

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:

- initially submitted by fax +44 171 388 0323 or e-mail 100536.2733@compuserve.com (where practicable). Always follow this up by posting the paper copy to us
 - 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.

The difficulties in assessing right ventricular function

SIR—In their editorial Oldershaw and Bishop argue that the high incidence of circulatory collapse in acute inferior infarction that is attributable to right ventricular dysfunction and is independent of arrhythmia and left ventricular dysfunction suggests that right ventricular dysfunction is more than a simple index of left ventricular filling pressure.¹

In 1987 we showed that both global and regional right ventricular dysfunction were related to the site of the left ventricular infarction.² In patients with inferior left ventricular infarction there was no correlation between the right and left ventricular ejection fractions, and right ventricular free wall motion was impaired. This suggests that in inferior myocardial infarction isolated right ventricular dysfunction is caused by infarction of the right ventricular free wall. In contrast there was a strong correlation between the extent of left and right ventricular dysfunction after anterior left ventricular infarction, suggesting that right ventricular dysfunction in this group is related to impairment of left ventricular filling.

It therefore seems that the mechanism for right ventricular dysfunction may vary with the site and extent of left ventricular dysfunction, and that right ventricular dysfunction may be an index of left ventricular filling pressure and hence right ventricular afterload in patients with extensive anterior myocardial infarction.

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- 1 Oldershaw P, Bishop A. The difficulties in assessing right ventricular function. *Br Heart J* 1995;74:99–100.
- 2 Caplin JL, Dymond DS, Flatman WD, Spurrell RAJ. Global and regional right ventricular function: dependence upon site of left ventricular infarction. *Br Heart J* 1987; 58:101–9.

Exercise ventilation after balloon dilatation of the mitral valve

SIR—We were interested to read the report of Banning *et al*¹ of an acute reduction (within 24 hours) of the \dot{V}_E/\dot{V}_{CO_2} slope after balloon dilatation of the mitral valve. Banning *et al* explain this observation by suggesting that the abnormally high \dot{V}_E/\dot{V}_{CO_2} slope is the result of ventilation/perfusion mismatching. They argue that with acutely improved haemodynamics this mismatch, and hence the \dot{V}_E/\dot{V}_{CO_2} slope, improves. The basis of this assumption is the observation in a subset of 10 patients that the \dot{V}_E/\dot{V}_{CO_2} slope is reduced from a mean of 41 to 36 within 24 hours. We are concerned, however, with the method used to obtain the \dot{V}_E/\dot{V}_{CO_2} slope in these patients. They were exercised to 75% of their pre-procedure maximal workload, and the \dot{V}_E/\dot{V}_{CO_2} slope was then calculated from this submaximal test. We have previously demonstrated that the \dot{V}_E/\dot{V}_{CO_2} relation is not uniformly linear, at least in chronic heart failure,² and that the value obtained for the overall \dot{V}_E/\dot{V}_{CO_2} slope is dependent upon the level of exercise completed.³ In our study the \dot{V}_E/\dot{V}_{CO_2} slope was at least 10% less if it was calculated from a submaximal (75% maximal) test. The observed reduction in \dot{V}_E/\dot{V}_{CO_2} slope at 24 hours could thus largely be explained by this artefact. If the 24 hour \dot{V}_E/\dot{V}_{CO_2} slope is spuriously lowered then it is interesting to observe that there would actually be a gradual reduction in \dot{V}_E/\dot{V}_{CO_2} slope rather than an acute drop, suggesting a peripheral rather than haemodynamic cause for the high \dot{V}_E/\dot{V}_{CO_2} slope in these patients. This hypothesis is also supported by the recently reported dissociation between haemodynamic improvement and exercise tolerance after balloon dilatation of the mitral valve.⁴

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- 1 Banning AP, Lewis NP, Elborn JS, Hall RJC. Exercise ventilation after balloon dilatation of the mitral valve. *Br Heart J* 1995;74: 386–9.
- 2 Clark AL, Poole-Wilson PA, Coats AJS. Relation between ventilation and carbon dioxide production in patients with chronic heart failure. *J Am Coll Cardiol* 1992;20: 1326–32.
- 3 Clark AL, Poole-Wilson PA, Coats AJS. Effects of motivation on indices of exercise capacity in chronic heart failure. *Br Heart J* 1994;71:162–5.
- 4 Douard H, Gilles YM, Choussat A, Broustet JP. Lack of correlation between haemodynamic and cardiopulmonary exercise capacity improvement after catheter-balloon mitral valvuloplasty. *Eur Heart J* 1995;16: 1375–9.

