Transthoracic repair of innominate and common carotid artery disease: Immediate and long-term outcome for 100 consecutive surgical reconstructions

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Purpose: **This is a review of 100 consecutive supraaortic trunk reconstructions (SAT) performed over 16 years.**
Methods: **There were eight innominate endarterectomies and 92 bypass procedures based on the thoracic aorta (n = 86) or proximal innominate artery (n = 6) in 98 patients 24 to 79 years of age. Indications included cerebrovascular ischemia in 83 and upper extremity ischemia in four. Thirteen patients were asymptomatic. An innominate lesion was bypassed in 78 cases. The left common carotid and left subclavian arteries required reconstruction in 38 and nine patients, respectively. Multiple trunks were reconstructed by direct bypass grafting in 35. Approach was via median sternotomy in 92, partial sternotomy in six, and left thoracotomy in two. Seven patients underwent concomitant cardiac surgery.**
Results: **Eight deaths and eight nonfatal strokes occurred, for a combined stroke/death rate of 16%. The operative mortality rate was 6% for SAT and 29% for SAT/cardiac operations. Perioperative complications included two asymptomatic graft occlusions, three nonfatal myocardial infarctions, seven significant pulmonary complications, three sternal wound infections, and one recurrent laryngeal nerve injury. Follow-up ranged from 1 to 184 months (mean, 51 ± 4.8 months). Eight patients were lost to follow-up. Twenty-one late deaths occurred. Two SATs required late revision. The cumulative primary patency rates at 5 and 10 years were 94% ± 3% and 88% ± 6%, respectively. The stroke-free survival rates at 5 and 10 years were 87% ± 4% and 81% ± 7%, respectively. Patients who survived beyond 30 days had a median stroke-free life expectancy of 10 years, 7 months (SE, 6%).**
Conclusions: **Direct reconstruction of complex symptomatic SAT lesions can be performed with acceptable death/stroke rates and with long-term patient benefit. Asymptomatic lesions in patients who have significant concomitant conditions should be managed with a less-morbid cervical or endovascular approach, even if long-term outcome of the latter is inferior. (J Vasc Surg 1998;27:34-42.)**

Severe occlusive disease of the supraaortic trunks (SAT) is managed by transthoracic, extraanatomic cervical or endovascular repair. The natural history of these lesions is not well known because they are relatively uncommon and because they are difficult to image using noninvasive technology (duplex, magnetic resonance angiography). There are, however, well-recognized ischemic manifestations of occlusive disease of the SAT in the hemispheric, ocular, vertebrobasilar, and arm territories that may lead to stroke or to digital gangrene. Repair of occlusive lesions of the SAT accounts for less than 10% of the operations performed on the extracranial arteries of cerebral destination.[1-3]

Over the past 16 years we have preferred direct, transthoracic reconstruction over cervical repair for innominate artery (IA) occlusion and for complex

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IA and common carotid artery (CCA) lesions in patients who are reasonable cardiopulmonary risks. Single CCA or subclavian lesions, in cases in which adequate inflow is available, have generally been corrected through a cervical approach. Lately, a few selected cases (not reported here) have been reconstructed using an endovascular technique.

Most papers that deal with the surgical treatment of SAT disease combine data from both cervical and thoracic approaches together.[4-8] Often, comparisons cannot be made between transthoracic and cervical repairs because the characteristics of the atherosclerotic lesions (multiple versus single, flow-restricting versus embolizing) fairly mandate one approach or the other. Single lesions of the subclavian artery or CCA are best treated with a cervical operation, whereas combined IA and left CCA disease often requires a thoracic approach. Likewise, embolizing lesions of the IA usually require transthoracic repair because the most appropriate exclusion of an embolic source may not be possible from a cervical approach. On the other hand, single, stenosing disease of the IA may be repaired via either a thoracic or a cervical operation, the latter via a contralateral bypass graft to the right CCA. The patient's cardiopulmonary risk, previous operations, radiation, or infection may also determine the choice of operation. A previous mediastinotomy performed for myocardial revascularization or the presence of severe pulmonary disease substantially increases the risk of a thoracic approach. In other cases, the problem may be corrected equally well by either a transthoracic or a cervical approach. It is pertinent then to know the short-term and long-term history of transthoracic, cervical, and endovascular repairs.

