

## LETTERS TO THE EDITOR

### Abnormal Cellularity in Asymptomatic Relatives of Patients With Idiopathic Dilated Cardiomyopathy

I read with great interest the study by Mahon et al. (1) demonstrating abnormal myocardial pathology in apparently asymptomatic relatives of patients with idiopathic dilated cardiomyopathy (DCM). This finding, of course, has great clinical import. The investigators state that their study “provides, for the first time, to the best of our knowledge, direct pathologic confirmation that relatives with LVE (left ventricular enlargement) have myocardial disease . . .”. Several years ago, histopathologic changes were reported in a smaller number of asymptomatic first-degree relatives of patients with DCM (2). That study claimed, more correctly, to have shown “for the first time that the myocardium in such relatives demonstrates abnormal cellularity”. The study was published in a nonobscure, English-language journal and, most surprisingly, Dr. Mahon was one of the co-authors!

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## REFERENCES

1. Mahon NG, Madden BP, Caforio ALP, et al. Immunohistologic evidence of myocardial disease in apparently healthy relatives of patients with dilated cardiomyopathy. *J Am Coll Cardiol* 2002;39:455–62.
2. McKenna CJ, Sugrue DD, Kwon HM, et al. Histopathologic changes in asymptomatic relatives of patients with idiopathic dilated cardiomyopathy. *Am J Cardiol* 1999;83:281–3.

## REPLY

The study to which Dr. McKenna refers (1), focusing principally on the evaluation of two markers of apoptosis and a single, unquantified marker of inflammation, was considered to be hypothesis-generating but by no means confirmatory. It was recognized to have been limited not only by an incomplete and insensitive evaluation of myocardial inflammation, but more importantly by very small numbers ( $n = 5$ ) and the lack of a control group. Accordingly, the investigators themselves quite deliberately and correctly concluded that “the observations described must be viewed as provocative but preliminary.”

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1. McKenna CJ, Sugrue DD, Kwon HM, et al. Histopathologic changes in asymptomatic relatives of patients with idiopathic dilated cardiomyopathy. *Am J Cardiol* 1999;83:281–3.

### Efficacy of Biventricular Pacing in Congestive Heart Failure

Linde et al. (1) reported in the *Journal* that biventricular pacing significantly improves clinical parameters in patients with congestive heart failure. In their study, the 6-min walking distance, New York Heart Association class, and quality of life improved with biventricular pacing. Objective measures of function, including the maximal O<sub>2</sub> consumption, left ventricular end-diastolic diameter, left ventricular end-systolic diameter, and left ventricular ejection fraction did not improve significantly. The investigators were only able to demonstrate subjective improvement but not any objective improvement with the biventricular pacing. Subjective improvement was previously shown by Linde et al. (2) to improve with sham pacing, suggesting that a significant placebo effect is associated with the insertion of a pacemaker. It is therefore possible that the subjective improvement in this group was a result of the placebo effect of pacemaker implantation rather than of the mode of pacing utilized.

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1. Linde C, Leclercq C, Rex S, et al. Long-term benefits of biventricular pacing in congestive heart failure: results from the MULTISite STimulation In Cardiomyopathy (MUSTIC) study. *J Am Coll Cardiol* 2002;40:111–8.
2. Linde C, Gadler F, Kappenberger L, et al. Placebo effect of pacemaker implantation in obstructive hypertrophic cardiomyopathy: PIC study group. *Am J Cardiol* 1999;83:903–7.

## REPLY

In their letter, Moshenyat and colleagues suggest that the one-year benefits by biventricular pacing in the MUSTIC trial reflect the placebo effect by pacemaker implantation because no significant one-year improvements in objective parameters were found (1). The MUSTIC trial was designed as a randomized single-blind crossover comparison between biventricular pacing and inactive pacing (sinus rhythm) or right ventricular pacing (atrial fibrillation) assuming a 10% improvement in 6-min walk and a 10% reduction in Minnesota score by three months of active (biventricular) treatment (2). The crossover results indicate a 20% improvement in 6-min walk and a 30% improvement in quality of life. Subsequent long-term follow-up indicates that these benefits were sustained in magnitude over a one-