also be treated. On the other hand, perfusing vessels in nonpulsatile aneurysms are observed for signs of aneurysm growth. If the etiology of the endoleak is unclear, we find ourselves in a quandary and undergo a detailed evaluation of the potential collateral vessels. We may rely on the expected decrease in aneurysm size to help delineate a treatment course, or we may perhaps even treat the endoleak merely to definitively establish the diagnosis. A nonpalpable aneurysm is treated in a more aggressive fashion than a palpable, nonpulsatile aneurysm. In this situation, we believe the risks of observation are largely unknown, and without knowledge of the extent of the physical forces on the aneurysm wall, the risks of observation may outweigh the risks of treatment. Quantifications of these forces are under investigation but have not yet entered into the clinical arena. Although some may criticize this protocol as being overly aggressive, we believe that the overall validity of endovascular repair will depend on long-term efficacy, which is, at this point, undefined.

Differences between this decision tree and published regimens include a means to determine the need for aggressiveness in patients with either undefinable endoleaks or those caused by perfusing vessels. Fundamental to this algorithm is that the follow-up physical examinations be coupled with radiographic evaluation and that a clinician evaluate the patient both before and after surgery, paying careful attention to the findings of the physical examination. Furthermore, this information should be noted in follow-up examination. Because endovascular grafting techniques have lead us down a highly technical path, we are, in a sense, recommending a step backward to reemphasize the role and need for the clinical examination.

REFERENCES


24/41/103790

Regarding “In situ replacement of infected aortic grafts with rifampicin-bonded prostheses: the Leicester experience (1992 to 1998)”

To the Editors:

In the article by Haye and colleagues (J Vasc Surg 1999;30:92-8), the authors referred to one of our publications and erroneously stated that we have suggested that a polytetrafluoroethylene graft may be used for in situ replacement of infected aortic prostheses.1 The paper that the authors refer to was addressing whether infected prosthetic grafts could be preserved successfully, but never suggested that a new prosthetic graft be used to replace a previously excited infected prosthetic graft. We have never suggested this as an option to treat infected prosthetic grafts.2 We believe that it is extremely important that readers of the article by Haye et al do not believe that we are in favor of placing a new prosthetic graft into an infected field, although the role of rifampicin-bonded prostheses may prove to be useful.

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REFERENCES


24/41/105672

Reply

We are pleased that Dr Calligaro notes that in situ replacement with rifampicin-bonded grafts may have a role to play in the management of graft infection. We accept that Dr Calligaro and colleagues1 have not previously recommended the replacement of infected prosthetic grafts with a further prosthetic graft, rather their selective preservation in extravascular graft infections. For intracavitary infections, the focus of our paper, the mainstays of treatment are graft excision followed by either in situ replacement or extranatomic bypass (EAB). We believe that in situ replacement overcomes a number of the problems of EAB, namely, stump blowout, poor flows, lower patency, and prolonged procedure time. The use of antibiotic-bonded grafts may
further increase the success of in situ replacement.

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REFERENCE

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Regarding “A rational algorithm for duplex scan surveillance after carotid endarterectomy”

To the Editors:
We read with interest the paper by Roth and colleagues regarding requirement for duplex surveillance following carotid endarterectomy.1 We have similarly looked at this issue and previously published in the Journal of Vascular Surgery.2 In contrast to Roth and colleagues, we followed a conservative path with respect to asymptomatic contralateral carotid artery disease and restenosis. We only treated patients who developed symptoms in association with a tight stenosis. We related development of ipsilateral or contralateral stroke during follow-up to the presence of contralateral carotid artery disease at the time of operation, in addition to the development of ipsilateral restenosis or progression of contralateral carotid artery disease.

We found no relationship between development of restenosis and ipsilateral stroke. Similarly, there was no relationship between the presence of tight contralateral stenosis and subsequent contralateral stroke, nor was there a relationship between the progression of contralateral artery disease and subsequent contralateral stroke. On the basis of this, our data would support a conservative approach to restenosis and contralateral carotid artery disease. We feel that long-term duplex surveillance following carotid endarterectomy is unwarranted on these data, and funds would be better spent elsewhere.

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REFERENCES


24/41/105674

Reply

The vascular surgery section at Charing Cross Hospital found no benefit from duplex surveillance after carotid endarterectomy in a prospective study of 305 patients studied at frequent (1 week and 3, 6, 9, 12 months) intervals during the first postoperative year and annually thereafter. As stated in their letter, “funds would be better spent elsewhere.” We agree in part with these conclusions and proposed a “rational” surveillance algorithm based on duplex scanning performed at operation, at 4 to 6 weeks after endarterectomy, and thereafter on the basis of carotid disease severity. When enrolled in a stroke prevention program after carotid endarterectomy, most patients do not need intensive duplex scan surveillance, if a policy of operation for high-grade internal carotid artery (ICA) stenosis is adopted. When the intraoperative duplex scan verifies a normal repair, the incidence of restenosis is low, less than 4% by life table analysis at 3 years. Thus approximately 80% of patients with a normal repair site and less than 50% contralateral ICA diameter reduction stenosis can be followed up by clinical examination and a duplex scan at 1- to 2-year intervals. Mr Greenhalgh and coauthors similarly noted in their paper that “restriction of follow-up to patients with >50% stenosis would have reduced to 78% the percentage of patients requiring duplex surveillance, and still identifying 8 of 9 patients who required endarterectomy.” Contralateral >50% ICA stenosis is a lesion with a propensity to progress in severity (approximately 10% incidence per year) and produce stroke or ICA occlusion. In our series, approximately 15% of patients with a normal repair had contralateral >50% stenosis, and duplex surveillance at 6-month intervals was adequate to detect asymptomatic disease progression. The carotid repair with residual or early appearing restenosis is at risk for progression to a high-grade lesion but occurs in less than 5% of patients. It seems prudent to me to evaluate these patients at 6-month intervals by duplex scanning. Duplex surveillance is one component of an effective stroke prevention program for patients after carotid endarterectomy. Control of atherosclerotic risk factors, lipid-lowering therapy, antiplatelet drug administration, and control of hypertension are equally important features of the patient’s surveillance program. In our experience, the risk of stroke was less than 1% per year in all patient groups, if the duplex surveillance is tailored to the individual patient and a policy of repair of high-grade (>75%–80% diameter-reduction) asymptomatic stenosis is adopted. In their paper, Mr Greenhalgh and associates did not define what duplex criteria indicated a high-grade stenosis that warranted consideration for further intervention. At the University of South Florida, according to our patient surveillance data and correlation studies with contrast arteriography, we recommend repair of a progressive atherosclerotic ICA stenosis in appropriately screened surgical candidates when end-diastolic velocity at the site of stenosis is more than 125 cm/s and the ICA:CCA ratio is more