With substantial experience limited to a few large medical centers, the indications for direct, transthoracic repair of the SAT have been derived from assumptions on the basis of the natural history of their atherosclerotic involvement. Survival and long-term patency data are limited after such reconstructions. To contribute to the study of risk/benefit on this subject, we undertook this retrospective analysis.

PATIENTS AND METHODS

Patients. Over a 16-year period (1981 through 1996), 100 transthoracic reconstructions for occlusive disease of the SAT were performed. Patient medical records were reviewed in detail, and extensive efforts were made to update graft patency and patient survival data.

The 100 reconstructions were performed in 98 individuals and included eight innominate endarterectomies and 92 aorta or IA-based bypass procedures. Fifty-six patients were female, and 44 were male. The mean patient age was 61 ± 9.5 years, with ages ranging from 24 to 79 years.

The vast majority of patients (98%) were treated primarily for atherosclerotic disease involving one or more of the SATs. One young patient required reconstruction after a traumatic dissection that resulted in symptomatic IA occlusion. One patient underwent an IA bypass procedure for a chronic and symptomatic occlusion that followed a remote cardiac embolic event.

Cerebrovascular ischemia, defined as hemispheric, monocular, or vertebrobasilar transient ischemic attack or stroke, attributable to SAT lesions occurred alone or in any combination in 83 patients (83%). Thirty patients (30%) had a hemispheric stroke before undergoing revascularization. In 14 patients (14%) a hemispheric stroke was the primary indication for operation. An additional 16 patients (16%) had a remote history of stroke (longer than 1 year).[2]

Arm symptoms were present in five patients (5%). Four of them had exclusively upper extremity ischemic symptoms. Two patients had limb claudication, whereas three patients had significant ulceration or necrosis of the finger(s).

Thirteen patients (13%) were asymptomatic. One of them was found to have a silent hemispheric infarct on a preoperative computed tomographic scan. Patient symptoms are summarized in Table I.

Forty-five patients (45%) had significant cardiac disease, and nine of them had previously undergone a median sternotomy for coronary revascularization (seven patients), valve repair (one), or resection of a ventricular aneurysm (one). Seventy-two patients (72%) were undergoing treatment for hypertension. Significant pulmonary dysfunction was present in 13 patients (13%). Fourteen patients (14%) had diabetes, and five patients had renal failure (5%). Three patients had undergone previous head and neck irradiation. Ninety-six patients (96%) had a significant smoking history. Forty patients (40%) had significant aortoiliac (12%) or femoropopliteal (28%) occlusive disease.

Before diagnosis and repair of their SAT lesions, 37 patients (37%) had undergone a total of 61 cerebrovascular reconstruction procedures. These operations included four transthoracic SAT operations, 42 carotid bifurcation endarterectomies, and 15 CCA, vertebral artery, or subclavian artery reconstructions.

Results of a preoperative computed tomographic scan of the brain were available in 72 patients (72%),

Table I. Clinical presentation and symptoms

	No. of patients
Ocular only	14
Hemispheric only	16
Vertebrobasilar only	25
Arm only	4
Hemispheric + ocular	9
Hemispheric + vertebrobasilar	18
Hemispheric + arm	1
No symptoms	13

of which 51 were normal (51%), 18 showed a hemispheric infarction (18%), and three showed an infarction in the vertebrobasilar territory (3%).

All 100 patients underwent preoperative four-vessel arteriography. The most common indication for diagnostic arteriography was the association of cerebral ischemic symptoms and a positive result of a duplex scan of the carotid bifurcation. Cervical or supraclavicular bruits, radial or temporal pulse deficits, and upper extremity blood pressure differential readings also contributed to the clinical diagnosis of SAT disease.

At angiography, severe disease was defined as stenosis >75% diameter or, in symptomatic patients, a deep ulceration within a plaque or the presence of thrombus within the arterial lumen. The IA was the vessel most commonly involved with disease. In 78 patients (78%) the IA had severe disease; 18 patients (18%) had occlusion, 36 patients (36%) had >75% diameter stenosis, and 24 patients (24%) had ulcerated or irregular atheromata that was thought to be the source of ipsilateral hemispheric events.

