefer nitroprusside infusion under these circumstances, although deepening the level of anes-

thesia will suffice in most instances. In the occasional patient who develops hypotension during the period of cross-clamping, the blood volume must be restored with whole blood or components. Vasopressors may be administered cautiously to maintain a physiologic level of blood pressure. For this purpose, we use either dopamine or phenylephrine (Neo-Synephrine, Winthrop-Breon Laboratories; New York, New York, USA). Since blood loss may be a problem during surgery, we occasionally use the cell-saver[21] or other methods of autotransfusion with or without cardiopulmonary bypass. Fresh frozen plasma and platelet transfusions, when necessary, are used to maintain blood volume and to enhance clotting.

## Graft Preclotting

In 1980, a new method of pretreating grafts was introduced to reduce graft-related hemorrhage.[22] Woven fabric Dacron grafts are soaked in the patient's own plasma and autoclaved to seal the interstices of the fabric and prevent bleeding. After approval by the FDA, grafts pretreated with bovine collagen will be available to provide a reliable, nonporous prosthesis.

## Surgical Technique

For the most part, operations for aortic dissection should be considered palliative in nature, since total removal is uncommon. The principal objective is to prevent rupture of the jeopardized segment, which is the primary cause of mortality. If the major portion of the aneurysmal sac is removed, this objective is accomplished. In some of the larger, chronic lesions, the aneurysm should be repaired because of encroachment upon such anatomic structures as the superior vena cava, pulmonary arteries, left main-stem bronchus, and left lung. In a relatively small number of patients, localized aortic dissections occur; surgical "cure" of such localized lesions may be accomplished by placing the anastomosis in normal aortic wall, proximal and distal to the diseased or dissected area.

### Dissection of the Ascending Aorta

For acute ascending aortic aneurysms, the operation is performed through a median sternotomy, with cannulation of the left femoral artery and the right atrium. "Open" repair of the distal portion of the dissection in the transverse arch is preferable to the standard method of cross-clamping and attempting repair of the aorta proximal to the aortic cross-clamp (Fig. 1). Repair of dissection at the aortic arch level usually involves no more than 20 minutes of circulatory arrest. The aorta should be completely transected distally to ensure that both layers of aortic wall involved in the dissection are included in the suture line. The proximal portion of the dissection may affect the attachment of the aortic valve and orifices. We try to repair the valve, if possible—especially when the valve is only modestly insufficient. When repair cannot be done, valve replacement may be necessary (Fig. 2). In most cases, the adventitial and intimal layers can be sutured together, thereby obliterating the false lumen. Reinforcement of the distal suture line with an external "wrap" by using a Dacron vascular graft or felt strip is both useful and advisable. Because distal dissection does not generally undergo extension after proximal repair, attention to the distal segment may be postponed safely for months or even years.

### Dissection of the Descending Aorta

Acute lesions of the descending aorta are repaired by using similar techniques (Fig. 3). At present, in our bypass techniques, we rely on expeditious repair rather than pumps or shunts to limit the period of circulatory interruption. Recently, we have advocated open distal repair for aneurysms of the descending aorta, and it appears as effective as open distal repair for aneurysms of the ascending aorta. When the dissection extends proximally in the transverse aortic arch, the proximal cross-clamp may be situated in the transverse aorta. In 1982, we reported on a series[23] of 30 consecutive patients in which we placed the clamp between the innominate and the left common carotid arteries. No patients in this series showed neurologic complications from temporary left common carotid occlusion.[19] Moreover, none of the patients demonstrated left ventricular strain due to the proximal level of the temporary occlusion, which allows blood to exit the heart only through the single innominate artery. Neither is the incidence of paraplegia or renal failure affected by the level of placement of the proximal clamp. We continue to use this technique without complications. The anesthesiologists must remain alert, however, to excessive elevations of blood pressure.

In lesions of the descending aorta, the condition of the abdominal aorta, where the visceral vessels have been separated from the true lumen, may be a cause for concern. In general, we have concentrated on removal of the point of origin and a major segment of the remaining dissected aorta, usually above the aortic hiatus. When the dissection extends into the abdominal aorta, the mediastinal pleura may be opened over the aneurysm, and repair done above the diaphragm. Any tissue adhering to the lung should not be dissected or divided, since this may lead to subsequent diffuse hemorrhage. Total replacement of the abdominal aorta with reimplantation of the vessels is not necessary and may not be practical, particularly in the acute lesion. In the chronic lesion, however, the abdominal aorta may be completely replaced.

