stiffness modulus \((r = 0.53; P < 0.001)\). Moreover, only \(E/E'_{\text{lat}}\) discriminated HFNEF patients from age-matched controls possibly because of aspecific ageing-induced elevation of \(E/E'_{\text{sept}}\). 6

In conclusion, the consensus document prefers \(E/E'_{\text{ave}}\) over \(E/E'_{\text{sept}}\) because both predict LV filling pressures equally well in patients with heart failure, 2 because use of \(E/E'_{\text{ave}}\) also reveals a value of 15 to be the optimal cut-off for detection of high LV filling pressures, 3 and because \(E/E'_{\text{sept}}\) fails to discriminate between HFNEF patients and controls. 5 It is evident that the recommendations of the consensus document will have to be prospectively tested in clinical practice and adjusted accordingly. A critical comparison between \(E/E'_{\text{ave}}\), \(E/E'_{\text{sept}}\), and \(E/E'_{\text{lat}}\) could be a valuable adjunct of such prospective testing.

References


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doi:10.1093/eurheartj/ehm381

Online publish-ahead-of-print 10 October 2007

Is subendocardial ischaemia present in patients with chest pain and normal coronary angiograms? A cardiovascular MR study

I read with interest the recent article by Vermeeltoft et al., 1 on the use of dynamic contrast enhancement in MRI to evaluate the possible presence of subendocardial ischaemia in patients with chest pain and normal coronary arteries (‘syndrome X’). Their conclusion, that they found no evidence of subendocardial hypoperfusion with adenosine stress in these patients, is in striking contrast to a previous report by Panting et al., 2 who found evidence of a failure of the perfusion of the subendocardium to increase significantly with adenosine in patients with syndrome X, using similar MRI methods. The question thus arises as to how to account for this apparent difference. There are several differences in the studies that may contribute to this apparent discrepancy.

A first difference is the reported presence of a prominent ‘dark rim artifact’ obscuring the endocardial region during the early enhancement phases in the Vermeeltoft study. While not explicitly commented on in the Panting study, the images presented in that article do not show prominent artifacts. While overall similar, the imaging equipment and techniques used in the two studies were different enough that there may have been a different level of artifacts. A second crucial difference in the studies is the lack of a normal control group in the Vermeeltoft study. While they state that ‘an ischaemia-related defect . . . shows a more sustained signal loss’ than the artifact, it seems quite possible that this artifact could have obscured a true subendocardial perfusion defect, if present. Without a normal comparison group, it is impossible to know how much of a role this may have played. Third, the stress protocols were different in the two studies: Panting et al. used a 6 min infusion of adenosine, whereas Vermeeltoft et al. only used a 3 min infusion. The lower incidence of chest pain noted in the Vermeeltoft study may be related to this, and this may account for some of the differences in the observations. Fourth, the patient population may have differed significantly between the two studies, as only 25% (5/20) of the Vermeeltoft study subjects showed significant horizontal or downsloping ST-segment depression in exercise electrocardiogram, whereas this was present in 100% of the Panting study patients.

In summary, there are enough differences between the two studies that their different conclusions do not necessarily imply a fundamental inconsistency in the role of ischaemia in syndrome X patients. Further well-controlled studies will be needed to clarify this issue.

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