The left CCA was severely diseased in 50 patients (50%) and was reconstructed in 38 cases (38%). The most common reason not to reconstruct the left carotid system was left internal carotid artery occlusion. The left subclavian artery was significantly diseased in 52 patients (52%) but was reconstructed in only nine patients (9%). Reconstruction was not performed for asymptomatic left subclavian artery disease.[3]

Sixty-three patients (63%) had multiple SAT lesions. All three SATs were diseased in 22 patients (22%), and two of three were significantly involved in 41 patients (41%). Only a single SAT vessel was involved with disease in 37 patients (37%), and it was the IA in 28 instances. There was arteriographic evidence of vertebral steal in 25 patients (25%). Thirteen patients with radiographic steal had vertebrobasilar symptoms. Twenty-five patients (25%) had significant (>60%) carotid bifurcation stenosis.

Surgical repair. Innominate endarterectomy was used only during the early years. Later, bypass grafting became the preferred technique. Our standard technique for transthoracic repair of the SAT has been described elsewhere.[9] Our policy for revascularization was to reconstruct any trunk that was presumed to be symptomatic and those anterior trunks that were severely diseased. Asymptomatic severe lesions of the left subclavian artery were not repaired unless it was needed for a left internal mammary myocardial revascularization procedure. Synchronous severe lesions of the carotid bifurcation were generally corrected at the time of operation if they were ipsilateral to the patient's SAT reconstruction or were potentially a cause for the patient's symptoms. Contralateral and severe carotid bifurcation lesions were corrected at another time based on their own merits.

The brain was a target for revascularization in all cases. There were 33 aortoinnominate bypass procedures (33%), 8 IA endarterectomies (8%), and 28 bypass procedures in which the graft originated from the ascending aorta (28%), with one branch to the right (10 grafts), left (eight), or both (10) carotid systems. In addition, there was one bypass graft from the descending thoracic aorta to both carotid bifurcations and one graft replacement of the transverse segment of the arch of the aorta with left CCA and subclavian branches.

There were 15 aortoinnominate bypass grafts with a side limb to the left carotid system (14 grafts) or to the left subclavian artery (one). There were six aortocarotid bypass grafts with a limb to a subclavian artery. There was a single aorta–left subclavian artery bypass graft and a single aortovertebral bypass graft to revascularize the brain through a single vertebral artery in two patients who had bilateral internal carotid occlusion. In addition, there were six bypass procedures based on the proximal IA: four to the right carotid artery, one to the left subclavian artery, and one bifurcated graft to the right carotid and subclavian arteries. In total, there were 57 straight grafts and 35 with multiple limbs.

The reconstruction was performed through a full median sternotomy in 92 cases, a partial median sternotomy in six, and a left thoracotomy in two. Because of previous cardiac or SAT interventions, in 13 patients (13%) the full median sternotomy was a redo procedure. A partial median sternotomy involving the upper half of the sternum was used in six IA endarterectomies performed for lesions of the distal half of the IA. In 10 patients the carotid bifurcation operation performed concomitantly with the SAT repair was a reoperation. Autologous vein was used

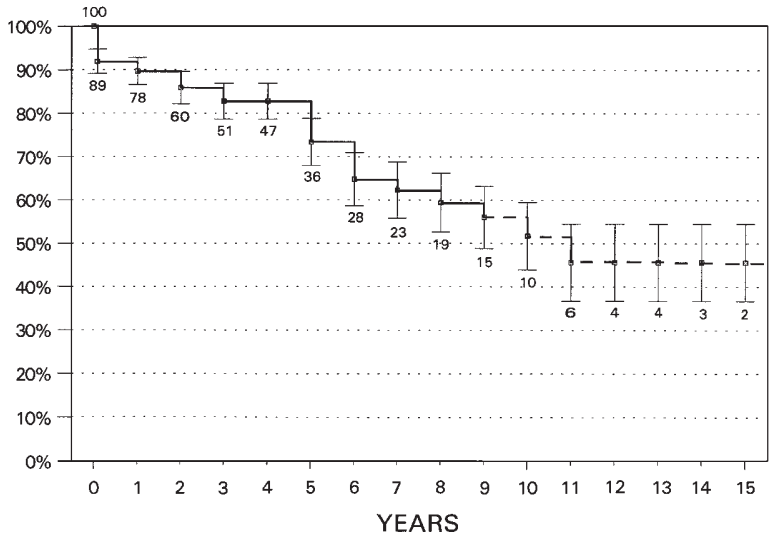

Fig. 1. Overall SAT cumulative survival data.

as conduit in two cases, expanded polytetrafluoroethylene was used in 66, and Dacron in 24.

