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Correspondence

Acute Exercise and Markers of Endothelial Injury

Sir

The recent study by Woodburn *et al.*[1] found no evidence of vascular endothelial damage following treadmill exercise in claudicants. While the authors' comments may be correct, we doubt that the study they present could in any way lend support to their conclusions. A number of groups have observed evidence of an inflammatory response after maximal exercise in claudicants. Whether this is of short or long-term clinical significance is unclear.

The authors report the results in a small number of patients, some of whom did not walk to their maximum capacity. The timing of the post-exercise sample was presumably based on the authors' previous work following angioplasty. Several clinical studies have used the treadmill model of claudication to define the time-scale of this inflammatory response. Maximal exercise followed by rest leads to free radical formation at 1 min,[2] neutrophil activation at 5 min,[3] neutrophil thromboxane production at 10 min, peaking at 60 min[3] and endothelial damage (urinary microalbumin excretion) at 60 min.[4,5] One would therefore expect an increase in vWF to occur much later than 2 min post-exercise. In fact, Edwards *et al.*[6] reported a significant increase in vWF after 60 min rest in claudicants, with no change in control subjects.

The authors report the effects of a single episode of walking and suggest that this has implications for the role of therapeutic exercise programmes in claudication, which are generally continued for 6 months or more. In fact, we found that regular exercise attenuates this inflammatory response over a period of time.[5]

The exercise-induced inflammatory response in claudicants is well reported. The significance, if any, of the phenomenon is unclear but this paper has done little to contribute to our understanding of this.

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Exercise in Patients with Intermittent Claudication

Sir,

The paper by Woodburn *et al.* will certainly stimulate discussion among clinicians and scientists interested in the effects of exercise in patients with intermittent claudication (IC).

Endothelial injury may lead to progression of POAD or acceleration of coronary/carotid or cerebral artery disease and, is therefore considered important in this patient group who are known to have an increased morbidity and mortality mainly due to adverse cardiac or cerebral events.[1] Endothelial behaviour is, however, only one aspect of a hierarchy of cellular and chemical interactions, also involving platelets neutrophils and cytokines, that occur when patients with IC exercise.

The authors omitted to mention that von Willebrand's factor and thrombomodulin have previously been measured as markers of endothelial damage in patients with IC and serum levels have been shown to rise significantly following exercise,[2,3] but

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these observations were made over different time periods than that chosen by the authors. Animal models of IC have also directly demonstrated endothelial swelling using electron microscopy.[4]

We would therefore suggest a more cautious conclusion. The results of Woodburn *et al.* relate to a narrow temporal window in a group of patients who were subjected to relatively gentle exercise when compared to that suggested by those using exercise as a treatment for IC.[5] The debate regarding adverse effects of exercise in patients with IC remains open.

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No reply received

Carotid Surgery

Sir,

We read with interest the article by Dr Melissano *et al.*[1] which describes how the costs of carotid surgery can be reduced by the limited use of arteriography, the routine use of local regional anaesthesia, the selective use of postoperative intensive care and early postoperative discharge.

We agree with the author's selective use of arteriography only in patients with questionable duplex scans and would like to support their view that a CT scan and a neurological assessment is obtained in all patients. This policy will undoubtedly save money and possibly reduce morbidity, although non-selective digital subtraction arch angiography is very safe and most carotid trials require its use.

With regard to the use of local regional anaesthesia, we doubt whether there is much saving to be made as an anaesthetist is still required to administer the anaesthetic and other drugs and to monitor the patient during the procedure. The authors state that 30 min of global operating room time was saved compared to general anaesthesia but give no data to support this figure. For instance, has it been possible to increase the number of cases per operating session as a result of this policy?

We agree that intensive care is rarely required after carotid surgery. However, the authors do not define what they mean by intensive care. The standard definition of an intensive care unit is one that is able to provide 24h ventilatory report. A high dependency unit is all that is required for these patients and most can just as well be cared for on a vascular ward with a special care facility. However, we are unhappy with the author's contention that continuous monitoring is not required in the immediate postoperative period. It is perhaps fortunate that they did not have any untoward events in their series. Other authors have not been so lucky, as the high rate of untoward events after major vascular surgery has been well documented.

Further savings were claimed to be due to early postoperative discharge with reduction in the average hospital stay from 10 to 5 days. However, the authors do not explain why patients previously needed to be kept in hospital for 10 days. In the U.K. a 5-day stay would be regarded as conventional with many centres having now reduced their stay to 2 or 3 days. Fig. 1 also demonstrates that the postoperative length of stay had fallen to 2 days by 1992 – 3 years before their new protocol was introduced!

Our main criticism is of the 103 (27%) patients operated upon in 1995 for asymptomatic disease. In the introduction, the authors state that several large multicentre trials have identified the patients with carotid atherosclerosis who may benefit from carotid endarterectomy. None of these trials, including the Asymptomatic Carotid Atherosclerosis Study, have demonstrated an unequivocal benefit for asymptomatic patients. Not operating on these patients would have saved 301172 Ecu according to the papers estimate of 2924 Ecu per operation. Whilst addressing the possible cost reductions entailed in an operative procedure are important, considerably larger savings might be made by more attention to patient selection.

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