cardiovascular disease. Prevention of an acute coronary event is certainly preferable to a relatively better outcome thereafter.

References


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Heinz Joachim Buettner
Interventional Cardiology
Herz-Zentrum Bad Krozingen
Su¨dring 15
D-79189 Bad Krozingen
Germany
Tel: +49 7633 4020
Fax: +49 7633 402409
E-mail address: achim.buettner@herzzentrum.de

Christian Mueller
Department of Internal Medicine
University Hospital Basel
Switzerland
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**Recurrent angina and the problem of inadequate/inappropriate revascularization**

Recurrent or persistent angina following revascularization procedures, whether this is CABG and/or PCI, is a relatively frequent, and surely challenging, clinical event. In a recent issue, Abbate *et al.* provided an effective and complete review on this problem; however, we believe that one further comment should be made regarding the 'coronary' causes of this clinical condition.

The concept behind every revascularization procedure is that evidence of inducible ischaemia in the myocardial territory downstream to a functionally significant stenosis can be demonstrated. However, it has to be acknowledged that there are at least two limitations to the clinical application of this concept in the real world: first, the spatial resolution of non-invasive tests, particularly in the case of multivessel disease. Secondly, while being considered the gold standard, coronary angiography is far from being a perfect tool for the investigation of stenosis. Angiography systematically underestimates eccentric stenoses, and, most importantly, provides no information regarding the functional importance of a coronary lesion (i.e. the existence of dynamic component and/or the cumulative haemodynamic effect of multiple or long lesions). The systematic use of more sensitive and specific tools, such as intravascular ultrasound and, particularly, the study of fractional flow reserve, dramatically reduces the quote of patients who would otherwise be categorized in the group of those with suspected ischaemia and no evidence of coronary artery disease at angiography."

From this perspective, until these techniques will be more systematically used, patients with recurrent angina will often happen to belong to one of two categories: (i) those who received treatment for lesions that were significant at angiography, but were not functionally significant (i.e. lesions with a fractional flow reserve >0.75) and (ii) patients who did not receive treatment for lesions that were functionally, but not angiographically, significant (i.e. fractional flow reserve <0.75). In the first group, the persistence of angina could be because of microvascular disease or other non-coronary causes as described by Abbate *et al.*; in the latter, to the failure to treat a source of ischaemia (i.e. inappropriate revascularization). We have to admit that such ‘failures’ are not remote possibilities as much as a daily clinical problem for interventional cardiologists. Therefore, we feel that the study of fractional flow reserve should be encouraged when re-evaluating patients for recurrent angina (Table 3 of Abbate *et al.*).

In sum, we would like to suggest that among the coronary causes of recurrent angina (Table 2 of Abbate *et al.*), besides incomplete revascularization, physicians should also be aware (and beware) of inappropriate revascularization. Admittedly, this condition is often the result of our incapacity, given the current technologies and the cost of more accurate technologies, to identify adequately the functional significance of angiplasty in moderate coronary stenoses.
limitations, were used to establish its clinical role.2 Actually, FFR had 76% sensitivity and 76% specificity in comparison to imaging studies.

Moreover, there is incomplete correlation between ICUS and FFR, as Takagi et al.3 reported that differences in ICUS minimal lumen area explain only 62% of the variability in FFR measurements.

Thus, clinical decision can be helped by non-invasive or invasive tests, but most complex decisions still rely on comprehensive clinical judgement. Nonetheless, appropriate supportive diagnostic tools should certainly be encouraged to resolve equivocal or uncertain circumstances.

References

Enoxaparin and ST elevation myocardial infarction
The ExTRACT-TIMI 25 investigators conclude that enoxaparin is superior to intravenous unfractionated heparin in patients with ST elevation myocardial infarction undergoing fibrinolysis, with either a fibrin-specific agent or streptokinase.1 Since intravenous unfractionated heparin has not been demonstrated to be superior to placebo in this context,2 the relevance of this finding to clinical practice is not clear. The investigators cite a review3 as evidence that enoxaparin is efficacious for patients with ST elevation myocardial infarction treated with fibrinolysis and aspirin. In fact, this review2 concludes with the statement 'Nor is there good evidence that, among patients who are given aspirin and fibrinolytic therapy, the routine addition of either intravenous or subcutaneous heparin produces any worthwhile improvement in outcome'.

The pertinent clinical question is: does the addition of enoxaparin to standard therapy (fibrinolysis plus aspirin) for ST elevation myocardial infarction improve outcomes? Unfortunately, this question remains unanswered. We cannot assume that intravenous unfractionated heparin is a surrogate for placebo. The small number of studies that have compared unfractionated heparin with placebo have been underpowered to detect any likely benefit either individually or as part of a meta-analysis.4 Therefore, whether unfractionated heparin is slightly better or worse than placebo remains unknown. The finding that enoxaparin is marginally superior to unfractionated heparin does not allow us to conclude that it is superior to placebo.

References