have been sicker than control patients. Patients with schizophrenia are at higher risk for medical illnesses than people in the general population. The risk of atherosclerosis and cardiac death in these patients is markedly higher and parallels the severity of the disease.6

Last but not least, one should mention the role of the treating physician in the unwanted occurrence of abnormal QTc prolongation and SCD, mostly resulting from torsade de pointes (TdP). There is no question that the most effective way of preventing complications due to drug-induced QTc prolongation is to avoid the use of these drugs. However, there are many QTc-prolonging drugs which have certain therapeutic advantages which the physician usually does not want to miss. In these cases, a systematic approach seems advisable. First of all, the physician prescribing such a drug must be familiar with the problem of drug-induced TdP. Moreover, the physician should also know that the risk for abnormal QTc prolongation and TdP varies between patients; although some patients may have a high propensity to the development of drug-associated TdP, the risk may be low in others. Risk factors for the occurrence of the arrhythmia have been identified (e.g. female gender, bradycardia, and hypokalaemia). Thus, although the role of regulatory authorities in pharmacology is crucial, we should not forget the role of the prescribing physician.

References
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percutaneous or surgical approach, depending on coronary anatomy. In fact, in all three trials supporting the benefit of invasive strategy vs. conservative strategy, independently from the possibility to identify and treat the culprit lesion, an aggressive revascularization to all coronary segments presenting with significant (>70% at visual estimation) stenosis was protocol-mandated. The relatively high number of patients who received coronary artery bypass grafting (CABG) at 1 year, among those in whom myocardial revascularization was found to be feasible and clinically indicated in the invasive arm of these trials (50% in FRISC II, 34% in TACTICS-TIMI 18, and 37% in RITA 3), may also indirectly confirm that a multivessel intervention was often accomplished. This is clearly more in line with a complete revascularization strategy rather than a culprit lesion-oriented approach. Yet, it is noteworthy that in the FRISC II and TACTICS trials, despite the fact that the majority of the surgical procedures were performed in patients with left main or multivessel disease early after infarction (<7 days), CABG was associated with a low-risk of mortality (=2%).

Thus, whether early intervention is undertaken in patients with NSTEMI, as currently recommended by ACC/AHA and ESC guidelines, any attempt to pursue a complete revascularization should be thoroughly carried out, well beyond and independently from the possibility to identify and treat the culprit lesion. Indeed, complete revascularization in this setting might be beneficial due to the deleterious progression of unstable plaques otherwise left untreated in the non-culprit vessel(s). The failure of current guidelines to address the issue of adequacy of coronary revascularization in patients affected by NSTEMI should be regarded as a potential source of incomprehension, and a position statement in this regard in the upcoming guidelines updates seems to be highly warranted.

References


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do:10.1093/eurheartj/ehi404
Online publish-ahead-of-print 29 June 2005

Complete myocardial revascularization: between myth and reality: reply

In the setting of unstable angina (UA) and non-ST elevation myocardial infarction (NSTEMI), by far, the most beneficial strategy is actually to identify patients at medium-high risk and treat them aggressively with intensive antiplatelet therapy, early angiography, and subsequent myocardial revascularization. This message is shared by most of the recent large-scale clinical trials that have been designed to address this issue. 1–3 Deferral of intervention for any reason does not improve the outcome of such patients. 4 Assumptions on the completeness of myocardial revascularization may only indirectly be inferred, and no absolute statement can be made in this direction on the pure basis of the prevalence of intervention in the aggressive arm.

In our review, we deliberately did not underline the necessity of a complete revascularization of patients with multivessel coronary artery disease (CAD), as the present evidence does not point in this direction. We agree that patients with UA/NSTEMI have several complex non-culprit lesions, 5 but this notion cannot be extrapolated to the belief that treatment of all plaques improves prognosis. The angiographic evaluation of non-culprit lesions in UA/NSTEMI patients is often inaccurate. The great advantage deriving from a percutaneous coronary intervention (PCI) strategy is that after the treatment of the culprit lesion, patients may undergo a functional non-invasive evaluation directed to unveil the haemodynamic significance of other lesions, although prediction of the risk of rupture remains elusive. Staged PCI might be the optimal strategy in UA/NSTEMI patients with multi-vessel CAD. 6, 7 Although it may increase catheterization laboratory occupancy, it could effectively decrease costs avoiding unnecessary treatment of all lesions.

References