

Correspondence

Prognostic significance of programmed ventricular stimulation in survivors of acute myocardial infarction

Sir,

We were most interested in the paper by Bhandari *et al* (1989;61:410-6) because we are engaged in a similar study. There are several points upon which we would like to comment.

Firstly, 10 out of 33 patients with inducible sustained arrhythmias were treated with potent antiarrhythmic agents. Though the drugs were chosen empirically and later stopped, the fact remains that several patients may have been protected from arrhythmic events during the high risk early post-infarction phase. Even if outcome was influenced in only a small number of patients, this could have significantly influenced the overall results, because there were only a small number of end points.

Secondly, as Bhandari *et al* themselves confirmed, inducible ventricular fibrillation is known to be a non-specific response to ventricular stimulation¹ and should not be included as a positive response in the overall analysis. Inducible ventricular tachycardia was, in fact, present in all but one of their patients who died suddenly. The predictive accuracy of inducible ventricular tachycardia is also known to be greater if only uniform ventricular tachycardia is regarded as important—particularly if an upper rate limit (for example < 250 beats per minute) is included.²

Thirdly, it should be further emphasised that Bhandari *et al*'s study included only patients who were at a relatively low risk of arrhythmic events (Killip class I or II). Most patients with documented spontaneous ventricular arrhythmias after infarction have significantly impaired left ventricular function. Arrhythmias may occur either as a primary electrical phenomenon or as the result of new ischaemic events; perhaps the latter are a more likely cause in those with small infarcts.

Finally, follow up was as short as four months in some patients, though sudden deaths occurred as late as 26 months. The predictive accuracy of the test may increase with longer follow up.

Our preliminary results from 75 unselected

patients including those with clinical evidence of major infarction tested with up to three extrastimuli³ show a higher sensitivity (6/6) and positive predictive accuracy (6/8) than in the study of Bhandari *et al*, in line with previous studies that included high risk patients.^{4,5} We found a high incidence (29%, 22 patients) of sustained inducible arrhythmias, often of a non-specific multiform type requiring DC cardioversion, which makes the test invasive and therefore unattractive.

We agree that programmed ventricular stimulation should not be used as a test in the routine assessment of patients after uncomplicated infarction. It may be useful, however, in the identification and selection of treatment in those judged on clinical and non-invasive grounds to be at high risk of death from arrhythmia, and of course in patients with late spontaneous sustained ventricular tachycardia, in whom empirical treatment may be ineffective and is possibly harmful.

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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 al*³ 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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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,