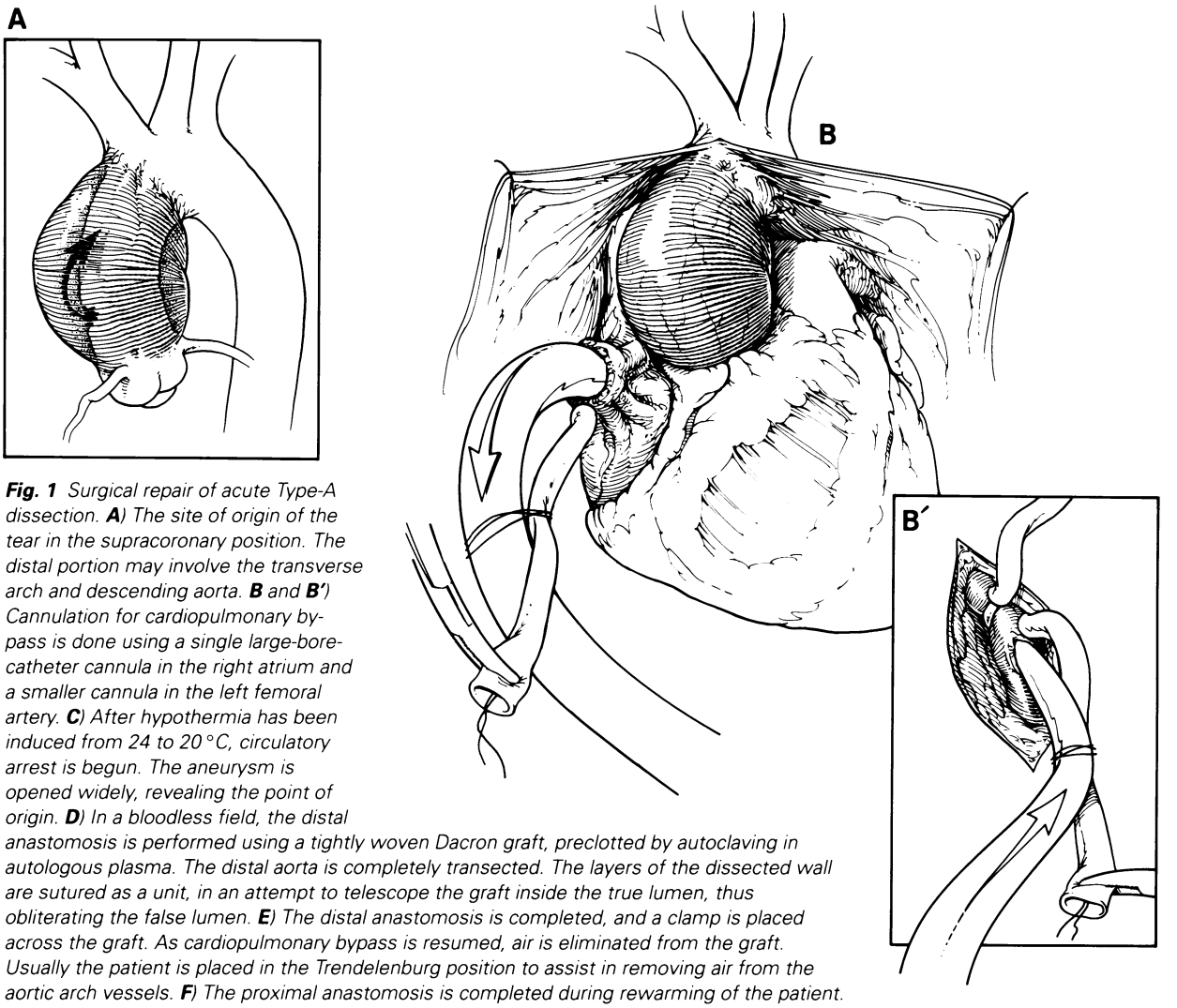


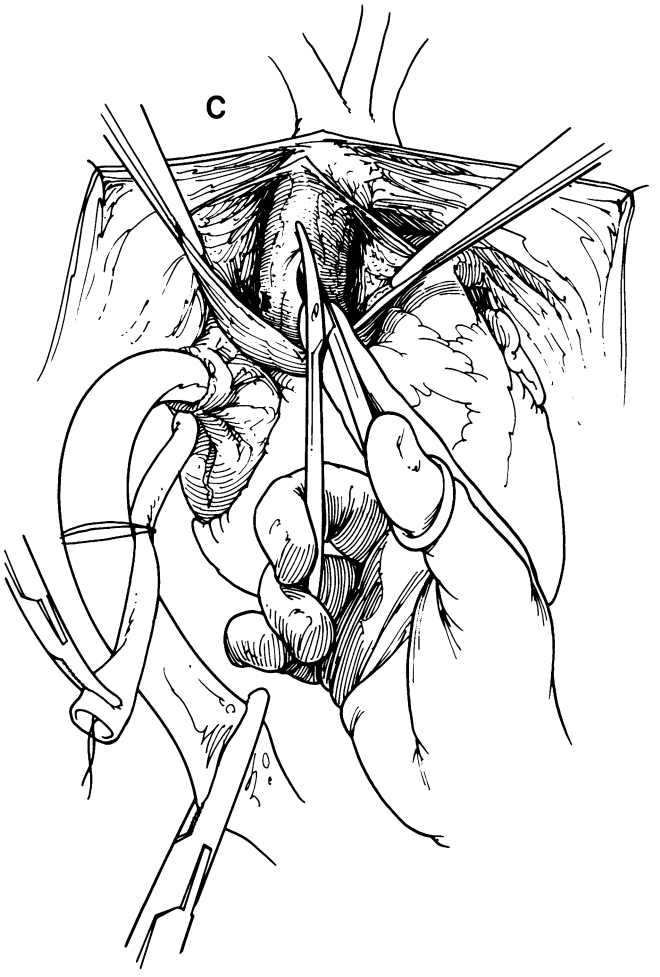

***Fig. 1*** *Surgical repair of acute Type-A dissection.* ***A****) The site of origin of the tear in the supracoronary position. The distal portion may involve the transverse arch and descending aorta.* ***B*** *and* ***B′****) Cannulation for cardiopulmonary bypass is done using a single large-bore-catheter cannula in the right atrium and a smaller cannula in the left femoral artery.* ***C****) After hypothermia has been induced from 24 to 20 °C, circulatory arrest is begun. The aneurysm is opened widely, revealing the point of origin.* ***D****) In a bloodless field, the distal anastomosis is performed using a tightly woven Dacron graft, preclotted by autoclaving in autologous plasma. The distal aorta is completely transected. The layers of the dissected wall are sutured as a unit, in an attempt to telescope the graft inside the true lumen, thus obliterating the false lumen.* ***E****) The distal anastomosis is completed, and a clamp is placed across the graft. As cardiopulmonary bypass is resumed, air is eliminated from the graft. Usually the patient is placed in the Trendelenburg position to assist in removing air from the aortic arch vessels.* ***F****) The proximal anastomosis is completed during rewarming of the patient.* ***G****) After cardiopulmonary bypass is discontinued and heparinization is reversed with protamine sulfate, the remaining aneurysmal wall is sutured around the graft. In dissecting lesions, adding a reinforcing cuff of Dacron is helpful to eliminate bleeding from suture holes and to support the distal anastomosis.*

*(From: Cooley DA,[11] with permission from the publisher.)*

If large intercostal arteries are present in the nondissected posterior wall of the aorta, we preserve them by tapering the graft appropriately, so that the distal anastomosis includes the large intercostal arteries, some of which may supply the spinal cord. Using open distal repair has expedited the surgical procedure and improved the outcome in patients with descending thoracic aneurysms. None of our patients has developed paraplegia since we began using this technique, but the reason for this positive result is not yet clear.