Seven patients (7%) underwent a concomitant cardiac operation at the time of SAT reconstruction. Six patients underwent revascularization of their coronary arteries, and one patient underwent concomitant mitral valve replacement. All of the patients who underwent a cardiac procedure initially had cerebrovascular symptoms that arose from their SAT lesions and were found to have severe cardiac problems on further evaluation.

There were two intraoperative acute bleeding events without adverse consequences that occurred while reopening the sternum. Both occurred in patients who had undergone at least one previous midsternotomy. One patient had a small tear develop in the right ventricle. In the other patient, a chronically occluded aortoinnominate artery bypass graft tore away from the ascending aorta. In both instances the bleeding was controlled by direct repair.

The brachiocephalic vein was divided in six cases to improve exposure. It was ligated in five cases and reconstructed in one.

In addition to the 100 direct SAT reconstructions, 43 additional procedures were performed in the same setting in 39 patients who had complex SAT disease and further atherosclerotic lesions downstream. Carotid bifurcation endarterectomy was performed concomitantly with SAT reconstruction in 26 patients (26%) either as a remote component to the operation or incorporated into the distal anastomosis of the bypass graft. A vertebral artery was transposed or bypassed in five cases (5%). A separate reconstruction was performed to repair a diseased subclavian artery (nine) or CCA (three) in 12 cases.

Blood loss averaged 866 ± 57 ml, and an average of 1.2 ± 0.17 units of blood were transfused. Postoperative angiograms were obtained for all patients during the index hospitalization before discharge.

The average duration of hospital stay was 10.5 ± 0.79 days, and patients remained in the intensive care unit for 4 days on average (SEM, 0.42).

RESULTS

The combined stroke/death rate was 16%. There were eight perioperative deaths (8%). Six deaths (6.4%) occurred in the 93 patients who underwent reconstruction of the SAT alone. Two deaths (28.5%) occurred in the seven patients who underwent a simultaneous cardiac/SAT procedure. Among the 93 patients who underwent reconstruction of the SAT alone, there were 13 asymptomatic patients (no deaths) and 80 symptomatic patients (six deaths). The statistical difference between these two groups is not significant ($p = 0.59$). A single nonfatal stroke occurred amongst the asymptomatic population.

Two deaths were directly related to cerebral infarction. One patient had a massive stroke on the seventh postoperative day in a distribution contralateral to the SAT reconstruction. This stroke was attributed to embolization from a known atrial thrombus in a patient with active fibrillation. The SAT repair was patent at the time of the patient's death. The second stroke death was the result of a

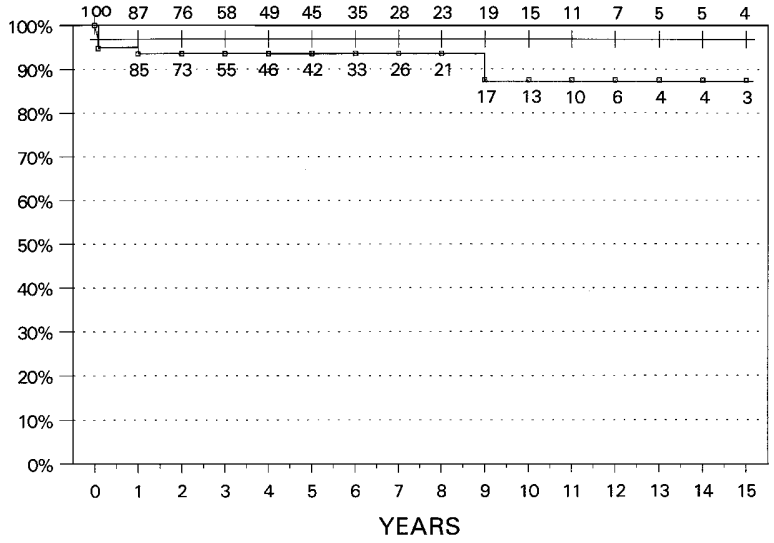

Fig. 2. SAT reconstruction patency data.

sudden, large intracranial bleed on the fourth postoperative day while the patient was ambulatory and ready for discharge. Before operation this woman was symptomatic and had an occluded aortoinnominate bypass graft that had been implanted elsewhere 11 years previously. Her left CCA and subclavian artery were occluded as well. We performed a second aortoinnominate bypass procedure. The postoperative cerebral hemorrhage appeared to be the result of cerebral hyperperfusion. Again, the bypass graft was patent at the time of death.

