ASK THE EXPERTS

Which Factors Have the Most Influence on Your Decision-Making Regarding the Treatment of Perforators?

Leading venous practitioners share their treatment algorithms for when it is appropriate to intervene on perforator veins and when it is not.

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Dilatation of perforating veins is commonly caused by the hemodynamic changes that occur with superficial venous insufficiency. Typically, enlarged perforators are noted in the calf and serve as a reentry point for reflux in the saphenous system. When the saphenous reflux is treated, the dilated perforators remain, occasionally resulting in the reversal of blood flow from the deep system back into the superficial. This is particularly true in concomitant deep venous insufficiency. This theory helps to explain why perforators are a common cause of recurrent varicose veins after treatment. However, perforators can also be the cause of primary superficial venous insufficiency. Although this is a less common presentation, these patients can be very symptomatic and typically have an extensive network of varicosities that arise from the incompetent perforator.

Because dilated perforating veins are commonly associated with superficial venous insufficiency, their mere presence alone is not an indication for treatment. Residual dilated perforators posttreatment in the calf that are not causing any symptoms can and should be left alone. Perforator treatment should only be offered to patients whose symptoms can be clearly attributed to the incompetent perforator. Making this determination can be very easy or very difficult. When perforators are the primary and only source of superficial venous insufficiency, there is little question that the symptoms are associated with the incompetent perforator. In the setting of multiple superficial venous reflux sources and deep venous insufficiency, it is much more difficult to determine if a specific incompetent perforator is the cause of the patient's primary complaint.

Currently, there are several minimally invasive perforator treatment options. These include ultrasound-guided foam sclerotherapy (UGFS) as well as endovenous ablation and adhesion. UGFS has been shown to be very effective in the treatment of perforators. Treatment results have demonstrated early closure rates as high as 98% and a 20-month follow-up closure rate of 75%.[1] Endovenous ablation with specialized radiofrequency devices and laser fibers has also

demonstrated acceptable short- and long-term results, with closure rates ranging between 61% and 95%.[2-5] When UGFS has failed, endovenous ablation was reported to be very effective, with closure rates of 85% to 89%.[5] Recently, cyanoacrylate adhesion in conjunction with a sclerosant was used to occlude perforators with a reported success rate of 100%. Asymptomatic cyanoacrylate extension into the deep veins was noted in 4.8% of patients, with a prolonged thrombophlebitis rate of 38.5%.[6]

Given the available treatment modalities, my preferred treatment method is UGFS. This is particularly true for residual, symptomatic, incompetent calf perforators. This technique is effective at closing the perforator and simultaneously eliminating many of the deep subcutaneous tributary branches and smaller perforators that likely play a role in the causation of symptoms. I reserve endovenous techniques for large-diameter perforators that have not resolved after UGFS. This is a rather uncommon scenario, as most patients' symptoms will improve and ulcers will heal with UGFS.

However, there are some notable exceptions to this rule of thumb. Primary perforator incompetence, although unusual, does present in typical patterns. One pattern is the presence of a large lateral thigh perforator giving rise to an extensive chain of varicosities that course along the lateral aspect of the thigh and calf into the foot. In my experience, the use of UGFS to address this problem often results in an extensively long recovery with thrombophlebitis and occasional persistent skin discoloration. Additionally, I have seen recurrence of varicosities secondary to the persistence of reflux in the perforator itself. Based on this experience, I currently use endovenous techniques to ablate the perforator and eliminate the chain of varicosities with ambulatory phlebectomies. This approach has achieved rapid resolution of symptoms with a durable and good cosmetic result.

Another primary perforator pattern is often noted in the posterior knee region. In these cases, a perforator arises from the popliteal vein, giving rise to varicosities that are typically noted in the knee crease and extend down the calf into the foot. Sometimes, this perforator has been confused with a small saphenous vein, but it has a separate insertion into the popliteal vein. In my experience, this perforator has been very difficult to close. UGFS is largely ineffective for this type of perforator. Endovenous ablation is more effective, but it too has a failure rate that appears to be higher than expected. For these cases, I use endovenous laser with slightly higher energies and simultaneous ambulatory phlebectomies. It is very important to distinguish this popliteal perforator from a sciatic nerve venous anomaly. These occur along the lateral aspect of the calf and do not directly connect to the popliteal vein but run parallel to the sciatic nerve. The use of an endothermal ablation device in this vessel will likely result in a nerve injury and should be strictly avoided.

