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 0523 or e-mail 100536.2733@compuserve.com (where practicable). Always follow this up by posting the paper copy to us.
- not to exceed 600 words and six references in length
- 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 transplantation1 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 reinnervation of the transplanted human heart occurs. Using injection of tyramine we and others showed sympathetic efferent reinnervation.2,3 We and others have failed to demonstrate parasympathetic reinnervation using autonomic function testing4 and intracoronary injection of contrast.5

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.6 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 could 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 remain in 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 suggest 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 strain phase of Valsalva manoeuvre.7 We suggest that the haemodynamic effects of squatting provide opportunities for the investigation of baroreflexes in other groups of subjects, 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.

SIR-Stephen L. HANSON
Regional Cardiothoracic Centre, Freeman Hospital, Newcastle on Tyne NE7 7DN


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

SIR,—Carlsson et al suggested that serum lipids should be measured 4 weeks after acute myocardial infarction. The risk of restenosis after coronary thrombosis because there were no significant differences between these values and those obtained within 24 hours of onset of symptoms. This may in fact not be valid if data are obtained during hospital stay. Measurements were taken 1 to 5 months after and not before thrombolysis, because thrombolyis itself may be associated with a small but significant fall in total cholesterol and low density lipoprotein cholesterol concentrations may not return to base line values until several months later. 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,1 each hospital should identify their best local practice of targeting patients for intervention. The previously recommended strategy of waiting to start lipid lowering treatment in patients who have had an infarction should be reconsidered as a consequence of this observation.2

MARY JANE BENNIE Department of Pharmacy, GUY'S Hospital, ST THOMAS'S TERRACE, LONDON SE 1 9RT

ing lipid lowering treatment. We fully agree that it is better to measure lipid concentrations before thrombolytic treatment. If early lipid estimations are not available, val-
ues obtained within 24 hours after admis-
sion or 4 weeks later are a sufficiently valid basis for reaching a decision about early lipid intervention.

ROLAND CARLSSON
Section of Cardiology,
Department of Medicine,
Central Hospital,
S-65185 Karlstad
Sweden

Aortic distensibility measured by mag-
netic resonance imaging in patients with Marfan’s syndrome

Sin,—Adams and colleagues’s reported aortic distensibility measurements based on assess-
ments by eye of magnetic resonance (MR) and echocardiographic images in 12 patients with Marfan’s syndrome. They suggest that the measurements are reproducible. Without reviewing these patients 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 ultrasonic probes used for echocardiogra-
yphy, and it would be valuable for such work, the maximum spatial resolu-
tion would be of the same order of magni-
tude as the change in aortic diameter being measured. Furthermore, the errors involved in the off-line measurement of dimensions from a scan are much less than those involved in actually performing the scan.3 Therefore data on the repeatability of the analysis procedure—or methods for reducing measurement error from repeat scans at the same visit—tell us very little, if anything, about the true repro-
ducibility 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 con-
sider the role of blood pressure. Adams et al measured the change in diameter (or area) at different cross-sections of the ascending and descending thoracic aorta but then applied blood pressure values recorded from the brachial artery to calculate aortic disten-
sibility. Blood pressure varies along the arter-
tial tree and amplification of the pressure pulse between central and peripheral arteries makes brachial pressure values an inaccurate measure of central aortic pressure.5 There can be absolute differences in systolic and pulse pressures of up to 20 mm Hg. This problem may be partly overcome by indi-
rectly determining an average elastic prop-
erty of the aortic wall based on pulse wave velocity (PWV) measurements.6 Such an approach does not require the blood pres-
sure at a particular aortic cross-section to be known.7 This is especially pertinent because aortic distensibility indices determined from PWV measurements have good repro-
ducibility (coefficient of variation <10%). A transfer function can also be used to calculate central aortic systolic and pulse pressures based on non-invasively deter-
ded data on pressure waveforms in the mesocirculation.

Kupari et al described the application of PWV or echocardiography for directly mea-
suring the change in aortic diameter (or area) between diastole and systole by eye are increasing. But not many papers report the reproducibility of each method for non-
invasively assessing aortic distensibility. Kupari et al reported a mean (SD) repro-
ducibility for their MR measurements in the ascending and descending aorta of 26.3
(23.3)% and 34.9 (32.1)% respectively.6 Iranol et al using echocardiography reported a reproducibility values of up to 23% in the ascending aorta.7 Dart et al also used echocardiography and, taking special care to obtain an image perpendicular to the aortic arch (to avoid measuring the change in diameter of an ellipse), obtained a mean reproducibility (SEM) in young, healthy, athletic men of 9.4 (2.9)% 8 Unfortunately, these latter data tell us very little, if any-
thing, about the reproducibility of the tech-
nique in older patients with stiffer arteries in whom the change in aortic diameter during the cardiac cycle would be much reduced (as would the reproducibility). In view of these data we do not find it sur-
prising 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.

E D LEHMANN
Academic Department of Radiology,
St Bartholomew’s Hospital,
London EC1A 7BE
K D HOPKINS
Academic Department of Medicine,
Whittington Hospital,
London N 19 GOSLING
School of Applied Science,
University of the South Bank,
London

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 mechanical properties. What 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 used for diagnostic purposes. 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 Stefansdottir et al have underlined, this relation is similar in patients with coronary disease and healthy men and the pulse pres-
sure measured indirectly by sphygmoman-
eter correlates well with the pressure mea-
sured directly by catheterisation of the ascending aorta. Our aortic distensibility deter-
mined non-invasively is closely related to that obtained using direct measurements.9 We chose to use brachial pressure rather than pulse wave velocity because brachial pressure is more widely available and easier to use.

JACQUELINE N ADAMS
Department of Cardiology
Glasgow Royal Infirmary,
Queen Elizabeth Building,
Alexandra Parade, Glasgow G31 7ER

STEVEN WALTON
Department of Cardiology
Abdern Royal Infirmary,
Forres Street, Aberdeen


Is aspirin safe for patients with heart failure?

Sin.—The prophylactic benefit of aspirin may have been overstated not only in coro-
ary heart disease, as Cates et al., 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,1 and that aspirin may sometimes be perceived to have a prophylac-
tic benefit because some NVAF patients may benefit from even non-carrier thrombo-
embolism that are more amenable to risk modification by aspirin than by warfarin.2

Furthermore, the risk/benefit profile of antithrombotic therapy might be more favourably disposed towards aspirin than towards warfarin in high-intensity3 than in low-intensity4 anticoagulant regimens. For thromboembolic prophylaxis, the principal disadvantage of aspirin is the unpredictabil-
5 Stroke Prevention in Nonrheumatic Atrial Fibrillation Investigators. Warfarin versus aspirin for pre-
vention of thromboembolism in atrial fibril-
6 Ziegler M, Schmeling S, Wellmann KE, et al. Warfarin in the prevention of stroke associ-

Am Heart J 1995;130:387–95.
3 Kanter MC, Tegler CH, Pearce LA, et al. Carotid stenosis in patients with atrial fibril-
5 Stroke Prevention in Nonrheumatic Atrial Fibrillation Investigators. Warfarin versus aspirin for pre-
vention of thromboembolism in atrial fibril-
6 Ziegler M, Schmeling S, Wellmann KE, et al. Warfarin in the prevention of stroke associ-
Serum lipids four weeks after acute myocardial infarction are a valid basis for lipid lowering intervention in patients receiving thrombolysis.

M. J. Bennie

Heart 1996 75: 213-214
doi: 10.1136/hrt.75.2.213-a