The remaining six deaths followed cardiorespiratory complications. One patient who underwent a concomitant and complicated coronary revascularization procedure had an intraoperative myocardial infarction. This patient could not be weaned from cardiopulmonary bypass and died in the operating room. A second patient who underwent a combined coronary/SAT procedure had an accidental air embolus during the operation and died on the first postoperative day. The remaining cardiorespiratory deaths occurred on postoperative days 2, 4, 8, and 9. The first two followed massive myocardial infarctions in patients with known coronary artery disease, the third was the result of a pulmonary embolus, and the fourth was precipitated by a cardiac arrhythmia.

Eight nonfatal strokes occurred during the perioperative period. One patient had occipital bleeding on the fourth postoperative day. The reconstruction was patent, and the intracranial hemorrhage was attributed to hyperperfusion. This patient was treated by surgical evacuation of the hemorrhagic tissue and recovered normal neurologic function in 2 weeks. Two of the eight strokes were a result of perioperative graft occlusion. The single aortovertebral bypass graft occluded on postoperative day 5; this was the result of vein graft impingement by adjacent structures in the thoracic outlet. The bypass graft was revised, and the mediastinal path was corrected. Hemianopsia developed as a result of a small occipital infarction. Another patient had thrombosis of an aorta–left CCA bypass graft on postoperative day three and had a minor stroke. The graft was replaced, and the patient's deficit had disappeared by the time of discharge. One patient had a stroke while at home on postoperative day 15. This stroke resulted from a known significant contralateral internal carotid artery stenosis that was scheduled to be repaired. Blindness and superior vena cava syndrome developed in one patient after repair of a large tear that occurred at the brachiocephalic-caval venous junction during exposure of the anterior mediastinum. A technically successful juguloatrial bypass procedure corrected the patient's superior vena cava syndrome, but her blindness persisted. Three postoperative strokes occurred for which no specific etiologic mechanism could be identified after emergency arteriography. All of the patients who had nonfatal strokes were included in long-term graft patency data and in survival analyses.

There were, in addition, two asymptomatic graft occlusions. One was cleared by thrombectomy and revised; the other (occlusion of a subclavian limb) was left untreated. The three patients who had grafts that failed and were revised within 30 days of

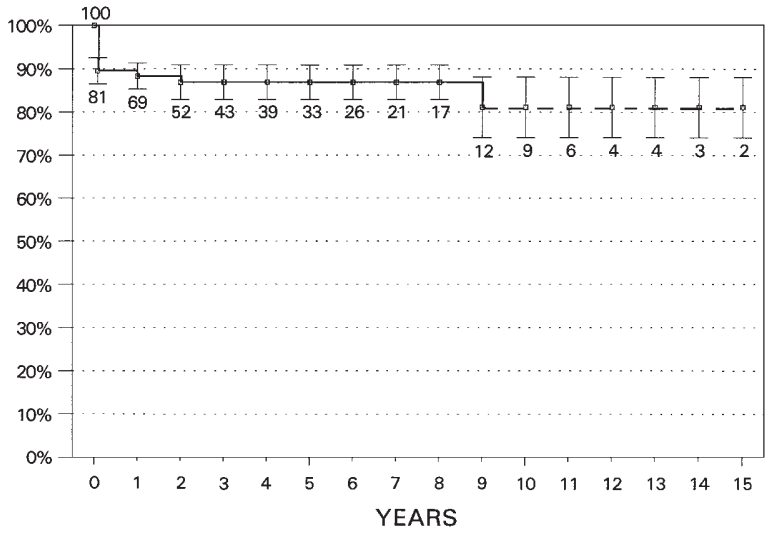

Fig. 3. SAT protection from stroke

operation were followed-up late and included in secondary patency data. The early patency of all reconstructions was documented by a postoperative angiogram.

There were three nonfatal perioperative myocardial infarctions (3%) in patients who had known cardiac disease. Seven nonfatal pulmonary complications (7%) were noted. These included one episode of respiratory failure, two pneumothoraces that required chest tube placement, two pleural effusions that required aspiration, one case of pneumonia, and one case of adult respiratory distress syndrome. Three of these pulmonary complications required prolonged (>3 days) ventilatory support. In total, 16 patients (16%) had perioperative cardiopulmonary morbidity or died.

