LETTERS TO THE EDITOR

The British Heart Journal welcomes letters commenting on papers that it has published within the past six months.

All letters must be typed with double spacing and signed by all authors.

No letter should be more than 600 words.

In general, no letter should contain more than six references (also typed with double spacing).

We studied 34 patients in sinus rhythm during dipyridamole stress and compared age-matched coronary disease patients who developed ischaemia, detected by echocardiographic and electrocardiographic criteria, with those who did not.1 Transmural flow was uninterpretable in three (8%) patients owing to fusion of the early and atrial waves. Doppler filling variables were insensitive in identifying patients with ischaemia induction even when this was severe. Our data strongly suggested that changes in ventricular loading and heart rate induced by dipyridamole masked the expected effects of myocardial ischaemia on the filling profile. Other workers have reached similar conclusions2 indicating that this approach cannot supplement wall motion analysis for the evaluation of patients with coronary artery disease. Despite reservations on interpretability, the data from Shahi et al are consistent with these observations and highlight the ease with which spurious differences between groups may emerge when numerous confounding variables are operative and group size is small.

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Two patients in the control group developed a single filling velocity in the study period but for only a maximum of two readings. In these patients the filling velocity of the early filling wave just before merging of the waves was taken to represent the filling velocity.

To allow for baseline temporal variability the mean value of five one minute recordings was taken to represent baseline value.

Regional wall motion abnormalities were not specifically assessed in our study but their importance as a more sensitive marker of ischaemia was discussed in the discussion section (p 270).

Reference 5 quoted by Dr Mazeika and Dr Oakley was cited in our paper together with its limitations. We cannot comment on reference 4 at present.

Although it was not possible for technical reasons to assess the presence of mitral regurgitation during myocardial ischaemia in our study, Dr Mazeika and Dr Oakley are correct to state that this may have affected our results. Ischaemia-induced mitral regurgitation would increase left atrial pressure and therefore decrease the isovolumic relaxation period and subsequently increase the transmural pressure gradient with a resulting increase in the early filling velocity and a possible decrease in the atrial filling velocity. It is therefore possible that this may be the reason for the difference in left ventricular filling velocities in groups 3A and 3B. Both at the end of the abstract and in the discussion section we stated that our observations could be attributable either to the degree of myocardial ischaemia or to different haemodynamic changes occurring during myocardial ischaemia.

Once again we would like to emphasise that the study was designed to observe the left ventricular filling characteristics during myocardial ischaemia and not to suggest that these changes in filling velocities could predict myocardial ischaemia in an individual patient (last paragraph p 269).

I hope these comments will help clarify the points made by Dr Mazeika and Dr Oakley.

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Circadian variation in the frequency of onset of chest pain in acute myocardial infarction

Sir,—Dr Thompson and colleagues make a convincing case in Leicester for a midnight peak in onset of myocardial infarction in addition to the well-known one at 8 am (British Heart Journal 1991; 65:177-8). They try to persuade us that meta-analysis of other studies would confirm this. Unfortunately they have omitted from their references perhaps the largest study of all. This was the collected data from the World Health Organisation Regional Office for Europe Heart Attack Registers of the 1970s covering some 10 000 events. The pooled data from these show a clear peak between 8 and 10 am on weekdays which is much sharper at 10 am on Saturdays and Sundays. There is also a bulge around 4-5 pm but no overall peak around midnight. The exception interestingly is a possible such peak on Saturday.

If the Leicester peak is not just a chance finding, and it is found in some cities but not others and may be related to Saturdays, one
Left ventricular filling dynamics during dipyridamole induced myocardial ischaemia.

P Mazeika and C M Oakley

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