Letters to the Editor

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
- Numbered by editor
- Include a self-addressed, stamped envelope
- 500 words in length (including references)
- Typed in double spacing (fax copies and paper copy only)

They may contain short tables or a small figure.

Squatting revisited: comparison of haemodynamic responses in normal individuals and heart transplant recipients

Sir—We read with interest the investigation by Hanson et al into the haemodynamic effects of squatting after heart transplantation and were impressed with the elegant demonstration that the effects of a squat on blood pressure and stroke volume are similar in heart transplant recipients and in normal subjects. We are surprised that Hanson et al consider the transplanted heart to be denervated at a mean of 16 months after operation. There is clear evidence that at least sympathetic efferent innervation of the transplanted human heart occurs. Using injection of tyramine we and others showed sympathetic efferent reinnervation.1 2 We and others have failed to demonstrate parasympathetic reinnervation using autonomic function testing3 and intracoronary injection of contrast.

Hanson et al conclude that the differences between normal controls and transplant recipients are due to denervation. While this may be true, there are alternative explanations for their findings. Cardiac transplant recipients are survivors of cardiac failure, and the absence of bradycardia in response to hypertension may be partly explained by persisting reduced central baroreflex sensitivity.4 The response of forearm vascular resistance in the transplant recipients is consistent with this. Thus the absence of bradycardia cannot be taken as evidence of vagal denervation.

The small increase in heart rate of the transplant recipients is also consistent with sympathetic reinnervation in the absence of parasympathetic innervation, and this increase may be stimulated by the effect of squatting, in a similar manner to the effect of sustained handgrip, rather than by a volume reflex. Most cardiac sensory nerves lie in the atria, and thus a significant number reach the recipient atrial cuff. It cannot therefore be concluded that any effect of atrial volume change on heart rate is direct.

We would also like to point out that this manoeuvre might be an excellent non-invasive measure of sino-aortic baroreflex sensitivity, because of the rapid change observed in systolic blood pressure, akin to that observed after the initial phase of a Valsalva manoeuvre.1 2 We suggest that the haemodynamic effects of squatting provide opportunities for the investigation of baroreflexes in other groups of patients, and may also be useful in measuring reinnervation after cardiac transplantation. All investigations of reflexes in cardiac transplant recipients should take the possibility of reinnervation into account.

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

Sir—We are aware of recent reports of partial sympathetic reinnervation of the transplanted heart. Following publication of your paper we had a discussion of this issue in our paper. Subsequently we deleted that portion to shorten the text and because we did not have data to verify the presence or absence of sympathetic reinnervation in our patients.

It is unlikely that residual impairment of arterial baroreflex sensitivity was a factor in our study. Previous studies from our laboratory showed normal sinoaortic baroreflex control of sympathetic vasomotor tone in heart transplant recipients during orthostatic stress.5 In addition, baroreflex control of the innervated autonomic was also normal.6 The small increase in heart rate observed in the heart transplant recipients during squatting is difficult to explain. We agree that it is not possible to exclude an autonomic component, perhaps caused by sympathetic reinnervation.

Finally, we fully concur with the suggestion that the heart rate and blood pressure responses to the squatting manoeuvre may be used to evaluate baroreflex function in various groups of patients.

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SIR,—Carlsson et al suggested that serum lipids should be measured 4 weeks after acute myocardial infarction. The measurement of serum lipids after thrombolysis because there were no significant differences between these values and those obtained within 24 hours of onset of symptoms.3 This may in fact not be valid if the correct measurements were taken after and not before thrombolysis, because thrombolysis itself may be associated with a small but significant fall in total cholesterol concentrations which may return to basal values within several months later.4 The danger of course is that some patients with spuriously low concentrations may be overlooked. Given that the 4S study clearly supports active intervention for secondary prevention,4 each hospital should identify their best local practice of targeting patients for intervention. The previously recommended cut-off for routine routine lipid measurement for lipid measurement when intravenous access is secured for thrombolysis is easily applied, yields acceptable baseline results, and helps to ensure that no patient with hypercholesterolaemia is missed.