Two patients (2%) required a return to the operating room for evacuation of a hematoma or drainage of a pericardial tamponade. Three wound complications occurred; all were sternal wound infections. Two were treated successfully with myocutaneous flaps, whereas one required graft removal and complex extraanatomic autogenous reconstruction using the superficial femoral artery. This patient is counted as a graft failure but was followed-up for purposes of complete survival data.

Other complications included a permanent vocal cord paresis after injury to the right recurrent laryngeal nerve (1%). Upper extremity swelling developed in six patients (6%); all occurred in patients who underwent ligation of the innominate vein. There was one episode of lower limb ischemia and major amputation in a patient who had heparin allergy.

Patient follow-up ranged from 1 to 184 months (mean, 51 ± 4.8 months). Thirty-four patients were available for follow-up at 5 years, and 10 patients were available at 10 years. Follow-up data on long-term graft patency was obtained by physical examination, by indirect Doppler waveform analysis of the carotid and upper extremity circulation, by direct duplex ultrasound interrogation of the reconstruction, or by angiogram.

Twenty-one patients (21%) died during follow-up. One patient died during a coronary artery bypass grafting procedure at 88 months, and one patient died during abdominal aortic aneurysm repair at another institution at 52 months. A fourth patient died of a stroke 16 months after an aorta–left carotid artery bypass procedure; it is not known whether the graft was patent at the time of death. There were seven late cardiac deaths at 30, 31, 48, 55, 55, 66, and 122 months. Three cancer-related deaths occurred at 18, 48, and 110 months, and one death from pneumonia occurred at 77 months. The remaing seven deaths from unknown causes occurred at 3, 4, 20, 60, 60, 106, and 120 months. It is known that the one death that occurred at 3 months took place at in-patient rehabilitation in the patient who had required treatment for adult respiratory distress syndrome. The overall patient survival rate was 73% and 52% at 5 and 10 years, respectively. Long-term overall patient survival statistics are summarized graphically in Fig. 1.

Two successful revascularization procedures were performed for late failures of a bypass limb, which caused transient ischemic attacks at 3 and 104 months after the operation. Fig. 2 shows primary and secondary patency curves for these SAT reconstructions.

Three patients required repeat sternotomy for coronary revascularization at 62, 88, and 166 months after undergoing SAT bypass grafting. Two patients had nonfatal strokes, and eight had transient ischemic attacks during late follow-up; all were attributed to carotid bifurcation or intracranial atherosclerotic disease contralateral or distal to the patient's SAT repair. In addition, six patients had persistent, recurrent, or new vertebrobasilar symptoms severe enough to require further intervention. Late reconstructions of the more distal extracranial cerebral vasculature was performed when indicated for symptomatic or asymptomatic disease. Nineteen patients (19%) required 22 late cerebrovascular operations for occlusive disease unrelated to SAT bypass failure. These included 14 carotid endarterectomies or replacements, four vertebral reconstructions, and four left subclavian to carotid transpositions. The ability for SAT reconstruction to protect patients from stroke is depicted in Fig. 3. Patients who survived beyond the 30-day perioperative period had a median stroke-free life expectancy of 10 years, 7 months (SE, 6%). Patency and survival curves were plotted in accordance with criteria established by the SVS/ISCVS.[10,11]

DISCUSSION

In this series of patients with complex disease involving the IA and CCA, direct reconstruction was aimed at revascularizing the brain. In the few cases in which the target vessel was a subclavian or a vertebral artery, the patients had bilateral ICA occlusions and the reconstruction was done to provide brain perfusion via the posterior circulation.

Most series that deal with the surgical treatment of patients with IA and CCA disease include patients who were treated using the cervical and transthoracic options available to the surgeon.[4-8] Our allocation of patients to one or the other method of treatment over the past 16 years has been generally based on cardiopulmonary risk and on the anatomic distribution of the atherosclerotic lesions. In patients with significant cardiopulmonary risk and patients who have previously undergone operation in the mediastinum, we prefer a cervical approach. Those with a single intrathoracic lesion of a CCA or subclavian artery are also managed with a relatively straightforward neck operation. On the other hand, complex IA lesions, particularly those that are embolizing or associated with left CCA lesions, we have preferentially treated by direct transthoracic exclusion bypass grafting.

