LETTERS TO THE EDITOR

Scope
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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 more than 400 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

Siri—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 sympathetically 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 recipient 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 is exaggerated by the effort 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 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 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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Suir—Carlsson et al suggested that serum lipids should be measured 4 weeks after acute myocardial infarction.3 We are surprised by the lack of reinnervation after cardiac transplantation because there were no significant differences between these values and those obtained within 24 hours of onset of symptoms.2 This may in fact not be valid if late measurements were taken after and not before thrombolysis, because thrombolysis itself may be associated with a small but significant fall in total cholesterol.3 The authors should confirm the serum lipid values until several months later.2 The danger of course is that some patients with spurious low total cholesterol values 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 strategy of measuring serum lipids for lipid measurement when intravenous access is secured for thrombolysis is easily applied, yields adequate 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:

Suir—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. The authors 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 of less than an hour after the time lost when patients wait 3-6 months before start-
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