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The Authors' reply We appreciate Dr West's comments on our paper[1]; he has given us the opportunity to clarify some points that seem unresolved.

First, we confirm that the analysis was conducted as intention-to-treat. We fully agree with Dr West that a lower rate of in-hospital death in the staged revascularisation (SR) compared with the culprit only revascularisation (COR) group is surprising. However, the study was not powered to address in-hospital mortality, rather the main study endpoint was a composite of all major adverse cardiac events (MACE) including cardiac and non-cardiac death, in-hospital death, re-infarction, re-hospitalisation for acute coronary syndrome and repeat unplanned coronary revascularisation. Thus, the imbalance of the in-hospital death rate between these two groups may be the result of an underpowered population for these events. Furthermore, we observed an unexpected lower percentage of patients treated with clopidogrel in the COR group (92%) than in the SR group (100%) that approached statistical significance. This may have increased the rate of in-hospital coronary complications (eg, stent thrombosis or re-infarction evolving to death) in the group randomised to COR.

Secondly, we used the MACE rates of the study of Qarawani *et al*[2] for sample size calculation because despite its non-randomised design, this was the most recently published study on the topic and enrolled patients in a recent era, thus providing results comparable with ours. In contrast, the study by Di Mario *et al*[3] was published in 2004 and the enrolment stopped well before 2003—that is, before the beginning of our study. Indeed, one of the strengths of our trial is that, given the relatively recent enrolment time, all percutaneous coronary interventions (PCIs) were performed using contemporary devices and treatments, thus providing an up-to-date picture of the current practice in ST-elevation myocardial infarction (STEMI). Furthermore, the study by Ijsselmuiden *et al*[4] was based on a randomised strategy but it excluded patients having PCI during acute myocardial infarction; therefore, this population is totally non-comparable with our study population of STEMI patients undergoing primary PCI.

Finally, we agree with the author that this is a very controversial field and that further larger multicentre trials are needed before there is enough evidence to change the current guidelines.

Luigi Politi, Giuseppe Massimo Sangiorgi, Maria Grazia Modena

Division of Cardiology, Policlinico Hospital of Modena, Modena, Italy

Correspondence to Luigi Politi, Division of Cardiology, Policlinico Hospital, via del Pozzo 71, Modena 41100, Italy; luigi.politi@unimore.it

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CT or MRI for post-procedural aortic stenting?

To the Editor We thank Drs Rosenthal and Bell for their insightful editorial comments[1] relating to our study.[2] There is little doubt that until we have longer follow-up on the incidence of potential adverse events following coarctation stenting, some form of advanced imaging is required, and this is endorsed by ongoing large follow-up studies.[3] Although we agree that with specific imaging techniques MRI may provide information regarding complications particularly in newer platinum stents, there are numerous reports demonstrating almost complete loss of signal with stainless steel stents when imaging with MRI.[4] Although the authors have demonstrated a case in which protrusion of the aortic wall is seen with MRI following a stainless steel stent, smaller aneurysms have been missed (A. Taylor, personal communication). In many countries including the USA, platinum stents are not available and stainless steel stents are used almost exclusively. MRI, as the authors point out, is less available than CT and this has implications for patient follow-up, requiring patients to travel to a specialised centre for imaging, and this has had implications on patient compliance in our region. As the authors also point out, MRI will not detect stent fractures; however, it is not true that it is only the complications of these that require intervention. It is our practice to re-stent in the setting of a circumferential stent fracture and this may be missed with MRI. Also, the ability of MRI to demonstrate increased flow velocity distal to the stent is very dependent on where the restenosis occurs within the stent and where the velocity sample is acquired below the stent. CT offers excellent in-stent imaging, allowing preprocedural planning of further intervention and limiting unnecessary catheter procedures. It is beyond argument that MRI offers more functional data on left ventricular dynamics; however, this requires time and cost and is not usually indicated in the setting of specific post-stent follow-up imaging. We fully accept the radiation doses associated with CT and the authors are correct to point out that this dose is cumulative; however, the ultimate goal of a screening tool should be to provide sensitive and specific data to guide further management. MRI may provide this in selected cases, but it is questionable whether it will do so over the general population and over the range of stents used in coarctation of the aorta, and thus we continue to advocate the use of CT with continued efforts to minimise radiation doses.

Damien P Kenny, Mark Hamilton, Rob Martin

Bristol Childrens Hospital, Bristol, UK

Correspondence to Dr Damien P Kenny, Bristol Childrens Hospital, Upper Maudlin Street, Bristol BS2 8BJ, UK; damienkenny@doctors.org.uk

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The Authors' reply

Luigi Politi, Giuseppe Massimo Sangiorgi and Maria Grazia Modena

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Notes