Perforator Vein Incompetence in CVD Patients

Understanding the characteristics and impact of perforator vein incompetence on patients with chronic venous disease.

BY ANTONIOS P. GASPARIS, MD, RVT, FACS, AND NICOS LABROPOULOS, PHD, DIC, RVT

Several investigators have shown the flow patterns and morphologic characteristics of perforator veins in healthy subjects and in patients with chronic venous disease (CVD). The complex nature of CVD, having many different factors associated with disease severity and deterioration, has made it difficult to elucidate the role of perforator vein incompetence. In this article, many of the important elements associated with perforator veins in CVD are analyzed.

ANATOMY AND NORMAL FUNCTION

There are approximately 150 perforator veins in each lower extremity. Using microinjection techniques, van Limborgh found approximately 60 perforator veins in the thigh, eight in the popliteal fossa, 55 in the leg, and 28 in the foot.[1,2] Of these veins, about 30 become incompetent and are identified in clinical practice (Figure 1).[3] Perforator veins connect superficial veins with deep veins by piercing the deep fascia, and that is why they are termed *perforator veins* (Figure 2).

Perforator veins should not be confused with communicating veins because the latter connect veins of the same system (ie, deep veins to deep veins). Most perforator veins have at least one subfascial bicuspid unidirectional valve that allows blood to flow from superficial veins to the deep veins (inward flow).[4] In the foot, the flow in perforator veins is bidirectional, whereas in the calf, the flow is mostly inward (flow direction from superficial vein to the perforator), although bidirectional flow has been reported.[4] Veins in the thigh area are much longer because they must cross a longer distance compared to those in the calf, ankle, and foot. The calf veins are often duplicated, with one limb connected to a muscular vein and the other into a deep axial vein.

In normal limbs, the outward flow (flow direction from the perforator vein to the superficial vein) is < 350 ms, and in the vast majority of them, it is even lower (< 200 ms)

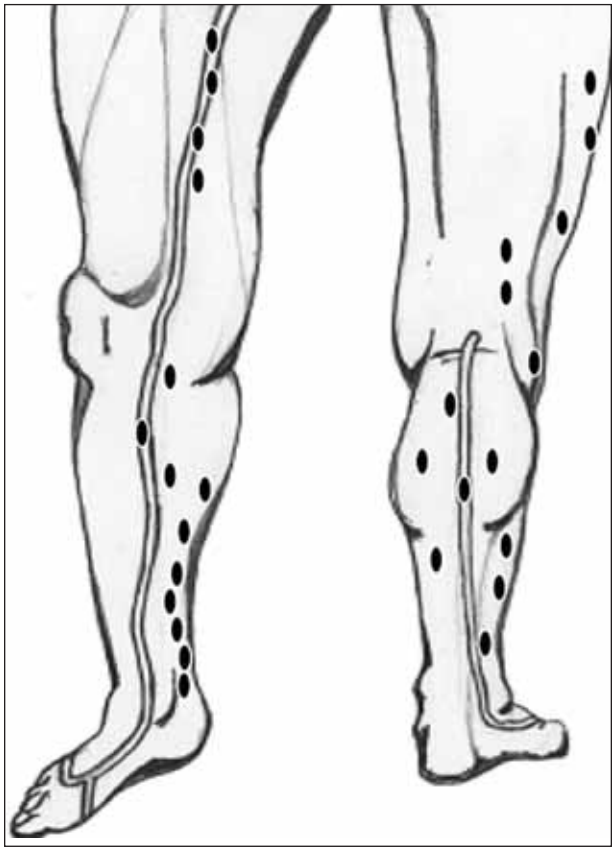

Figure 1. The lower extremity with bony, muscular, and skin landmarks showing the most common locations where perforator veins become incompetent. The prevalence of reflux is more common in the medial aspect of the calf.

