Comparison of 12-lead and computer-analysed 3-orthogonal-lead electrocardiogram in coronary artery disease

Sir,

The data presented by Murray et al. (1976, 38, 773) do not in my opinion show that the 3-lead-orthogonal electrocardiogram performed more favourably than the 12-lead system. By using formulae recommended by Thorner and Remein (1963) it can be shown that the differences in sensitivity and specificity proportions are not statistically significant. However, I suggest that, in any case, the calculation of these values was based on data from biased samples. The standard of comparison of the 2-leading systems used by Murray et al. was the coronary arteriogram, carried out on 102 patients, 77 of whom were categorised as 'ischaemic' and 25 as 'non-ischaemic'. Both the 12- and 3-lead electrocardiograms classified the 102 patients into 4 categories, 'normal', 'ischaemic', 'left ventricular hypertrophy' and 'left bundle-branch block'. Bias was introduced by excluding from the calculations of sensitivity, specificity, and index of merit, the 10 and 13 patients, respectively, diagnosed by the 12- and 3-lead systems as 'left ventricular hypertrophy' and 'left bundle-branch block'. If these are included, the denominators for the calculation of sensitivity and specificity for both systems are 77 and 25, respectively, the numerators remaining as before. The following results are obtained (Table).

The differences in sensitivity and specificity values are, once more, not statistically significant. The only conclusion, in my opinion, which can be drawn from the indices of merit, which are equal at 0·34 on a possible scale of 0 to 1, is that the performance of both systems in the detection of coronary artery disease is equally poor.

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This letter was shown to Dr. Murray and his co-authors who reply as follows.

Sir,

Thank you for the opportunity of replying to the letter from Dr. Rosamond Gruer concerning our article on 12-lead and computer analysed 3-orthogonal-lead electrocardiograms in coronary artery disease.

We think that Dr. Gruer is wrong in suggesting that our data on sensitivity and specificity are biased. The interpretation of T wave abnormalities on an electrocardiogram can be difficult since they may be the result of a number of factors—and not simply myocardial ischaemia. Classical teaching separates T wave abnormalities into two categories—primary and secondary. Primary T wave changes are the result of repolarisation abnormalities which may be caused by myocardial ischaemia, electrolyte imbalance, etc. Secondary T wave changes are a consequence of some other cardiac abnormality such as left ventricular hypertrophy or left bundle-branch block. The difficulty is, however, that some T wave abnormalities may be a combination of the two. In our analysis we thought it advisable to separate the two classes of T wave abnormalities as far as possible and our paper was essentially concerned with the diagnostic accuracy of interpere-

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tation of primary T wave abnormalities thought to be caused by myocardial ischaemia.

We did examine the possibility that left ventricular hypertrophy might be accepted as an index of myocardial ischaemia and we provided in the paper alternative estimates of sensitivity and specificity of 3- and 12-lead electrocardiograms in diagnosing coronary artery disease based on this hypothesis. Tracings showing left bundle-branch block were ignored. The 3-orthogonal lead electrocardiogram had an index of merit of 0.51 and the 12-lead electrocardiogram correspondingly had an index of merit of 0.47. These indices are not statistically significantly different. If in addition left bundle-branch block is considered as an index of coronary artery disease corresponding indices are 0.49 and 0.45. If left ventricular hypertrophy and left bundle-branch block are not regarded as being indices of myocardial ischaemia, as implied by Dr. Gruer, such electrocardiographic reports are, therefore, taken as compatible with normal coronary arteries. Thus sensitivity falls to 66 per cent and 62 per cent for 3- and 12-lead electrocardiograms, respectively. Dr. Gruer’s figures for specificity, however, are wrong. Two patients, one with 3- and 12-lead electrocardiographic evidence of left ventricular hypertrophy and the other with left bundle-branch block, had normal coronary arteries. These patients must be included in the numerator as well as the denominator for the calculation of specificity if left ventricular hypertrophy and left bundle-branch block are not indices of coronary artery disease.

Thus:

\[
\text{Specificity} = \frac{\text{No. of patients with no electrocardiographic evidence of coronary artery disease}}{\text{No. of patients with no coronary arteriographic evidence of coronary artery disease}}
\]

\[
= \frac{(17+2)}{25} \quad \text{or} \quad \frac{(18+2)}{25}
\]

\[
= (76\%) \quad \text{3-lead} \quad \text{or} \quad (80\%) \quad \text{12-lead}
\]

This results in indices of merit being 0.42 for both lead systems and not 0.34 as indicated by Dr. Gruer.

Because left ventricular hypertrophy and left bundle-branch block neither clearly indicate the presence of coronary artery disease nor suggest the contrary, it seemed best to us to adopt the approach presented in our paper where the groups are treated separately. To include left ventricular hypertrophy and left bundle-branch block as indices of myocardial ischaemia in a population with chest pain does in our opinion bias data towards a high sensitivity (81% against 77%) at the expense of decreased specificity (68% against 74%). To exclude left ventricular hypertrophy and left bundle-branch block as indices of myocardial ischaemia in a similar population introduces bias against a high sensitivity (66% as against 77%). Specificity is essentially unchanged (76% as against 74%). These figures apply to the 3-lead electrocardiogram; similar trends apply to the 12-lead electrocardiogram data. To avoid bias, we presented our results in two ways and the conclusion of the paper was unchanged, as indeed it still is if one takes Dr. Gruer’s view.

Our research has always aimed at showing ‘that the 3-lead electrocardiogram is clinically as useful as the 12-lead when interpretation is made by automated methods’ (Macfarlane et al., 1971). The present study does not alter this view and our conclusion with respect to the diagnosis of coronary artery disease remains as before viz ‘This favourable performance provides further justification for the routine use of 3 orthogonal lead electrocardiography’.


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Reference