Reconstruction of the superior vena cava: Benefits of postoperative surveillance and secondary endovascular interventions

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Purpose: Superior vena cava (SVC) reconstructions are rarely performed; therefore the need for surveillance and the results of secondary interventions are unknown.

Methods: During a 14-year period 19 patients (11 male, 8 female; mean age 41.9 years, range 8 to 69 years) underwent SVC reconstruction for symptomatic nonmalignant disease. Causes included mediastinal fibrosis (n = 12), indwelling foreign bodies (n = 4), idiopathic thrombosis (n = 2), and antithrombin III deficiency (n = 1). Spiral saphenous vein graft (n = 14), polytetrafluoroethylene (n = 4), or human allograft (n = 1) was implanted.

Results: No early death or pulmonary embolism occurred. Four early graft stenoses or thromboses (spiral saphenous vein graft, n = 2, polytetrafluoroethylene, n = 2) required thrombectomy, with success in three. During a mean follow-up of 49.5 months (range, 4.7 to 137 months), 95 imaging studies were performed (average, five per patient; range, one to 10 studies). Venography detected mild or moderate graft stenosis in seven patients; two progressed to severe stenosis. Two additional grafts developed early into severe stenosis. Four of 19 grafts occluded during follow-up (two polytetrafluoroethylene, two spiral saphenous vein graft). Computed tomography failed to identify stenosis in two grafts, magnetic resonance imaging failed to confirm one stenosis and one graft occlusion, and duplex scanning was inconclusive on graft patency in 10 patients. Angioplasty was performed in all four patients with severe stenosis, with simultaneous placement of Wallstents in two. One of the Wallstents occluded at 9 months. Repeat percutaneous transluminal angioplasty was necessary in two patients, with placement of Palmaz stents in one. Only one graft occlusion and one severe graft stenosis occurred beyond 1 year. The primary, primary-assisted, and secondary patency rates were 61%, 78%, and 83% at 1 year and 53%, 70%, and 74% at 5 years, respectively.

Conclusion: Long-term secondary patency rates justify SVC grafting for benign disease. Postoperative surveillance with contrast venography is indicated in the first year to detect graft problems. Endovascular techniques may salvage and improve the patency of SVC grafts. (J Vasc Surg 1998;27:287-301.)

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Obstruction of the superior vena cava (SVC) is most frequently caused by primary or metastatic mediastinal malignancies. Mediastinal fibrosis is the most frequent cause of nonmalignant occlusion, accounting for 10% to 20% of all patients who have SVC syndrome.^{1,2} Central lines, indwelling catheters, and pacemaker lines are additional causes of thrombosis. Revascularization of the SVC can be achieved by endovascular techniques³⁻¹⁹ or by surgical reconstruction.²⁰⁻³² Endovascular techniques such as percutaneous transluminal angioplasty (PTA) and stent placement with or without thrombolysis have been used preferentially for palliation

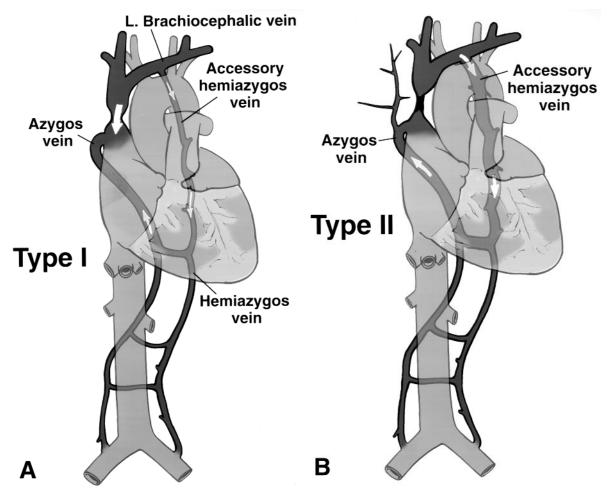


Fig. 1. A, Type I: high-grade superior vena cava stenosis but still normal direction of blood flow through superior vena cava and azygos veins. Increased collateral circulation through hemiazygos and accessory hemiazygos veins. **B**, Type II: greater than 90% stenosis or occlusion of superior vena cava with normal direction of blood flow through the azygos vein.

of symptoms in malignant disease.^{12,17} However, primary venous stenting has been performed with increasing frequency in patients with benign SVC syndrome.^{7,8,10,11,15,16,19} At our institution surgical treatment has been used since the mid-1980s to treat patients with symptomatic SVC syndrome.^{25,28,29,31,32} In some of these patients endovascular techniques have been used as secondary interventions to salvage failing SVC grafts. This report evaluates the late outcome of surgical reconstructions in SVC syndrome caused by benign disease and focuses on the benefits of postoperative surveillance and secondary endovascular interventions in maintaining graft patency.

METHODS

Between November 1, 1983, and December 31, 1996, 19 consecutive patients underwent surgical reconstruction for SVC syndrome caused by nonmalignant disease (Table I). Eleven men and eight women with a mean age of 41.9 years (range, 8 to 69 years) were evaluated. All had persistent signs and symptoms of SVC obstruction (mean, 22 months; range, 4 to 72 months) despite medical treatment and physical measures to decrease venous congestion of the head and neck (Table II). The most frequent signs of SVC syndrome were head or neck swelling (100%) and the presence of visible large chest-wall collateral veins (89%). The most frequent

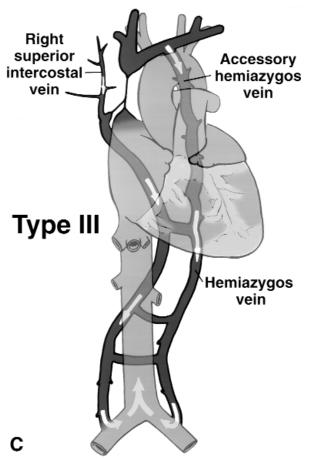


Fig. 1. C, Type III: occlusion of superior vena cava with retrograde flow in both azygos and hemiazygos veins.

symptoms included a feeling of fullness in the head or neck (74%), dyspnea on exertion or orthopnea (63%), and cough (32%).

