

Correspondence

Radionuclide measurements of diastolic function for assessing early left ventricular abnormalities in the hypertensive patient

Sir,
Caruana *et al* reported changes in the radionuclide measurements of diastolic cardiac function in hypertensive patients (1988;59:218-26). Although I agree completely with their findings I am concerned about the interpretation of these findings. In their summary they state that "The occurrence of diastolic abnormalities when systolic function is still normal may mark an early stage in the development of hypertensive heart failure." This statement, and the introduction to the paper suggest the presence of an intrinsic cardiac problem, evident as a reduced compliance.

No proof for such a cardiac abnormality is yet available. On the contrary, it has been shown that the changes in the indices of diastolic function and their "improvement" by certain drugs are secondary to alterations in loading conditions, as well as to other factors such as the sympathetic tone.^{1,2} The finding that the impairment of indices of diastolic function precedes even the development of myocardial hypertrophy³ also supports the hypothesis that these changes are secondary to alterations in loading conditions. I believe that this point should have been emphasised.

Adam Schneeweiss,
Possbachhöhe 10,
6204 Taunusstein 4 (Seitzenhahn),
Federal Republic of Germany.

References

- 1 Fouad FM. Left ventricular diastolic function in hypertensive patients. *Circulation* 1987;59:218-26.
- 2 Plathnick GD, Kahn B, Rogers WJ, Fisher ML, Becker LC. Effect of postural changes, nitroglycerin and verapamil on diastolic left ventricular function as determined by radionuclide angiography in normal subjects. *J Am Coll Cardiol* 1988;12:121-9.
- 3 Antonelli G, Sabato G, Tota F, *et al*. Left ventricular diastolic filling in subjects with hypertensive pressure response to handgrip and normal myocardial mass [Abstract]. *Eur Heart J* 1988;9(suppl 1):81.

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

Sir,
In our study we carefully defined and validated radionuclide techniques for measuring diastolic left ventricular function. We also showed that the peak filling rate did not separate controls from those with hypertension as previously suggested.^{1,2} There was considerable overlap of indices of diastolic function between controls and patients with hypertension; this was not found between controls and those with chronic stable angina.³ Thus the finding of diastolic dysfunction in hypertensive patients should be interpreted with caution.

Indeed, we feel that the last paragraph of our summary is valid. The hypertensive group was divided into two, one with normal and the second with abnormal electrocardiographic changes. Blood pressure was not significantly different in these two groups. Despite equal "loading" conditions, patients with abnormal electrocardiography had worse diastolic left ventricular function. Therefore, some other factor other than hypertension produced these abnormal findings. In the absence of clinically significant coronary artery disease one may, therefore, postulate that some hypertensive patients are liable to intrinsic diastolic left ventricular dysfunction, which may be related to calcium overload and left ventricular hypertrophy. Finally, Dr Schneeweiss's comments are further invalidated, because he alludes to a paper where alterations in diastolic function were induced by short term intervention with drugs in normal volunteers⁴ rather than in hypertensive patients. This fact has little or no bearing on our study.

A Lahiri,
E B Raftery,
Northwick Park Hospital and Clinical Research Centre,
Watford Road,
Harrow,
Middlesex HA1 3UJ.



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A Schneeweiss

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