## Complications

During repair of a descending aortic dissection, the most serious potential complication is paraplegia; therefore, a principal concern during operation is avoiding spinal cord injury. The variable anatomy and segmental derivation of the spinal cord circulation is known.[24,25] The blood supply of the spinal cord is derived from segmental radicular arteries that feed the anterior spinal artery and the twin dorsal spinal arteries. Intercostal arteries supplying the middle thoracic segments are often ligated from within the aneurysm, thereby reducing collateral circulation. The portion of the spinal cord most susceptible to injury is supplied by the large, unpaired arteria radicularis magnus, or the great radicular artery, described by Adamkiewicz.[26] Although this artery may originate anywhere between the T-8 and the L-3 levels, it arises somewhere between T-9 and T-12 in 75% of patients. The tenuous collateral anastomosis of the anterior spinal artery in the midthoracic region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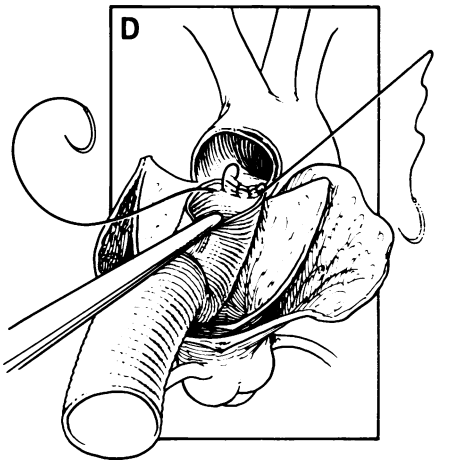
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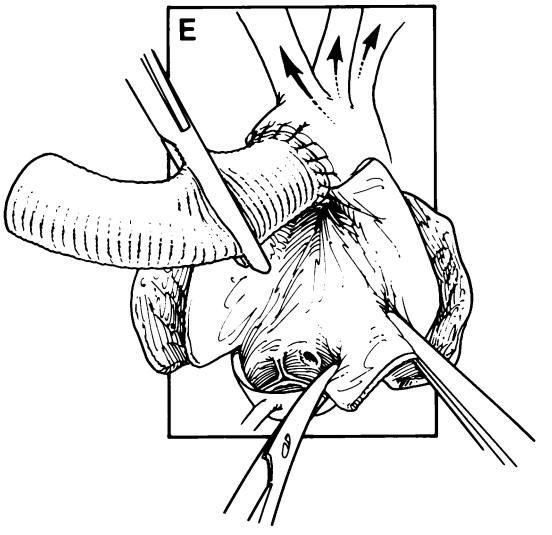
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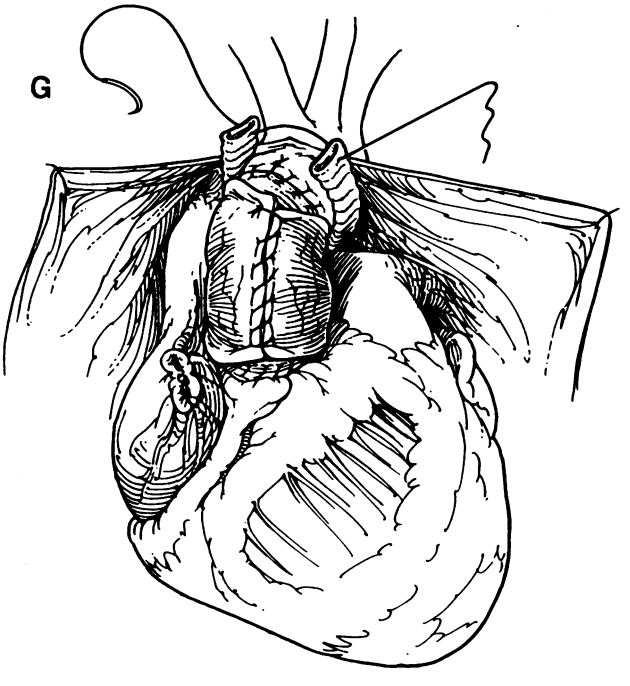
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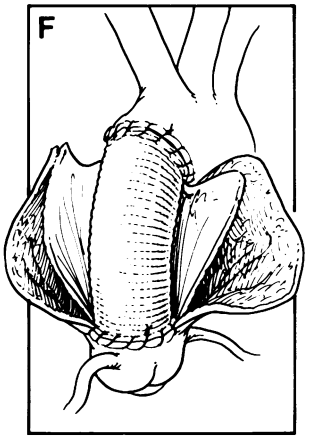
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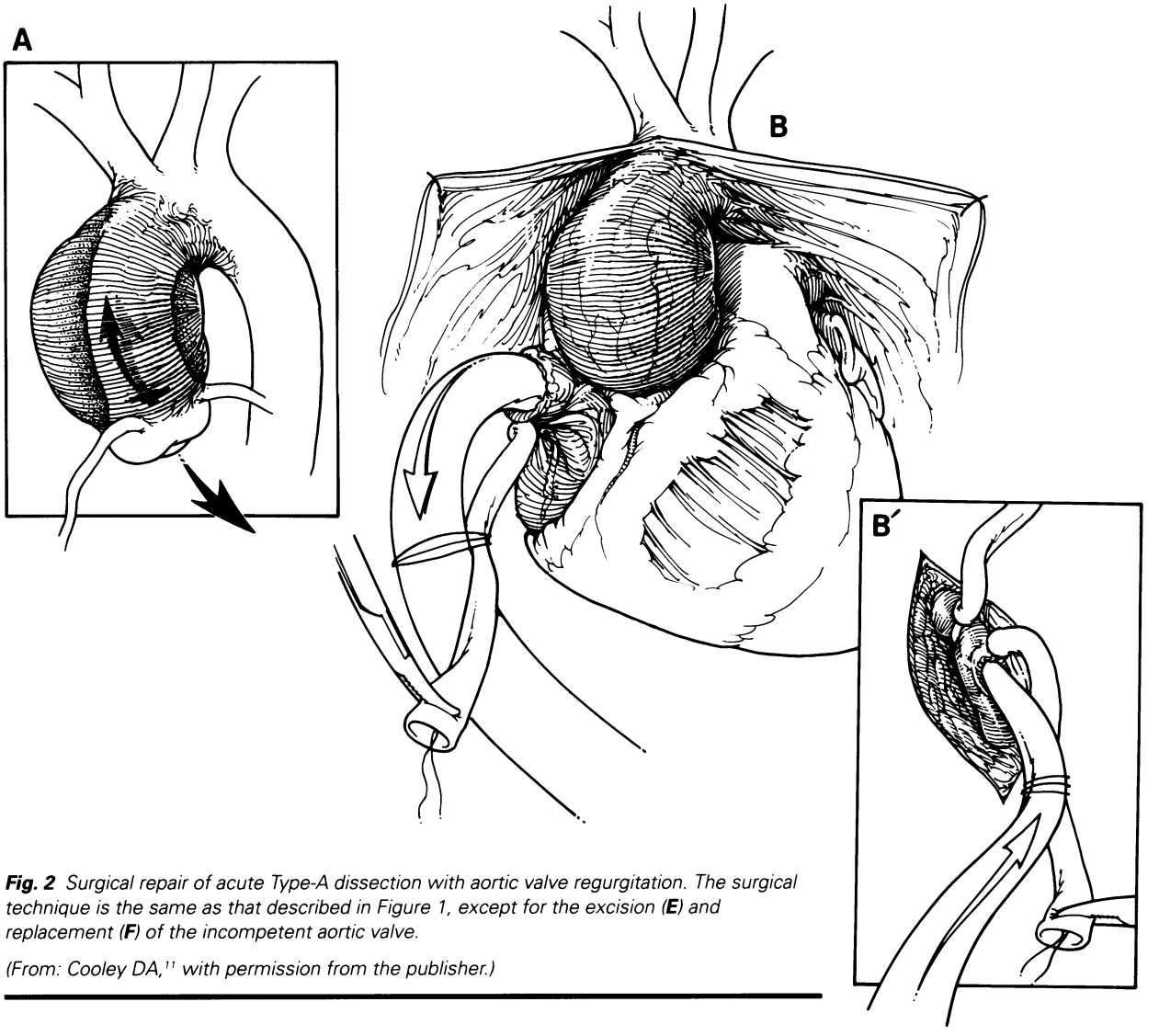