The most frequent cause (Table I) of SVC syndrome was mediastinal fibrosis in 12 patients (63%) and a history of histoplasmosis in four. Indwelling foreign bodies (Hickman catheter, pacemaker wire, central line placement, and ventriculoatrial shunt) caused SVC thrombosis in four patients (21%). One patient had an attempted SVC replacement at another institution after early thrombosis of a Wallstent occurred. Another patient had a Palmaz stent placement for a type III lesion (Table III) in another hospital that resulted in early recurrent symptoms and thrombosis, and the patient was transferred to our institution for surgical reconstruction.

Preoperative imaging, in addition to biplane chest roentgenography, included bilateral upper

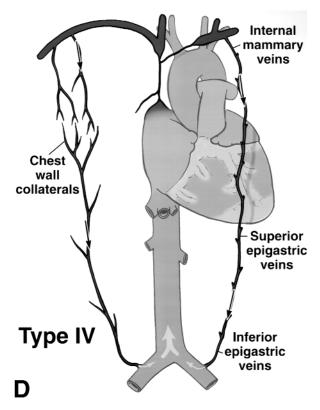


Fig. 1. D, Type IV: extensive occlusion of superior vena cava, innominate and azygos with chest wall and epigastric venous collaterals.

extremity venography in 19 patients (100%) and duplex scanning of the internal jugular veins in 12 (63%). Seventeen patients (89%) underwent computed tomography (CT) of the chest, four (21%) underwent magnetic resonance imaging, and four (21%) underwent echocardiography. Five patients underwent mediastinoscopy before SVC reconstruction.

With the classification scheme of Stanford and Doty,³³ on the basis of the severity of SVC occlusion and the extent of collateral venous circulation, our patients were classified into four types (Table III, Fig. 1).

The technique of autologous spiral saphenous vein graft has been described in detail previously.^{28,31} In brief, the saphenous vein is removed and opened longitudinally. We excise the valves and wrap the saphenous vein around a 32F or 36F polyethylene chest tube. The spiral vein graft is prepared by

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| Patient no. | Age (yr)/ sex | Cause | Location of graft | Type of graft | Early graft complications |
|----------------|------------------|------------------------------|--------------------------|-----------------|-----------------------------------|
| 1 | 36/M | Ventriculoatrial shunt | Left-IJV-RAA | SSVG | _ |
| 2 | 56/M | Pacemaker electrode | Right IVJ-RAA | SSVG | _ |
| 3 | 43/F | Mediastinal fibrosis* | Right IJV-left IV-RAA | SSVG | Graft thrombosis† (partial) |
| 4 | 27/M | Mediastinal fibrosis* | Left IV-RAA | SSVG | _ |
| 5 | 69/M | Pacemaker electrode | Right IJV-RAA | SSVG | _ |
| 6 | 8/M | Antithrombin III deficiency | IV-RAA | PTFE | Graft thrombosis† |
| 7 | 46/F | Idiopathic venous thrombosis | Right IJV-RAA | PTFE | Graft thrombosis† |
| 8 | 30/F | Mediastinal fibrosis* | Left IV-RAA | SSVG | _ |
| 9 | 41/F | Mediastinal fibrosis* | Left IV-RAA | Human allograft | _ |
| 10 | 65/F | Mediastinal fibrosis* | Left IV-RAA | SSVG | — |
| 11 | 44/M | Mediastinal fibrosis* | Left IJV-RAA | SSVG | _ |
| 12 | 36/M | Mediastinal fibrosis* | Left IV-RAA | SSVG | _ |
| 13 | 37/F | Central line placement | Left IJV-RAA | SSVG | _ |
| no- | | | | | |
| 14 | 46/M | Idiopathic venous thrombosis | Right IVJ-RAA | PTFE | _ |
| 15 | 40/M | Mediastinal fibrosis* | Left IV-right IJV-SVC | SSVG | Graft thrombosis† |
| 16 | 24/M | Mediastinal fibrosis* | Right IV-left IV-SVC | SSVG | _ |
| 17 | 45/M | Mediastinal fibrosis* | IV-SVC | SSVG | _ |
| 18 | 60/F | Mediastinal fibrosis* | Right IV-SVC | SSVG | _ |
| 19 | 44/F | Mediastinal fibrosis* | Left IV-RAA | PTFE | _ |

Table I. Clinical data of 19 patients with superior vena cava syndrome caused by nonmalignant disease

IJV, Internal jugular vein; RAA, right atrial appendage; IV, innominate vein; D scan, Duplex scanning; MRA, magnetic resonance angiography; SSVG, spiral saphenous vein; PTFE, externally supported expanded polytetrafluoroethylene. *History of histoplasmosis.

†Thrombectomy with revision.

\$\$coring system suggested by the Subcommittee of the Joint Vascular Societies on Reporting Standards in Venous

Disease³⁵: +3 = complete relief of symptoms, +2 = marked relief of the symptoms, +1 = mild clinical improvement, 0 = no clinical improvement.

suturing the edges of the saphenous vein with running 6.0 or 7.0 monofilament nonabsorbable sutures. Harvesting vein from the groin to the knee will result in an approximately 10 cm long spiral saphenous vein graft.

Reconstruction with autologous spiral saphenous vein graft (SSVG) was performed in 14 patients (74%); 12 had straight SSVGs, and two had bifurcated grafts. Of the straight grafts, the site of the proximal anastomosis was the internal jugular vein in

five patients and the innominate vein in seven. In one patient (case 16) the left innominate vein was reimplanted into a right innominate vein. One limb of each of the two bifurcated grafts originated from the internal jugular vein, and the other originated from the innominate vein. The site of the central anastomosis was the right atrial appendage in 10 patients (71%) and the SVC in four (29%).

