popliteal artery entrapments reported in the Journal. We continue to believe that this condition, both anatomic and functional, is a frequently underdiagnosed cause of symptoms in the young, athletic claudicant.

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Regarding "The use of endovascular stents in the treatment of penetrating ulcers of the thoracic aorta"

To the Editors:

We read with interest the article by Brittenden et al titled "The use of endovascular stents in the treatment of penetrating ulcers of the thoracic aorta."[1] We were very surprised to note that the authors mentioned three times in this paper that the endovascular repair of penetrating thoracic aortic ulcers has not previously been reported.

Indeed, we have recently published an article titled "Penetrating atherosclerotic aortic ulcer of the descending thoracic aorta: treatment by endovascular stent-graft."[2] In this article, we report the results and the follow-up of endoluminal treatment in four patients with a penetrating aortic ulcer. Furthermore, Michael D. Dake et al previously reported their experience in five patients included in an article titled "Transluminal placement of endovascular stents-grafts for the treatment of descending thoracic aortic aneurysms."[3] Let's give credit when credit is due.

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Reply

We thank Dr Murgo and Dr Golzarian for their interest in our article. Our case reports describe two patients with ruptured penetrating ulcers who did not have aneurysms. We have clearly stated that endovascular repair has been performed for aneurysms and dissections in the past and are aware of the paper from Dake, as well as more recent publications.[1] In all of these cases, stents were deployed for patients with aneurysms, a small proportion of which were possibly secondary to penetrating ulcers. The natural history of penetrating ulcers remains unclear, and their ability to give rise to true, as opposed to pseudoaneurysmal formation, has not been proven. We therefore feel that our patients represent a discrete subgroup. At the time of writing the case report, a full MEDLINE search was performed, and the article by Murgo S et al, entitled "The use of endovascular stents in the treatment of penetrating ulcers of the thoracic aorta," was not identified at this time. However, we admit that this should be acknowledged as the first report of this procedure to have reached publication.

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Regarding "Photoplethysmography and calf muscle function after subfascial endoscopic perforator ligation"

To the Editors:

I would like to comment on the paper by K. A. Illig and co, "Photoplethysmography and calf muscle function after subfascial endoscopic perforator ligation" published in December of your journal (J Vasc Surg 1999;30:1067-76). I want to mention some inaccuracies in this article.

1. The duplex scan examination was performed in a 10-degree Trendelenburg's position. Under these circumstances no precise diagnosis of venous reflux can be made. The examination must take place in an erect position (ie, under the influence of gravitation). In addition, the presented diagnostic methods (physical examination, duplex scan), when used in Trendelenburg's position, cannot reliably differentiate primary from secondary varicose veins.

2. High ligation and stripping of the incompetent greater saphenous vein, high ligation, and stripping of the lesser saphenous vein, and SEPS were performed in most patients. This cohort represents a mixed material including elimination of reflux in the greater and lesser saphenous veins and interruption of the calf perforators. It is a scientific misinterpretation to describe the obtained results as a consequence of the subfascial endoscopic perforator ligation only. Unfortunately, many other authors make the same mistake.
3. The authors have classified the VRTs as "uninterpretable," if the calf could not empty below the baseline at the end of each tiptoe maneuver. In reality, such findings represent a severe venous disturbance. When the results given in Table I are evaluated from this point of view, then postoperatively six patients showed an amelioration, 11 patients a deterioration, and 13 patients remained unchanged when compared with the preoperative values. Therefore, a conclusion can be drawn that this treatment deteriorated the venous hemodynamics in 36% of cases, and an amelioration was achieved in only 20% of cases. This is certainly no positive result. The authors conclude, on the contrary, that the clinical results are satisfactory and that the plethysmography is a poor test for the assessment of reflux.
4. In the discussion from the auditorium, an important question was discussed, namely, whether the SEPS procedure is reimbursed and is billed as a perforator ligation. The most important question, however, was not asked: namely, whether the incompetent calf-perforating veins really play a role in the pathogenesis of the chronic venous insufficiency. The answer is NO! It is a proved fact (*a*) that the insufficient calf-perforating veins are not the cause of the chronic venous insufficiency,[1] (*b*) that the selective ligation of them does not improve the venous hemodynamics,[2-4] and (*c*) that the selective elimination of the saphenous reflux in patients with primary varicose veins and insufficient calf perforators repairs the venous disturbance and restores normal hemodynamic conditions in spite of the persistence of insufficient calf perforators.[1] In my paper published in 1996 I showed that in most patients with primary varicose veins and chronic venous insufficiency, the preoperative plethysmographic parameters (refill time t-90 and t-50 as well as refill volume, obtained with strain gauge plethysmography) were equal to zero (ie, severe venous disturbance, but according to the criterion of Illig and co "uninterpretable"). After selective elimination of the saphenous reflux (high ligation, no stripping, no ligation of the perforating veins), the parameters in nearly all patients returned to normal values. This is the proof that saphenous reflux and not insufficient calf perforators are responsible for the venous disturbance in primary varicose veins.

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Reply

Dr Recek's letter addresses two general issues: methodology of testing and underlying philosophy of pathogenesis and treatment.

We believe that the presence or absence of reflux can be reproducibly documented with the patient in 10 degrees of Trendelenburg. We are, however, in agreement that the upright position is the most physiologically sound, especially for the quantification of reflux (valve closure time). The "severe venous disturbance" he describes is, we believe, an artifact of a poor test and not indicative of any "real" hemodynamic change.

Dr Recek does not believe that the perforating veins play a role in the pathogenesis of chronic venous disease. We obviously disagree, and recognize that persuasive evidence can be cited on both sides of the argument. Whether or not ablation of incompetent perforators offers benefit over superficial ablation alone (or, indeed, nonoperative care) will only be answered by well-organized prospective randomized trials.

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