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

Sir,—Dr Bennie refers to a study from Chua et al including 26 male patients. Chua et al reported a 0-4 mmol/l fall in serum cholesterol concentration from a pre-streptokinase treatment concentration of 7-0 mmol/l. They concluded that serum cholesterol concentrations may be underestimated when they are measured after streptokinase treatment. However, the clinical importance of this underestimation of the lipid concentration is of less importance than the time lost when patients wait 3-6 months before start-
Aortic distensibility measured by magnetic resonance imaging in patients with Marfan's syndrome

Sin,—Adams et colleagues' reported aortic distensibility measurements based on assessments of eye of magnetic resonance (MR) and echocardiographic images in 12 patients with Marfan's syndrome. They suggest that the measurements are reproducible. Without repeating the measurements on a separate occasion and repeating their analyses we do not see how they can conclude this. They do not specify the oscillation frequency of the ultrasound probe used for echocardiography, which is critical for such work, the maximum spatial resolution would be of the same order of magnitude as the change in aortic diameter being measured. Moreover, the errors involved in the off-line measurement of dimensions from a scan are much less than those involved in actually performing the scan. Therefore data on the repeatability of the analysis procedure—or methods for errors from repeat scans at the same visit—tell us very little, if anything, about the true reproducibility of the measurement techniques.

In any discussion of the validity of direct measurements of distensibility at a particular aortic cross-section it is important to consider the role of blood pressure. Adams et al measured the change in diameter (or area) at discrete cross-sections of the ascending and descending thoracic aorta but then applied blood pressure values recorded from the brachial artery to calculate aortic distensibility. Blood pressure varies along the arterial tree and amplification of the pressure pulse between central and peripheral arteries makes brachial pressure values an inaccurate measure of central aortic pressure. There can be absolute differences in systolic and pulse pressures of up to 20 mm Hg. This problem can be partly overcome by indirectly determining an average elastic property of the aortic wall based on pulse wave velocity (PWV) measurements. Such an approach does not require the blood pressure at a particular aortic cross-section to be known.

This is especially pertinent because aortic distensibility indices determined from PWV measurements have good reproducibility (coefficient of variation <10%). A transfer function can also be used to calculate central aortic systolic and pulse pressures based on non-invasively determined data on pressure waveforms in peripheral arteries. We describe the application of MRA or echocardiography for directly measuring the change in aortic diameter (or area) between diastole and systole by eye are increasing. But not many papers report the reproducibility of such methods for non-invasively assessing aortic distensibility. Kupari et al reported a mean (SD) reproducibility for their MR measurements in the ascending and descending aorta of 26.3 (23-3)% and 34.9-32.1%, respectively. It will be necessary to repeat aortic imaging on the same patient in order to determine reproducibility values of up to 25% in the ascending aorta. Dert et al also used echocardiography and, taking into account the magnitude of the procedure, obtained a mean reproducibility (SEM) in young, healthy, athletic men of 9.4 (2.9)%. Unfortunately, these latter data tell us very little, if anything, about the reproducibility of the technique in older patients with stiffer arteries in whom the change in aortic diameter during the cardiac cycle would be much reduced (as would the associated distensibility).

In view of these data we do not find it surprising that Adams et al found that individual values for aortic distensibility varied by up to a factor of five depending on whether MR or echocardiographic data were used.

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This letter was shown to the authors, two of whom reply as follows:

Sin,—Magnetic resonance imaging is an established non-invasive method of assessing aortic distensibility and stiffness. We have shown in our study that aortic images obtained by this technique can be produced quickly and simply in patients with Marfan's syndrome and are responsive to antithrombotic controls. Measurements made on these images can be reproducibly assessed by independent observers. We did not perform further scans on a second occasion and therefore cannot comment further on the reproducibility of this technique.

We agree that brachial pressure is lower than that measured directly. However, as Stefanidis et al note, the underestimation is similar in patients with coronary disease and healthy men and the pulse pressure measured indirectly by sphygmomane-
ter correlates well with the pressure measured directly by catheterisation of the ascending aorta. The non-invasively determined values are a reflection of both the ascending aorta and the thoracic aorta.

Is aspirin safe for patients with heart failure?

Sin.—The prophylactic benefit of aspirin may have been overstated not only in coronary heart disease, as Christopher et al suggest, but also in thromboembolism related to non-valvar atrial fibrillation (NVAF). In NVAF, this overstatement may be the result of failure to recognise that warfarin cannot prevent all thromboembolic events in all patients all the time, and that aspirin may sometimes be perceived to have a prophylactic benefit because some NVAF patients are on warfarin and thromboembolism that are more amenable to risk modification by aspirin than by warfarin.

Furthermore, the risk/benefit profile of antithrombotic treatment might be more favourably disposed towards aspirin than towards warfarin in high-intensity than in low-intensity anticoagulant regimens. For thromboembolic prophylaxis, the principal disadvantage of aspirin is the unpredictability of its Embolism Task Force. Cardiogenic brain embolism. Arch Neurol 1986;43:71-84.
