The second concern is the difference in the incidence of ischaemic heart disease between the groups. In addition, chronic ischaemia is usually present in all patients with coronary artery disease. If there was more myocardial ischaemia in one group compared with another, or if the group included more patients with valvular disease, this would affect the ischaemia-reperfusion injury. Patients who had a degree of postoperative myocardial infarction could also adversely affect the clinical results.

We believe that it would only be possible to demonstrate that i.v. vitamin E supplementation would improve the biochemical markers of myocardial injury and the haemodynamic parameters following cardiac surgery if diabetics were excluded from the study and more appropriate patients selected.

S. Canbaz
E. Duran
Editme, Turkey

Editor—Thank you for the opportunity to reply to Drs Canbaz and Duran. The authors state that many experimental and clinical studies suggest that vitamin E has a strong antioxidant capacity and improves myocardial reperfusion injury. As stated in our paper (Introduction), previous clinical studies with oral vitamin E are contradictory and have not confirmed the promising results of animal experiments. The papers they quote did not deal with patients, specifically not with perioperative cardiac surgical patients. We agree with Drs Canbaz and Duran that studies in diabetic rats support the hypothesis that diabetes may modify the response to injury.

Our aim was to investigate cardiac surgical patients as they are scheduled in our daily practice (median EuroSCORE > 6.5), to give ample amounts of parenteral vitamin E, and to study whether any biochemically and/or clinically measurable change in outcome was detectable. Based on our relatively small sample size and the above mentioned inclusion criterion, it is not possible to discuss any probable benefit of parenteral vitamin E supplementation for a subgroup of patients, for example non-diabetic coronary artery disease (CAD) patients, as suggested by the authors of the letter. The percentage of patients with CAD in our placebo group was almost twice as high as in the treatment group, and the incidence of Q-wave infarction on day 6 was five and two patients, respectively. Therefore, the incidence of these events was similar.

In addition, the distribution of patients between groups with slightly more CAD patients in the placebo group would have potentiated any theoretical vitamin E related treatment effect, but no difference was found.

A. Lassnigg
Vienna, Austria

Ischaemia-reperfusion studies and diabetes mellitus

Editor—We read with interest the article by Lassnigg and colleagues1 that evaluated the effects of i.v. vitamin E supplementation in cardiac surgery on oxidative stress. Their study shows that normalization of plasma vitamin E concentrations with parenteral vitamin E emulsion does not affect biochemical markers of myocardial injury and clinical outcome after cardiac surgery. In contrast, many experimental and clinical studies suggest that vitamin E has a strong antioxidant capacity and improves myocardial reperfusion injury.2

Our main concern about this study is the lack of the exclusion criteria in it. Both groups entered into the study include different numbers of diabetic patients. It is well-known that both the ischaemic injury and the oxidative stress following reperfusion injury are greater in the presence of diabetes mellitus.3,4 Diabetic patients face an increased risk of ischaemic events and organ damage from, for example, myocardial infarction and low output syndrome, and they may mount an inordinate response to ischaemia and reperfusion.5 Hyperglycaemia is a potent stimulus for endothelin-1 production, and it has been hypothesized that increased endothelin-1 in diabetes might represent an important mediator of endothelial dysfunction in patients with the disease.5 Leukocytes also play a central role in ischaemia-reperfusion injury.6 Diabetes induces an exaggerated inflammatory response to ischaemia reperfusion, manifest as a greater accumulation of adherent and emigrated leukocytes which express adhesion molecules, and a larger increase in albumin extravasation.7 It is generally accepted that diabetes increases ischaemia-reperfusion injury and has worse clinical and laboratory outcomes.

6 Tsujikawa A, Kiryu J, Nonaka A, et al. Leukocyte-endothelial cell...
interactions in diabetic retina after transient retinal ischemia. Am J Physiol Regul Integr Comp Physiol 2000; 279: 980–9


DOI: 10.1093/bja/aeg579