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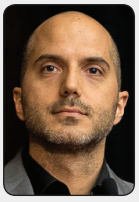

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Perforator assessment and treatment modalities remain some of the most challenging and interesting topics in modern phlebology. The idea of a perforating system draining unidirectionally from the surface toward the deepest compartments of the leg has been incorrectly described for decades. Back in 1952, Hojensgard and Sturup had already reported pressure values of the great saphenous vein and the posterior tibial vein, showing that the role of perforators was as vessels balancing the pressure gradients in between the superficial and deep systems.[1]

Perforator diameter has been recognized as an important factor to take into consideration in the indication for treatment: a caliber ≥ 3.5 mm and localization beneath a healed or open venous ulcer should lead to treatment, according to international guidelines. Nevertheless, it must be recognized that the indication only has a class IIb level of evidence.[2,3] Indeed, perforating veins > 3.9 mm have a high probability of being incompetent, but it has also been demonstrated that one-third of incompetent perforators have a smaller diameter.[4]

The same definition of perforator incompetence can generate controversies. According to the traditional definition, an incompetent perforating vein is one with a diastolic flow lasting > 0.35 or ≥ 0.5 seconds, based on different guidelines.[2,5] Nevertheless, the current sonographic analysis is limited by the fact that the single-sample volume assessment is dependent on vessel tortuosity.

Last year, our group demonstrated that by using an innovative software that provides a simultaneous assessment of 256 sample volumes, independent of vessel tortuosity, a discrepancy was detected between the net flow direction and the current incompetence definition. Indeed, a perforator showing an outward diastolic flow lasting > 500 milliseconds presented a net systolic-diastolic outward flow in only 13.9% of cases (95% confidence interval [CI], 9%–20.1%). On the contrary, a perforator with an outward diastolic flow lasting < 500 milliseconds presented a net inward flow in 96.4% of cases (95% CI, 93.2%–98.3%).[6]

Based on the previously mentioned data, in my practice, I carefully report perforator calibers and diastolic outward flow lasting > 0.35 seconds, as per the guideline recommendation; but even more, I focus on the net flow direction and the eventually clinically correlated signs and symptoms. In terms of treatment, I give an indication for treatment to thigh perforators clearly exhibiting a net outward flow and feeding a truncal incompetence with overload of the superficial system. Below the knee, I believe that treatment is only indicated if the perforator with a net outward flow represents the leaking point feeding a venous hypertension scenario, with related signs and symptoms. In my opinion, an indication for treatment solely based on the caliber and/or the outward flow can lead to the risk of overtreatment. Perforating system hemodynamics should not be reduced to the outward diastolic flow of just one of the more than 100 vessels belonging to a system meant to equalize the pressure between the superficial and deep networks.

In conclusion, the main factors I take into consideration whenever giving an indication for perforating vein treatment are a net outward flow and evidence of related signs and symptoms of venous hypertension. Future investigations reporting on a deeper analysis of perforator hemodynamics and related clinical impact remain some of the most interesting and fascinating topics in modern phlebology.

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When I treat perforator disease, I adhere to the Society for Vascular Surgery (SVS) and American Venous Forum (AVF) guidelines. They state that a perforating vein needs to be ≥ 3.5 mm, reflux for 500 milliseconds or longer, and be in the vicinity of an active ulcer (CEAP [clinical, etiology, anatomy, and pathophysiology] C6) or healed ulcer (CEAP C5).[1] The most compelling scenarios for treatment are those with an open ulcer with a large, refluxing perforator that feeds a network of varicosities under the ulcer. In these situations, I like to ablate the perforator with the VenaCure 1,470-nm, 400-µm laser (AngioDynamics), as it is technically easy to advance into the perforator and it has good closure results. After this, usually in the same sitting, I foam the tributaries with 1% polidocanol physician-compounded foam to completely treat the varicosities because I think it makes the perforator more likely to close and increases resistance to flow. The situations where I will wait to foam, or will only foam part of the tributaries, are when the patient has very thin, damaged skin under a large network. In those patients, I have seen skin necrosis over a large body of thrombosed veins. If this is a consideration, then treatment of varicosities should have a more gradual timeline. For ulcer patients, I ablate any refluxing superficial axial veins and correct any central venous occlusive disease prior to perforator treatment.

For patients with CEAP C5 disease, I will treat perforators if there are new symptoms, including pain, itching,

swelling, or new varicosities in the area of the healed ulcer. For these, I will usually foam initially, as this is technically facile, well tolerated by patients, and usually effective. Many in this population have reoperative disease, and the perforating veins may be partially occluded or very tortuous.

I occasionally treat patients who have CEAP C3 or C4 disease. In this population, I treat perforators that are large (usually > 4 mm) with reflux and are in the vicinity of symptoms. Common symptoms that provoke treatment include focal pain where the perforator supplies superficial varicosities, focal skin disturbances such as rash or discoloration where the perforator exits, or large symptomatic varicosities supplied by a perforator.

