Correspondence

Br Heart J 1982; 47: 186–7

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 pre-

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 prevent-
ing 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 inade-
quate regimen in the later years of study therefore
cannot influence the incidence of ventricular fibril-
ation 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 clear-
ance 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 clear-
ance 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 deter-
bined 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.

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cal use of rapid method for determining serum levels

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

Sir,

In our study we were concerned with the use of li-
nocaine 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
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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.

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