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).

Heart rate variability in left ventricular hypertrophy

Sin,—Mandawat et al in their paper (Br Heart J 1995;73:139–44) showed that heart rate variability was significantly reduced in patients with left ventricular hypertrophy secondary to hypertension or aortic valve disease. In addition they showed that heart rate variability was not affected by β blocker treatment. However, they made no comment on the effect of other antihypertensive agents such as thiazide diuretics and certain calcium antagonists such as nifedipine which can cause reflex sympathetic activation and hence reduce heart rate variability. Nearly 60% of their patients were taking diuretics. We have recently found in a study of Chinese patients with hypertension that thiazide treatment was associated with reduced heart rate variability assessed by nonspectral and spectral methods, compared with a 4 week control during which potassium replacement treatment alone was given (unpublished). Like Mandawat et al we found a significant reduction in the SD of all NN intervals over 24 (SDNN) and in addition root mean square of difference of successive RR (rMSSD), proportion of adjacent RR's more than 50 ms different (pNN50), with reduced low frequency and high frequency spectral power.

Though left ventricular hypertrophy may well be independently associated with reduced heart rate variability, we were surprised that no comment is made about the possible effect of treatments, other than those of β blockers, on heart rate variability in hypertensive subjects. In view of the possibility of an increase in sudden death in hypertensive patients taking thiazide diuretics we would be interested to know whether Mandawat et al have any comparable data on the relation of thiazide treatment to heart rate variability in hypertensive subjects with left ventricular hypertrophy.

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This letter was shown to the authors, one of whom replies on behalf of his co-authors as follows:

Sir,—The observation by Sanderson and Tomlinson of reduced heart rate variability during treatment with a thiazide diuretic raises the interesting possibility of altered cardiac autonomic tone as a potential mechanism for the increased risk of sudden death associated with thiazide treatment. Unfortunately this suggestion is neither supported nor refuted by our study. Most of our subjects were receiving a combination of antihypertensive drugs (mean 2±5). The inclusion of a diuretic with the regimen did not influence indices of heart rate variability (SD of all NN intervals over 24 h; triangular index; and SD of the mean NN interval for all 5 min segments of a 24 h recording), whether analysed as any diuretic drug (n = 54) or, more specifically, as a thiazide preparation (n = 32 of 82) (heart rate variability corrected for RR interval). Similarly, neither calcium antagonist (n = 29) nor angiotensin converting enzyme inhibitors (n = 21) affected time heart rate variability in these subjects with left ventricular hypertrophy. During prospective follow up of this cohort over 9±3 years, all-cause mortality was reduced by β blocker treatment but was unaffected by diuretic treatment (unpublished).

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Risks and results of surgery

Sin,—We congratulate Professor Tom Treasure on his interesting and far-reaching article.1 However, we suggest that the objectives in this instance should not be limited merely to “redressing apparent differences in results by correcting for differential casemix”. Much more important potential benefits await the widespread introduction of an appropriate means of risk adjustment from observational databases.2 These include an ability in coronary artery surgery to produce predictive data comparable to randomised controlled trials.

Risk stratification on the basis of preoperative, perioperative, and postoperative variables is the cornerstone of any realistic comparisons but the system chosen must be robust and not open to manipulation or subjective elements.3 The best system available for the task must be used. A system described in 1990 has been endorsed by the Association of Cardiothoracic Anaesthetists of Great Britain and Ireland (ACTA).4 This ACTA system has been demonstrated to perform better (with improved receiver operating characteristic curves) than the Parsonnet system.5 In this comparison the explanatory components of the Parsonnet and ACTA scoring systems were subjected to multiple logistic regression (MLR) analysis.6 The MLR package selected the ACTA items rather than the Parsonnet items. Moreover the subjective elements of the Parsonnet data set (“catastrophic state” and “FTCA crash”) were not selected as explanatory of mortality or prolonged stay in the intensive therapy unit (ITU) in this analysis.

There are other systems available, one of which (from New York) recently gained considerable publicity for the claims made for it and the criticism it has received.7 8 This system and the Parsonnet system both contain subjective elements, which in the Parsonnet system are not good explainers of outcome but can be used subjectively to increase the score a patient achieves.

The Parsonnet and New York state data sets confine their outcome analysis to death in hospital, but there are more valuable measures of the processes undertaken in different centres. Length of ITU stay (included in the ACTA system), length of hospital stay, and costs estimated in hospital cost are also important outcomes that can be estimated before cardiac surgery and are valuable both to the patient and to the centre undertaking the surgery.

The data becoming available from this type of observational study are increasingly valuable in addressing the planning needs of healthcare purchasers worldwide. Conclusions drawn from such observational studies with casemix adjustment have been recognised as to be as valuable as randomised controlled trials. Acceptance of a proven inferior system would considerably retard the laudable objectives of Professor Treasure and others.

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Percutaneous balloon dilatation of the mitral valve in critically ill young patients with intractable heart failure

Sir,—Patel and colleagues demonstrated the important role of emergency percutaneous

balloon dilation of the mitral valve using the Inoue technique in young patients who are critically ill because of severe mitral valve stenosis.1 We note that the procedure was performed while three of their patients were on ventilator. Three patients had atrial fibrillation, but it is not clear whether this group overlapped with the patients who required ventilatory support.

In an earlier study percutaneous balloon dilation of the mitral valve was associated with a 2–4% incidence of arterial emboli.2 We believe that patients at high risk for embolic events should have transoesophageal echocardiographic (TOE) screening before valve dilation because it has been shown that TOE is significantly superior to transthoracic imaging in detecting of important left atrial thrombus.

