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.

4 Kienzle MG, Ferguson DW, Birkett CL, Myers GA, Berg WJ, Mariano DJ. Clinical, haemodynamic and sympathetic neural correlates of heart rate variability in congestive heart failure. *Am J Cardiol* 1992;69:761–7.

Dipyridamole and dobutamine for myocardial perfusion imaging

SIR,—Kumar and colleagues conclude that dipyridamole is better than dobutamine during thallium myocardial perfusion tomography.[1] We also prefer to use a vasodilator routinely (we use adenosine) and we reserve dobutamine for patients who are unable to exercise and in whom adenosine is contraindicated.[2 3] Kumar and colleagues give several reasons to justify their conclusion, including greater stress perfusion scores in the lateral wall and apex of the left ventricle when dipyridamole is used. In the light of a recent editorial pointing out the importance of rigorous statistical methods in biomedical research,[4] we question whether the conclusion and hence the title of the paper is valid.

Without a prior hypothesis of regional differences it is not appropriate to make multiple statistical comparisons of individual segments. Analysis of variance is the preferred test statistic, using a nonparametric method (Kruskal-Wallis) given the discontinuous nature of the scoring system, followed by an appropriate post hoc test for individual segments only if there is evidence of heterogeneity. Any regional differences detected in this way should then be tested prospectively in a separate group of patients. The claimed segmental difference between the two forms of pharmacological intervention is unlikely to be real because there is no plausible reason why these segments should differ from the remainder of the myocardium. Kumar *et al* make no attempt to explain this anomaly.

They also claim a better correlation of perfusion score with a score derived from the *x* ray angiogram when dipyridamole rather than dobutamine is used. There is no description of the statistical methods used in this analysis, and therefore the validity of this claim cannot be judged from the data provided.

We therefore suggest an alternative conclusion: that the null hypothesis of equivalence in efficacy for dipyridamole and dobutamine cannot be rejected, and that practical matters such as cost and duration of protocol should determine which is used in individual circumstances.

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1 Kumar EB, Steel SA, Howey S, Caplin JL, Aber CP. Dipyridamole is superior to dobutamine for thallium stress imaging: a randomised crossover study. *Br Heart J* 1994; 71:129–34.
2 Pennell DJ, Underwood SR, Swanton RH, Walker JM, Ell PJ. Dobutamine thallium myocardial perfusion tomography. *J Am Coll Cardiol* 1991;18:1471–9.
3 Pennell DJ, Underwood SR, Ell PJ. Safety of dobutamine stress for thallium-201 myocardial perfusion tomography in patients with asthma. *Am J Cardiol* 1993;71:1346–50.
4 Altman DG. The scandal of poor medical research. *Br Med J* 1994;308:283–4.

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

SIR,—We sought statistical advice from a professional statistician on the methods used in our paper. It was suggested that Student's *t* test was appropriate for comparison of perfusion scores obtained with the two pharmacological stressing agents. The original hypothesis was that there was no difference in the effect of the two agents on myocardial perfusion and we therefore expected no difference in perfusion scores. We did do multiple analysis and agree with Dr Underwood and Professor Wood that this may have been the cause of the segmental differences.

Because dobutamine and dipyridamole induce abnormalities of perfusion by different mechanisms there may be a "true" difference in their ability to produce segmental hypoperfusion. The correlation of perfusion score with angiographic score was performed by linear regression according to the methods in Draper and Smith (*Applied regression analysis*. 2nd ed. New York: Wiley, 1981;84).

Our study undoubtedly showed that studies with dipyridamole are cheaper, better tolerated, and less time consuming than dobutamine studies, without any loss in the ability to detect abnormal myocardial perfusion.

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Long-term results of the corridor operation for atrial fibrillation

SIR,—The corridor operation for atrial fibrillation[1] is an ingenious operation in which the surgeons isolate the left and right atrial free walls from the atrial septum, leaving a corridor of contiguous tissue between the sinus and atrioventricular nodes, thereby permitting chronotropically responsive atrioventricular conduction.

Unfortunately, because both atria remain in fibrillation, the corridor procedure fails to address the two major consequences of atrial fibrillation—namely, the loss of atrial transport function and thromboembolism. Therefore, it seems that the corridor procedure has no advantage over His bundle ablation and currently it is a major cardiac surgical procedure. I note that in the series of 36 patients reported by van Hemel *et al* His bundle ablation was performed and a pacemaker implanted in five patients "in whom the corridor operation was unsuccessful".

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1 van Hemel NM, Defauw JJAMT, Kingma JH, Jaarsma W, Vermeulen FEE, de Bakker JMT, Guiraudon GM. Long-term results of the corridor operation for atrial fibrillation. *Br Heart J* 1994;71:170–6.

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

SIR,—We welcome the opportunity to respond to Professor Cheng's concern.

According to the Frank-Starling law, right atrial contraction and right filling pressures are the main determinants of cardiac function[1]; in addition, diminished or absent left atrial contraction is not associated with alteration of cardiac function.[2] The normal heart acts as a suction pump during normal diastole. So, at least in the normal heart, the atrial contribution to cardiac function is negligible.[3]

Because chronotropic sinus node function is the main determinant of increasing cardiac output during exercise,[4] preservation of the physiological chronotropic response is one of the aims of the corridor concept. The maintenance of native chronotropic sinus function prevents impaired exercise tolerance and avoids the lifelong dependency on a pacemaker that is one of the consequences of His bundle ablation.[5] Our long-term results show that sinus node function remained undisturbed in most of our patients.

During the corridor operation the left atrial appendage, which is commonly the origin of atrial thrombosis in patients with atrial fibrillation, is excised. Possibly, resection of the left atrial appendage alone could prevent systemic emboli in patients with lone atrial fibrillation. This measure has not been tried in patients in whom atrial fibrillation continues after catheter ablation of the His bundle for rate control of drug refractory atrial fibrillation. Their risk of thromboembolism is not negligible.

Though corridor surgery did not suppress atrial fibrillation in all our patients, postoperative atrial fibrillation occurred only in the left atrium and never de novo in the corridor. The operation was sometimes unsuccessful because we failed to create a persistent conduction block between the left atrium and the coronary sinus. This is why some of our patients needed His bundle ablation. Such surgical failures require technical improvement, but they do not detract from the corridor concept.

Currently, it is impossible to make a valid comparison between a non-selective and less invasive procedure such as His bundle catheter ablation[6] and selective surgery for atrial fibrillation.[7] This is because of the differences in selection criteria, patient population, and the end point of treatment. The comparison is not even scientifically valid, because atrial fibrillation is a multifactorial protean disease.[8] In the face of excellent long-term results, we strongly believe that surgical procedures for atrial fibrillation will become a well-established treatment in some subgroups of patients—for example, those who do not want to be dependent on a pacemaker and those who need cardiac surgery for other reasons.

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1 Robinson TF, Factor SM, Sonnenblick EH. The heart as a suction pump. *Scientific American* 1986;254:84.