***Fig. 2*** *Surgical repair of acute Type-A dissection with aortic valve regurgitation. The surgical technique is the same as that described in Figure 1, except for the excision (**E**) and replacement (**F**) of the incompetent aortic valve.*

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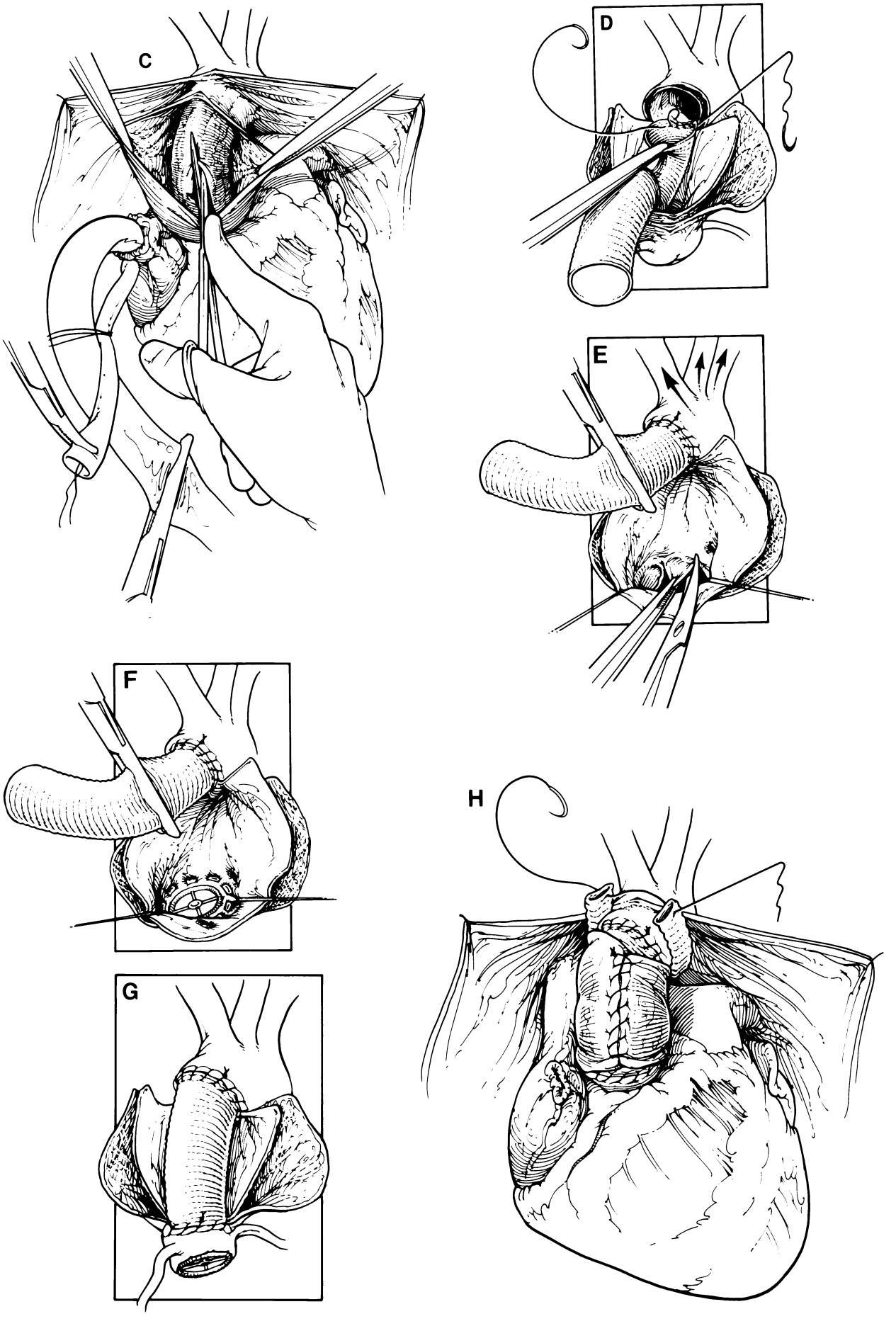
places segments of the spinal cord in jeopardy during hypotension or aortic occlusion if distal aortic pressure falls abruptly. Vasodilators used to control hypertension have been shown to reduce spinal collateral circulation.[27] A rise in cerebrospinal fluid pressure may further impede spinal circulation.[15,16] Thrombi, atherosclerotic debris, and intimal dissection may occlude spinal arteries or hinder their being detected during operation.