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

SIR—Further analysis of our data,¹ with direct comparison of equivalent exercise levels, showed a persistence in the difference in the \dot{V}_E/\dot{V}_{CO_2} slope before and 24 hours after balloon dilatation of the mitral valve (mean 40 to 36, $P < 0.05$).

The relation between ventilation and carbon dioxide is known to vary with the severity of heart failure.² The finding of a 10% difference in \dot{V}_E/\dot{V}_{CO_2} slope calculated from a submaximal test was derived from a group of patients with predominantly mild

to moderate heart failure (mean peak \dot{V}_{O_2} 19ml/kg/min, mean \dot{V}_E/\dot{V}_{CO_2} 33).³ Our patients had more severe functional limitation (peak \dot{V}_{O_2} 12ml/kg/min, \dot{V}_E/\dot{V}_{CO_2} 41) and previous work suggests that in such patients the increase in \dot{V}_E/\dot{V}_{CO_2} slope is linear and that it is seen from the outset of exercise. Data in these severely affected patients are therefore less likely to be affected by reductions in patient motivation and exercise duration during a submaximal test.

We have previously shown that changes in cardiac output can acutely alter the \dot{V}_E/\dot{V}_{CO_2} slope in patients with impaired resting left ventricular function.⁴ Similar changes do not occur in patients with normal ventricles. These data, taken with our current data, suggest that in patients with chronic heart failure and increased \dot{V}_E/\dot{V}_{CO_2} slope, the \dot{V}_E/\dot{V}_{CO_2} slopes and therefore ventilation/perfusion matching can be altered acutely and that these changes are likely to reflect changes in the distribution of pulmonary flow rather than alterations in skeletal muscle metabolism.

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- 1 Banning AP, Lewis NP, Northridge DB, Elborn JS, Henderson AH. Perfusion ventilation mismatch during exercise in chronic heart failure: an investigation of circulation determinants. *Br Heart J* 1995;74:27–34.
- 2 Banning AP, Lewis NP, Elborn JS, Hall RJC. Exercise ventilation after balloon dilatation of the mitral valve. *Br Heart J* 1995;74: 386–9.
- 3 Clark AL, Poole-Wilson PA, Coats AJS. Relation between ventilation and carbon dioxide production in patients with chronic heart failure. *J Am Coll Cardiol* 1992;20: 1326–32.
- 4 Clark AL, Poole-Wilson PA, Coats AJS. Effects of motivation of indices of exercise capacity in chronic heart failure. *Br Heart J* 1994;71:162–5.

NOTICES

With much sadness the Council and administrative staff of the British Cardiac Society were informed of the death, after a long illness, of Jennie Lodge on 24 January 1996. Jennie had worked tirelessly for the Society for many years, up to and including the 1995 annual meeting in Harrogate, as Senior Administrator.

The 1996 Annual Meeting of the **British Cardiac Society** will take place at the Scottish Exhibition & Conference Centre, Glasgow from 7 to 9 May.

The **XXth South African Congress of Cardiology** will be held at the Wild Coast Sun, South Africa from 6–10 October 1996.

The **ETRO/ISSFAL** meeting on lipids, membranes and thrombosis: fundamental basis of cardiovascular disease and its dietary prevention will be held at the University of Maastricht, The Netherlands from 10–13 July 1996. For further information please contact Mrs Silvia de Bruin, Congressbureau Scem (tel: +31 345 576 642; fax: +31 345 571 781).