The identification of predictors of sudden death after acute myocardial infarction and the subsequent demonstration that implantation of a cardioverter defibrillator (ICD) reduces total mortality in this high risk population has stimulated interest in the clinical utility and economic feasibility of primary prevention of sudden death. Thus, the study by González et al is highly relevant. The question whether primary angioplasty for acute myocardial infarction reduces or influences sudden death predictors and the number of ICD implants has significant clinical implications.

Although it is known that primary angioplasty involves less deterioration in left ventricular function and longer short- and medium-term survival than other treatments, the effect of early reperfusion on the appearance of arrhythmogenic substrates associated with life threatening arrhythmia is unknown. Given that reperfusion promotes the viability of some muscle fibers in scar tissue, it could facilitate the development of re-entry circuits. Obviously, the greater the number of viable fibers, the greater the probability that such circuits will appear. However, preserving ventricular function could prevent the onset of fibrosis and diminish ventricular wall tension and ventricular remodeling. Thus, it is reasonable to question whether early reperfusion would favor the appearance of arrhythmogenic substrates, or prevent their development. Do the authors address this issue? The answer is «No»; they do not address this directly. This would have required an electrophysiological study of every patient to assess whether sustained tachycardia could be induced—or the use of noninvasive risk markers, eg. by analyzing for the presence of late potentials using signal averaged electrocardiography. This non-invasive technique would have provided valuable information on the presence of slow conduction zones related to myocardial infarction. The data provided by the authors do not clarify the effect of angioplasty on the arrhythmogenic substrate: a) the incidence of non-sustained ventricular tachycardias is not significantly lower than that found in historical controls in the literature, and b) the data based on the inducibility of sustained ventricular tachycardias are not significant, mainly due to the small number of patients who underwent ventricular stimulation. Thus, we can infer that the effect of primary angioplasty on ICD implantation rates in patients with acute myocardial infarction is mainly due to its impact on ventricular function, rather than to possible effects on the arrhythmogenic substrate.

The study by González et al concludes that a defibrillator is indicated in only 5% of the patients who undergo primary angioplasty. However, although this implant rate is apparently lower than that found in the literature in similar patients, the lack of comparisons with a control group of patients who undergo reperfusion by fibrinolysis or who do not undergo reperfusion makes it difficult to draw definite conclusions. Despite these limitations, the authors have provided extremely valuable information. The percentage of patients with acute myocardial infarction treated by primary angioplasty who require an implantable defibrillator is considerably less than expected and affordable by the Spanish national health system. Systematic evaluation following myocardial infarction should aim to identify those patients who will benefit from a defibrillator.

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