Four patients received an externally supported expanded polytetrafluoroethylene (ePTFE) graft,

| Patency at discharge | Late complications | Endovascular treatment | Last imaging | Graft patency documented by imaging | Clinical out come score‡ |
|-------------------------|--|--|----------------------|--|--|
| Patent | _ | | Venogram | Patent at 5 mo | +2 at 5 mo, lost to follow-up |
| Patent | _ | | CT scan | Patent at 72 mo | +3 at 96 mo |
| Patent | 3 restenoses (at 37, 51 and 71 mo) | PTA (×3) | Venogram | Right limb not visualized by CT at 3 mo; <i>both</i> (!) limbs Patent at 72 mo | +2 at 95 mo |
| Patent | — | — | Venogram | Patent at 73 mo | +3 at 88 mo |
| Patent | _ | _ | Duplex scan | Patent at 65 mo | +3 at 84 mo |
| Patent | _ | _ | MRA | Patent at 23 mo | +2 at 24 mo, lost to follow-up |
| Patent | _ | _ | Duplex scan | Occluded at 5 mo | 0 at 37 mo |
| Patent | _ | _ | MRA | Patent at 52 mo | +2 at 52 mo |
| Patent | _ | _ | Duplex scan | Patent at 22 mo | +3 at 28 mo |
| Patent | _ | _ | Venogram | Patent at 17 mo | +3 at 18 mo |
| Patent | 3 restenoses (at 3, 5, and 6 mo) | PTA at 3 mo, PTA with Palmaz stents at 5 and 6 | Venogram | Patent at 14 mo | +2 at 20 mo |
| Patent | 1 restenosis (at 10 mo) | Treated with PTA+Wallstent | Venogram | Patent at 17 mo | +3 at 17 mo |
| Patent | _ | _ | Venogram | Patent at 109 mo | +2 at 109 mo Death (bron- chogenic carci- ma) |
| Patent | — | — | CT scan | Patent at 57 mo | +3 at 137 mo |
| 1 limb occluded | _ | _ | CT scan | l limb patent at 4 mo, 1 limb occluded | +1 at 8 mo |
| Patent | 1 restenosis (at 5 mo) | Treated with PTA+Wallstent | Venogram | Occluded at 9 mo | +1 at 9 mo |
| Patent | — | | Venogram | Patent at 49 mo | +3 at 59 mo |
| Patent Patent | | | Venogram Venogram | Patent after surgery Occluded at 26 mo | +3 at 45 mo +2 at 26 mo |

two originating from the internal jugular vein and two from the innominate vein. The central anastomoses was performed with the atrial appendage in all four patients.

One additional patient with severe symptoms of SVC who had concomitant orthotopic liver transplantation received a human iliocaval allograft implanted from the left innominate vein to the right atrial appendage. This patient was reported recently.³²

The Kaplan-Meier method was used to calculate primary, primary-assisted, and secondary graft patency rates.³⁴

All except two patients were receiving oral anticoagulation at discharge. Low molecular weight heparin was used in one patient, and dipyridamole was given to the remaining patients to decrease thrombotic complications.

RESULTS

No early deaths or pulmonary thromboembolism occurred. Four early reinterventions were necessary for stenosis in one patient and for occlusion in three. The stenosis occurred in a bifurcated SSVG; the occlusions occurred in one limb of a bifurcated SSVG and in two PTFE grafts. A fifth patient required reoperation for evacuation of a mediastinal hematoma. The 30-day primary patency rate was 79% (15 of 19), and the secondary patency rate was 95% (18 of 19). One limb of a bifurcated SSVG graft reoccluded.

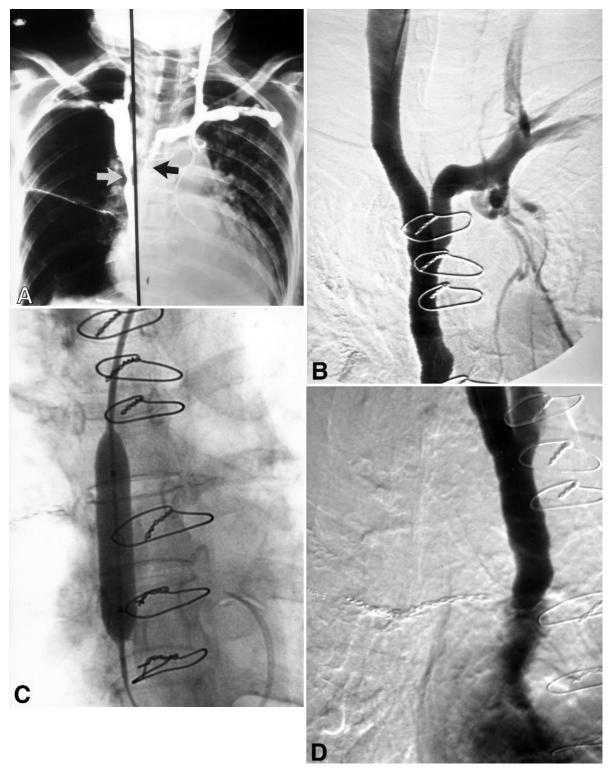
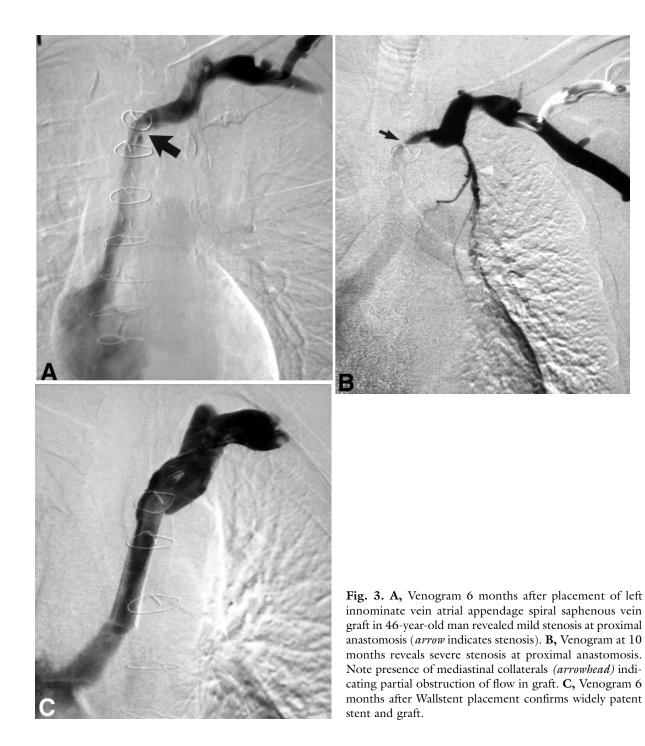


