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, interface of susceptibility change, and Gibbs ringing. 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. 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. 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-E CG and SPECT is an accepted method. All our patients with an abnormal exercise ECG had an increase of their subendocardial MPI, and a normal MPI.

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


Ilse Vermeltfoort
VU University Medical Centre Nuclear Medicine and PET Research de Boelelaan 1117
1007 MB Amsterdam
The Netherlands
Tel: +31 20 4444214
Fax: +31 20 4443090
E-mail address: I.vermeltfoort@vumc.nl

Pieter Raijmakers
Department of Nuclear Medicine & PET Research
VU University Medical Centre de Boelelaan 1117
1007 MB Amsterdam
The Netherlands

Mark Hofman
Department of Physics and Medical Technology
VU University Medical Centre de Boelelaan 1117
1007 MB Amsterdam
The Netherlands

Bert van Rossum
Department of Cardiology
VU University Medical Centre de Boelelaan 1117
1007 MB Amsterdam
The Netherlands

E-mail address: m.hofman@vumc.nl

Acute chromosomal DNA damage after radiation exposure

We read the article by Andreassi et al. dealing with the impact of radiation exposure on acute chromosomal DNA damage in human lymphocytes with great interest. The authors reported significantly increased chromosomal damage in lymphocytes by revealing increased percent of micronucleus in samples of second and 24th hours after radiation exposure in invasive cardiovascular procedures compared to baseline levels. In our opinion, some points of this work are not sufficiently clear.

Authors revealed significantly increased percent of micronucleus in samples of second and 24th hours after the procedure compared to baseline levels in percutaneous coronary intervention, peripheral transluminal angioplasty, and cardiac resynchronization therapy groups, and the discussion of the current report was chiefly based on the influence of radiation exposure on acute chromosomal DNA damage. However, in order to reveal a pathophysiological link between radiation exposure and acute chromosomal DNA damage, the correlation between change in chromosomal DNA damage and magnitude of radiation exposure such as fluoroscopy time, effective dose, or dose-area product would have been assessed.

Percutaneous coronary interventions have been reported to be related with myocardial ischaemia evidenced by post-procedural elevated cardiac enzymes. Myocardial ischaemia has been reported to be a cause of acute chromosomal DNA damage. Evaluating the influence of ischaemia on acute chromosomal DNA damage by analysing the correlation between the magnitude of chromosomal DNA damage and the serum levels of cardiac markers in percutaneous coronary intervention group would aid in excluding a potential cause of acute chromosomal DNA damage.

Finally, but not least importantly, in the present report authors could not find significant change in acute chromosomal DNA damage in coronary angiography group inconsistent with a recent report revealing significantly increased lymphocyte DNA damage in 54 patients undergoing coronary angiography.