Letter by Ferrari et al Regarding Article, “Long-Term Arrhythmia-Free Survival in Patients With Severe Left Ventricular Dysfunction and No Inducible Ventricular Tachycardia After Myocardial Infarction”

To the Editor:

We read with great interest the article by Zaman et al1 on the role of programmed ventricular stimulation (PVS) in post–myocardial infarction (MI) patients with depressed left ventricular ejection fraction. In our opinion the article has important limitations. First, left ventricular ejection fraction was assessed only 4 days after MI and therefore it is possible that the subsequent left ventricular function recovery contributed to the favorable outcome observed in the study population. Secondly, the significant differences in baseline characteristics between PVS-positive and PVS-negative patients could per se justify the difference in clinical outcome, irrespective of the results of PVS. Finally, implantable cardioverter-defibrillator programming with a ventricular tachycardia zone at a low heart rate (167 bpm) has likely overestimated the arrhythmic risk in PVS-positive patients. Despite these limitations, the article has the great merit to renew the interest on the predictive role of PVS.2 We previously published our own experience3 on 106 consecutive late post-MI patients with left ventricular ejection fraction <40%, who underwent PVS and were followed for 24 months. The primary end point of the study, the combination of arrhythmic death and ventricular fibrillation requiring implantable cardioverter-defibrillator shock, occurred in 24% of PVS-positive patients versus 3.5% of PVS-negative patients (P=0.002). These figures are very similar to the 2-year arrhythmic event rates reported by Zaman et al, 24% and 1%, respectively.

Although Zaman et al’s and our own article focused on different study populations (early versus healed post-MI patients), both led to the conclusion that PVS might still have a role, especially because of the similar and high negative predictive value of this test (96% in our and 97.5% in Zaman et al’s study). Programmed ventricular stimulation was considered to be futile mostly on the basis of the Multicenter Automatic Defibrillator Implantation Trial (MADIT) II definition of positive PVS,5 significantly different from that used by the same authors in MADIT-I.

At variance with the MADIT-II substudy, both Zaman et al’s and our study enrolled consecutive patients and used a very careful methodology for PVS, including repetition of PVS if the initial induction was negative1 and repetition of each protocol that induced nonsustained ventricular tachycardia lasting ≥10 beats,1 respectively. This attention has likely contributed to the high negative predictive value.

Zaman et al’s and our study are unlikely to be enough to revitalize the use of PVS in the risk stratification of post-MI patients with left ventricular dysfunction. However, their combined data warrant the planning of a multicenter prospective trial assessing the predictive value of PVS in this setting. We appreciate that it will be impossible to obtain private funding for this but it should be the role of the scientific community to unite forces for such an endeavor. As Dr Buxton reminds us, it is hard to undo a lie.2 If the lack of predictive role of PVS is indeed not true, we should all feel the responsibility to undo this lie, even if it will be hard to do so.

Disclosures

Dr De Ferrari reports receiving fees as a Member of the Steering Committee of a trial sponsored by Boston Scientific. Dr Rordorf reports receiving speaker fees from Medtronic and St. Jude Medical. Dr Landolina has speakers’ bureau appointments and an advisory board relationship with Medtronic and other device companies.

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References

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