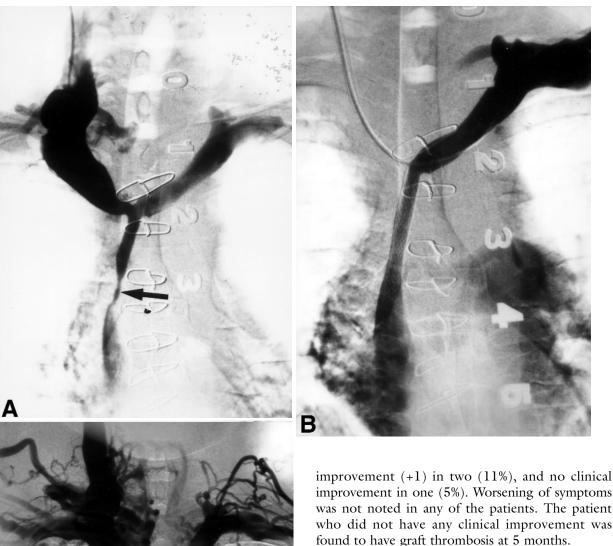
Fig. 2. A, Postoperative venogram of 43-year-old woman after bifurcated spiral saphenous vein graft implantation reveals bilateral stenosis (*arrows*) in limbs of bifurcated graft. B, Venogram at 37 months reveals widely patent limbs of bifurcated graft. Lysis of thrombus in graft occurred while patient was receiving oral anticoagulation. C, Percutaneous transluminal angioplasty of stenosis at the atrial anastomosis. D, Venogram confirmed successful angioplasty of anastomotic stenosis with no residual gradient.



During a mean follow-up of 49.5 months (range, 4.7 to 137 months), one late death occurred as a result of bronchogenic carcinoma at 9 years in a female patient who had a patent SSVG and no symptoms of SVC syndrome at the time of her death (case 13). Three patients were lost to follow-up at 5, 8, and 24 months, respectively (Table I). The median follow-up for the last imaging studies was 23

months, whereas the median follow-up of clinical outcome was 37 months.

The late clinical outcome was assessed with a classification scheme suggested by the Subcommittee of the Joint Vascular Societies on Reporting Standards in Venous Disease.³⁵ Complete relief of symptoms (+3) was obtained in nine patients (46%), marked relief (+2) in seven (33%), only mild clinical



Postoperative imaging confirmed nonsignificant (<50%) stenosis in seven patients, six within the first year. In three patients stenosis was noted before hospital dismissal, in two at the first postoperative visit, and in the last at 34 months. Two of these patients progressed to severe stenosis, requiring endovascular treatment at 3 months and 37 months after surgery (cases 3 and 11; Table I). Two additional patients had severe stenosis. All four patients with severe stenosis had recurrent symptoms at the time of presentation. Three stenoses developed within 1 year after surgery (at 3, 5, and 10 months, respectively), and the fourth patient had severe recurrent stenosis at 37 months. This patient (case 3) underwent early thrombectomy for high-grade stenosis caused by thrombus deposition throughout the entire graft. The patient was discharged with mild graft stenosis, and at 3 months the right limb of the bifurcated graft could not be seen with CT scan. Oral antico-

Fig. 4. A, Venogram at 5 months in 24-year-old man reveals severe stenosis (*arrow*) in right innominate vein superior vena cava spiral saphenous vein graft. Left innominate vein was reimplanted into saphenous vein graft. B, Venogram after placement of Wallstent (10×60 mm) shows good flow through the graft. C, Venogram at 9 months confirms occluded graft and stent.

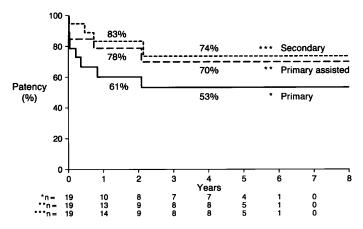


Fig. 5. Cumulative patency rates of 19 bypass grafts used for superior vena cava reconstruction.

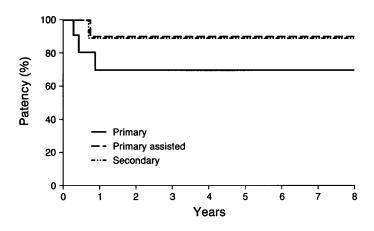


Fig. 6. Cumulative patency rates of 12 straight spiral saphenous vein grafts used for superior vena cava reconstruction.

agulation was maintained, and both limbs of this bifurcated SSVG were widely patent on venography at 37 months. However, recurrent stenosis at the atrial anastomosis required balloon angioplasty. The incidence of early and late stenosis in our total experience was 37% (7 of 19).

All four patients with high-grade stenosis underwent PTA, stenting, or both. Recurrent stenosis in case 3 was initially treated first with PTA at 37 months. Repeat PTAs were performed at 51 and 71 months because of recurrent symptoms. This patient currently has minimal residual symptoms at 95 months after surgery (Fig. 2).

PTA performed at 3 months for early restenosis was not successful in a 44-year-old man with SSVG to prevent recurrence of symptoms (case 11). PTAs with placement of Palmaz stents (Johnson and Johnson Interventional Systems) at 5 and 6 months at the referring hospital were successful, with patent graft documented at 14 months and significant clinical improvement documented at 20 months.

A third patient with symptomatic stenosis of a straight SSVG at 10 months (case 12) was treated with PTA and placement of a Wallstent. Venography at 17 months revealed no pressure gradient and a widely patent graft (Fig. 3) with excellent clinical result. The fourth patient with an SSVG had high-grade stenosis at 5 months after surgery. This SSVG had the left innominate vein implanted into the side of a right innominate vein-SVC graft, and revisions for kinking were performed during initial implantation. Treatment was attempted with PTA and placement of a 10 mm self-expanding metallic stent (Wallstent, Medivent). The pressure gradient of 26 mm Hg was decreased to a residual gradient of 16 mm Hg. Reocclusion with recurrence of symptoms was documented at 9 months after surgery (Fig. 4). After stent placement all

Table II. Signs and symptoms of SVC syndrome in19 patients with benign disease

| Signs | No. of patients | % |
|-------------------------------------|--------------------|-----|
| Head and neck swelling | 19 | 100 |
| Large chest wall venous collaterals | 17 | 89 |
| Arm swelling | 11 | 58 |
| Facial cyanosis | 8 | 42 |
| Symptoms | | |
| Feeling of fullness in head or neck | 14 | 74 |
| Dyspnea on exertion or orthopnea | 12 | 63 |
| Headaches | 7 | 37 |
| Visual problems, painful eyes | 5 | 26 |
| Cough | 6 | 32 |

patients underwent full anticoagulation with heparin and then with warfarin.

