LETTERS TO THE EDITOR

Scope
Heart welcomes letters commenting on papers published in the journal in the previous six months. Topics not related to papers published earlier in the journal may be introduced as a letter: letters reporting original data may be sent for peer review.

Presentation
Letters should be:
- not more than 600 words and six references in length
- typed in double spacing (fax copies and paper copy only)
- signed by all authors
They may contain short tables or a small figure. Please send a copy of your letter on disk. Full instructions to authors appear in the January 1999 issue of Heart (page 104).

Increased dispersion of ventricular repolarisation during stress test induced ischaemia

Sir,—We read with interest O’Sullivan et al’s article “Abnormal ventricular activation and repolarisation during dobutamine stress echocardiography in coronary artery disease” in the January 1999 issue of Heart. They may contain short tables or a small figure. Please send a copy of your letter on disk. Full instructions to authors appear in the January 1999 issue of Heart (page 104).

We found that QT dispersion at rest was proportionally higher in the CAD group (table 1).

Our understanding from these studies under ischaemic conditions, is that non-homogeneity of repolarisation, and therefore QT dispersion, would be expected to increase further. O’Sullivan et al found mean QT dispersion values in patients with CAD and healthy subjects of 98 ms and 82 ms, respectively. Data from many studies suggest that QT dispersion at rest varies from 30–60 ms in healthy subjects and 60–80 ms in patients with CAD. Therefore, O’Sullivan et al’s findings in conflict with many other studies and further work is required to investigate the effects of stress test induced ischaemia on QT dispersion.

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This letter was shown to the authors, who reply as follows:

We thank Dr Aytemir and colleagues for their comments. In fact, effects on QT dispersion formed only a minor part of our study, and we note that there are differences between our results and those subsequently appearing in the literature. We believe that these differences are methodological. As stated, our aim to determine all ECG intervals objectively using previously developed algorithm on digitised records. In all three studies quoted by Aytemir et al, measurements were made manually from the ECG as printed in analogue form, and leads were arbitrarily excluded from the analysis. As we included data from all 12 leads in all subjects studied, it is not surprising that we obtained larger values for QT dispersion.

Table 1 QT dispersion (QTD) and change of QTD during exercise in patients with CAD and healthy controls

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<th>CAD</th>
<th>Controls (n = 40)</th>
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<tr>
<td></td>
<td>Rest</td>
<td>(n = 58)</td>
</tr>
<tr>
<td>RR interval</td>
<td>852 (72)</td>
<td>870 (42)</td>
</tr>
<tr>
<td>QTD</td>
<td>33 (8)</td>
<td>52 (7)</td>
</tr>
<tr>
<td>Exercise</td>
<td>430 (65)</td>
<td>438 (52)</td>
</tr>
<tr>
<td>RR interval</td>
<td>32 (10)</td>
<td>73 (16)</td>
</tr>
<tr>
<td>QTD Change in QTD</td>
<td>2.0 (2)</td>
<td>4.0 (5)</td>
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Values are mean (SD).

While QT dispersion measurements appear to have shown some promise in predicting ventricular tachycardia or mortality after a previous myocardial infarction, this is not a uniform finding. The use of QT dispersion is based on the idea that spatial differences in depolarisation within the heart translate into differences between ECG leads. Theoretical backing for this idea is lacking, and convincing evidence that apparent QT dispersion is anything other than the instantaneous electrical vector being perpendicular to the lead in question has not been produced. While the possibility of dispersion of depolarisation within the myocardium remains an important one, we concur with recent expressed opinion that methods in current use based on QT dispersion are not reliable enough to detect it. Nevertheless, if it were possible to demonstrate a correlation between dispersion of mechanical activity, which can readily be detected by simple non-invasive methods, and QT dispersion, this might go some way towards validating the electrocardiographic approach. We were unable to do so.


Ten year follow up of patients referred for CABG from a single DGH

Sir,—Bathgate and Irving have wisely pointed out that the 10 year overall clinical results of coronary bypass can be disappointing. However, patients with severe class IV angina experienced pronounced symptom relief, allowing return to work in 91 of 100 consecutive bypassed cases, and an apparent improvement in survival with 85% alive a decade later. It seems that bypass is more beneficial in this group of more severely symptomatic patients, especially in those with advanced triple vessel or left main fixed disease when left ventricular function is impaired. Medical management alone generally will serve for those with milder symptoms and with less pronounced one, two or three vessel disease, while angioplasty is likely to be helpful in resistant patients.

W R ROGERS
Division of Cardiology, Good Samaritan Hospital, Portland, OR 97210, USA


This letter was shown to the authors, who reply as follows:

We agree with Dr Rogers that the greatest benefit of coronary bypass surgery is in those whose angina is particularly severe. However, our long term follow up continues to indicate that no patient with impaired left ventricular function survived even five years. This is likely to be related to the different population studied. When advising patients about bypass surgery, it is important to use the results that are most pertinent rather than extrapolating from clinical trial data.


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