therapy are relevant to the study but, unfortunately, were published after the study period.

We hope that Dr Schouten and colleagues may be able to add to our understanding by using the Revised Cardiac Risk Index and the presence or absence of cardioprotective medication as criteria in a similar study.

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Regarding "A prospective study of subclinical myocardial damage in endovascular versus open repair of infrarenal abdominal aortic aneurysms"

We read with interest the article by Abraham et al (J Vasc Surg 2005;41:377-81) reporting increased levels of cardiac troponin (cTn) T in 9% of patients after elective endovascular repair and 25% of patients after elective open repair of infrarenal abdominal aortic aneurysm. These findings are similar to our own, in which increased levels of cTnI were detected in 10 (29%) of 35 patients after elective open aortic reconstruction.[1] Because our study was performed in a unit that did not perform endovascular abdominal aortic aneurysm repair at the time, it seems unlikely that the authors' suggestion that institutional unfamiliarity with open aortic surgery, or more advanced arterial disease in patients unsuitable for endovascular repair, can adequately explain the higher incidence of myocardial injury in the open repair group. One possible explanation for the findings may be related to the fact that there was a higher incidence of previous myocardial infarction in patients treated by endovascular repair (41%) compared with open repair (22%). There is considerable evidence to support the use of antiplatelet agents, β-blockade, and statin therapy in reducing the incidence of early and late myocardial infarction and cardiovascular deaths in patients undergoing major vascular surgery.[2-5] One would expect a cardiologist to have been involved in the management of myocardial infarction in these patients and, therefore, best medical therapy to have been commenced. It is possible that such medical optimization may have contributed to the reduction in myocardial injury associated with endovascular repair. We would be most interested to know whether the authors have information on the relative use of best medical therapy in their two groups of patients.

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Reply

We thank the readers for their comments, and we were very interested to see that they have obtained a similar incidence of cTnI elevation in 29% of patients after elective open aortic reconstruction at a unit that did not perform endovascular abdominal aortic aneurysm repair at the time. This, in our opinion, would confirm our results that there is a statistically and almost certainly clinically significant difference in the incidence of subclinical myocardial damage after the two types of infrarenal abdominal aortic aneurysm repair. In our two groups of patients, the preoperative management did not include routine β-blockade and statin therapy. Although this was not prospectively documented, medical optimization was more likely to have taken place in the open group in view of the degree of severity of the planned procedure. It is quite unlikely that medical optimization would have contributed to the reduction in myocardial injury associated with endovascular repair.

Our study protocol, as approved by the local ethics committee, dictated analysis of all samples in batches in a way that was not related to the day-to-day management of the individual patient. This was to avoid unnecessary interventions based on the results of a serum analysis that under normal circumstances would not take place according to the best current practice of that time, because troponin measurement is not a routine part of postoperative patient care.

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Regarding "Ultrasound findings after radiofrequency ablation of the great saphenous vein: Descriptive analysis"

In the recent article by Sergio Salles-Cunha et al,[1] the authors suggest a very high neovascularization rate after radiofrequency ablation (RF) of the great saphenous vein (GSV), which does not correspond to our own experience. The authors describe small vessel networks (SVN), which covers without discrimination all vessels smaller than 2 mm in the surrounding tissue of the treated GSV, including muscular, collateral, and tributary veins and their satellite arteries. The high prevalence of these SVN elements in the groin area and at the thigh level is interpreted as the result of a process similar to the neovascularization described after GSV ligation and stripping. However, without a controlled assessment of

