INVITED COMMENTARY

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Carlie Chaplin once said that simplicity is not a simple thing. We seem to forget this when treating patients with varicose veins. Population studies showed that only one-third of these patients has saphenous reflux, yet elimination of saphenous reflux when it is present is believed to be curative, and removal of the saphenous vein from the circulation remains the mainstream intervention. Excellent short-term results of high ligation with or without stripping of the saphenous vein convinced many surgeons that recurrences result from the errors in surgical technique. Over time, however, it became clear that other factors play an important, if not the major, role in development of recurrent varicose veins and recurrent saphenous reflux. Surgical trauma induces inflammation and angiogenesis. Along with hemodynamic forces, these processes lead to reconnection of remaining distal saphenous vein and other superficial veins to the deep venous system. It is especially demonstrative in cases of high ligation without stripping, when neoveins form large collaterals around the ligated segment, sometimes indistinguishable in appearance from the saphenous vein itself.

Endovascular thermal ablation, which has replaced most open procedures, faces the same challenge. Similar to high ligation, segments of saphenous vein can reconnect to the femoral, or common femoral veins. In addition, the saphenous vein itself can recanalize.

Thermal damage results in coagulation necrosis of cells in the venous wall, denaturation of collagen, and, in many cases, acute thrombosis of a residual lumen. What happens later is less defined and more variable. Inflammatory response, fibrosis, angiogenesis, and recanalization differ significantly in activity, magnitude, and timing among the patients and the technologies that are used for vein ablation.

The acute effects of thermal ablation on venous wall have been studied extensively. In addition to the acute damage, the study by Ashpitel et al describes more longitudinal mechanism of activation of cell apoptosis. In an ex vivo experiment, more evenly distributed thermal energy produced the same effects as a conical laser beam, but at lower energy levels. Obviously, the advantages of a controlled experimental environment are counterbalanced by uncertainty of extrapolation of the finding to a clinical situation. How much of the increased expression of the apoptosis markers translates into a progressive degeneration of the media and consequent fibrosis remains unknown, as well as if activation of this process is sufficient for effective vein fibrosis. The influence of the thrombus in the residual lumen, and the inflammatory hyperemia in vasa vasorum on the longer term wall degeneration, as well as many other components of the biological reaction to the thermal injury of the intima remain to be studied. Despite these open questions, the article by Ashpitel et al contributes to increase awareness of the concept that initial thermal injury of venous endothelium is sufficient for long-term vein ablation, is a gross simplification. It also introduces an intriguing question—can acute damage to the venous wall be minimized without compromising the long-term outcome of the treatment?

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