Regarding “Stent fractures in the Hemobahn/Viabahn stent graft after endovascular popliteal aneurysm repair”

It was with great interest we read the article by Tielliu et al., from Groningen, pioneers in the development of endovascular treatment. They reported their experience of using stent grafts to treat patients with popliteal artery aneurysms (PAAs). Sixty-four patients with 78 PAAs were treated and monitored for a median of 50 months. The authors are to be congratulated that their follow-up was so meticulous, including yearly examinations with duplex imaging and radiographs. Stent fractures were reported in 17% of the treated PAAs. The only significant risk factor for stent fracture development was younger age, most likely a proxy for a high activity level. Occlusion of the reconstruction occurred in 39% when a stent fracture was identified, compared with 25% among those without a stent fracture; this difference was not significant.

We also had an interest in the treatment of PAAs and performed a nationwide study on 517 patients operated on for 717 PAAs. The main findings were that preoperative thrombolytic therapy was associated with better runoff and a lower amputation rate when the patients presented with acute ischemia, that lifelong surveillance was warranted due to the high risk of developing new aneurysms, and that the surgical technique used was important for the long-term outcome.

When we re-examined 190 patients with 239 operated-on PAAs after a median of 7 years, we identified a clinical problem that has been underestimated previously: late expansion due to a phenomenon similar to that of type II endoleak after endovascular aneurysm repair. Among the patients operated on with a medial bypass and ligation of the popliteal artery above and below the aneurysm, this problem occurred in 33% and was symptomatic in 88% of those affected. Many of these patients need a reoperation, illustrated by the patient in the Fig., in whom a large PAA developed 8 years after ligation and bypass. This problem was virtually nonexistent after an operation with a posterior approach, when the branches are ligated, and this is presently our preferred technique for open surgery, whenever feasible.

Late expansion is also a potential problem after endovascular repair. Unfortunately, only 26 of the patients in our cohort were operated on with an endovascular technique, most of them with short follow-up. Thus, we are curious if Tielliu et al. have encountered this problem after endovascular repair of PAAs. They write: “In one case where a single stent-graft had been used, an endoleak was found due to disruption of the graft material.” However, no data are presented on the possible expansion of the PAA. Did they measure the diameter of the PAA on duplex examination? How often did the diameter increase, and how often was that associated with symptoms? If they did identify patients with late expansion after endovascular repair, was that more common after those with longer follow-up, as was the case after open repair with a medial approach?

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Reply

We thank Björck and colleagues for their interest in our study and their thoughtful comments and relevant questions. Their nationwide study on 717 popliteal artery aneurysms (PAAs) confirms the known problem of aneurysm expansion after open PAA repair. In total, 33% (57 of 174) of PAAs that were operated by the medial approach and monitored for a median of 7 years showed late expansion as a result of a type II-like endoleak. This was symptomatic in 88% (50 of 57) and resulted in reoperation in 14% of those affected.

The authors acknowledge that in 42% of the cases with a long bypass (originating from the common femoral artery or the prox-
nal superficial femoral artery) no ligation or only distal ligation was performed. This opens the way for back bleeding from collaterals and growth of the aneurysm. Ongoing growth of the aneurysm after open PAA repair has also been described in other reports, with a similar incidence.3,4 Mehta et al4 reported 10 of 26 patients (38%) showing flow in the aneurysmal sac after a follow-up of 38 months, with 23% demonstrating growth of the aneurysm and 12% ruptures.4

The importance of both proximal and distal ligation as close to the aneurysm as possible is therefore now generally recognized. But even then, collaterals originating from the aneurysm itself can be the source of continuous sac flow. No single report describes similar effects after endovascular PAA repair. However, this is an important question.

In this respect, we reviewed our data regarding endoleak and aneurysm growth for the cohort of 78 PAs. Of the 78 PAs, 22 were followed-up in other hospitals, and of the remaining 56 patients, 18 experienced stent graft occlusion. Therefore, data on endoleak and growth for 38 PAs were immediately available from our own hospital files up to this date. In 21 patients (55.3%), the aneurysm proved to have disappeared on duplex ultrasound imaging, and in 13 (34.2%), the aneurysm had shrunk. In another two patients (5.3%), a type II endoleak was seen on duplex, but the aneurysm did not grow. Finally, two other aneurysms (5.3%) with an endoleak had grown initially, from 32 to 42 mm and from 28 to 41 mm. They were treated with ultrasound-guided percutaneous thrombin injection, although they were asymptomatic, and have had a stable diameter since then. This means that growth of the aneurysm only occurred in 5% of the patients after endovascular repair. This figure is even lower than the 8.3% (2 of 24) mentioned by Ravn et al2 with a posterior approach in open surgery.

The explanation for the low incidence of type II endoleak and aneurysm growth after endovascular PAA repair is probably that the stent graft fills the lumen of the PAA and prevents back bleeding in the landing zone due to apposition of the stent graft to the wall of the vessel. It is clear that collaterals originating from the aneurysm itself still can give rise to an endoleak, but apparently this is rarely happening.

References

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Regarding “Inherent problems with randomized clinical trials with observational/no treatment arms”

The unusual conclusions in the article by Buckley et al1 that life-threatening diseases like abdominal aortic aneurysm (AAA) “should not be evaluated using RCTs containing an observation/no treatment arm” and that “intent-to-treat analysis . . . should not be used in this situation” were based on the authors’ belief that high crossover rates are an “inherent weakness” of these studies. This belief stems from a misunderstanding of the term “crossover.” A crossover occurs when a patient receives the alternative intervention rather than the randomly assigned intervention, and represents a serious protocol violation which, if sufficiently frequent, can obscure the trial results. The Aneurysm Detection and Management (ADAM) trial2 compared two strategies in patients with an AAA 4.0 to 5.5 cm: immediate open repair vs surveillance with repair of AAA that enlarged to 5.5 cm or greater. Patients in the surveillance group who had repair when the AAA enlarged to 5.5 cm or greater were not crossovers, they were treated according to protocol and, therefore, contribute to the valid comparison of the outcomes of the two strategies. The statement by Buckley et al regarding the ADAM trial that “61.6% of those randomized to ultrasound surveillance crossed over to open repair” is incorrect. As reported in the text and Figure 1 of the ADAM manuscript, less than 10% of the surveillance group had repair off-protocol and represent crossovers.

Apart from Buckley et al, there is near universal agreement that randomized trials remain “the gold standard in evaluating healthcare interventions” and that intent-to-treat analysis remains an essential aspect of their validity.3 Randomized trials established the benefit of coronary artery bypass surgery and carotid endarterectomy,5 and there is no “inherent” reason why AAA repair should be studied any differently.

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Reply

The primary purpose of the authors’ article was to identify potential problems, which can occur when large numbers of patients in randomized abdominal aortic aneurysm (AAA) clinical trials cross over from no treatment to treatment. We feel these cross overs obfuscate some of the trial’s conclusions. Despite comments to the contrary, the predominant reasons for trial participants to