Three additional graft occlusions occurred during follow-up. One was the early reocclusion of one limb of a bifurcated SSVG (case 15). Another patient with extensive type IV SVC syndrome (case 7) had occlusion of a jugular vein-atrial appendage PTFE graft. Because of the extensive occlusive disease and small jugular vein, a 6-mm graft had to be used in this patient. After successful early thrombectomy was performed, the graft reoccluded at 5 months. Another 10 mm PTFE graft occluded at 26 months after surgery (case 19).

The long-term secondary patency rate was 100% (three of three) in patients with type I disease, 67% (two of three and four of six, respectively) in patients with types II and III, and 85% (six of seven) in patients with type IV disease. Primary, primary-assisted, and secondary patency rates of all grafts were 61%, 78%, and 83% at 1 year and 53%, 70%, and 74% at 5 years, respectively (Fig. 5). Straight SSVGs had a 70% primary patency rate and a 90% secondary patency rate at both 1 and at 5 years (Fig. 6).

During follow-up of the 19 patients, 95 imaging studies were performed, averaging five per patient (range, 1 to 10). Forty-five venograms were obtained and documented mild or moderate stenosis in eight studies (seven patients) and severe graft stenosis in eight grafts or limbs (four patients). Of the noninvasive examinations, 22 CT scans, three magnetic resonance imaging examinations, and 19 postoperative duplex scan examinations were performed. CT failed to identify graft stenosis in two of seven cases and was inconclusive in one. Magnetic resonance imaging failed in two out of two cases (one stenosis, one occlusion), and duplex scanning was inconclusive on graft patency in 10. In none of the patients could duplex scanning visualize the graft

Table III. Venographic classification of 19 patients with SVC syndrome according to Stanford and Doty³³

| Туре | No. of patients | % |
|---|--------------------|-----|
| I. Stenosis (<90%) of SVC with patency and antegrade flow of azygos-right atrial pathway | 3 | 16 |
| II. >90% stenosis or occlusion of SVC with patency and antegrade flow in azygos-right atrial path | 3 | 16 |
| III. >90% stenosis or occlusion of SVC with reversal of azygos blood flow | 6 | 32 |
| IV. Occlusion of SVC and one or more major caval tributary including azygos systems | 7 | 37 |
| Total | 19 | 100 |

in the mediastinum. Patency of the internal jugular veins with good flow velocities and associated respiratory variations were indirect evidence of graft patency.

DISCUSSION

Surgical treatment of SVC syndrome has documented long-term success in both benign and malignant disease.²⁰⁻³² Of the available autologous material, SSVG has been used most frequently for bypass grafting.^{20,24,25,28} Advantages of SSVG are that it is autologous material and that graft diameter can be matched to the size of the internal jugular or innominate veins. Potential disadvantages include the time needed to prepare the graft (45 to 90 minutes), limitations in lengths, and that endothelial injury during preparation may increase thrombogenicity. We had early thrombosis in two bifurcated SSVGs, and thrombectomy with revision failed to salvage one limb in one graft. Severe symptomatic stenosis developed within the first few months in three SSVGs and at 37 months in another. Patency of three grafts could be salvaged by endovascular techniques. Our study confirmed that straight SSVGs have excellent long-term patency rates, with 11 of 12 grafts patent during an average follow-up of 50 months. The only occluded straight SSVG required revision immediately after placement because of kinking caused by reimplantation of the left innominate vein into the graft. Subsequent placement of a 10 mm Palmaz stent in the stenotic graft failed to maintain patency.

Good long-term results with SSVG used for treatment of patients with nonmalignant SVC syndrome were also reported by Doty.²⁰ In a group of nine patients, seven with mediastinal fibrosis, two

| First author (yr) | No. of patients | Sex/age (yr) | Cause | Type of lesion* | Treatment | Patency at last imaging | Clinical outcome score† |
|-------------------------------------|--------------------|-----------------|--|--------------------|--|---|---|
| Sunder ¹¹ (1992) | 1 | F/68 | Pacing electrode | IV | Lysis (UK)+PTA | ? | +3 (12 mo) |
| Sherry ⁴ (1986) | 1 | F/82 | Pacing electrode | Ι | РТА | Restenosis at 15 mo | +3 after repeated PTA |
| Grace ⁸ (1991) | 1 | F/53 | Pacing electrode | Ι | РТА | ? | +3 (6 mo) |
| Lindsay ¹⁵ (1994) | 1 | M/63 | Pacing electrode | III | Lysis+PTA+Wall- stent (16 mm) | Thrombus in stent at 6 mo. | +3 (6 mo) |
| Dodds ² (1994) | 1 | M/44 | Mediastinal fibrosis | II | PTA+Palmaz stents (12&15 mm) | Patent after thrombolysis at 8 mo | +3 (8 mo) |
| Kastner ¹⁸ (1995) | 1 | M/45 | Pacing electrode | Ι | РТА | Patent at 6 mo | +2 (6 mo) |
| Walpole ⁶ (1988) | 1 | M/71 | Pacing electrode | II | РТА | Patent at 9 mo | +2 (9 mo) |
| Frances ¹⁶ (1995) | 1 | M/70 | Pacing electrode | III | TA+Wallstent 16 mm | Patent at 6 mo | +3 (6 mo) |
| Rösch ¹² (1992) | 2 | F/58 | Postradiation fibrosis | III | Gianturco-Rösch self-expandable Z-stents | Mild stenosis at 2 mo | +3 (11 mo, death of unrelated cause) |
| | | ? | Mediastinal fibrosis | II | Gianturco-Rösch self-expandable Z-stents | ? | +3 (12 mo) |
| Dondelinger ¹⁹ (1996) | 20 | ? | Central venous catheter, 16; mediastinal fibrosis, 1; idiopathic thrombosis, 3 | ? | PTA + Wallstent or Gianturco stent | l failure; 6 redo PTA with stents | Primary patency rate, 72% |

Table IV. Endovascular treatment of nonmalignant SVC syndrome

*See Table III.

graft occlusions were noted at 5 and 12 months after surgery. Seven of nine patients had excellent clinical results at a mean follow-up of 82 months. These results support our policy to continue to use SSVG as the graft of choice in these patients. However, increasing success with superficial femoral vein as an arterial conduit has resurrected this autologous graft for large vein reconstructions as well. Recent reports on good early results indicate that when available, autologous femoropopliteal vein shows promise for replacement of large central veins.³⁶ Still, the morbidity of harvesting a deep vein in patients with thrombotic potentials and venous thrombosis elsewhere in the body is not well known.

