


This letter was shown to the authors, who reply as follows:

Sir,

As Dr Cripps and his colleagues note (and as we emphasised in our article) the results of our study are applicable only to clinically stable patients with uncomplicated myocardial infarctions and the finding ought not be extrapolated to patients with complicated infarctions where the risk of future arrhythmic events is higher and the predictive accuracy of programmed ventricular stimulation may be better. Their study1 and others including our own,2–4 support the clinical use of programmed ventricular stimulation in such patients.

Until recently there were few data on the clinical significance of inducible ventricular fibrillation in survivors of acute myocardial infarction. The results of our study and those of Denniss et al3 clearly show that inducible ventricular fibrillation is a non-specific response of little clinical significance in these patients. Therefore we agree that this arrhythmia should not be included as a positive end point in existing or future prospective trials of programmed stimulation in survivors of recent myocardial infarction.

In our article we commented on the limitations imposed by the empirical antiarrhythmic treatment started in our first 10 patients with inducible sustained ventricular arrhythmias. The minimum length of follow up was at least 12 months in all but five patients, who were lost to follow up between four and eight months after the index infarction.

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References


Timing of treatment with oxygen radical scavengers and its influence on reperfusion injury

Sir,

In their editorial on oxygen radicals and myocardial damage (1989;61:4–8), Burrell and Blake pointed out that proof that there is specific, reperfusion-mediated damage depends on the administration of oxygen radical scavengers at the moment of reperfusion rather than before the onset of ischaemia. This is a valid point, but the reader may be left with the impression that the hypothesis of a specific reflow injury remains to be tested because the editorial does not refer to the results of several pertinent published reports.

In the classic study by Jolly et al, administration of superoxide dismutase and catalase towards the end of the ischaemic period proved as effective in reducing infarct size as pretreatment.1 Furthermore, in other studies administration of "anti-free radical" agents at the moment of reflow, after the ischaemic episode,
The authors reply

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