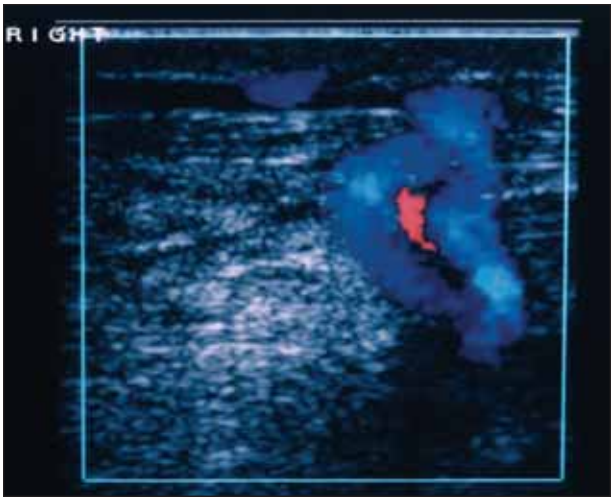

Figure 2. A duplicated perforator vein is shown in the medial aspect of the calf. The perforator pierces the deep fascia and connects the superficial veins with deep veins. An artery having opposite flow direction is seen between the two veins. Typically, perforator veins are accompanied by an artery that is smaller (ie, the vein and a nerve that is smaller than the artery).

(Figure 3).[5] Outward flow of > 350 ms has been defined as reflux.[5] Because this outward flow of 350 ms duration is short, as well as the fact that other veins have a cutoff value of > 0.5 s (with the exception of common femoral, femoral, and popliteal veins, which have a cutoff of > 1 s), most centers use a duration > 0.5 s as a cutoff for perforator reflux. Most normal perforator veins have a subfascial diameter of < 3 mm. The best cutoff diameter for reflux was identified to be 3.9 mm.[4] Another study set this cutoff at 3.5 mm.[6] Although the larger diameter indicates the presence of reflux with high certainty, one-third of refluxing perforator veins have a smaller diameter than these cutoff points.[4] This is not unique and has been often observed in the saphenous veins as well.[7]

CHARACTERISTICS AND FUNCTION OF PERFORATOR VEINS IN CVD PATIENTS

Incompetent perforator veins are often found in patients with CVD. The number and diameter of incompetent perforator veins increases with disease severity.[4] It has also been shown that the diameter of a normal perforator vein increases with CVD deterioration.[4] The diameter of a perforator vein is greater below the fascia because the lumen cannot expand as much at the collagenous deep fascia (Figure 4).

In another study, in addition to increased diameters, perforator vein incompetence was characterized by significantly higher mean and peak flow velocities, volume flow, venous volume outward displacement, and a lower flow pulsatility.[8] Reflux in perforator veins occurs only in the presence of superficial vein reflux that is in continuity with the affected perforator veins.[4,8,9] In patients with deep vein incompetence, refluxing perforator veins sustain further hemodynamic impairment.[4,8,10] Outward flow in perforator veins is greater when there is triple-system incompetence compared to reflux confined to superficial veins and perforator veins.[4,8-10] Reflux in all three systems—superficial, perforator, and deep—is more common in CVD patients with skin damage.[8,10,11]

Reflux in perforator veins develops through the superficial veins, first in an ascending manner, because venous wall disease extends from superficial veins to perforator veins. Typical examples of this are perforator veins in the medial thigh connecting to the great saphenous vein (GSV) and posterolateral thigh perforator veins connecting to the local superficial vein plexus. Second, this development occurs in a