In our experience, the most important determinants of the incidence of paraplegia and renal failure have been ischemic cross-clamp time and location and extent of the distal aneurysm. In a recent study,[17] we reported that the incidence of paraplegia was less than 4% if the spinal cord ischemic time was less than 30 minutes, and that the risk of paraplegia increased directly with the cross-clamp time (Table I). When the entire distal aorta was involved, the incidence of paraplegia increased to 9%, compared to 3% for the proximal aorta. In both laboratory and clinical studies, Symbas[28] and others[29] have observed that 30 minutes of aortic occlusion is well tolerated and results in a low incidence of paraplegia. In our experience, we have found that most aneurysms can be removed with no more than 45 minutes' interruption of flow through the descending aorta (and often with less than 30 minutes' interruption of flow).[17] We believe that our usage of heparin also reduces the incidence of ischemic damage during aortic cross-clamping.

## Alternative Techniques for Preventing Ischemic Injury to the Spinal Cord

Concern over paraplegia has existed since the earliest days of aortic surgery in the 1940s, when coarctation of the aorta was 1st successfully resected.[30,31] Fortunately, in coarctation, the extensive collateral blood supply that develops around the occlusion protects the spinal cord from ischemic damage. During aortic resection of aneurysms, however, the collateral supply is insufficient to provide protection during prolonged temporary occlusion. In 1957, we described methods for excising aneurysms of the descending thoracic aorta and proposed a technique of controlled extracorporeal circulation,[32] using a bypass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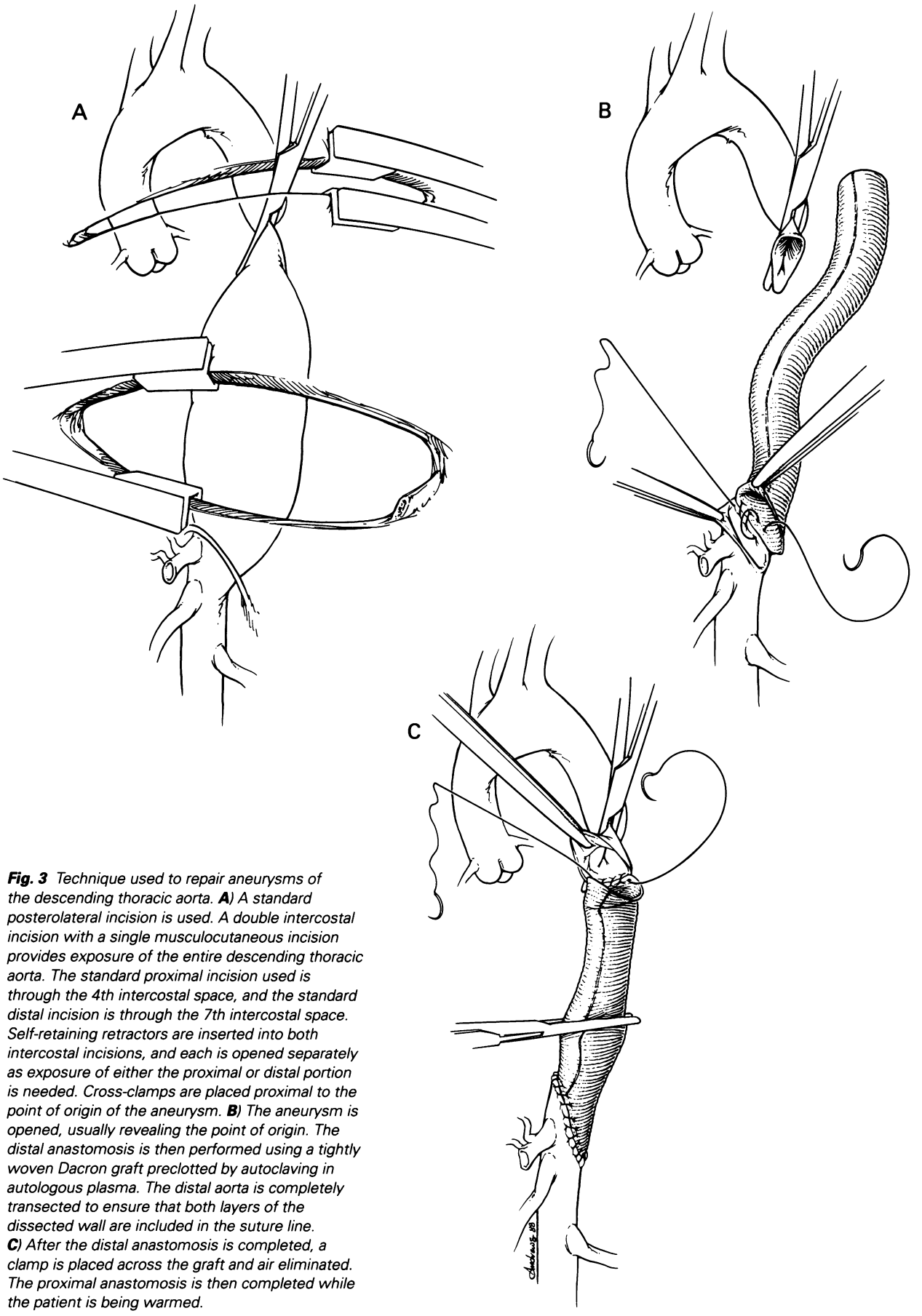