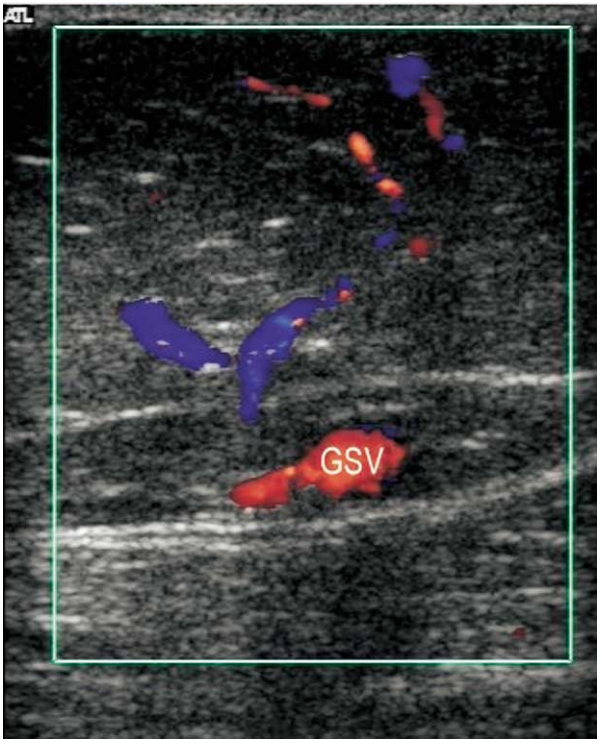

Figure. Pretreatment duplex image showing collateral veins and arteries close to the refluxing great saphenous vein *(GSV)* trunk.

the preoperative extent of the SVN, the possibility of pre-existing SVN cannot be excluded, especially when the duplex ultrasound method was not identical before and after surgery, with postoperative setting optimization meant to maximize sensitivity.

In fact, many small vessels can be identified with ultrasonography before any treatment, and there is no reason that RF ablates them. For example, the external pudendal artery visible in contact with the GSV termination in preoperative ultrasound examination remains discernible after RF, in contact with the occluded GSV. Similarly, it is common to visualize arterioles close to the GSV during ultrasonography-guided sclerotherapy (Figure). Considering these arterioles as SVN, they cannot be attributed to neovascularization. Regarding the veins, it is known that the saphenofemoral junction (SFJ) tributaries, which are also covered by the SVN definition, remain patent after RF in more than 90% of cases.[1] Therefore, it is not surprising that the authors found a lower prevalence of SVN when a high ligation was performed. It would have been helpful for the authors to provide a more anatomically precise SVN classification. It is difficult to assign an identical pathologic effect to vessels so anatomically different as arterioles and refluxing or not refluxing veins. In our experience, refluxing veins observed after RF—excepting the cases of recanalization or nonocclusion—involve only the low SFJ tributaries or the groin ganglionic veins.

The persistence of a short patent segment at the termination of the GSV is a known pattern, consistent with the endovenous therapeutic concept that allows physiologic drainage of the SFJ collaterals toward the deep system.[2] However, the prognosis of GSV termination recanalization in case of associated high ligation should be more pejorative because of the possibility of physiologic drainage of the recanalized GSV is suppressed. The rate of GSV trunk recanalization in this study (35%) is much higher than previously reported (9.5% at 2 years and 11.5% at 4 years).[2,3] Knowing that only 20% of the GSVs with partial occlusion or nonocclusion are refluxing, it is not obvious that the network including the feeding vein, the recanalized GSV, and the draining vein observed by the authors was inconsistent with a physiologic circulation, especially in the absence of reflux.

Because the endovenous treatment concept is very different from conventional surgery, it is mandatory to attentively assess the results of RF. Ensuring an identical preoperative and postoperative ultrasound method to establish consistent conclusions is also necessary.

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Reply

We did not suggest a very high neovascularization rate after radiofrequency ablation (RFA) of the great saphenous vein. We did discuss a possible connection between what other authors called *neovascularization* and what we prefer to name *small vessel networks.* A major reason why we selected *small vessel network* was to avoid the connotation associated with the concept of neovascularization. Our intention was also to critique the use of the term *neovascularization* without proof that these vessels did not exist before. For one reason, existing small vessels may dilate in response to an inflammatory process and may constrict after the inflammation subsides. Clinically, thrombosis may occur in the postoperative period in untreated segments proximal and distal to the segment exposed to the RFA and also within the treated segment. Furthermore, patients have complained of localized pain or discomfort starting months after a successful RFA. This localized pain was associated with a visible vein valve sinus either totally thrombosed or partially recanalized; pain was associated with a thrombus that seemed hypoechoic enough to be considered recent. There is growing evidence associating the appearance of small vessels, including arterialized flow, with thrombus.

