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".¹ In that study patients with coronary artery disease (CAD) had an increased QTc interval but not QT dispersion during positive dobutamine exercise stress testing. QT interval is a measure of the duration of ventricular repolarisation,² and is sensitive to myocardial ischaemia. Therefore, the authors suggest that there are repolarisation abnormalities in patients with CAD during positive dobutamine stress testing. The interlead variation of QT interval in the 12 lead ECG referred to as QT dispersion reflects heterogeneity in the duration of myocardial repolarisation.³ O'Sullivan *et al* showed that QT dispersion was not heart rate dependent and did not change with increasing heart rate in control subjects. They found that QT dispersion decreased, but not significantly, during positive dobutamine stress testing in patients with CAD; they did not discuss QT dispersion as a marker of repolarisation abnormalities.

In contrast to O'Sullivan *et al*, many investigators have found increased QT dispersion during exercise induced ischaemia in patients with CAD.³⁻⁵ In one study, myocardial ischaemia induced by incremental atrial pacing caused an acute increase in QT dispersion.³ Furthermore, increased QT dispersion was found during positive stress testing in patients with CAD.^{4,5} We also performed a study related to QT dispersion during positive exercise test in patients with CAD. Our study group comprised 58 patients with CAD and 40 healthy volunteers. We found that QT dispersion at rest was profoundly increased in the CAD group compared with controls, and the percentage change in QT dispersion with exercise was significantly 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.³ Therefore, O'Sullivan *et al*'s findings are in conflict with most other studies and further work is required to investigate the effects of stress test induced ischaemia on QT dispersion.

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- 1 O'Sullivan CA, Henein MY, Sutton R, *et al*. Abnormal ventricular activation and repolarisation during dobutamine stress echocardiography in coronary artery disease. *Heart* 1998;79:468–73.
- 2 Kautzner J, Malik M. QT interval dispersion and its clinical utility. *PACE* 1997;20:2625–40.
- 3 Sporton SC, Taggart P, Sutton PM, *et al*. Acute ischaemia: a dynamic influence on QT dispersion. *Lancet* 1997;349:306–9.
- 4 Stoltzney LN, Pai RG. Value of QT dispersion in the interpretation of exercise stress test in women. *Circulation* 1997;96:906–10.
- 5 Roukema G, Singh JP, Meijis M, *et al*. Effect of exercise induced ischemia on QT interval dispersion. *Am Heart J* 1998;135:88–92.

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 we stated, it was our aim to determine all ECG intervals objectively using previously developed algorithms 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

	Controls (n = 40)	CAD patients (n = 58)	p value
Rest			
RR interval	852 (72)	870 (42)	0.08
QTD	33 (8)	52 (7)	0.001
Exercise			
RR interval	430 (65)	438 (52)	0.4
QTD	32 (10)	73 (16)	0.001
Change in QTD (%)	2.9 (2)	40 (5)	0.001

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.¹ 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 recently expressed opinion² 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.

- 1 Zabel M, Klingenhoben T, Franz MR, *et al*. Assessment of QT dispersion for prediction of mortality or arrhythmic events after myocardial infarction. *Circulation* 1998;97:2543–50.
- 2 Coumel P, Maison-Blanche P, Badilini F. Dispersion of ventricular repolarisation. Reality? Illusion? Significance? *Circulation* 1998;97:2491–3.

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.¹ 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 88% alive a decade later.²

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.

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- 1 Bathgate AJ, Irving JB. Ten year follow up of patients referred for coronary artery bypass grafting from a single district general hospital. *Heart* 1997;78:584–6.
- 2 Rogers WR, Wysham DN. Coronary bypass for acute rest angina; 10 year follow up. *Br Heart J* 1982;47:365–8.

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 indicated 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.