Furthermore, perhaps some lipoprotein (a) phenotypes do not contribute to thrombogenesis and as a result, some patients with high lipoprotein (a) levels may not be at ‘high risk’ for thrombogenesis: some evidence for such a dissociation has already been provided by population comparisons. For example, Afro-Caribbeans, despite having lower rates of coronary artery disease[6], have higher lipoprotein (a) levels than white caucasians, despite having lower rates of coronary artery disease[6].

Thus, our group has been unable to show significant interactions between lipoprotein (a) and thrombogenesis in patients with left ventricular dysfunction, despite this patient group being at high risk for sudden death and thrombus-related complications. If any relationship does exist, it may be weak and masked by the strong genetic influence on lipoprotein (a) levels and phenotypes.

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Stress-induced ST-segment elevation on Q leads after myocardial infarction

We read with great interest the paper by Mezlis et al[1] which relates stress (exercise and dobutamine)-induced ST-segment elevation on Q-leads shortly after myocardial infarction to myocardial viability. According to previous works[3], it is suggested that this finding is a good predictor of myocardial viability and late improvement in the infarcted area. Some points should be clarified before accepting this conclusion:

No mention is made in the paper of the recent controversy on the significance of this electrocardiographic sign. The authors seem to think that current literature links exercise-induced ST-segment elevation on Q-leads with myocardial viability; obviously it is not the case[5].

As in a previous report[7] with similar results, the study group of Mezlis et al. is composed of patients with small infarctions (score index 1.3–1.68) and a high probability of viable myocardium (all patients were referred to revascularization by the cardiologist; 82% of cases had a positive exercise test; contractible reserve or remote ischaemia was observed in 98% of patients). Data about baseline ST segments are not reported; in the work of Margonato et al[2], patients with baseline ST-segment elevation (in general, those with more severe systolic dysfunction and less viability[4,5]) were excluded. Taking into account the lack of patients with severe dysfunctions (those in whom a viability study is indicated) and the highly selected study group (patients referred to revascularization by the cardiologist in charge, probably because of evidence of either ischaemic or viable myocardium) the relationship between stress-induced ST-segment elevation and myocardial viability, suggested by the authors, could not be generalized to all post-infarction patients.

We recently analysed the same issue in 51 consecutive patients shortly after myocardial infarction[1]. Regional wall motion, contractible reserve, left ventricular volumes and late improvement were quantitatively determined. Patients with baseline ST-segment elevation (n=36) were not excluded. As in previous works, exercise-induced ST-segment elevation was not a predictor of contractible reserve[4,5]; moreover, patients without ST-segment elevation had a greater response to dobutamine. Nevertheless it was not related to late improvement or remodelling. Cases without baseline ST-segment elevation but with exercise-induced ST-segment elevation had the greatest improvement with
dobutamine; the profile of these patients is probably similar to the groups analysed by Mezlis et al. and Margonato et al. Unfortunately this was a small subgroup (8% of cases in our consecutive patients) with small dysfunctions and a high pre-test probability of viability.

The apparently discordant conclusions regarding the significance of stress-induced ST-segment elevation probably reflects differences in patient populations[3]. When considering patients post-infarction as a whole, this finding does not seem to be related to late improvement, particularity in cases with severe dysfunctions[5] (those in whom it would be useful). The results of Mezlis et al. should be considered in selected populations (small infarctions, high probability of ischaemia) with a similar profile to their study group.

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A reply

Dr Bodi’s comments based on his previous study[1] are certainly interesting and useful; however, his first statement seems rather unjustified, as the controversy regarding the significance of ST segment elevation in the literature is mentioned in the very first sentence of our study. It is obvious that our work was based on the hypothesis, supported by recent data[4–5], that stress-induced ST segment elevation after acute myocardial infarction may indicate viability. In an attempt to clarify some of the issues raised in his letter, some points should be stressed.

• The absence of patients with severe left ventricular dysfunction in our study, due to ethical considerations, is mentioned extensively in the limitations section. We agree with Dr Bodi that viability studies are of greater importance in this subgroup of patients, so we carefully avoided extrapolating our results to all post-infarction patients. However, in a recent study, Schneider et al.[4] investigated patients with significant left ventricular dysfunction (wall motion score index of 3·1–3·0) and they concluded that exercise-induced ST segment elevation yields a high probability of improvement of myocardial function post revascularization.

• In our opinion, the discordant results regarding the significance of exercise-induced ST segment elevation in the early post-infarction period, reflect not only differences in patient selection but also mainly differences in methodology. The mechanism of functional recovery after revascularization may be different to that of spontaneous recovery of function. The latter, although not representative of pure stunning, is associated mainly with stunned myocardium, whereas the former represents mainly hibernation, which is associated with greater evidence of myocardial ischaemia. Therefore, our results are not necessarily applicable to different settings, such as spontaneous recovery of function.

• In conclusion, current literature does not justify using exercise-induced ST segment elevation as a screening test for revascularization for all post-infarction patients, but only for patients to whom we refer in our study, especially for those with a first, uncomplicated myocardial infarction. Therefore, the conclusions Dr Bodi draws from our study, as well as from other similar studies, could be misleading. We believe that there is now enough data to support the hypothesis that if the patients included in the study by Bodi et al.[1] were revascularized, the conclusion of the latter could be totally different.

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