References

1 Melissano G, Castellano R, Mazzitelli S, Zoppei G, Chiesa R. Safe and cost-effective approach to carotid surgery. *Eur J Vasc Endovasc Surg* 1997; **14**: 164–169.

Authors' Reply

We would like to thank Dr Munro for the interest he has shown in our paper[1] and for his comments. We are sure that Dr Munro is aware that an increasing number of institutions are using duplex ultrasound as the single independent preoperative test; on the other hand, non-selective digital subtraction arch angiography, which is safer than selective views, lacks the detail that is needed, particularly in unclear cases.

In order to be reliable, duplex ultrasonography should be performed on good quality equipment, by qualified personnel and the duplex criteria for identifying critical stenosis should be validated. The lack of these points has probably contributed to the poor correlation between duplex ultrasonography reported in some multicentre trials such as NASCET.[2]

The 33 min time saving that we recorded and reported in Table 1, employing regional anaesthesia, is clearly explained by the fact that the patient doesn't need to be awakened and extubated at the end of the procedure and that the anaesthesiologist may perform the anaesthetic block in a different room while the operating room is cleaned up and prepared for the next procedure. Similarly we perform most of our peripheral and phlebologic procedures under epidural or spinal anaesthesia with a significantly increased number of cases per operating session. We think that this policy should be highly recommended to all centres in which this anaesthetic skill is available.

Our definition of intensive care unit is indeed the standard one, and this facility was available every time but was required very seldom in this series. As we stated in the patients and methods section, all our patients are monitored (with two lead ECG-D2, V5-, pulsed arterial pressure and continuous SpO_2) in the immediate postoperative period for at least 3h. We assume that a careful nerve-sparing dissection, an accurate reconstruction of the carotid bifurcation, the greater haemodynamic stability that is granted by regional anaesthesia,[3] the ability to detect from the history and the perioperative behaviour the patients at greater risk for haemodynamic instability and the presence of skilled staff on the surgical ward may all play a role, besides fortune, in the prevention of untoward postoperative events.

As far as the total length of stay is concerned, we do agree that a 5-day stay is still too long; in particular our preoperative stay is far too long. We stated in the Discussion section that ideally the patient could be admitted on the same day of surgery. We are working on this and we commend the centres that already reduced the total stay to 2–3 days. Interestingly, McCollum *et al.*[4] recently reported that the mean hospital stay in 709 patients undergoing carotid surgery in the U.K. and Ireland under 59 different surgeons was 7.1 days (1–91). As Fig. 1 demonstrates, it took us a few years to lower the postoperative stay down to 2 days. Further shortening is an even slower process; according to our protocol in 1995 we were able to send patients home on the first postoperative day in less than half of the cases.

The indication for carotid surgery in asymptomatic patients is a very interesting topic that has been addressed in a large number of papers and that will still be much discussed until unequivocal data are available. McCollum[4] reported that British surgeons, who are considered rather conservative in this respect, operated 8.1% of their patients with asymptomatic stenosis, 8.9% with controlateral symptoms and 2.4% with vertebro-basilar symptoms. Unfortunately this topic has little to do with our paper. We do agree with Dr Munro that patients should be selected with the utmost attention; in particular each surgeon should be aware of his own results when suggesting a procedure especially to an asymptomatic patient. Our mortality and morbidity rate in asymptomatic patients is well below 1%, we would probably be even more cautious in considering this indication if our complication rate was higher.

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Hyperhomocysteinaemia

Sir,

I note with interest the recent case report by I. V. Mohan and colleagues regarding hyperhomocysteinaemia and aneurysm formation.[1] My recent presentation at the Surgical Research Society demonstrated a significant relationship between hyperhomocysteinaemia and abdominal aortic aneurysms.[2] The age-matched control group gave a reference range for total fasting homocysteine of 8.86–14.25 μmol/l using high performance liquid chromatography with electrochemical detection. In 14 patients with isolated abdominal aortic an-

eurysms and no clinical evidence of other vascular disease we found 11 (79%) with hyperhomocysteinaemia (homocysteine >14.25 μmol/l). To date we have found no significant difference in B_{12} and folate levels in these patients. Results for the methylene tetrahydrofolate reductase transition are awaited. Previous studies have found hyperhomocysteinaemia in up to 39% of patients with vascular disease[3] but not in aneurysmal disease. In addition to the anecdotal reports and further references quoted in this case report, recent work has demonstrated the induction of a serine elastase by homocysteine lending additional evidence to the putative role of homocysteine in aneurysm formation.[4]