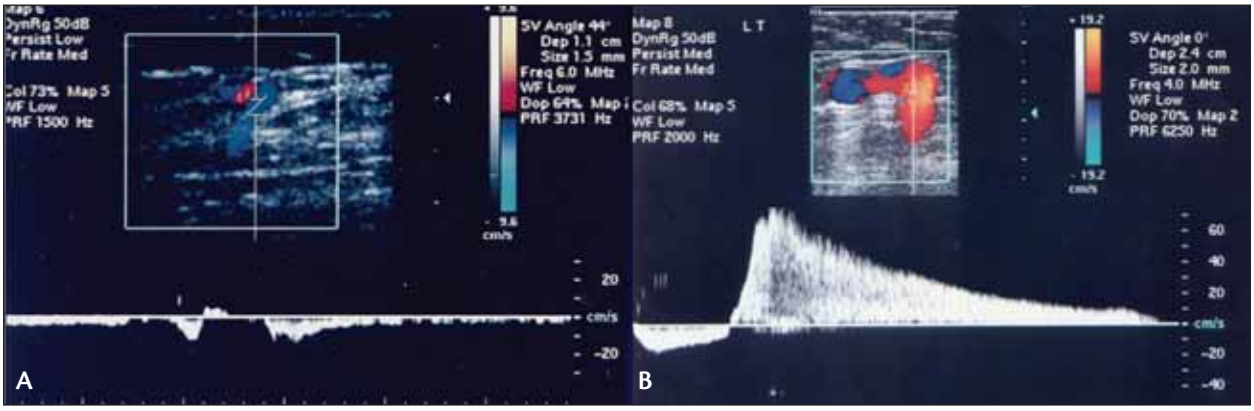

Figure 3. Evaluation of reflux in a perforator vein using duplex ultrasound. The patient is assessed in the standing position, and augmentation is performed distal to the perforator vein. A normal perforator vein at the medial aspect of the calf (A). During compression, inward flow is enhanced while at the release of compression, a short duration of outward flow is seen followed by prolonged inward flow. A large incompetent perforator vein at the medial aspect of the thigh (B). At the release of the compression, high-velocity, prolonged outward flow is detected. Reflux is also seen in the superficial vein connecting to the perforator.

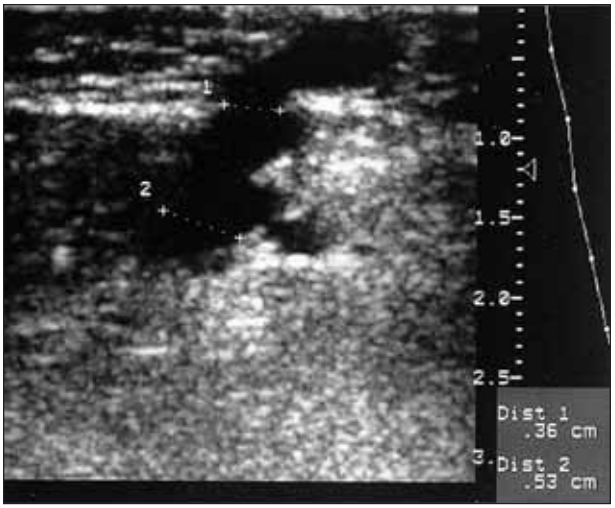

Figure 4. An incompetent perforator vein at the medial aspect of the calf. The perforator vein measures 3.6 mm at the level of deep fascia and 5.3 mm below it. The deep fascia does not allow the same degree of dilation to occur in the perforator veins. Similar anatomic configuration is also seen in normal perforator veins.

descending manner where refluxing veins empty into reentry perforator veins. Over time, reentry perforator veins dilate and become incompetent. An example of this type of perforator vein reflux development is calf perforator veins that empty blood from the refluxing saphenous tributaries into deep veins.

Reflux in perforator veins can develop in both manners but also in new locations where new reflux develops in superficial veins. Over time, the refluxing perforator veins affect the deep veins, which may become incompetent as well.[4,9,11,12] The available information on perforator reflux development has been misinterpreted by multiple cross-sectional studies that have associated perforator veins as the cause for the superficial vein reflux.

In a prospective study, we identified the three patterns previously described by which once-competent perforator veins become incompetent (Figure 5). The ascending development of reflux into perforator veins from previously competent segments of superficial veins was more prevalent (47.4%) but comparable to the reentry type. A smaller number of incompetent perforator veins were detected in new locations that previously did not have reflux in any system.[9] Because there was no deep vein reflux in this location, and in the absence of other serial examinations, the reflux development in the new sites could have been either ascending or descending. Nevertheless, in all the types, perforator vein reflux was always associated with reflux in the superficial veins connected to them.

