Reduction of Vein Graft Intimal Hyperplasia by ex vivo Treatment with Desferrioxamine Manganese

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We read with interest the paper by Hagen et al. [1] which implied that a single ex vivo treatment with desferrioxamine manganese reduces intimal hyperplasia in experimental vein grafts. This is an interesting observation but we feel that the design of this study severely limits its potential. The authors have chosen an experimental vein graft model in which a reversed segment of the external jugular vein is placed as an interposition bypass graft into the ipsilateral common carotid artery of rabbits. Unfortunately, no information is given regarding the flow rates or shear stresses which developed in these grafts after implantation. Changes in blood flow and shear stress have been shown to be important determinants of the complex process of intimal hyperplasia in both experimental prosthetic [2, 3] and vein grafts [4]. In the authors' study [1], it may be that the changes in intimal thickness they observed between their control and experimental grafts merely reflects the response of the vessel wall to different flow conditions. These concerns, and the fact that it has recently been shown that endothelial and smooth-muscle cell function in these experimental grafts do not correlate with the responses seen in human bypass grafts [5] raises serious doubts as to the relevance of this model to the clinical situation. As the authors point out, they have previously shown that the administration of aspirin and dipyridamole also reduces intimal hyperplasia in this model [6], but these agents have not been shown to be effective clinically [7]. We feel that it is important that all the factors which may contribute to the development of intimal hyperplasia (including hemodynamic changes in vein grafts) be controlled for before the claim can be made that a single intervention or treatment reduces this complex process.

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