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).

Heart rate variability and cardiac failure

EDITOR,—We read the recent important editorial by Lombardi and Mortara with interest, and we would like to comment on several points raised.

As the authors state, spectral analysis of short term recordings of heart rate variability (HRV) is of limited value in patients with cardiac failure. Long term recordings in patients with cardiac failure contain a large amount of noise, artefact, ectopic activity, and non-stationary heart rate fluctuations, and spectral measurements are unreliable under these circumstances.1 We believe that studies of HRV in patients with cardiac failure are most reliable when confined to time domain techniques, which are highly reproducible in patients with cardiac disease.2 Most early studies of HRV and risk stratification were small, retrospective, inadequately sized, and most of the patients enrolled had very severe cardiac failure. These patients can be risk stratified by a physician using simple echocardiography in routine clinical practice, and this was reflected in the protocol for UK-HEART.4 We are surprised that Lombardi and Mortara did not consider these straightforward issues relating to the M mode derived mean ejection fraction in UK-HEART in their editorial.

The simple take home message from UK-HEART is that ambulant outpatients with mild-moderate cardiac failure (who are difficult to risk stratify using conventional techniques) and a mean SDNN of < 100 ms (37.8% of our patients) have an unfavourable prognosis (annual mortality rate 16.8%). Patients with an SDNN of > 100 ms have a good prognosis (annual mortality rate 5.5%). In multivariate analysis, SDNN is the most powerful predictor of death from progressive heart failure. This may reflect the presence of major neuroendocrine dysfunction in patients with a low SDNN, which has deleterious effects on ventricular geometry leading to a decline in pump function and progressive heart failure.

In contrast to Lombardi and Mortara, we believe that the results of UK-HEART are eminently applicable to a very large proportion of patients, and that 24 hour electrocardiography with measurement of SDNN has a useful role in the assessment of patients with cardiac failure.

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FOR THE UK-HEART STUDY GROUP

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1 Lombardi F, Mortara A. Heart rate variability and cardiac failure. Heart 1998;82:133–14

Table 1 Relative risk of cardiac death according to age for a decrease in SDNN

<table>
<thead>
<tr>
<th>SDNN</th>
<th>Survivors</th>
<th>Non-survivors</th>
<th>p Value</th>
<th>RR of cardiac death (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age &lt; 50 (n = 47)</td>
<td>110.5 (58.0)</td>
<td>79.9 (57.5)</td>
<td>0.009</td>
<td>1.036 (1.002–1.072)</td>
</tr>
<tr>
<td>Age &gt; 50 (n = 75)</td>
<td>96.0 (32.5)</td>
<td>79.6 (25.0)</td>
<td>0.02</td>
<td>1.027 (1.003–1.053)</td>
</tr>
</tbody>
</table>

p value based on proportional hazards model.

Risk ratios (RR) are calculated for a decrease in SDNN equal to 1 ms.

hour ECG recording was assessed in 122 patients with IDC (WHO criteria; mean age 50 years; range 18–72; 94 men; mean (SD) left ventricular ejection fraction 34 (12)%). Patients had conventional treatment with angiotensin converting enzyme (ACE) inhibitors, diuretics, and digoxin. With a mean (SD) follow up of 54 (39) months, 18 patients died from cardiac causes. Using multivariate analysis (proportional hazard model) only reduced SDNN (p = 0.003); increased mean pulmonary artery pressure (p = 0.04), and ventricular tachycardia during 24 hour ECG recording (p = 0.04) predicted cardiac death. Relative risk for cardiac death was calculated for a decrease in SDNN in two groups of patients aged < 50 (n = 47) or > 50 (n = 75) (table 1). For a decrease in SDNN of 1 ms, relative risk was one third higher in patients aged < 50 compared to patients aged > 50.

The fact that a decrease in HRV is a more powerful predictor of risk in young patients is of particular interest, as these patients probably have the most to gain from additional drug treatment, an exercise programme, or both, or for whom heart transplantation could be considered. The usefulness of HRV for such implication remains to be evaluated in large studies.

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These letters were shown to the authors, who reply as follows:

We read with interest the letter by Nolan and Fox on behalf of the UK-HEART study group concerning our recent editorial.1 Whereas there is a general agreement on the interpretation of previous studies and on the limits of spectral analysis of short term recordings, these authors suggest that the results of UK-HEART2 were not adequately interpreted.

The reason for such apparent discrepancy may be the fact that the results of UK-HEART were published in full only in October 1998 and therefore well after the acceptance of our editorial. Nevertheless, we were aware of the importance of this prospective study and quoted and discussed the results that had been published in abstract form. The complete results of UK-HEART indicate that a reduced SDNN identifies a group of ambulant outpatients with mild–moderate heart failure with an unfavourable prognosis because of the progression of the disease. However, it is noteworthy that these data, as reported in our editorial, may be not applicable to patients with a more severe impairment of ventricular function. The possibility, suggested by Nolan and Fox, of an overestimation of the value of ejection fraction by the methods used in their study does not facilitate an appropriate appraisal of the severity of the disease. Indeed, according to the figure recently published on Circulation4 nearly 25% of the patients enrolled in UK-HEART had a left ventricular ejection fraction > 50%.

An additional point that deserves further investigation is why a reduced SDNN is unable to identify cardiac failure patients who are victims of sudden cardiac death. Moreover, the finding that a cut off value of 100 ms was effective in stratifying mortality risk of these patients is likely to reflect, in our opinion, the design of the study, which was based on ambulant outpatients rather than those restricted to hospital. This cut off is higher than the value reported to stratify patients with a recent myocardial infarction in the post-thrombolysis era and suggests a possible significant influence of non-neural factors in the determination of HRV parameters in chronic heart failure.

Fauchier et al provide interesting comments on the possible clinical utility of a high negative predictive value of preserved HRV (SDNN > 100 ms) to identify a subgroup cardiac failure patients at low risk.1 Unfortunately, good clinical indicators of favourable outcome have been extensively reported in the literature, while the identification of high risk patients remains the most challenging and important objective. As to the adjucitive role of aging in determining a reduction in HRV parameters, we are in agreement with Fauchier et al’s observation that age has to be considered when determining the prognostic role of HRV. The results described in the letter (table 1) suggest that reduced HRV may be a more powerful predictor of cardiac death in younger rather than in older patients; however, a significant difference between the two relative risks is, at the moment, not proved and needs to be confirmed in larger studies. Nevertheless, the correlation between reduction in HRV and increased cardiac mortality described by Fauchier et al leaves unsolved the question of why a depressed HRV is unable to identify cardiac failure patients at risk for sudden cardiac death.