

3. The significant recoil after simple dilatation warrants a stent insertion in all cases following venoplasty.
4. Stents should be inserted well into the IVC to prevent recurrence of central stenosis.
5. The definite objective preoperative test to use for selection for this procedure needs to be defined.

CASE OF SECONDARY DEEP VENOUS DISEASE

**Robert L. Kistner, MD
Straub Clinic and Hospital
Honolulu, Hawaii, USA**

This 47-year old healthy male was referred with a 4-year history of chronic venous ulceration of the right lower extremity due to post-thrombotic disease of the femoral-popliteal-tibial veins.

The problem began 4 years ago with the spontaneous onset of itching, discoloration, and a small sore on the posterior aspect of the calf. Swelling and chronic aching with dependency were progressive, and the ulcer grew to a large, encircling lesion of the entire circumference of the calf. Treatment by a series of 8 physicians entailed many medications during this time, but elastic support was not prescribed. No objective tests were done. During this time the problem was progressive.

A clinically apparent DVT of the SFV was diagnosed one year ago and treated with heparin-coumadin. Workup for a hypercoagulable state was negative.

3 months prior to this referral, he consulted a new physician who diagnosed severe deep venous reflux and prescribed elevation of the leg, pneumatic compression and pumping of the leg, and limitation of activity. His ulcer closed over the next three months.

The dominant symptoms were aching and swelling of the leg, and venous claudication with walking. The need to pump his leg interfered with his occupation as a traveling sales person. He had to stop his hunting activities. The problem was that the treatment prescription to control the ulceration required a life-style change that was incompatible with his desired way of life, and with his occupation.

P.E.: Healthy, strong 47-year old male. 5'10" tall, 230 lbs. Normal physical examination including arterial pulses, except for venous findings in RLE: Right calf 1" larger than left; large discoloration encircling the leg, 6" in length on post-lat and 2" on medial sides; no induration of skin, good turgor. V.V. in leg, mild.

Doppler: Mild local reflux in Posterior tibial vein, local and sustained 4-second Valsalva reflux in the popliteal vein. Late onset reflux in GSV, as found with perforator reflux. Normal phasic flow in common femoral vein.

Phase II.

Duplex scan: Occlusion of SFV to adductor canal.

PFV-popliteal connection, with 2 second reflux

SFV-popliteal collateral, with 4 second reflux

Incompetent greater saphenous vein, with low velocity 4-second reflux

Popliteal, crural, and short saphenous reflux, low velocity, 4 second duration

Incompetent, 2.0 mm perforators, medial and lateral lower calf, single

Summary: Post-thrombotic extremity with mixed obstruction and reflux. Occlusion of entire SFV, and low velocity reflux in all veins.

APG: O.F.-normal at 50%

VFI - 2.72 ml/min

2.10 ml/min with superficial occlusion

VV - 75 ml

Ejection Vol - 58 ml Ejection Fraction - 77%

RVF - 51%

Summary: Normal outflow and reflux volumes. Calf muscle pump satisfactory. Elevated RVF

Venous Pressure:

AVP: GSV: sustained high pressure without fall while walking

Dorsal toe vein: Normal fall to 30mm HG, rapid rise (<10 sec)

Arm-foot: Resting: <4 mm. Hg. Difference

Post hyperemia: 7 mm. Hg difference

Summary: Sustained venous hypertension and rapid return to baseline (Difference between GSV and toe tracings not explained)

Consistent with deep venous obstructive and reflux disease

Summary of Phase II workup and case analysis:

Findings diagnostic of post-thrombotic disease with elements of reflux and of obstruction shown on duplex. Physiologic confirmation of significant obstruction and reflux lacking in APG. Venous pressure consistent with venous disease, both obstructive and reflux.

Further workup needed to find a way to improve his symptoms since his present way-of-life was unsatisfactory.

Phase III.

Ascending venogram: Ascending flow preferentially by superficial veins, even with tourniquet at the ankle. Ankle and upper calf tourniquets required to force flow into severely distorted tibial, popliteal, and SFV-PFV veins. Popliteal vein distorted, becoming obstructed above popliteal space.