The composition of the series of patients with IA/CCA disease published in the literature varies substantially.[4-9,12-16] The rate of comorbidity in our series is significantly higher than in the other reports reviewed here. There are also substantial differences in the percentage of asymptomatic patients (13% in our series) in published reports, ranging from 8% to 32%.

The deaths in our series were primarily cardiopulmonary. In fact, one of the two stroke deaths also had cardiac cause—embolization of a large atrial thrombus. Our experience is similar to that reported by Vogt et al.[7] and by Kieffer et al.,[13] who also list cardiopulmonary complications as their most frequent morbidity, but it differs from that of Crawford et al.,[6] who reported stroke as the most common complication and cause of death. The mortality rate of transthoracic repair varies in the reported literature from 0% to 14.7%.

In the small subset described in our series that underwent cardiac and SAT repair simultaneously, the mortality rate was 29% (two of seven). Although the numbers are limited, should this data preclude us from performing both repairs simultaneously? The causes of death in these two patients were, in one patient, an accidental air embolus caused by a perfusion error and, in the other, a complex coronary repair that resulted in an intraoperative myocardial infarction. None of these problems were related to the associated SAT procedure, and other authors have not reported a high mortality rate from the synchronous performance of SAT and coronary repair. We believe that these two procedures should be combined when there are operative indications to repair both SAT and coronary lesions.

We have traditionally viewed disease of the IA and CCA in the same light as disease of the carotid bifurcation. The composition and surface appearance of atheromatous plaques is similar in both locations, and the mechanisms of brain injury (flow restriction and embolization) are the same. Regarding our indications for surgical correction, we have slightly stricter criteria for operating on asymptomatic lesions of the SAT (>75% diameter stenosis) than we do for operating on asymptomatic lesions of the carotid bifurcation. We necessarily take into account the increased magnitude and risk attending the former operation. The ACAS study has shown prospectively the benefits of operating on asymptomatic lesions of the internal carotid artery with >60% diameter stenosis when the combined morbidity/mortality rate is less than 3.0%.[17] The life expectancy of the patients in our series is similar to that of patients with carotid bifurcation disease—

approximately 50% of them are dead 10 years after surgical repair, mostly from cardiac disease. Although none of the asymptomatic patients in this series died, their number is too small to be statistically valid. In considering indications for operation, we should apply the mortality rate of the entire group (with and without symptoms), which was 6.4% and consisted mostly of cardiac problems. Even if we assume that the asymptomatic patients with IA/CCA disease may have less-advanced coronary disease than those who have symptomatic IA/CCA lesions, the risk of transthoracic repair probably negates the long-term stroke-sparing benefit it has over their limited life expectancy. On the other hand, in patients who have ischemic brain symptoms, a direct transthoracic reconstruction is an effective curative operation that at 5 years has a cumulative primary patency rate of 94% and a stroke-free survival rate of 87%.

Cervical repair of atherosclerotic SAT disease may be inappropriate when the lesions are complex or multiple; however, when a cervical operation is possible, the patient mortality rate is less than 2%. Limited experience with endovascular repair anticipates that its long-term results may be inferior to those obtained by direct anatomic bypass reconstruction. Long-term patency goals, however, are less relevant in a population in which 50% will not survive a decade. The immediate morbidity and mortality rates of endovascular repair remain to be determined.

CONCLUSION

Direct transthoracic repair of lesions of the IA and CCA is not recommended in patients without neurologic symptoms because the morbidity rate of the operation is probably greater than that of the natural course of the atherosclerotic disease. In these patients, consideration should be given to cervical surgical repair or to endovascular techniques that carry a lower perioperative mortality rate, even if their long-term patency and stroke-free survival rates are inferior or unknown. Patients who have symptomatic lesions of the IA (with/without associated CCA disease) benefit from direct transthoracic repair and can expect long-term protection from stroke.