***Fig. 3*** *Technique used to repair aneurysms of the descending thoracic aorta.* ***A****) A standard posterolateral incision is used. A double intercostal incision with a single musculocutaneous incision provides exposure of the entire descending thoracic aorta. The standard proximal incision used is through the 4th intercostal space, and the standard distal incision is through the 7th intercostal space. Self-retaining retractors are inserted into both intercostal incisions, and each is opened separately as exposure of either the proximal or distal portion is needed. Cross-clamps are placed proximal to the point of origin of the aneurysm.* ***B****) The aneurysm is opened, usually revealing the point of origin. The distal anastomosis is then performed using a tightly woven Dacron graft preclotted by autoclaving in autologous plasma. The distal aorta is completely transected to ensure that both layers of the dissected wall are included in the suture line.* ***C****) After the distal anastomosis is completed, a clamp is placed across the graft and air eliminated. The proximal anastomosis is then completed while the patient is being warmed.*

**TABLE I.** Incidence of Paraplegia and Renal Failure Related to Cross-Clamp Time

| Time (min) | No. of Patients* (N=260) | Paraplegia (%) | Renal Failure (%) |
|---|---|---|---|
| 0-15 | 8 | 0 | 0 |
| 16-30 | 142 | 3.5 | 4.2 |
| 31-45 | 90 | 10.0** | 7.8 |
| 46-60 | 16 | 12.5** | 6.3 |
| > 60 | 4 | 25.0** | 0 |
| Average | — | 6.5 | 5.4 |

* Excludes 3 patients who died in the immediate post-operative period.
** $p < 0.025$ (compared with cross-clamp time ≤ 30 minutes).

(From: Livesay JJ, et al,[17] with permission from the Society of Thoracic Surgeons.)

from the left atrium to a femoral artery, with a roller pump in the circuit. Since then, many techniques have been proposed, including 1) atriofemoral bypass with an interposed mechanical pump or with an oxygenator in the circuit;[33] 2) femoral-femoral bypass, in which blood removed from 1 common femoral artery is passed through a bubble oxygenator, and is returned via a pump to the opposite common femoral vein;[34] and 3) the use of a Gott large-bore bypass tube[35,36] (Sherwood Medical Industries; St. Louis, Missouri, USA), which may be inserted into the subclavian artery, inserted directly into the ascending or transverse aortic arch proximally, or placed below the level of the aneurysm distally. Recent reports indicate that the Gott tube may be interposed between the apex of the left ventricle and the lower half of the body.[37] We have used all of these methods in the past, and have observed no substantial difference between the results obtained with supportive shunts and those achieved with simple aortic cross-clamping.[17]

In a recent experimental study performed at our institution using pigs, hypothermic regional perfusion proved to be an effective technique for protecting the spinal cord during ischemia.[10] As much as 45 minutes of ischemia was tolerated when this technique was used in experimental animals. The method also allowed us to control the mean pressure and blood flow through the aortic segment. The technique has not yet been explored clinically.

Recently, we have modified our system of femoral-femoral bypass to prevent pulmonary and bleeding complications (Figs. 4A and 4B). Use of a double-lumen endotracheal tube allows the left lung to be collapsed, thus preventing pulmonary injury. Bleeding from the graft is prevented with proper preclotting techniques. Standard femoral-femoral bypass enables better cardiopulmonary regulation and provides an easy method of allowing for autotransfusion with use of the cardiotomy reservoir. The temperature can be controlled. If full cardiopulmonary support is needed, aortic cannulation with left ventricular venting can be done using standard "Y" connectors (Fig. 4C). Despite all of these modifications, emphasis is still placed upon expeditious removal of the aneurysm and restoration of pulsatile flow.[11]

## Postoperative Management

After surgery, optimal circulatory equilibrium must be maintained. Measurement of renal function and urinary output provides a good index of the general circulatory status. Deficits in blood volume must be monitored by measuring central venous and systemic pressure. Because of the extensiveness of the operation and tissue dissection, patients with aortic dissection have a greater tendency to bleed than those who have undergone most other operative procedures. Hypotension may lead to spinal cord ischemia, and neurologic deficits may develop unexpectedly during the 1st 48 hours after operation; intensive care unit personnel should be alerted to this possibility. Heavy sedation should be avoided. If blood pressure falls below normal, it should be corrected immediately by administering vasopressors and increasing the blood volume. Prolonged hypotension may lead to renal tubular necrosis and renal failure. We depend upon expeditious surgical repair to minimize the period of ischemia caused by occlusion.