Large GSV, main outflow tract from the calf.

The LSV (SSV) ended in a Giacomini vein which ascended to join the saphenofemoral junction. There was no connection between the LSV and the popliteal.

Normal CFV-Iliac-IVC.

No significant calf or thigh perforator veins seen.

Descending venogram: Upright examination with Valsalva revealed:

Non-visualization of SFV

Reflux in distorted PFV down through PFV-popliteal branch into popliteal vein, and then reflux down into anterior tibial and muscular veins. One large lateral branch of the CFV (common femoral vein) showed a competent valve.

GSV showed slight reflux in thigh only. (Valve leaflets seen in GSV.)

Analysis of workup:

Life-style limiting venous claudication, pain, and swelling in an otherwise healthy and athletic 47 year old male. Healed ulceration and significant skin changes. The problem was due to post-thrombotic disease which had virtually wiped out the deep venous return in the deep veins. Most of the obstruction was in the lower thigh, extending down through the popliteal into the calf veins. Reflux of greater

or less degree was present in all veins except two, and these were the Giacomini vein and the lateral branch of the PFV. The GSV and the perforator veins were not of great importance in the process. The dominant symptoms which limited his lifestyle appeared to be due to poor emptying of the calf. Outflow from the calf was limited by deep vein obstruction in the SFV, poor collaterals, and reflux in the PFV. Outflow channels were the GSV, the Giacomini extension of the SSV, and possibly the competent lateral branch of the PFV (Fig. 1).

Figure 1

Diagram of pre-operative condition determined by duplex scan and venography. A, upper end Giacomini vein with competent valve. B, junction of LSV with Giacomini vein at popliteal level, showing obliteration of branch to popliteal vein. C, incompetent profunda femoris vein with large communication to popliteal vein. D, permanently occluded superficial femoral vein. E, lateral branch of common femoral vein with competent valve.

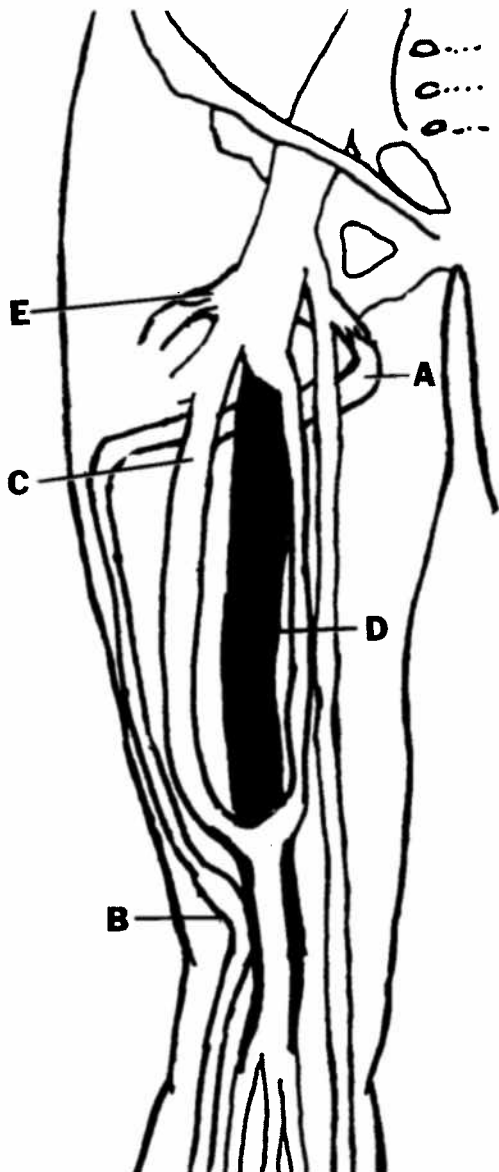
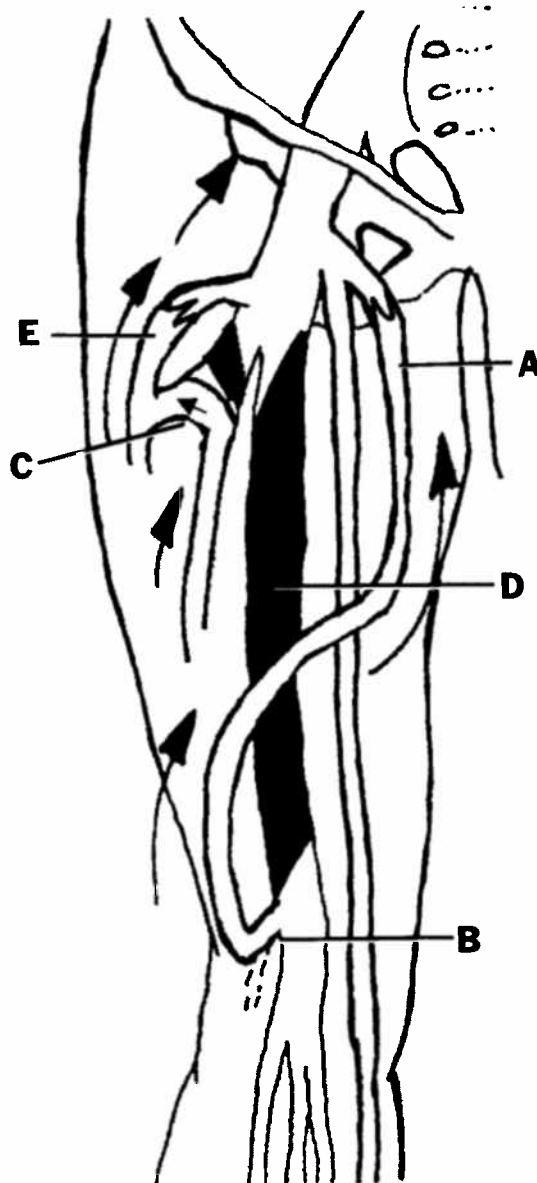