Early failure of grafts implanted in the venous system can be caused by technical failure resulting in stenosis of the graft at the anastomosis or extrinsic compression by the thoracic inlet or the sternum. Low flow may also result in early thrombosis of prosthetic grafts. Intimal hyperplasia and recurrent venous thrombosis have been causes of late graft failure. Therefore in patients who have anticoagulation abnormalities, lifelong anticoagulation is warranted.

Of the available prosthetic materials, externally supported expanded PTFE continues to be the best conduit. Grafts with less than 10 mm internal diameter, however, have poor patency rates because low flow results in thrombus deposition on the thrombogenic surface of a prosthesis. One of our two occluded prosthetic grafts was 6 mm in diameter. Moore and Hollier²⁷ reported six patients with nonmalignant central vein occlusion treated with PTFE grafts. Five of these patients, however, had brachial arteriovenous fistula placed to improve flow through the prosthetic grafts, indicating that high flow is required to keep prosthetic grafts patent in the venous system. All grafts had documented patency at 1 year, and all patients had excellent clinical results at a mean follow-up of 30 months.

Our data indicate that contrast venography is the most reliable study to document stenosis in central vein grafts, and duplex scanning will only provide indirect evidence of patency. Contrast venography has been performed in all patients before discharge and is recommended again at 3 to 6 months for postoperative surveillance even in patients who have no symptoms. We have to admit, however, that currently available data do not support the use of routine venography in all patients. Most problems occurred in our study within the first year, and two patients with mild to moderate stenosis progressed to severe stenosis. Follow-up venography in patients with documented stenosis is warranted even after 1 year. It is noteworthy, however, that all patients with severe stenosis had recurrent symptoms, and of the three late occlusions only one patient had no warning symptoms before occlusion. In patients with renal insufficiency, magnetic resonance angiography has become our second choice. Our experience is accumulating with magnetic resonance angiography with gadolinium enhancement, but artifacts from pacemakers in many of these patients make followup with this examination problematic.

Consideration of endovascular therapy as an adjunctive measure to improve results of surgical SVC reconstruction is important. In our series endovascular therapy was useful in a small group of patients; however, it resulted in increased patency rates and symptomatic improvement in three of four patients treated. All four patients who had symptomatic severe stenosis during follow-up underwent PTA, and three had stent placement.

Three basic stent types have been used most frequently for treatment of SVC syndrome: the Gianturco Z stents (with the Rosch-modification), the Wallstent, and the Palmaz stent. Advantages of the Z stent are that it can be custom-built to appropriate length and it is available in larger sizes (16 mm and greater), which makes it more conducive for the treatment of patients with central vein obstruction. However, stent migration has occurred with Gianturco Z stents, especially with the unibody design. Rösch et al.¹² successfully added small barbs and used multibody design to prevent stent migration.

Wallstents have been used successfully to treat patients with SVC obstruction.¹⁶ The 16 mm or larger diameter stents have only become recently available in the United States. The advantage of this stent is its flexibility, and in our patient it allowed placement along the curved extent of an SSVG. The disadvantage of the Wallstent is the decreased expansile force compared with Z stents or Palmaz stents.

Palmaz stents are widely used in the arterial bed, but their use for SVC syndrome has also been reported.^{2,9} The advantage of the Palmaz stent is the precision with which it can be placed and the ability to increase the diameter as needed at deployment. Its disadvantage is the lack of flexibility.

In patients with malignant disease short-term palliation is the goal, and endovascular techniques have been quite successful. In nonmalignant SVC syndrome, however, reconstruction with reliable longterm patency is required. Currently available literature on endovascular techniques in this group of patients is limited to case reports or small series (Table IV), and results on patency beyond 1 year have usually not been reported. Recently presented data of Dondelinger and Trotteur,¹⁹ however, are promising, because these authors performed stenting in 26 patients with large vein occlusion caused by benign disease and in 20 with SVC syndrome. The secondary patency rate in the entire group was 80% after a mean follow-up of 16 months. Although additional data are needed, the observations of these authors support the preferential use of stenting in patients with types I and II SVC obstruction caused by benign disease. Primary surgical treatment in our experience should be offered to all patients with more extensive benign SVC occlusion (types III and IV) or if endovascular attempts fail in less severe disease.

CONCLUSION

Long-term secondary patency rates justify SVC grafting for benign disease. In our experience straight SSVGs have performed the best and continue to be our graft of choice for this operation. Postoperative surveillance and contrast venography before discharge and again at 3 to 6 months is indicated to detect graft stenosis. Beyond the first year patients should undergo contrast venography when symptoms develop. Endovascular techniques with PTA and stenting are useful, and they can salvage and improve the patency of SVC grafts.

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REFERENCES

 Parish JM, Marschke RF, Dines DE, Lee RE. Etiologic considerations in superior vena cava syndrome. Mayo Clinic Proc 1981;56:407-13.