What seems surprising is that relatively modest increases in plasma homocysteine seem to be so significant, as demonstrated by a three-fold increase in myocardial infarction.[5] The problem remains not only in understanding how homocysteine causes vascular disease, but in its relevance. It is now almost 30 years since homocysteine was first implicated in vascular disease. Numerous studies have established it as a risk factor which is modifiable, yet despite the ease with which homocysteine levels can be lowered by dietary supplementation, we have no long-term prospective data on the influence of homocysteine lowering treatment on progression of vascular disease. Such a study would answer the question about the possibility of managing small aneurysms with vitamin supplementation. Furthermore, studies on the mechanism of homocysteine induced vascular damage using a reproducible *in vitro* model are required to elucidate the role of homocysteine in atherogenesis.

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References

1 Mohan IV, Adam DJ, Kurian KM, Vaughan Ruckley C. Isolated external iliac aneurysm associated with hyperhomocysteinaemia. *Eur J Vasc Endosvasc Surg* 1997, **14**: 506–508.
2 Caldwell S, Martin SC, Hilton AC, Bartlett WA, Jones AF, Mosquera DA. Hyperhomocysteinaemia is associated with abdominal aortic aneurysms. *Brit J Surg* 1998; abstract in print.
3 Taylor LM Jr, Defrang RD, Harris JE, Porter JM. The association of elevated plasma homocyst(e)ine with progression of symptomatic peripheral arterial disease. *J Vasc Surg* 1991; **13**: 128–136.
4 Jourdheuil-Rahmani D, Rolland PH, Rossset E, Branchereau A, Garcon D. Homocysteine induces synthesis of a serine elastase in arterial smooth muscle cells from multi-organ donors. *Cardiovascular Research* 1997; **34**: 597–602.
5 Stampfer MJ *et al.* A prospective study of plasma homocyst(e)ine and risk of myocardial infarction in U.S. physicians. *JAMA* 1992; **268**: 877–881.

No reply received

Rifampicin-Soaked Grafts

Sir,

We read with interest the article by Torsello and Sandman regarding the use of antibiotic-bonded grafts in vascular graft infection. They show that the *in situ* technique using a rifampin-bonded graft can be an effective strategy with good mid-term results. After a mean follow-up of 33 months, only one recurrence and one death by sepsis was found among 12 patients treated with a gelatin-coated Dacron prosthesis soaked with rifampin (60 mg/ml). Recent clinical and experimental studies have suggested an expanded role for an antibiotic-bonded vascular prosthesis in the treatment of established graft infections, particularly when the graft is impregnated with gelatin or collagen, leading to drug accumulation and prolonged release. Despite the use of different concentrations of rifampin in which grafts have been immersed (ranging from 1 mg/ml to 60 mg/ml), this procedure has been associated with excellent results in animal models[2–4] and in some sporadic clinical cases. Until now 16 patients receiving antibiotic-bonded grafts for vascular infections have been reported.[5–8] Although the simplicity of this bonding technique greatly enhances its clinical applicability, our experience suggests that the use of *in situ* replacement of infected vascular prosthesis with rifampin-soaked vascular grafts does not avoid the risk of recurrent infections.

Between 1993 and 1995, five patients with vascular infections were treated using rifampin-soaked Dacron grafts at our institution. For impregnation, the grafts were soaked in a saline solution containing 600 mg rifampin (2 mg/ml) at room temperature for at least 15 min before the implantation. Two patients had infected aortic aneurysms. One of them had a piece of a coronary catheter embedded into thrombus and aortic wall 3 years before. Because of suspected infection, a collagen-sealed graft (Hemashield; Meadox Medicals, Inc., Oakland, NJ, U.S.A.) was soaked with rifampin before the implantation. Cultures from the catheter, thrombus and aortic wall grew *Staphylococcus epidermidis*. A CT scan at 48 months showed no evidence of residual infection, and the patient did well after surgery. The other patient underwent emergency