Figure 2

Diagram of surgical procedure. A, upper end of Giacomini vein with competent valve utilized as outflow for the popliteal vein. B, lower end of Giacomini vein disconnected from LSV and anastomosed to popliteal vein to serve as popliteal outflow. C, incompetent PFV disconnected from CFV and transposed to the competent lateral branch of the CFV. D, permanently occluded SFV. E, Competent valve in lateral branch of CFV.



DISCUSSION

DR. O'DONNELL: That was an excellent presentation, and represents one of the largest experiences with this problem.

DR. GLOVICZKI: Well, this is an extremely complex case, no question about it, and I may follow a little bit what Mike was suggesting, try to stay on the conservative side before I would decide on any type of deep vein reconstruction. I'm even thinking of some kind of a May-Husni operation to use the saphenous vein to bypass the obstruction.

DR. O'DONNELL: Do you think they're missing something in the iliac segment?

DR. GLOVICZKI: I certainly would look very, very carefully into that venogram. I don't have large experience repairing deep vein valve incompetence of the profunda femoris vein, that's another option that could be done in this case.

DR. DEPALMA: Tom Wakefield pointed something out that Mike Dalsing emphasized too, that this patient may be suffering from repeated episodes of thrombophlebitis. I would seriously consider long-term anticoagulation and Heparin or low molecular weight Heparin therapy with elevation to see if these would improve his symptoms. If not a surgical intervention can be considered. Dr. Husni was a friend of mine in Cleveland. I did six of these operations. None of them worked so I quit. So I don't see why the May-Husni procedure would help this man, and if the saphenous thrombosed he'd lose his outflow tract.

DR. PERRIN: Conservative treatment and anticoagulation. I have been very disappointed by saphenotibial bypass. I have performed I would say 20 years ago 25 or something like that, and the results were not good. May in Austria and Chris in Germany have the same poor results.

DR. GARCIA-RINALDI: I'd like to propose to Dr. Dalsing that maybe the approach of taking the entire circumference of the vein has been your problem. We have approached this problem by taking a monocusp patch made of the pulmonary artery of human beings, cryopreserved, and we have implanted this in over 50 cases. They've been followed for up to five years. We have not had a single case of thrombosis even though these patients go home on aspirin, and I would propose that as an alternative to the entire circumference replacement. There seems to be a mechanism not known to me that protects this particular patch from thrombosis if you leave the posterior aspect of the host of vein.