In our articles, we also made the distinction between recanalization and reflux. The term *recanalization* was preferred to avoid the clinical inference associated with the concept of reflux. At early stages of recanalization, clinical reflux is commonly absent. This recanalization can be temporary and limited in length. (A reason why recanalization was higher in this series than previously reported may be that we considered recanalization segments as short as 1 or 2 cm occurring at any level.) An unstable cycle of thrombosis, inflammation, thrombolysis, recanalization, reduction of inflammation, low-flow condition, and then new thrombosis has been suspected. Eventually the recanalization either becomes wide enough to be part of a local network of small vessels or thrombosis dominates, and, with thrombus aging, the vein segment becomes atretic and atrophic and eventually disappears as such, becoming unrecognizable by ultrasonography.

We also have described small vessel networks associated with telangiectasias. It is not surprising to detect small vessel networks in these patients before or after treatment of the saphenous vein.

Patients have described the appearance of new telangiectasias after saphenous vein treatment, RFA, or stripping. We have connected the appearance of such new telangiectasias to the small vessels surrounding thrombosed, treated segments of the saphenous vein, mostly large vein valve sinuses. Small vessel networks can also be noted around thrombus inside the canal previously occupied by a stripped saphenous vein. Rather than dismiss the findings of small vessel networks noted after treatment of the saphenous vein, we should investigate whether and when the presence of these channels has a clinical effect. The first step was to recognize the presence of such small vessel networks.

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Regarding: Carotid endarterectomy in patients with chronic renal insufficiency: A recent series of 184 cases

We read with interest the article by Ascher et al (J Vasc Surg 2005;41:24-9) about early outcome of 184 internal carotid artery endarterectomy procedures in 166 patients with chronic renal insufficiency (CRI). These patients with CRI (serum creatinine level >1.5 mg/dL) accounted for 27% of all patients undergoing endarterectomy, a proportion much higher than in our own experience of 4.6% based on the same criterion (146/3157 patients during 1990 to 2002). We believe that the higher risk of postoperative death associated with severe CRI, as shown by Ascher et al, should limit the indication for surgery, especially in asymptomatic patients on dialysis. Many vascular surgeons have intuitively applied that restriction. However, as shown by Reil et al,[1] the neurologic risks associated with endarterectomy are similar in patients with CRI and normal renal function. Rather, it is cardiac complications that are the most serious threat for death in these patients.

Regretfully, Ascher et al failed to give details about the five deaths that occurred in their series. However, in the meeting discussion accompanying the article, the authors stated that two deaths were due to myocardial infarction, one due to multiple organ failure triggered by an unknown cause, one due to bronchopulmonary aspiration (neurologic status not specified), and one due to postoperative stroke. This stroke-related death shows that it would have been useful to do a thorough analysis of mortality rather than to speculate about the potential role of protamine or hemodynamic disturbances observed after dialysis sessions.

The article and meeting discussion also revealed an ambiguity towards cardiac work-up. In the article, the authors suggest that rigorous cardiac assessment is necessary even in asymptomatic patients. In the discussion, they argue against such work-up by stressing the morbidity of "open heart" surgery. It is unclear why they would rule out the possibility of treating coronary artery lesions screened by percutaneous angioplasty.

Patients with CRI must be considered a high-risk group for carotid endarterectomy because of mortality rather than stroke risk. In this regard, they may be better candidates for carotid stenting, like those patients with advanced heart disease included in the SAPPHIRE study.[2] Further study would be necessary to demonstrate the safety and long-term efficacy of such an approach. Until then, our approach is to be very conservative in recommending carotid endarterectomy for patients with CRI, especially the asymptomatic cohort.

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Reply

Thank you for your comments regarding our paper concerning carotid endarterectomy in patients with chronic renal insufficiency (CRI). We agree with you that a more thorough assessment of the cause of death postcarotid endarterectomy in these patients would have been of value. However, upon reviewing the medical records of the patients who died postoperatively, we were unable to uncover a common denominator. We did speculate about the role of protamine and also the hemodynamic disturbances that may occur during hemodialysis, because these are known facts associated with increased mortality.

It is our opinion that coronary bypass surgery in patients with severe CRI should be limited because of the associated increased mortality and morbidity. Whether percutaneous coronary artery balloon angioplasty is of value to these patients remains to be proven.

Also, we agree with your comments that carotid endarterectomy should not be liberally used in asymptomatic patients with severe CRI. It will be interesting to see whether carotid artery balloon angioplasty and stenting would be of benefit for this cohort of patients in the long-term.

We appreciate your taking the time to read our paper and ask reasonable questions.

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