From the data in our study and the observations in other reports of patients with primary CVD, it would be safe to assume that reflux in perforator veins occurs only in the presence of incompetent superficial veins.[9,13] As the local hemodynamic conditions change over time and as intravenous pressure increases, the perforator vein diameter increases, therefore rendering the perforator vein valve

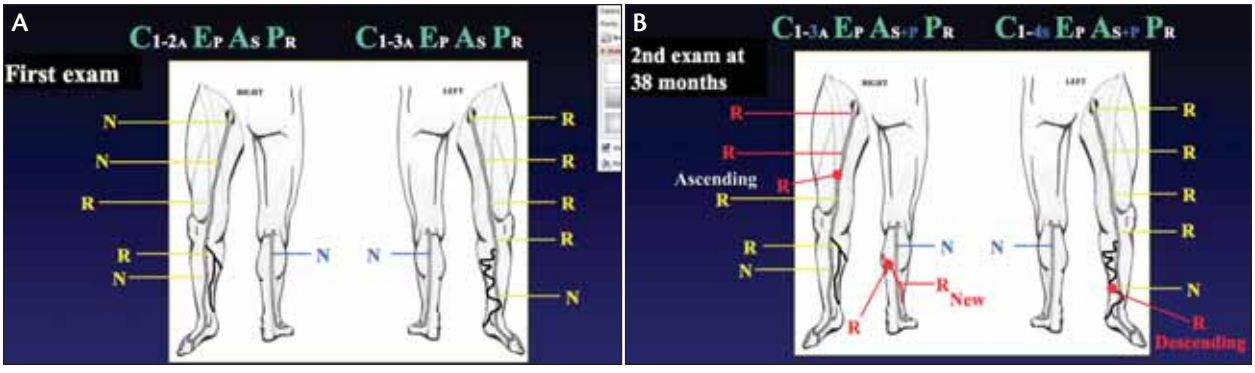

Figure 5. The different patterns of perforator reflux development. These data are from a 52-year-old woman who had three pregnancies and a positive CVD family history from both parents (A). During the initial examination of the right limb, she had reflux in the GSV at the knee and posterior calf tributary. On the left limb, she had reflux in the GSV from the saphenofemoral junction to the knee and in the posterior arch. There was no reflux found in any perforator at this time. During the second examination at 38 months on the right limb, she developed ascending reflux in the GSV, and a perforator vein at the thigh, and a new reflux site in a posterior calf tributary and a mid-calf perforator (B). On the left limb, she developed a reentry type of reflux in a medial perforator vein from the posterior arch. The diameter of the GSV enlarged by 2 mm, and the calf tributaries became more prominent. She had clinical deterioration in both limbs, from CEAP (clinical, etiologic, anatomic, and pathophysiologic) class 2 to 3 in the right limb and from 3 to 4A in the left limb. The small saphenous and deep veins were normal in both examinations. The red dot indicates perforator vein reflux. The red letters and lines indicate the new sites of reflux in the second examination (N= normal; R = reflux).

incompetent. This may or may not occur in combination with primary venous wall disease.

Our study also demonstrated that only a few incompetent perforator veins were seen in association with reflux in the deep vein connected to them. A very important observation was that in all cases, deep vein reflux was not present at the time of the first ultrasound study, when the perforator vein connected to them were still competent. Superficial vein reflux was present in all of these sites, suggesting that deep vein reflux is not required for development of perforator vein incompetence in primary CVD. Rather, deep vein reflux could develop as a result of increased flow from the incompetent superficial veins through the perforator vein, the diameter of which has increased.

EFFECTS OF TREATMENT ON PERFORATOR VEIN FUNCTION

As discussed, perforator veins are more involved with CVD deterioration. A multivariate regression analysis model indicated that perforator vein reflux was a significant factor for CVD severity, notwithstanding the conspicuous contributing effects of superficial vein and deep vein involvement.[13] Perforator vein incompetence was significantly associated with aging, superficial vein reflux or deep vein reflux, or recurrence of superficial vein reflux, whereas the number of refluxing perforator veins depended on the presence of superficial or deep vein reflux regardless.[13]