DR. RAJU: I think I am not fully understanding what you are saying.

DR. GARCIA-RINALDI: But I'm not proposing a model. I'm telling you I've done it.

DR. RAJU: I am familiar with your earlier work. I urge you to publish your latest results.

DR. GARCIA-RINALDI: If you take a monocusp and you place it properly, you will get competence. The problem we've seen or the problem we've had in some of these patients is the actual sizing of the prosthesis because we have had to use whatever LifeNet will send us. However, the bioprosthesis comes as a patch, not as a pulmonary artery trunk. So we are implanting a patch, and this we tested to be totally competent at the time of surgery by releasing the proximal clamp and placing it in the common femoral vein.

DR. DALRING: I really have no specific information how synthetic grafts fail. I feel that the problem with cryopreserved tissue is probably some low-grade rejection response. Certainly if

you do not aggressively anticoagulate these patients or if the anticoagulation levels drop or if the patient fails to take his medication, the allograft will fail. I know, at least in animal models, that if you place any kind of synthetic in the venous systems that they'll fail. So I'd be interested to see your data because I would expect to see different results.

DR. TRIPATHI: My first question is for Dr. Dalsing. There is a high incidence of incompetence of your cryopreserved vein valve at six months. I also notice that there is no external support for your vein. Have you tried external support and are the results better with external support?

DR. DALRING: We have not tried external support. I can tell you a little bit about what happens to these veins when they fail. It doesn't seem to be a dilation problem. They seem to fibrose. So I think putting an external support around them would probably make little difference.

DR. TRIPATHI: My second question is for Dr. Raju. It has been seen that nearly 30 to 50 percent of all axillary veins are incompetent. Do you always use axillary veins for femoral vein valve transplant or do you look for superficial femoral vein and popliteal veins of the contralateral limb whenever there is a duplication?

DR. RAJU: No, I don't think you should go to the contralateral limb. You are dealing with a disease which is bilateral in a large percentage of cases. Incompetent axillary vein can be repaired before you insert it in large number of cases.

DR. O'DONNELL: Using the external valvuloplasty technique that Bob has developed, we usually take it from the nondominant arm.

DR. TRIPATHI: My last statement is a comment about the iliac angioplasty. Learning from our techniques of iliac angioplasty, especially at the aortoiliac junction, we found that unless you use a kissing balloon technique you are going to decrease the lumen of the opposite side.

DR. RAJU: Not true in the veins.

DR. TRIPATHI: That's what I want to know, whether you are using a kissing balloon technique, and whether you are evaluating the opposite side common iliac vein after doing the ipsilateral common iliac angioplasty, especially when you are advocating putting the stent way up into the IVC?

DR. RAJU: We have not looked at the opposite side in every case. There has been no problem with the opposite side. I don't think the kissing balloon is necessary in the venous system because of lack of rigidity. It's not the same thing as in the artery.

DR. HASANIYA: This question is for Dr. Dalsing. Do you think that the failure rate could be related to other immune process and do you think in your experience that immune suppression might help decrease the failure rate?

DR. DALRING: I can only tell you a little bit about the arterial system where there seems to be cytotoxic T cells that causes much of the problem. So there has been some suggestion that immunosuppressive drugs directed to this T cell population may improve results. I have not had any personal experience with this, but I do believe it is being carefully considered by those involved with cryopreserved tissues.

DR. THORPE: This case represents, I think, a good example of how video phlebography, if you will, could help us understand this patient's problem because by looking at the still images, I can't tell exactly where the stasis is. Is it mostly in the calf, or mostly in the

thigh, or is there any clearance problem through the iliac, as well? Hopefully, in the future, we'll advance towards phlebography with digital imaging. I'd like to ask Dr. Neglen if you've used duplex velocities in the femoral before and after therapy, particularly after stent placement, to assess the flow? This might help you determine whether or not you've got enough stent opening to remain patent.

