LETTERS TO THE EDITOR

- *The British Heart Journal welcomes letters commenting on papers that it has published within the past six months.*
- *All letters must be typed with double spacing and signed by all authors.*
- *No letter should be more than 600 words.*
- *In general, no letter should contain more than six references (also typed with double spacing).*

Evidence of inadequate investigation and treatment of patients with heart failure

SIR,—We read with interest the audit by Clarke *et al*[1] and agree that patients with heart failure may not receive optimum treatment, despite increasing evidence for the benefits of treatment with ACE inhibitors.

This is of particular concern because heart failure is one of the commonest causes of admission to hospital in the United Kingdom.[2] We recently conducted a prospective survey of patients admitted to our city centre district general hospital over a three month period (March to May 1994) to determine the distribution by ethnic group of patients admitted with heart failure and the pre-admission treatment of these patients. We admitted 185 patients (84 male and 101 female; mean (SD) age 73·7 (11·3)) with clinical evidence of heart failure: of these 141 (77%) were white, 14 (7%) black or Afro-Caribbean, and 30 (16%) Asian. The proportions of whites, blacks, and Asians in the population served by our hospital are 83%, 10%, and 7% respectively, so Asian patients tended to be overrepresented ($\chi^2 = 4{\cdot}8$, $p = 0{\cdot}09$). This is consistent with the higher incidence of coronary heart disease in this ethnic group. White patients were, however, significantly older (mean age 75·8 y (10·4) than black patients (71·0 (13·3)) and Asian patients (66·3 (11·1)) (oneway ANOVA $F = 11{\cdot}3$, $P < 0{\cdot}0001$). In addition, women with heart failure were generally older than men (76·5 (11·1) *v* 70·4 (10·6); unpaired *t* test, $P = 0{\cdot}0002$).

Because atrial fibrillation is commonly found in patients with heart failure who require acute medical admission,[3] we also investigated the prevalence of this arrhythmia in our patients. We found that 46 (25%) of our 185 patients had atrial fibrillation, a proportion that is consistent with a previous study from London.[4]

Clarke *et al*[1] also report an underutilisation of ACE inhibitors, with only 17% of their patients with heart failure taking these drugs. We found a higher proportion of ACE inhibitor use among our patients. On admission, 106 (57%) of these patients were receiving treatment for heart failure; and of these, six (3%) patients were receiving ACE inhibitors alone and 41 (22%) were taking ACE inhibitors and diuretics in combination, with 59 patients (31·7%) taking only diuretics. This gives a total of 47 (44·3%) patients taking ACE inhibitors. We therefore agree that physicians may still need to alter their prescribing habits in favour of using the ACE inhibitors to treat patients with heart failure.

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1 Clarke KW, Gray D, Hampton JR. Evidence of inadequate investigation and treatment of patients with heart failure. *Br Heart J* 1994; 71:584–7.
2 Dargie HJ, McMurray J JV. Diagnosis and management of heart failure. *BMJ* 1994; 308:321–8.
3 Lip GYH, Tean KN, Dunn FG. Treatment of atrial fibrilation in a district general hospital. *Br Heart J* 1994;71:92–5.
4 Parameswara J, Poole-Wilson PA, Sutton GC. Heart failure in district general hospital. *J R Coll Physicians Lond* 1992;26:139–42.

Heart rate variability and clinical cardiology

SIR,—We welcome the increasing interest in heart rate variability (HRV) shown by recent articles.[1-3] As recognition of abnormal patterns of HRV in cardiovascular disease grows, so does the need to understand and interpret the patterns in the context of the disease studied. To measure the value of any test both its clinical and pathological significance must be known. This is rarely possible without a detailed knowledge of the physiological basis of the measurement in normal situations. Power spectral analysis of HRV signals has the advantage of providing the opportunity of understanding the physiological meaning of the frequency components of the HRV signal. Respiration is recognised as the key determinant of the high frequency (HF) peak mediated through the parasympathetic pathways, but interpretation of the peaks in the lower frequency domains is controversial. The low frequency (LF) peak at 0·1 Hz has been used as an index of sympathetic activity, though it is not exclusively affected by this, whereas the regulatory mechanisms for the very low frequency (VLF) peak below 0·03 Hz are poorly understood.