CONCLUSION

Although the hemodynamic contribution of perforator veins seems important, clinical studies have failed to show that their treatment is necessary. It has been shown that in the absence of deep vein reflux, surgical correction of superficial vein reflux corrects perforator vein insufficiency in most limbs.[14] More importantly, a prospective, randomized study of 200 ulcerated limbs in 170 patients did not show a benefit from perforator surgery.[15] A very recent prospective, randomized trial showed that adding perforator vein treatment to superficial venous surgery was safe and effective in eliminating incompetent perforator veins in patients with a venous ulcer. However, this did not have any clinical benefit at 1 year.[16] ■

Antonios P. Gasparis, MD, RVT, FACS, is Director of Stony Brook Vein Center and Associate Professor of Surgery, Division of Vascular Surgery, Stony Brook University Medical Center in Stony Brook, New York. He has disclosed that he holds no financial interest related to this article. Dr. Gasparis may be reached at (631) 444-2040; antonios.gasparis@stonybrook.edu.

Nicos Labropoulos, PhD, DIC, RVT, is Director of the Vascular Laboratory and Professor of Surgery, Division of Vascular Surgery, Stony Brook University Medical Center in Stony Brook, New York. He has disclosed that he holds no financial interest related to this article. Dr. Labropoulos may be reached at (631) 444-2019; nlabrop@yahoo.com.

1. van Limbergh J. L'anatomie du systeme veineux de l'extremite inferieure en relation avec la pathologie variqueuse. Folia Angiol. 1961;8:240-257.
2. van Limbergh J. Anatomie der venae communicantes. Zentralbl Phlebol. 1965;15;4:268-271.
3. Labropoulos N, Mansour MA, Kang SS, et al. New insights into perforator vein incompetence. Eur J Vasc Endovasc Surg. 1999;18:228-234.
4. Pirner F. Die Bedeutung der insuff. V. perforans fur die Krampfadeoperation. Chir Prax. 1963;7:112-119.
5. Labropoulos N, Tiongson J, Pryor L, et al. Definition of venous reflux in lower extremity veins. J Vasc Surg. 2003;38:793-798.
6. Sandri JL, Barros FS, Pontes S, et al. Diameter reflux relationship in perforating veins of patients with varicose veins. J Vasc Surg. 1999;30:867-874.
7. Labropoulos N, Kokkosis AA, Spentzouris G, et al. The distribution and significance of varicosities in the saphenous trunks. J Vasc Surg. 2010;51:96-103.
8. Delis KT, Husmann M, Kalodiki E, et al. In situ hemodynamics of perforating veins in chronic venous insufficiency. J Vasc Surg. 2001;33:773-782.
9. Labropoulos N, Tassiopoulos AK, Bhatti AF, Leon L. Development of reflux in the perforator veins in limbs with primary venous disease. J Vasc Surg. 2006;43:558-562.
10. Labropoulos N, Delis K, Nicolaides AN, et al. The role of the distribution and anatomic extent of reflux in the development of signs and symptoms in chronic venous insufficiency. J Vasc Surg. 1996;23:504-510.
11. Labropoulos N, Patel PJ, Tiongson JE, et al. Patterns of venous reflux and obstruction in patients with skin damage due to chronic venous disease. Vasc Endovascular Surg. 2007;41:33-40.
12. Delis K. Leg perforator vein incompetence: functional anatomy. Radiology. 2005;235:327-334.
13. Stuart WP, Lee AJ, Allan PL, et al. Most incompetent calf perforating veins are found in association with superficial venous reflux. J Vasc Surg. 2001;34:774-778.
14. Delis K. Perforator vein incompetence in chronic venous disease: a multivariate regression analysis model. J Vasc Surg. 2004;40:626-633.
15. van Gent WB, Hop WC, van Praag MC, et al. Conservative versus surgical treatment of venous leg ulcers: a prospective, randomized, multicenter trial. J Vasc Surg. 2006;44:563-571.
16. Nelzén O, Fransson I; Swedish SEPS Study Group. Early results from a randomized trial of saphenous surgery with or without subfascial endoscopic perforator surgery in patients with a venous ulcer. Br J Surg. 2011;98:495-500.