- Dodds GA, Harrisson JK, O'Laughlin MP, Wilson JS, Kisslo KB, Bashore TM. Relief of superior vena cava syndrome due to fibrosing mediastinitis using the Palmaz stent. Chest 1994;106:315-8.
- Charnsangavej C, Carrasco CH, Wallace S, Wright KC, Ogawa K, Richli W, et al. Stenosis of the vena cava: preliminary assessment of treatment with expandable metallic stents. Radiology 1986;161:295-8.
- Sherry CS, Diamond NG, Meyers TP, Martin RL. Successful treatment of superior vena cava syndrome by venous angioplasty. AJR 1986;147:834-5.
- Putnam JS, Uchida BT, Antonovic R, Rösch J. Superior vena cava syndrome associated with massive thrombosis: treatment with expandable wire stents. Radiology 1988;167:727-8.
- Walpole HT, Lovett KE, Chuang VP, West R, Clements SD. Superior vena cava syndrome treated by percutaneous transluminal balloon angioplasty. Am Heart J 1988;115:1303-4.
- Spittell PC, Vliestra RE, Hayes DL, Higano ST. Venous obstruction due to permanent transvenous pacemaker electrodes: treatment with percutaneous transluminal balloon venoplasty. Pacing Clin Electrophysiol 1990;13:271-4.
- Grace AA, Sutters M, Schofield PM. Balloon dilatation of pacemaker induced stenosis of the superior vena cava. Br Heart J 1991;65:225-6.
- 9. Solomon N, Wholey MH, Jarmolowski CR. Intravascular stents in the management of superior vena cava syndrome. Cathet Cardiovasc Diagn 1991;23:245-52.
- Chatelain P, Meier B, Friedli B. Stenting of superior vena cava and inferior vena cava for symptomatic narrowing after repeated atrial surgery for D-transposition of the great vessels. Br Heart J 1991;66:466-8.
- Sunder SK, Ekong EA, Silvalingam K, Kumar A. Superior vena cava thrombosis due to pacing electrodes: successful treatment with combined thrombolysis and angioplasty. Am Heart J 1992;123:790-2.
- Rösch J, Uchida BT, Hall LD, Antonovic R, Petersen BD, Ivancev K, et al. Gianturco-Rosch expandable Z-stents in the treatment of superior vena cava syndrome. Cardiovasc Intervent Radiol 1992;15:319-27.
- Dake MD, Semba CP, Enstrom RJ, Skeens JL, Venugopal C, Wittich GR. Percutaneous treatment of venous occlusive disease [abstract]. J Vasc Interv Radiol 1993;4:42.
- Kishi K, Sonomura T, Mitsunaze K, Nishida N, Yang R-J, Sato M, et al. Self-expandable metallic stent therapy for superior vena cava syndrome: clinical observations. Radiology 1993;189:531-5.
- Lindsay HSJ, Chennells PM, Perrins EJ. Successful treatment by balloon venoplasty and stent insertion of obstruction of the superior vena cava by an endocardial pacemaker lead. Br Heart J 1994;71:363-5.
- Frances CM, Starkey IR, Errington ML, Gillepsie IN. Venous stenting as treatment for pacemaker-induced superior vena cava syndrome. Am Heart J 1995;129:836-7.
- Furui S, Sawada S, Kuramoto K, Inove Y, Irie T, Maakita K, et al. Gianturco stent placement in malignant caval obstruction: analysis of factors for predicting the outcome. Radiology 1995;195:147-52.
- Kastner RJ, Fisher WG, Blacky AR, Bacon ME. Pacemakerinduced superior vena cava syndrome with successful treatment by balloon venoplasty. Am J Cardiol 1996;77:789-90.
- Dondelinger RF, Trotteur G. Expandable stents in the venous system. Abstracts of the International Symposium, Leuven, Belgium. December 14, 1996.

- 20. Doty DB. Bypass of superior vena cava: six years' experience with spiral vein graft for obstruction of superior vena cava due to benign and malignant disease. J Thorac Cardiovasc Surg 1982;83:326-38.
- Bergeron P, Reggi M, Jausseran J, Huet R, Ferdani M, Martelet JP, et al. Our experience in superior vena cava surgery [in French]. Ann Chir: Chir Thorac Cardiovasc 1985;39:485-91.
- 22. Herreros J, Glock Y, Fuente A, Gil A, Arcas R, Llorens R, et al. The superior vena cava compression syndrome. Our experience of twenty-six cases [in French]. Ann Chir: Chir Thorac Cardiovasc 1985;39:495-500.
- Ricci C, Benedetti VF, Colini GF, Gossetti B, Mineo TC. Reconstruction of the superior vena cava: 15 years' experience using various types of prosthetic material. Ann Chir 1985;39:492-5.
- 24. Doty DB. Bypass of superior vena cava: fifteen years' experience with spiral vein for obstruction of superior vena cava caused by benign disease. J Thorac Cardiovasc Surg 1990;99:889-96.
- Gloviczki P, Pairolero PC, Cherry KJ, Hallett JW. Reconstruction of the vena cava and of its primary tributaries: a preliminary report. J Vasc Surg 1990;11:373-81.
- Dartevelle PG, Chapelier AR, Pastorino U, Corbi P, Lent B, Cerrina J, et al. Long-term follow-up after prosthetic replacement of the superior vena cava combined with resection of mediastinal-pulmonary malignant tumors. J Thorac Cardiovasc Surg 1991;102:259-65.
- Moore WM Jr, Hollier LH. Reconstruction of the superior vena cava and central veins. In: Bergan JJ, Yao JST, editors. Venous disorders. Philadelphia: W. B. Saunders; 1991. p. 517-27.
- Gloviczki P, Pairolero PC, Toomey BJ, Bower TC, Rooke TW, Stanson AW, et al. Reconstruction of large veins for nonmalignant venous occlusive disease. J Vasc Surg 1992;16:750-61.
- Bower TC, Nagorney DM, Toomey BJ, Gloviczki P, Pairolero PC, Hallett JW, et al. Vena cava replacement for malignant disease: is there a role? Ann Vasc Surg 1993;7:51-62.
- Magnan PE, Thomas P, Giudicelli R, Fuentes P, Branchereau A. Surgical reconstruction of the superior vena cava. Cardiovasc Surg 1994;2:598-604.
- Gloviczki P, Bower TC, McKusick M, Pairolero PC. Superior vena cava syndrome: endovascular and direct surgical treatment. In: Gloviczki P, Yao JST, editors. Handbook of venous disorders. London: Chapman & Hall; 1996. p. 580-99.
- Rhee RY, Gloviczki P, Steers JL, Krom RAF, Kitabayashi K, Wiesner RH. Superior vena cava reconstruction using an iliocaval allograft. Vasc Surg 1996;30:77-83.
- 33. Stanford W, Doty DB. The role of venography and surgery in the management of patients with superior vena cava obstruction. Ann Thorac Surg 1986;41:158-63.
- Kaplan EL, Meier P. Nonparametric estimation from incomplete observations. J Am Stat Assoc 1958;53:457-81.
- Porter JM, Rutherford RB, Clagett GP, Cranley JJ Jr, O'Donnell TF, Raju S, et al. Reporting standards in venous disease. J Vasc Surg 1988;8:172-81.
- Hagino RT, Bengtson TD, Fosdick DA, Valentine RJ, Clagett GP. Venous reconstructions using the superficial femoral–popliteal vein. J Vasc Surg 1997;26:829-37.