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DISCUSSION

Dr. Luis A. Queral (Baltimore, Md.). Dr. Berguer has just presented a clear, concise, and well-documented paper outlining his 16-year experience with largely transsternal supraaortic trunk reconstructions. I congratulate him and his colleagues for this achievement. The surgical mortality rate, as noted, was 6.4% when reconstructions were carried out alone and 29% when combined with a cardiac operation. An 8% operative stroke rate was reported. The long-term patency rate of the reconstruction was a commendable 94% at 5 years and 88% at 10 years, with a stroke-free survival rate of 87%. There is no question regarding the durability of the reported traditional surgical approach. However, I would question the relevancy of long-term goals in a population in which 50% of the patients will not survive a decade. Is long-term patency worth the significant operative mortality rate of 6.4%, or even that of 29% when combined with cardiac surgery? Can a perioperative stroke rate of 8% be improved on? Is it, in fact, tolerable? What is the alternative? What does an endovascular approach offer in these complicated cases that Dr. Berguer reported on?

Well, let's examine the patients treated and their lesions to determine their suitability for endovascular repair. The 78 patients who were treated for innominate artery occlusive disease had obstructions in 18 cases—I don't know the length of these obstructions, but I assume that most of them were short, as is usually the case—36 of these patients had innominate artery stenosis >75%, and 24 of these patients had an ulcerated innominate artery or irregular atheromatous plaques.

There were 50 patients with severe left common carotid artery disease, 38 of whom underwent reconstruction, and 52 patients with subclavian artery disease underwent nine reconstructions. Even when you exclude a percentage of the patients with innominate artery occlusions, it appears that the vast majority of patients in this series could have been treated by placement of a stent at the site of the offending lesion. This endovascular approach, when performed in an operating room setting with a cervical cutdown allowing retrograde deployment and avoiding embolization, has a much lower morbidity and mortality rate and a hospital stay approximately 9 days shorter.

I do realize that stenting the supraaortic trunks may not be as durable, but is it not justified considering the markedly shortened lifespan of most of these patients? Why not treat all patients initially with endovascular stenting and perform the highly morbid traditional surgical approach in cases of recurrence or failure? We as vascular surgeons have a unique opportunity to change our approach to patients who have supraaortic trunk occlusive disease.

Dr. Ramon Berguer. I agree with Dr. Queral that for asymptomatic patients an endovascular procedure certainly seems like a more reasonable option. I think if we consider the natural history of the disease in individuals who are actively symptomatic from innominate and carotid disease, the option of an operation that carries a 6.4% mortality rate is not a bad one. After all, we have very little or no information on the outcome of endovascular procedures in patients who are actively symptomatic.

Dr. Malcolm O. Perry (Dallas, Tex.). Dr. Berguer, I enjoyed the presentation very much. It was elegant as always. I have one question for you. Those of us who perform these complex procedures, like you, prefer a single entry from the ascending aorta and the attachment of branches as needed. You mentioned one patient who died of hyperperfusion syndrome several days later. We have all seen that problem. Do you have any advice for us in these operations, that is, should one stage them or do all of the grafts at once? It has been my practice to correct all of them at one sitting. And I have seen this problem. Could you enlighten us to what you think may be the mechanism of this syndrome, and would we do better to stage them rather than to perform the complete operation at the first sitting?

Dr. Berguer. As we all know, strokes[6] occur usually on days 3 through 5. I don't know that it is preventable by the means that we're using now, but we are controlling the systolic pressure to the point where we will not let it rise above 110, and we're giving steroids to these patients. The steroids have certainly been proven to control edema in the spinal cord in trauma. Because there is a certain similarity between the spinal cord tissue and the tissue we are discussing here, I thought it reasonable to give these patients steroids.

Whether to reconstruct one trunk or three trunks, I'm not sure that there is a terrible amount of difference. I'm not sure there is any difference whatsoever in the intracranial pressure when you reconstruct an innominate artery or an innominate artery and a left common carotid artery. I think the end pressure that you get is just about the same.

Dr. Thomas S. Riles (New York, N.Y.). I appreciated the paper very much, Dr. Berguer. I always learn a great deal from your presentations. In the strokes that you reported, you found the cause in all but three. I'm curious to know whether these were strokes that could have been caused by poor perfusion during the time of the reconstruction or clamping of the artery. For that reason, I ask, do you have any means of monitoring the cerebral blood flow, and if you find that there is diminished blood flow during that period of time, do you use a shunt?

Dr. Berguer. No, I don't use any method of monitoring the blood flow. We have used a shunt if we have to clamp the only patent vessel available for reconstruction, but otherwise we don't. I think the strokes were probably caused by debris from either the graft or the anastomotic site. They were very discrete. I obtained an angiogram but found absolutely nothing wrong with the grafts. I presume they were caused by embolic disease, but I am not certain about it.