Editorial

Heart rate/ST slope

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In 1979 Berenyi *et al* first proposed that the rate of change of ST segment with respect to heart rate (the HR/ST slope) might be an indicator of the amount of prejudiced myocardium as judged by the coronary arteriogram.¹ This suggestion has been extensively studied particularly by the Department of Cardiovascular Studies at The University of Leeds. In 1982 these workers reported an excellent correlation between the slope and the number of main vessels with at least 75% stenosis, with no overlap of the slope ranges for one, two, and three vessel disease.²

Because heart rate is an important determinant of myocardial oxygen consumption it seems logical to propose that a relation between the degree of ischaemia and the heart rate could be used to assess the severity of coronary disease. A direct relation between the maximum ST depression in a single electrocardiographic lead and the degree of ischaemia is more difficult to accept since it assumes that all areas of the left ventricular myocardium are equally "accessible" to one of the 13 leads monitored and that the same amount of ischaemic myocardium anywhere in the heart will produce the same ST depression.

The arterial supply to the left ventricle is variable and equivalent stenoses in two arteries do not necessarily produce the same amount of ischaemic myocardium. The most obvious example of this is the extreme variability in the size and number of branches of the right coronary artery. Coronary calibre also varies from time to time and it would be surprising if there was a given level of critical stenosis (such as 75%). There are important additional problems posed by the known inaccuracies of visual

interpretation of angiograms.

Nevertheless, had the findings been reproducible the test would have been extremely valuable for determining which patients should be referred for angiography. Other workers, however, have not been able to reproduce the results. Some found no correlation at all^{3,4} and Okin *et al* reported a limited relation that identified patients with triple vessel disease.⁵

Kligfield *et al* recently summarised their own data and reviewed the arguments. They concluded that the heart rate/ST slope is better than standard electrocardiographic criteria for both the detection of coronary disease and the determination of the presence of triple vessel disease.⁶ They acknowledged that highly selected populations of patients with known coronary disease or those with "normal" hearts were investigated. They did not provide data to demonstrate that the test is able to select prospectively patients with important coronary disease from a suspect population. They criticised other studies that did not find a good correlation between the slope and the extent of coronary disease,⁴ generally because patients in the populations studied included a variable number who had had a previous myocardial infarction. The early reports from Leeds, however, did not mention whether or not the patients had had previous infarction² and since infarction was not a reason for exclusion there was presumably the usual admixture of patients.

On page 512 of this issue of the *British Heart Journal* the Leeds group report a group of patients with ischaemic heart disease. In two thirds of them the heart rate/ST slope failed to predict the severity of the demonstrated coronary artery disease. The Leeds workers suggest that this is because they were tested early after acute myocardial infarction. They say the underestimates were the result of left ventricular scarring producing less ischaemic myo-

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cardium for a given stenosis. The overestimates were attributed to increased left ventricular volume, as judged by the cardiothoracic ratio, causing "more ischaemia" than would otherwise have occurred.

It may well be that the early response to exercise after infarction is modified by myocardial factors. These studies were usually performed 10 days to three weeks after the infarction and used submaximal testing.^{7,8} Serial exercise testing after infarction has demonstrated that the findings change and for instance "ischaemic" ST depression at three weeks has a different prognostic significance than that occurring 11 weeks after infarction.⁹ The patients reported were studied for a mean of >34 days after the acute myocardial infarction, which more closely resemble the "late" tests reported by others. It seems more likely therefore that these observations, which are in line with those of other workers, are the result of the expected variability of a biological system.

The controversy therefore remains. It is agreed by all, however, that the original high hopes that a non-invasive method of determining the exact extent of coronary disease had been found have not been realised. The mere fact that reliable investigations have been unable to reproduce the results casts considerable doubt on the value of the technique. Much emphasis has been laid on the need to follow the test protocol exactly but despite doing this at least one group still failed to confirm the findings.⁴ The test cannot therefore be recommended at this stage for general use because there are doubts about its reliability and practical difficulties with both its performance and analysis.

References

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