DR. NEGLEN: Yes. All the patients have duplex ultrasound before and after surgery. Many of them do have decreased augmentation and a lack of respiratory changes before which is abolished by the stenting. However, I think ultrasound is too insensitive a test to detect slight borderline stenosis. For example, the cases of hyperplasia were not detected by ultrasound. They were detected by venogram, and they didn't have any symptoms whatsoever.

DR. GLOVICZKI: Peter, this is a wonderful series that you presented, but the indication seems to be different from our conventional indications of reconstruction for obstruction. I mean, you only had 18 percent of the patients who had greater than two millimeters of mercury pressure difference at best. That to me looks like maybe the circulation in most of these patients is sufficient and you did not really have functional obstruction in most of your patients. So what did you base your indications on?

DR. NEGLEN: Well, as I told you, we're trying to find the actual venous pressures which detect significant stenosis. I think the biggest problem is that in a supine position on an operating or radiology table, it is difficult to increase the venous flow enough to detect significant obstruction. It's a low flow/low pressure area as we talked about yesterday. The question is when you have collaterals, does that mean you have compensated the outflow obstruction, or does it mean that collaterals actually indicate that you have an ongoing outflow obstruction? So these are the kinds of questions we are trying to resolve. Unfortunately, there is no correlation between the intraoperative findings, the preoperative findings pressure-wise, and the postoperative clinical results. Unfortunately, with present diagnostic methods there are patients who have normal pressures in the presence of stenosis, who experience very good results post stenting.

DR. GLOVICZKI: You had ankle-arm pressure measurements? Or did you have any type of outflow obstruction on plethysmography?

DR. NEGLEN: Oh, yes. These patients were fully investigated with ankle-arm pressures and hyperemia pressures and so on. As I showed, the pickup rate for the proximal stenosis by reverse pressure testing, even if they are severe, is not very high. Even though we have been proposing the arm/foot hyperemia test to be the best available test presently, we don't think it is good in all situations,

and we're still looking for a better test.

DR. DALRING: I just have one question for Peter too. How do you inject the papaverine?

DR. NEGLEN: Intra-arterially at the level of the femoral artery.

DR. OSMAN: I am one of those unfortunate souls who occasionally have to see 50 patients on a Tuesday afternoon. I have a question to Professor Raju. I may have misheard him, but I understood in this last case that he would have stripped the long saphenous vein which I thought in this particular patient may have made things a lot worse. Did I mishear you or is that the case?

DR. RAJU: No, you didn't misunderstand me. We presented data yesterday to show that you can do it in similar type of situations without any clinical mal sequelae and oftentimes with clinical improvement if there is significant reflux. The paper was in Surgery last year.

SURGICAL MANAGEMENT

DR. KISTNER: We didn't want to operate on the patient, but the patient would not leave without having something done. He said, "My quality of life is so impaired, do whatever you can do. I'm not leaving here until you do something." This led to surgery, even though there were few encouraging findings for a surgical approach. We did have extensive venography of the pelvis and there was nothing abnormal in the iliac vein, so proximal obstruction was not part of the syndrome. We analyzed the case as being severely obstructed in the thigh, compounded by post-thrombotic reflux. Please recall that the patient had a Giacomini vein visualized on the ascending venogram. We disconnected the popliteal termination of the lesser saphenous and moved it down to a more advantageous soft spot on the popliteal vein; this converted the Giacomini vein, which had a valve in it, to an outflow tract for the calf to help the obstruction. We left the greater saphenous vein intact because it was a good outflow tract for the leg. The really non-standard thing we did was to interrupt the profunda femoris vein which was refluxing, and perform an end-to-side transposition between the PFV and the lateral femoral branch because this lateral femoral vein provided a valved outflow for the profunda femoris vein. These procedures were thought to be low risk. The patient post-op felt improved right away. He's now three years post-op. On duplex scan done elsewhere, both of these reconstructions are patent. I spoke to him on the phone this weekend because he lives elsewhere. The patient has returned to hunting. He's back to full-time work. He discarded his pump. He continues to use stockings. At least for three years to this point, he has obtained the result he was seeking.