remain in the study if medical therapy failed and the end point of angina was reached. Angina, unfortunately, is a subjective measure. Irrespective of how “objective” the investigators were in determining the success or failure of antianginal therapy, the use of a subjective end point may have inadvertently introduced bias into the trial. It is difficult to draw proper conclusions from such a trial when large crossover rates are allowed.

With respect to the European trial by Schoefield et al. (2), the number of sites with irreversible segments was adjusted for baseline, and for repeated within-patient, between-site measures. Therefore, to suggest that a “doubling” of fixed defects in the medical therapy group implies enhanced perfusion in the TMR group is inappropriate, especially because a subgroup analysis of the same TMR patients showed no improvement in myocardial perfusion with PET scanning (3).

To conclude, we believe that the reported benefits of TMR, even out to five years, may be related to the placebo effect. It is the most plausible mechanism of action, given the lack of concrete evidence to the contrary. A properly powered, blinded, sham-controlled surgical trial of TMR could certainly settle this issue. In the absence of such a trial, however, more studies using new perfusion imaging modalities must be conducted to elucidate the true value of this technique.

Mehrdad Saririan, MD
Mark Eisenberg, MD, MPH, FACC
Jewish General Hospital
McGill University
Divisions of Cardiology and Clinical Epidemiology
3455 Cote Ste Catherine Rd.
Suite A118
Montreal, Quebec H3T1E2
Canada
E-mail: marke@epid.jgh.mcgill.ca

REFERENCES

Mitochondrial Dysfunction in Heart Failure

I read with interest the report in the Journal by Scheubel et al. (1). Although their conclusion that a depression in the activity levels of the respiratory enzymes complex I activity in human failing myocardium is not due to disturbed mitochondrial gene expression is misguided. However, I do agree that the protective role of drug treatment against mitochondrial DNA damage remains to be proven.

Jose Marin-Garcia, MD
The Molecular Cardiology and Neuromuscular Institute
75 Raritan Avenue
Highland Park, New Jersey 08904
E-mail: tmci@att.net
doi:10.1016/S0735-1097(03)00494-1

REFERENCES

REPLY

We thank Dr. Marin-Garcia for the interest in our study (1). He is right in stating that our citation of his study (2) “indicating disturbed mitochondrial gene expression” is inaccurate. We regret such inaccuracies, which must have occurred during several reformulations of our text. The reasons for the reduced activities in complex III and complex V in the pacing-induced cardiac failure indicate disturbed mitochondrial gene expression is misguided. However, I do agree that the protective role of drug treatment against mitochondrial DNA damage remains to be proven.

Robert J. Scheubel, MD
Department of Cardiothoracic Surgery
Martin-Luther-University Halle-Wittenberg
Ernst-Grube-Str. 40
D-06097 Halle (Saale), Germany
E-mail: robert.scheubel@medizin.uni-halle.de
doi:10.1016/S0735-1097(03)00488-1

REFERENCES