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The treatment indications for refluxing perforators have been a challenging debate for many decades. Generally, I separate refluxing perforators in primary disease from those present in recurrent venous disease, the latter requiring treatment in most cases. In primary disease, it is important to distinguish whether the perforator is connected to a refluxing saphenous vein or if it is the source of refluxing tributary varicosities by itself.

It is important to note that in primary disease, a dilated and refluxing perforator vein connecting a refluxing saphenous vein to the deep vein system does not necessarily need treatment. Frequently, those perforators are medial calf perforators or gastrocnemius perforators, and their volume overload and dilation are part of the private circulation caused by the adjunct refluxing saphenous veins. Therefore, particularly in early clinical stages (CEAP C2 or C3), a wait-and-see strategy for 3 to 6 months after treatment of the saphenous vein is justified to find out if the missing blood reflux finally causes recompensation of the originally dilated perforator vein.

However, even in primary disease, if the skin above the perforator shows dermatitis, pigmentation, or dermatosclerosis, representing CEAP C4 disease or higher, it becomes a different story. Then, the perforator reflux has to be eliminated during the same session when the saphenous vein reflux gets abolished. It is clear that treatment of refluxing perforators should happen. Likewise, treatment of refluxing perforators should be performed if they are not connected to refluxing saphenous veins, including popliteal, lateral thigh, or lateral calf perforators, for example. This can be true for primary or recurrent venous disease.

For treatment of refluxing perforators, I personally prefer to use endovenous laser. A device that has water-absorbed 1,320-nm Nd:YAG in combination with a < 600-µm bare fiber allows direct puncture of any perforator with an 18-gauge needle and immediate placement of the fiber at the fascia level.

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The bottom line for me when deciding if I should treat a perforator vein is whether or not I believe it to be a major contributing source of my patient's symptoms. In my clinical experience (and supported by the literature), refluxing perforator veins, even those that are sizable, often no longer reflux after ablation of incompetent truncal veins. In this situation, they are not pathologic perforator veins but are "reentry" conduits from an incompetent superficial system into the deep system. Such perforator veins are common and rarely require treatment.

The SVS/AVF guidelines are clear in their definition of a "pathologic perforator" as a perforator vein that is ≥ 3.5 mm, demonstrates reflux (outward flow of ≥ 500 milliseconds), and is located beneath/adjacent to

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a healed or open venous ulceration.[1] In patients such as these with advanced venous disease (CEAP C5, C6, and sometimes C4b), in accordance with the guidelines, I will typically treat the pathologic perforator veins at the same time as when I perform a truncal ablation. In cases of advanced disease in patients who have previously undergone either truncal vein stripping or ablation, perforator treatment may be the primary treatment modality, usually accompanied by ultrasound-guided sclerotherapy in the ulcer bed varicosities or varicosities in the area of damaged skin.

There are exceptions for me to the general rule of only treating perforator veins in advanced clinical classes. Some patients with CEAP C2 or C3 disease will present with a large perforator vein that is the primary source of their varicose veins, and in this circumstance, I believe that treatment is justified. Classic locations for this type of perforator vein include the popliteal fossa, posterior thigh, and mid-thigh. Mid-thigh perforators are usually connected to a refluxing great saphenous vein, and often, the great saphenous vein more cranial to them is competent. In essence, they are like a saphenofemoral junction.

The method of perforator vein treatment is another decision I make after determining that the vein warrants treatment. If the patient is also undergoing a saphenous ablation, I will usually use the same ablation modality for the perforator vein that I used for the saphenous vein. Both radiofrequency and endovenous laser ablation are FDA approved for perforator treatment, and I have used both modalities. Overall, I find the endovenous laser technique (recently FDA approved after the SECURE trial) to be simpler than the radiofrequency technique, as it can be performed with a much smaller-profile device—either with a 4-F microcatheter sheath or through a 21-gauge needle. I have also used cyanoacrylate adhesive off-label to close perforator veins at the same time as cyanoacrylate ablation of saphenous trunks. The procedure is straightforward, and I have been encouraged by my initial experience with this newer technology. Ultrasound-guided sclerotherapy (with liquid or foam) is one of the simplest techniques for perforator treatment, but in my hands, the closure rates have been suboptimal with larger veins.

Overall, closure rates and clinical success rates for perforator vein treatment are lower than what is typical for truncal ablation. Managing patient expectations, selecting veins that truly warrant treatment, and using a technique that the practitioner is familiar with are all important aspects of successful perforator treatment. ■

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