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DISCUSSION

Dr. Larry H. Hollier (New York, N.Y.). First of all, I would like to congratulate Dr. Alimi on a very nice presentation. Dr. Gloviczki and his colleagues at Mayo Clinic are also to be congratulated for a superb article.

The large patient volume that Mayo Clinic has and their excellent ability to provide long-term follow-up has provided us with some very valuable data demonstrating the efficacy of superior vena caval reconstruction. As many of you recall, for many years we went under the assumption that major central venous reconstruction had very little value and had a very high failure rate. I think most of us now are quite convinced that superior vena caval reconstruction does have a very valuable role to play in treating patients who have quite disabling symptoms.

I was interested to see that they used so many spiral vein grafts in the reconstruction of these obstructions. It has been my impression for a long time that the external supported PTFE grafts had a distinct advantage in these central venous reconstructions. I noticed also that you did not include the data on patients with malignant disease, where we have included that as we reviewed these studies and found there that very clearly external support was of some added value.

In a previous study performed by myself and some of my cardiology colleagues, Chris White and Steve Ramey and others, we compared the central venous reconstructions with primary endovascular procedures. And looking at the 1-year patency rate of the surgical reconstructions, we had approximately an 87% primary patency rate. If we looked at the 1-year patency rate of the balloon angioplasty patients, that was down to approximately 35%. However, when we looked at the patients for long-term combined patency, both secondary and assisted patency, we found that out to 2 years the patency rate in both groups was still in the mid-80% range and that surgical reconstruction did not add any additional increase in patency compared with those patients treated with endovascular procedures alone if you allowed secondary balloon angioplasty or stenting in these patients.

So my first question to the authors is how frequently are you using endovascular procedures as the primary treatment of central venous obstruction? And of those in whom you have done primary endovascular procedures, what has been the patency rate of those patients?

Another question is that most of the rapid development of symptoms of central venous obstruction we have seen has been related to patients receiving chronic hemodialysis with an atrioventricular access in the same arm where you might have an innominate obstruction. How many of your patients were undergoing chronic dialysis during these times of reconstruction, and how many of the stenoses were related specifically to catheterinduced obstruction in patients receiving chronic dialysis?

Again, the final question I would ask specifically is do you still believe that there is an added value for the use of spiral vein grafts compared with PTFE grafts, and how would you compare that with the endovascular techniques?

Dr. Yves S. Alimi. Thank you, Dr. Hollier, for your thoughtful comments. Your expertise in this field is well recognized. I have the following answers for your questions.

In patients with malignant disease, short-term palliation is the goal and endovascular technique has been quite successful. In nonmalignant superior vena cava syndrome, however, reconstruction with reliable long-term patency is required. Currently available literature on endovascular procedures in this group of patients is limited to case reports of small series, and results on patency beyond 1 year have usually not been reported.

We saw recently the data of Dondelinger, however, which are promising because his group performed stenting in 26 patients with large vein occlusion caused by benign disease including 20 patients with superior vena cava syndrome. The secondary patency rate in the entire group was 80% after a mean follow-up of 16 months. Also, additional data are needed. Observations of this author support a preferential use of stenting in patients with type I and II benign disease.

Concerning the question on dialysis, we had no patient on dialysis during reconstruction, so this series is a little bit different than the one you published.

The third question was about the benefit of spiral saphenous vein graft. In our point of view, spiral saphenous vein graft has definite advantages; it is autologous material, and the graft diameter can be matched to the size of the internal jugular or innominate vein. Potential disadvantages include the time needed to prepare the graft, limitation in length, and that endothelial injury may occur during preparation, which may increase thrombogenicity. In our series we had the best results with spiral saphenous vein graft, and it remains our first choice.

Dr. Jorg D. Gruss (Kassel, Germany). I enjoyed your paper very much and want to point out that there is a very elegant extraanatomic alternative treatment for decompressing the superior caval vein. We just implant the greater saphenous vein into the infraclavicular subclavian vein. If necessary, we can do this on both sides. The results of this simple procedure are convincing because of the high pressure gradient. Thank you.

Dr. G. Patrick Clagett (Dallas, Tex.). I rise to point out that there is an alternative vein graft and that is the superficial femoral vein. It is a large caliber vein that is readily available, straightforward to harvest, and associated with minimal venous morbidity. We have used this in two patients with SVC syndrome, one for malignant and one for benign disease, with a follow-up of 3 years and 1 year. They are both widely patent, and it seems to be an excellent choice for this problem.

Dr. Laurens R. Pickard (Houston, Tex.). You men-

tioned one of your patients who required repeated balloon angioplasty successfully, and it made me wonder if you have a policy now on when you go to a stent after an initial balloon angioplasty for stenosis, why you would have repeated balloons without a stent?

Dr. Alimi. We use stents selectively in patients with an extrinsic compression of central veins or in those with recurrent stenosis after the first PTA. We always measure pressure above and below the stenosis. If the pressure gradient is still high after the PTA, most patients undergo stent placement.

Dr. Peter Gloviczki (Rochester, Minn.).Just one additional comment to the site and the choice of the stent. If the stenosis is within the graft, then we would certainly

prefer angioplasty and stenting. If it is right in the right atrial appendage or at that anastomosis, our radiologist may be a little bit reluctant to place a stent because of failure of distal displacement of the stent.

And the other comment that I would like to make is that we have shifted a little bit during the last decade, and certainly in type I and type II lesions where there is only a short segment of stenosis, we too perform primary stenting and that is our first choice. But most of the patients who undergo reconstruction now are either failure of previous stent placement or they have extensive venous occlusion that is chronic and not suitable for thrombolysis or stenting.