We have successfully carried out Inoue balloon mitral valve dilation in two patients (aged 61 and 59 years). Both had atrial fibrillation with severe left ventricular failure secondary to critical mitral stenosis and could not be weaned from intravenous inotropes or ventilatory support. In both patients transoesophageal assessment before the procedure showed spontaneous contrast in the left atrium but no left atrial thrombus.

The mitral valve echocardiographic scores (Wilkins et al3) were 8 and 10. A significant reduction was obtained in the transmural end diastolic gradient (50% and 80% respectively) and there was a 2–3 fold increase in the mitral valve area on echocardiography. Both patients were successfully weaned off ventilatory support within 24 hours of valve dilation.

We suggest that percutaneous dilation of the mitral valve using the Inoue balloon technique is as valuable in older patients who cannot be weaned from assisted ventilation as a result of critical mitral stenosis as it is in younger patients. Older patients are more likely to be in atrial fibrillation and should have transoesophageal echocardiography.

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This letter was shown to the authors, one of whom replies as follows:

SrR,—I appreciate the concern voiced by Davis and his colleagues regarding the role of transoesophageal echocardiography to exclude left atrial thrombus before percutaneous balloon dilation of the mitral valve.

At our hospital we have stopped performing routine transoesophageal echocardiographic assessment because in our experience the yield, following on a carefully performed transesophageal cross sectional echocardiographic study, was low. This may be because most of our patients are young (mean age 30 years) and in sinus rhythm. Only 16% of our patients undergoing balloon dilation of the mitral valve have transoesophageal echocardiography. I do not, however, underestimate its important role in elderly patients who are in atrial fibrillation.

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1 Patel JJ, Dyer RB, Pillay R, Munclinger MJ, Minha AS. Do all patients undergoing Inoue balloon mitral valotomy need transoesophageal echocardiography (TOE) to exclude left atrial thrombus? [Abstract] J Am Coll Cardiol 1995; 89A.

Strut fracture of the convexo-concave Björk-Shiley mitral valve prosthesis

Sir,—Goodfellow et al (Br Heart J 1995;73:301) recommend an overpenetrated chest radiograph in posteroanterior and lateral or oblique views if strut fracture is suspected. This takes time when there is no time. Also, in a breathless patient with pulmonary oedema it would be difficult to acquire the high quality penetrated views required to show absence of the occluder.

Sudden total mitral regurgitation transforms a person from normal health to desperate straits virtually instantaneously. In a patient with this prothetic valve, for practical purposes, there is no differential diagnosis.

Valve thrombosis has a more gradual onset and is less disasrrous, with auscultatory signs of mixed stenosis and regurgitation. Loss of a mitral occluder produces pulmonary oedema without murmurs (no pressure gradient between ventricle and atrium). Prosthetic clicks are absent in both.

Echocardiography is fast and fully informative but should only be done en route to the operating room.

Goodfellow et al successfully treated their patient. The x rays which they showed were taken with the chest open on the operating table.

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

SrR,—Professor Oakley is of course correct in her comments about our dramatic image in cardiology showing an intraoperative chest x ray with the disc occluder of a mitral Björk-Shiley convexo-concave valve lying within the aorta. The other x ray showed the fractured strut lying within the femoral artery. If a patient who is known to have a convexo-concave (CC) valve presents with sudden cardiovascular collapse and pulmonary oedema to a centre with cardiothoracic surgery, then of course the valve must immediately be replaced.

Many patients with implanted Björk-Shiley CC valves have not been identified and therefore do not carry information stating that they have this valve. Most patients presenting to hospital with acute pulmonary oedema do not present to one that has cardiac surgery immediately available and with an experienced cardiologist to see that individual in that casualty department and make the diagnosis. Patients with prosthetic valves in situ who develop sudden pulmonary oedema should always of course be suspected of having valve dysfunction but this may also have other causes such as dihiscence secondary to endocarditis, or may be related to ventricular dysfunction, for example associated with arrhythmia.

The absence of prosthetic clicks in a patient in extremis in a noisy accident and emergency department may be apparent only to very experienced observers. Cardiac surgeons would be unnecessarily overwhelmed if all such patients were transferred on presentation for immediate valve replacement.

There is a need for immediate and accurate diagnosis of outlet strut fracture and Hiratzka et al in a review of the subject have recommended "the single most expeditious study that should be readily obtainable in virtually any hospital or emergency treatment centre and that is highly effective in making the diagnosis of outlet strut fracture is an overpenetrated chest radiograph, preferably in two views (posterior anterior and lateral or oblique)." We believe that this remains sound advice. Patients presenting with cardiovascular collapse caused by strut fracture of the valve may need immediate ventilation and a penetrated chest x ray may then permit the diagnosis.

An intraoperative x ray can help identify the displaced components of the prosthesis and facilitate their removal, as was the case in the patient presented in our illustration.

Almost 100 000 patients are seen annually in the casualty department in our hospital and there are many cases of acute pulmonary oedema. The diagnosis may be obvious to an experienced cardiologist looking at a single illustration after the diagnosis has been made, but what is needed is straightforward advice for those dealing with a common medical emergency with an uncommon but catastrophic cause, and we believe that advice to obtain a penetrated radiograph in such cases remains sound.

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Percutaneous balloon dilatation of the mitral valve in critically ill young patients with intractable heart failure.

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