ACE inhibitors decrease AMI risk and are antithrombogenic

As previously described, ACE inhibition may modify not only atherogenesis and plaque vulnerability but also triggering mechanisms responsible for disease onset.229 For example, the renin-angiotensin system may interact with fibrinolytic function,254 and ACE inhibition may influence endogenous fibrinolysis, resulting in a reduced thrombotic response to plaque disruption.255 Importantly, ACE inhibition also seems to reduce mortality and reinfarction in the presence of ?-blocker therapy, suggesting an independent therapeutic effect.228

ACE activity may contribute to the development of coronary artery disease and myocardial infarction,225 and ACE inhibition seems to reduce the risk of major ischemic events (reinfarction, cardiac death, and possibly unstable angina) by about 22% in patients with low ejection fractions,226 227 228 probably via multiple beneficial mechanisms.229 ACE inhibitors may influence both atherogenesis (plaque vulnerability) and triggering mechanisms responsible for disease onset (plaque disruption, thrombosis, and/or vasospasm). The latter are discussed below in the section on trigger reduction. The hypothesis that these drugs are antiatherogenic and prevent or slow progression of coronary artery disease is now being tested in clinical trials.