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

We would like to thank Professor Axel for the comments related to our study. The dark-rim artifact in cardiac first pass perfusion images has been related in the literature to a number of potential sources; cardiac motion of the heart walls,1 interface of susceptibility change,2 and Gibbs ringing.3 The intensity of the artifact due to motion is determined by the temporal resolution of the acquisition in combination with the image resolution, k-space order, and extent of motion. Spatial and temporal resolution and k-space order were comparable or better for our study compared to the study by Panting et al.4 The influence of the interface of susceptibility is determined by the applied pulse sequence technique, the echo time, voxel size, and contrast dose. Both studies used a spoiled gradient echo pulse sequence and similar settings for the contrast agent application. The settings for the voxel size and echo time were actually slightly better in our study. Finally, Gibbs ringing is mainly determined by the spatial resolution; again this was not worse in our study compared to the study of Panting et al. Considering this discussion on the imaging techniques applied in both studies, we do not expect to have significantly more dark-rim artifacts in our study compared to the study by Panting et al.

The goal of our study, however, was to measure the extent and frequency of a possible decreased subendocardial perfusion reserve with CMR in patients with syndrome X and not to evaluate possible artifacts. Despite the presence of artifacts, we found a clear and significant rise of the myocardial perfusion index (MPI) in the subendocardial region of all our patients without any evidence of subendocardial ischaemia. Imaging of a control group would have increased our understanding of artifacts but would not change the findings in our patients.

We dispute the argument of Axel that a shorter adenosine infusion may account for the differences between both studies. Maximum coronary flow occurs at an average of 84 s with a range of up to 125 s following the onset of intravenous adenosine infusion.5 Therefore, we consider our adenosine protocol sufficient to induce a steady state of maximal hyperaemia. This is illustrated by the 82% increase of the subepicardial MPI during adenosine infusion in both our patients and the patients group of Panting et al.

Finally, we agree with Axel that patient selection is different from the earlier study. In our study, more patients with syndrome X had an abnormal myocardial SPECT result, whereas in the study of Panting et al. more patients showed an abnormal ECG during exercise. However, the selection of syndrome X patients using both exercise-ECG and SPECT is an accepted method.6 All our patients with an abnormal exercise ECG had an increase of their subendocardial MPI, and a normal MPRI.

In conclusion, we consider it unlikely that the differences mentioned fully explain the contrast of results between the two studies. We agree with Axel that more studies are necessary.

References

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angioography. Small sample size of the coronary angiography group (n = 9) in the present report might be the cause of this insignificance.

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Online publish-ahead-of-print 7 September 2007

Albuminuria and heart failure: is it an albuminuria or the hypertension?

Ingelsson et al. elegantly emphasized albuminuria as a risk factor for heart failure in elderly hypertensive men and present thought-provoking speculative mechanisms. However, the association between albuminuria and heart failure was seen in untreated hypertensive men compared to treated hypertension, suggesting that treated hypertensive men had advanced hypertension and thereby hypertensive heart disease with natural progression to heart failure, and therapy likely attenuated both the progression of albuminuria and heart failure. In the 40 untreated hypertensive men who developed heart