DISCUSSION

Dr Albeir Mousa (Charleston, WV). I have two questions. The first, what size perforator you have treated, and how far from the ulcer location that can still contribute ulcer pathology?

Dr Ellen Dillavou. We follow the Society for Vascular Surgery (SVS) guidelines for pathologic perforators; so, a perforator of ≥3.5 mm is considered pathologic and so we will inject anything of that size or larger.

Dr Mousa. So about two-thirds of your cohort was resistant to treatment. And I notice you keep following them. Did you evaluate the central venous system, iliac vein, like any venous outflow studies to delineate this issue?

Dr Dillavou. Yes, we perform complete duplexes of all of our patients initially and then as clinically indicated. So, if we performed our intervention and the ulcer heals, we continue that effective treatment for recalcitrant venous ulcers. J Vasc Surg 2011;54:737-42.


Dr Magnusson. The thing of that size or larger.


Dr Harlander-Locke. That was a great talk. I think this is real-world data, and this is what you expect to see in the population you are presenting. However, it is a bit misleading to indicate that the treatment of the perforators by the foam is what is causing the ulcers to heal. I believe that the foam does the job by closing all the refluxing tributaries and particularly the microvascular tress, which is actually responsible for the development of the ulcer. And in your population in particular it is evident, because only one-third of your patients had deep vein outflow obstruction, such as with chronic DVT or dampened femoral waveforms on ultrasound, we would then perform a central venous study.

Dr Nicos Labropoulos (Stony Brook, NY). That was a great talk. I think this is real-world data, and this is what you expect to see in the population you are presenting. However, it is a bit misleading to indicate that the treatment of the perforators by the foam is what is causing the ulcers to heal. I believe that the foam does the job by closing all the refluxing tributaries and particularly the microvascular tree, which is actually responsible for the development of the ulcer. And in your population in particular it is evident, because only one-third of your patients had deep vein outflow obstruction.
disease. So it is clear that the foam was successful to that extent because you were able to treat a significant number of veins within and around the ulcer.

Dr Dillavou. I completely agree, and one advantage of foam is that it does treat the network of varicosities. I think this is one tool. Dr Lawrence has elegantly demonstrated that ablation of perforators also increases ulcer healing, so the foam perforator ablation has value. This is just one part of the ulcer treatment package.

Dr Kathleen Gibson (Bellevue, Wash). The question I had, and I might have missed in your talk, what was your compression regimen after treatment? Did you track patient compliance? And if so, did that have any effect on either ulcer healing or the success of thrombosis of the perforator, or do you think that that is an important piece in the healing and in the success of your procedure?

Dr Dillavou. Our standard regimen is to put patients in a compressive stretch wrap for 24 hours after foam sclerotherapy. Or if they are in an Unna boot, we put the Unna boot on immediately after the treatment and leave that on for 3 days, or a week, however long the patient leaves the Unna boot on.

I do think it is important, and in general our patient population was very compliant with compression. Unfortunately, the largely retrospective nature of this study didn’t allow us to control for each type of compression, and there was a lot of bouncing back and forth with patients between different types of Unna boots and short stretch bandages, etc. But they were all in high-grade compression. We are very aggressive with that and, by and large, the patients are compliant.

Dr Gregory Moneta (Portland, Ore). I have a question about the status of the patients prior to when you began treating them. You said that they were all under maximum medical management. Were these patients de novo patients when they presented to you? If not, and if they were under your continuous management, why do you wait 2 years to treat them?

Dr Dillavou. We get a large number of patients referred from other institutions. And the reason it was only 2 years of compressive therapy prior to intervention for many patients is because although they would report a history of many years of ulcer, they were not under our care at that time. And so we started tracking the study from when we knew they were getting adequate compression and medical therapy. Much of the medical and compressive therapy was done in our clinics.

This study also reflects a change in our practice patterns: becoming more aggressive with elimination of refluxing perforators and correction of outflow obstruction, and that has happened over the last few years. Prior to the mid-2000s we were less aggressive in our practice and had a large number of patients who were treated with compression only.

Dr Alan Dietzek (Danbury, Conn). I was curious, how did you select your sclerosant regimen for these patients? Do you use a higher concentration if you don’t get a thrombosis of the perforator the first time? Did you see a difference when you switched to polidocanol from Sotradecol (AngioDynamics, Latham, NY)? And have you considered the combined use of foam sclerotherapy and heat ablation for those perforators that don’t thrombose with sclerotherapy only?

Dr Dillavou. We switched from Sotradecol to polidocanol as a group. We made a total change in our practice in May 2010 because we felt that polidocanol was safer and better tolerated than Sotradecol. We did not see a difference in thrombosis rates or complications between Sotradecol and polidocanol. Initially we were using 3% polidocanol for all ultrasound-guided perforator injections because we felt that that would be more effective. But then after the Varisolve trial (BTG International Inc, West Conshohocken, Pa) results showed that there was not a significant difference between 1% and 3%, we then downgraded to 1% and we have not seen a difference in our thrombosis rates or complications. And so now 1% polidocanol is the standard that we use.

Based on these results, we have become more aggressive with heat ablation of perforators. So anyone with a 5-mm perforator, the obese men on Coumadin (Bristol-Myers Squibb, Princeton, NJ), and those who fail ultrasound-guided injection, all get a heat ablation, and those we are doing as a combined heat ablation and chemical ablation at the same time hopefully through the same access site.