The LF power content is often regarded as reciprocal to the parasympathetic HF peak. From this the concept of the LF/HF ratio has emerged as an index of sympathovagal balance, though it is not a pure measure nor strictly quantitative. As Malliani *et al* point out LF is determined not only by sympathetic activity but also by vasomotor, baroreceptor, and some parasympathetic inputs[1]; with the relative influences of each depending on the conditions at the time of electrocardiographic recording.

Twenty four hour ambulatory recordings are not the most reliable recordings for the study of HRV because the stationary conditions necessary for spectral analysis are unlikely to be met and moreover it is not possible to control for several factors that influence sympatho-vagal balance such as posture, respiration, circadian rhythms, sleep/wakefulness, or exertion. This is of particular importance in assessing LF and VLF peaks because infrequent (and non-physiological) changes will create a band of noise merging with other signals. One explanation for the decreased HRV found by Fei *et al*[3] in survivors of sudden cardiac death compared with controls could be that the survivors had 24 hour ambulatory recordings made when they were inpatients and were therefore less active than the controls.

The study of patients will be less subject to confounding influences under controlled conditions and in a more stable environment. Longer periods of recording rather than the conventional 5–10 minutes will allow more detail to be extracted from the VLF peak. Patients studied at rest and under test conditions—for example, controlled respiration or tilt—may allow a better quantification of the power spectra under conditions where the predominance of one or more inputs to HRV is achieved. Furthermore HRV should not be regarded as a standard in isolation. The comparison of other measures of sympathetic and parasympathetic function such as noradrenaline concentrations, baroreceptor sensitivity, and microneurography[4] with HRV measures should provide fuller confirmation of the value of HRV as index of altered sympatho-vagal balance and shed further light on the patho-physiological pathways governing the clinical state. A parallel assessment of clinical correlates is complementary to this, to relate abnormal HRV profiles to functional, haemodynamic, and neurohumoral abnormalities and to the risk of ventricular arrhythmias and sudden death.

Two examples of the complexities of HRV analysis that significantly affect its clinical interpretation are the effect of high level exercise and severe heart failure. Both conditions are associated with dramatically enhanced sympathetic drive and vagal withdrawal. Both mild exercise and mild heart failure are associated with an increase in LF/HF ratio because of an important reduction in absolute HF power and a smaller percentage reduction in LF power. In vigorous exercise and severe heart failure, however, LF falls to zero and the LF/HF ratio as a sympathetic measure would suggest vagal predominance. Such a change has been described in patients with low output shock who are in intensive care. In other words, whereas the LF/HF ratio increases with mild sympathetic activation it decreases once that activation becomes extreme. Thus what is true as a generalisation may be very misleading in specific cases. Care must be taken to understand the meaning of a test before interpreting the result, especially when the physiology underlying the measurement is still poorly understood.

Thus although we support the increasing interest in the clinical value of the study of HRV, our knowledge of its physiology remains so poor that we may be overlooking the most important part of its message.

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1 Malliani A, Lombardi F, Pagani M. Power spectrum analysis of heart rate variability: a tool to explore neural regulatory mechanisms. *Br Heart J* 1994;71:1–2.
2 Malik M, Camm AJ. Heart rate variability and clinical cardiology. *Br Heart J* 1994;71:3–6.
3 Fei L, Anderson MH, Katritis D, Sneddon J, Stratters DJ, Malik M, Camm AJ. Decreased heart rate variability in survivors of sudden cardiac death not associated with coronary artery disease. *Br Heart J* 1994; 71:16–21.