Lignocaine therapy in myocardial infarction

Sir,

The study of Pentecost *et al.*¹ on lignocaine therapy in the management of myocardial infarction may easily lead to the general conclusion that lignocaine is of more harm than benefit to the patient. In our opinion this conclusion cannot be drawn from the data presented.

Firstly, in this study one has to distinguish between ventricular fibrillation complicating pump failure and primary ventricular fibrillation. It is known from experience, that lignocaine is less effective in preventing the former than the latter. It is not clear from the description how many patients had pump failure, but judged from the stated hospital mortality (50%), it was probably a considerable number.

Secondly, the dosage of lignocaine in the first and second period of investigation was clearly below that proven to be effective.² Withholding such an inadequate regimen in the later years of study therefore cannot influence the incidence of ventricular fibrillation and thus only proves that inadequate treatment with lignocaine is no better than no treatment at all. The observed incidence of ventricular fibrillation (8 to 9%) in all three study periods is equal to the expected incidence in untreated patients in a coronary care unit.³

Thirdly, patients with compromised hepatic clearance remain suitable for treatment. After a loading period, gradual tapering down of the doses creates a plateau level, thus avoiding blood levels at which major toxicity occurs. When in doubt hepatic clearance can be predicted by indocyanine green clearance⁴ and plasma levels can be determined quickly by enzyme immuno-assay.⁵

There is therefore no reason to withhold lignocaine treatment in any patient at risk for fear of major toxicity.

Pentecost's point that in ventricular fibrillation complicating pump failure the final outcome is determined by the extent of myocardial muscle loss and not by the initial success of defibrillation is well taken. In situations of suboptimal coronary care, however, or in the prehospital phase of acute myocardial infarction, adequate lignocaine treatment to prevent primary

ventricular fibrillation may prove effective, safe, and indeed lifesaving.

Rudolph W Koster and Donald C Harrison,
Department of Cardiology, Academisch Medisch Centrum, University of Amsterdam, Amsterdam, Netherlands; and Department of Cardiology, Stanford University, Stanford, California 94305, USA.

References

- 1 Pentecost BL, De Giovanni JV, Lamb P, Cadigan PJ, Evemy KL, Flint EJ. Reappraisal of lignocaine therapy in management of myocardial infarction. *Br Heart J* 1981; 45: 42-7.
- 2 Lie KI, Wellens HJ, van Capelle FJ, Durrer D. Lidocaine in the prevention of primary ventricular fibrillation. *N Engl J Med* 1974; 291: 1324-6.
- 3 Meltzer LE, Kitchell JB. The incidence of arrhythmias associated with acute myocardial infarction. *Prog Cardiovasc Dis* 1966; 9: 50-63.
- 4 Zito RA, Reid PR. Lidocaine kinetics predicted by indocyanine green clearance. *N Engl J Med* 1978; 298: 1160-3.
- 5 Haywood LJ, Claiborne K, Walberg C. Lidocaine: clinical use of rapid method for determining serum levels (abstract). *Am J Cardiol* 1979; 43: 360.

This letter was shown to Dr Pentecost who replies as follows.

Sir,

In our study we were concerned with the use of lignocaine to suppress ventricular extrasystolic activity, not with routine prophylaxis. The former practice is still followed in many hospitals where the suppression of "warning arrhythmias" is regarded as effective in the prevention of ventricular fibrillation. Our data suggest that this belief may not be justified. The important consideration concerning lignocaine dosage in this context is surely that the dose used was sufficient to suppress extrasystolic activity. In the review we did not distinguish between primary and

secondary ventricular fibrillation because we were reporting what happened in routine clinical practice rather than describing a formal trial conducted within a highly selected group of patients. The distinction between primary and secondary ventricular fibrillation is, moreover, somewhat artificial.

Your correspondents appear to be advocating routine lignocaine infusion for all patients with myocardial infarction. Even if their reassurances concerning the use of this drug are justified, and relatively few hospitals will have access to indocyanine green clearance and plasma lignocaine levels, there is

no available evidence of benefit upon which to base such a therapeutic policy. Even the trial which appears to be the spring-board for the enthusiast's approach to lignocaine prophylaxis based its message upon less than 30% of all patients presenting with suspected myocardial infarction—that is the population studied was highly selected. The conclusions cannot be extrapolated to all patients.

B L Pentecost,
The General Hospital, Steelhouse Lane,
Birmingham B4 6NH.