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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## REFERENCES

1. Abraham N, Lemech L, Sandroussi C, Sullivan D, May J. A prospective study of subclinical myocardial damage in endovascular versus open repair of infrarenal abdominal aortic aneurysms. J Vasc Surg 2005;41:377-81.
2. Chaikof EL, Fillinger MF, Matsumura JS, Rutherford RB, White GH, Blankensteijn JD, et al. Identifying and grading factors that modify the outcome of endovascular aortic aneurysm repair. J Vasc Surg 2002;35:1061-6.

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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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## REFERENCES

1. Haggart PC, Adam DJ, Ludman PF, Bradbury AW. Myocardial infarction following aortic surgery: a comparison of cardiac troponin I and creatine kinase ratio. Br J Surg 2001;88:1196-200.
2. Poldermans D, Boersma E, Bax JJ, et al. The effect of bisoprolol on perioperative mortality and myocardial infarction in high-risk patients undergoing vascular surgery. N Engl J Med 1999;341:1789-94.
3. Poldermans D, Bax JJ, Kertai MD, et al. Statins are associated with a reduced incidence of perioperative mortality in patients undergoing major noncardiac vascular surgery. Circulation 2003;107:1848-51.
4. Kertai MD, Boersma E, Westerhout CM, et al. Association between long-term statin use and mortality after successful abdominal aortic aneurysm surgery. Am J Med 2004;116:96-103.
5. Durazzo AES, Machado FS, Ikeoka DT, et al. Reduction in cardiovascular events after vascular surgery with atorvastatin: a randomized trial. J Vasc Surg 2004;39:967-75.

doi:10.1016/j.jvs.2005.05.064

## 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