## Results

At the Texas Heart Institute, we operated on 2,101 patients for ascending or descending aortic aneurysms between 1961 and 1989. Of these patients, 925 had documented dissecting aneurysms. (In our early years of record-keeping, we often did not distinguish between types of repair; therefore, we have based this analysis only on the 925 definite cases of dissection.)

In the 925 patients, dissections were located in the ascending aorta in 573 (62%) and in the descending aorta in 352 (38%). Most patients (86.6%) were over 40 years of age, and nearly half (45%) were over 60 years of age. Most of our patients were men (73.1%). Of the series, 44 patients (4.8%) had more than 1 aneurysm. Aortic valve replacement was done in 190 patients (20.5%), and 75 (8.1%) required coronary artery bypass at the time of aneurysmal repair.

Total early mortality was 21%. Early mortality for patients with aneurysms in the ascending aorta was 20.9%; early mortality for patients with aneurysms in the descending aorta was 18.5%. Patients who required total replacement of the aortic arch had the

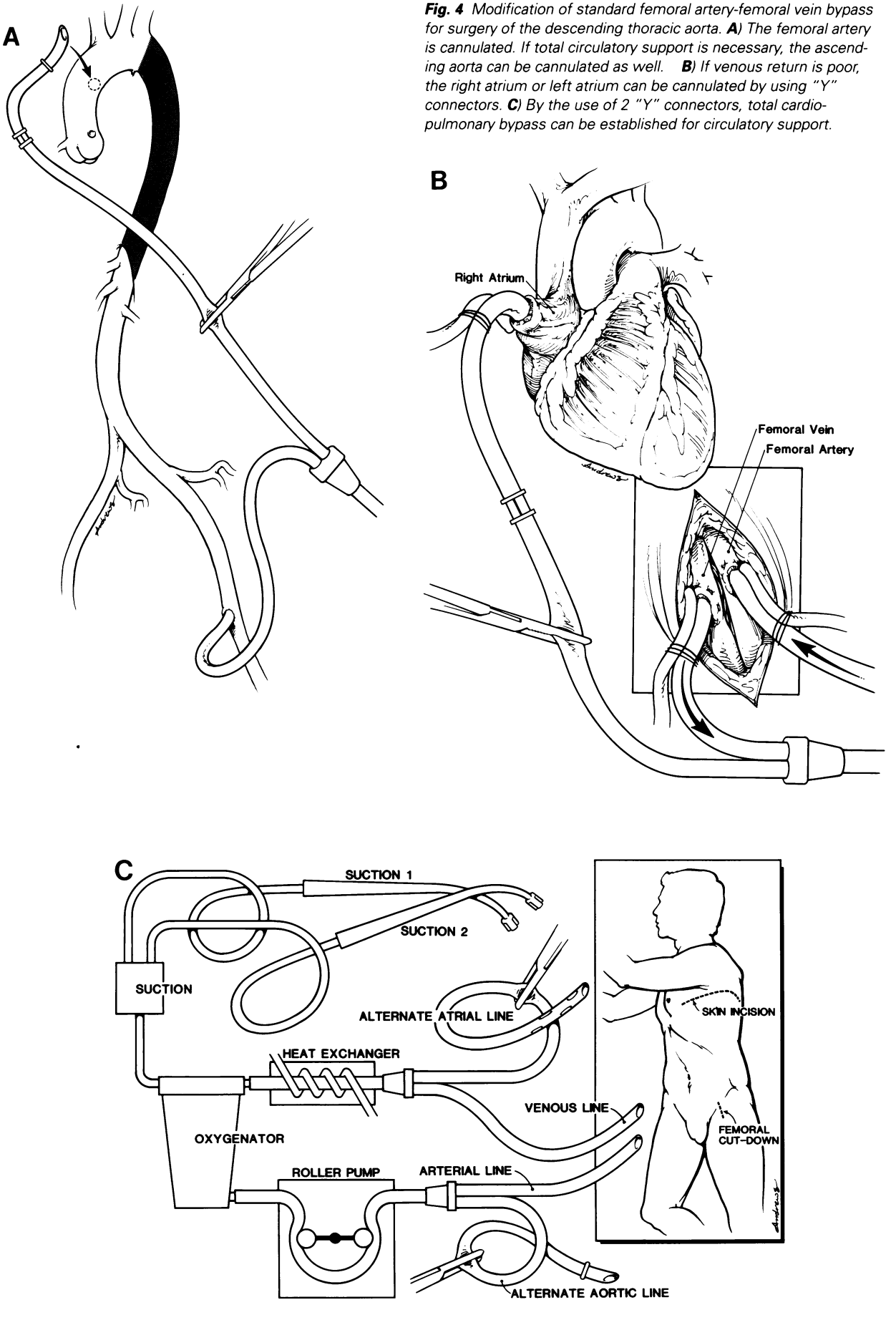


***Fig. 4*** *Modification of standard femoral artery-femoral vein bypass for surgery of the descending thoracic aorta.* ***A****) The femoral artery is cannulated. If total circulatory support is necessary, the ascending aorta can be cannulated as well.* ***B****) If venous return is poor, the right atrium or left atrium can be cannulated by using "Y" connectors.* ***C****) By the use of 2 "Y" connectors, total cardiopulmonary bypass can be established for circulatory support.*

