Letters to the Editor

A reply

We thank Dr Ronnevik for his comments on our article[1]. Early works, such as the one published by Ronnevik and Von der Lippe[2] are very valuable because they alert us to the real value of exercise testing.

One of the main conclusions of the study of Ronnevik et al[2], namely that work capacity is associated with poor outcome in patients suspected of having myocardial infarction, fits well with our results and has been illustrated by the early meta-analysis[3], as pointed out by Ashley and Froelicher[4]. Furthermore, the weak prognostic value of the presence of ischaemia during the test has been assessed both pre and post the thrombolytic era[1,3]. Our study, of a large unselected population with confirmed myocardial infarction[1], adds to current knowledge demonstrating that prognostic associations are present after 15 years follow-up. Similar to the cohort followed by Ronnevik et al.[2], the inability to perform the test is an even stronger predictor of poor outcome than information obtained during the test.

In our opinion it is worth highlighting the poor prognosis of patients unable to perform an exercise test[1,2] shortly after a myocardial infarction so that more can be done to improve and prolong their prognosis.

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References


Disent from the consensus on the redefinition of myocardial infarction

Many clinicians will agree with the views of the dissent[1] that the consensus redefinition of myocardial infarction[1] ignores many fatal cases, will cause diagnostic confusion and chaos in mortality and morbidity statistics, and will result in problems for insurance and subsequent employment of surviving patients.

Most clinical diagnoses of myocardial infarction have been based on the ‘two out of three’ criteria using total creatine kinase (CK) activity as the serum marker. The reasons for the lack of specificity of CK are well understood, and the two out of three criteria are sufficiently robust to allow conformity of diagnostic criteria among different observers. Consequently nearly all knowledge derived from clinical trials and observational studies of myocardial infarction has been derived from these criteria.

As well as removing some fatal cases (because troponin levels do not become elevated until about 6 h after the onset), reliance on troponin will add many new ones. Quite apart from elevations caused by coronary angioplasty, unstable angina is now more common in hospitals than myocardial infarction, and troponin but not CK levels are raised in 30–40% of these[3,4]. The pathogenesis of these fatal cases which will now be labelled myocardial infarction is almost certainly different from that of completed infarction as previously defined.

As the consensus document states[2], tiny infarcts occurring after angioplasty are probably caused by microemboli from disruption of an unstable atheromatous lesion. Patients who