Reply

We thank Dr Walker and his colleague for their kind interest in our recent publication. Their approach to treating the remaining popliteal aneurysm after exclusion and before bypass is a novel one. I must presume that this technique was carried out through a standard medial approach in which the entire aneurysm sac could not be opened completely and the feeding vessels ligated directly. Baum et al\(^1\) have shown that inducing complete sac thrombosis with glues or coils is a useful technique for eliminating type II endoleaks and halting aneurysm growth following endovascular repair for abdominal aortic aneurysm. Similarly, with popliteal aneurysms, if all source back-bleeding can be staunched at the time of aneurysm repair, it may be safe to presume that further aneurysm growth will not occur.

Although this procedure may have been successful in this single patient in 3-week follow-up, it may not be appropriate to declare this a complete success and “an effective technique for improving the exclusion of popliteal aneurysms” just yet. Other investigators have observed that, when feeding and outflow arteries are not ligated or coiled directly, blood flow into the sac and continued aneurysm growth (or bleeding, if the sac is left open) can still occur. In addition, this type of therapy will not likely treat symptoms from mass effect that may occur in patients with large popliteal aneurysms. We would suggest that patients with popliteal aneurysms who undergo this type of treatment should still be followed clinically and with serial scans so that we might know that this technique for eliminating sac perfusion is a durable one.

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REFERENCE


Regarding “Regarding ‘Veterans Affairs (VA) Cooperative Study #362’”

Recently, M.J.D. Tangelder et al, on behalf of the steering Committee of the Dutch BOA Study, commented on VA Coop Study #362 in a letter to the editor (J Vasc Surg 2003;38:629). We would like to respond to their comments and recommendations.

First, the two studies are not comparable as the VA trial evaluated a different hypothesis in an attempt to reduce the risk of bypass occlusion. A treatment program in which both platelet function and fibrin formation were simultaneously modified (warfarin combined with aspirin) was evaluated in the VA trial while the BOA study evaluated warfarin or aspirin independently.\(^1\)\(^2\) Second, the doses of aspirin (325mg = VA and 80mg = BOA) and warfarin (target INR = 1.4 to 2.8 in the VA trial and 3.0 to 4.5 in the BOA study) were different. Finally, the demographics of the study populations were different: women represented 36% of the BOA study group as compared with <1% in the VA trial, and the vein bypasses in the VA trial were at a more distal site (12% AK, 26% BK, 52% crural and 10% pedal) than those in the BOA study (46% AK, 34% BK, 18% crural and 2% pedal).

The mean length of follow-up was 38 months in the VA trial as compared with 21 months in the BOA study. The assisted primary patency (LTA) of the bypasses at 3 years in the vein group for patients treated with aspirin alone was similar (72% for BOA and 75% for VA). The BOA study did demonstrate a benefit (82% patency) for warfarin treatment alone as compared with low-dose aspirin while in the VA trial the patency (75%) was similar in both the warfarin plus aspirin group and the aspirin alone group. The increased warfarin dosage in the BOA study may have accounted for the benefit seen, but the lower dosage of aspirin could also have been a factor.

For prosthetic bypasses, the aspirin-treated patients had a somewhat similar patency (60% for BOA and 64% for VA); however, the warfarin plus aspirin—treated group had an improved patency of 72% in the VA trial as compared with 55% in the warfarin alone-treated patients in the BOA study. Hence, we cannot agree with Tangelder et al’s recommendation that “for patients with prosthetic bypass grafts, ASA remains the best antithrombotic treatment, worldwide,” but suggest that a combination of aspirin and warfarin may be better than aspirin alone.

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REFERENCES