**Table II.** Early Mortality by Age Group in Dissecting Aortic Aneurysms (Texas Heart Institute, 1961 through 1989)

| Age Group (years) | Total Number of Patients (n=925) | Early Patient Mortality | |
|---|---|---|---|
| | | Number (n=194) | Percent |
| 0-19 | 13 | 1 | 7.7 |
| 20-29 | 47 | 6 | 12.8 |
| 30-39 | 64 | 5 | 7.8 |
| 40-49 | 161 | 29 | 18.0 |
| 50-59 | 231 | 39 | 16.9 |
| 60-69 | 275 | 63 | 22.9 |
| 70-79 | 128 | 48 | 37.5 |
| ≥ 80 | 6 | 3 | 50.0 |

highest mortality (68.4%) of our series. Women had a slightly higher early mortality (24.9%) than men (19.5%). Early mortality increased as the ages of our patients increased (Table II). In patients from 70 to 79 years of age, early mortality was 37.5%; in patients over 80, early mortality was 50%.

The characteristics of our patient population have changed since 1961 when this series began. The patients we see today are sicker and older, and there are more women; yet our mortality has decreased. We compared ages of patients who underwent operation between the years 1978 through 1983 and between 1984 through 1989 and found a statistically significant difference ($p < 0.01$) between the groups. The number of patients less than 60 years of age decreased from 58.8% to 46.6%, whereas the number of patients over 70 years of age almost doubled, from 12.4% to 22%. The number of patients under 70 years of age decreased from 87.6% to 78.0%. Before 1977, early mortality in patients from 60 to 69 years of age was 28.7%, whereas from 1977 through 1989, mortality in this same age group was 19.9%. Early mortality in our young patients (0 to 29 years) also decreased dramatically, from 19.0% to 7.7%. Although not as substantial, decreases were noted in all other age groups as well.

## Summary

Dissection of the thoracic aorta is a well-described entity. Dissection can occur in the normal aorta, although most cases involve underlying medial degeneration: a result of either atherosclerosis or cystic medial necrosis. Damage results in tearing of the intima, with formation of a false channel in the diseased media. The forces of pulsation and ventricular ejection produce stress on the aorta, causing the false channel to extend in a retrograde or antegrade direction. The result is an aneurysm with a wall thickness dependent upon the depth of penetration into the media. In addition to the intimal exit site, an intimal reentry site may occur at any level of the distal aorta or aortic branches. Antegrade or retrograde dissection may result at all levels. The clinical picture depends on the aortic branches involved, the size and direction of the dissection, the duration of symptoms, and the location of the initial intimal tear. Dissecting aneurysms of the thoracic aorta must be distinguished from true aneurysms, which are contained by all the layers of the aortic wall, and from acute or chronic traumatic aneurysms, which rarely undergo dissection to any great extent.[8]

To further complicate the clinical picture, a dissecting aneurysm may rupture into the free pleural space, lung parenchyma, pulmonary artery, or bronchus. It may also compromise the great vessels; rupture the ascending aorta; or rupture into the pericardium, producing pericardial tamponade. Dissection may extend into the sinus of Valsalva, compromising aortic valve support and causing aortic valve incompetence, or it may extend further into a cardiac chamber, causing an aortic-cardiac chamber fistula. Right or left coronary occlusion may cause acute myocardial infarction and heart failure. Antegrade rupture or compromise of distal aortic branches can also occur. The clinical picture is indeed complex, and surgical treatment must be tailored to each individual's clinical state.

Although a few patients with descending aortic dissection may be managed medically, indications for immediate or emergency surgery include medical failure, expansion of the aneurysm, and rupture. Medical failure is indicated by persistent pain or unstable blood pressure. Expansion of the aneurysm may be heralded by increasing pain, mediastinal widening, or loss of the peripheral pulse. This expansion may be associated with neurological deficits, the presence of a new aortic-insufficiency murmur, or evidence of compromise to the visceral vessels, including renal failure or abdominal pain and tenderness. Patients must be carefully observed for appearance of these signs, which would indicate that immediate surgical intervention is necessary. If rupture does occur, the signs include persistent pain, shock, evidence of cardiac tamponade, or bloody pleural effusion. Hemoptysis may indicate the onset of an aorto-bronchial fistula with rupture of the aneurysm into the pulmonary parenchyma.

Overall survival rates for patients with untreated acute dissections are dismal—only 5%; however, 10-year survival rates are approximately 50% for patients whose aneurysms are treated (Fig. 5). Of late deaths, half are related to development and rupture of the

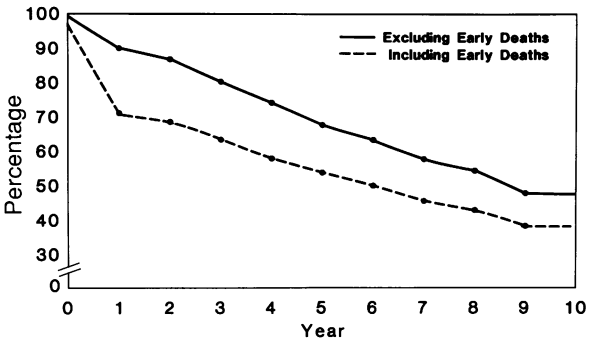


***Fig. 5*** *Actuarial survival of patients with dissecting aortic aneurysms operated on at the Texas Heart Institute from 1961 through 1989.*

aneurysm. Operative mortality has decreased to approximately 20%. Improvements in surgical management—for example, repair using the open technique with hypothermia and circulatory arrest—have been responsible for the improved results in treatment of aortic dissections.

Because of a multitude of factors, the optimal treatment of aortic dissection varies widely, and frequent complications must be expected in patients who are treated surgically, as well as in those who are managed nonsurgically. Thus, in cardiovascular surgery, no decisions are more challenging than those encountered in the management of acute dissecting aneurysm.

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