smoking history; (2) onset before the age of 50 years; (3) infrapopliteal arterial occlusions; (4) either upper limb involvement or phlebitis migrans; and (5) superficial phlebitis; and (6) exclusion of arteriosclerosis, diabetes, true arteritis, proximal embolic source, and hypercoagulable states. Typical arteriographic patterns have been described that are suggestive, but not pathognomonic. Besides, we agree with you that Buerger’s disease commonly affects males but females can be rarely involved.4

Measurement of ABI was done with technique described by Stewart et al.5 According to this measurement; no sound = 0 point, monophasic flow: 1, and if there is triphasic/biphasic flow: 2 point. We think that higher ABI value measured in present study depend on measurement technique.

M. Inan
Orthopaedic and Traumatology Department, Inonu University, Elazig yolu 10. km, 44900 Malatya, Turkey
E-mail address. minan@inonu.edu.tr

References

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Regarding: ‘Sapheno–femoral Junction Reflux in Patients with a Normal Saphenous Trunk’ by N. Labropoulos and Coll

I found much interesting statistical data in this paper, but some points were not very clear.

Incompetence at the sapheno–femoral junction (SFJ) associated with competence of the great saphenous vein (GSV) has only route though which it may flow. This a via reflux in the anterior accessory saphenous vein (AASV). This occurs when an AASV is present (40% of normal subjects), combined with incompetence of the ostial valve (terminal valve) of the GSV but competence of the pre-ostial valve (pre-terminal valve) of the GSV. Reflux from the SFJ takes the route of the AASV (where valves must also be incompetent) being prevented in the GSV by the competent pre-terminal valve.

This is confirmed by Fig. 1A and E of the paper, which both show an AASV. However, this route of reflux is not discussed in the text. This pattern has been described several times both in recent English and French literature, but I could find no citation of these papers. Do the authors agree with the conclusions of these papers or do they hold alternative opinions concerning the routes of venous reflux?

This discussion impinges on treatment strategies since there is a rationale for eliminating the AASV without ligation of the SFJ. This allows normal GSV emptying without permitting reflux in this vein since its valves are competent. I do not believe that it is appropriate to ligate the SFJ in such circumstances since this would create an obstruction to normal physiological GSV emptying and risk thrombophlebitis in the GSV. Finally, it is not clear to me what a ‘varicose’ SFJ is: how do you define ‘very dilated’?

S. Ricci
Corso Trieste 123, Ambulatorio Flebologico Ricci, 00198 Rome, Italy
E-mail address. varicci@tiscali.it

References

Re: Emboli Rate During an Early After Carotid Endarterectomy After a Single Preoperative Dose of 120 mg Acetylsalicylic Acid—A Prospective Double-blind Placebo Controlled Randomized Trial by Tytgat et al. Eur J Vasc Endovasc Surg 2005;29:156–161

I read with interest the paper by Tytgat et al. that the single dose of 120 mg acetylsalicylic acid does not influence the embolic rate as assessed by transcranial Doppler during carotid artery surgery. The conclusion seems valid. As the authors quote our study as a background and also compare our results and why they differ a point of clarification seems proper. The authors state that ‘in carotid surgery, a single dose of ASA administered the evening before surgery has been reported to reduce neurological events’. Our study was, however, not designed to look at a single dose but the patients were treated for 6 months and the evaluation of neurological outcome was performed during that period. I agree with the authors that it is of theoretical interest to analyse how a single dose of ASA preoperatively influences Doppler detected perioperative embolization, but our aim was to analyze whether half a year of ASA prophylaxis influenced the clinical outcome during a period when the risk of neurological complications is highest—and in fact it did.

D. Bergqvist*

Department of Surgical Sciences, Section of Surgery, University Hospital, 123, SE-751 85 Uppsala, Sweden

E-mail address. david.bergqvist@surgsci.uu.se

References


*Corresponding author. David Bergqvist, Professor of Vascular Surgery, Department of Surgical Sciences, Section of Surgery, University Hospital, 123, SE-751 85 Uppsala, Sweden.