or a continuous effect. That is, is it necessary to get just above a certain level of inhibition of platelet aggregation to obtain clinical efficacy while balancing safety, or is it a more linear relationship, in which greater platelet inhibition generally results in better efficacy? Is the appropriate analogy warfarin or statins? The study of the relationship between platelet function and ischemic events, as well as bleeding, will most likely remain a fertile area of investigation for years to come.

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Rosuvastatin in Older Patients with Systolic Heart Failure

TO THE EDITOR: The Controlled Rosuvastatin Multinational Trial in Heart Failure (CORONA), as reported by Kjekshus et al. (Nov. 29 issue),[1] failed to show a reduction in major vascular events with the use of rosuvastatin in older patients with systolic heart failure. One explanatory variable may be a reduction in coenzyme Q10, which is known to be caused by statins, as the authors mention in their introduction. Coenzyme Q10 is important in mitochondrial electron transport and energy generation, and depletion of coenzyme Q10 in myocardial tissue has been correlated with an increased clinical severity of heart failure.[2] More recently, a meta-analysis of coenzyme Q10 intervention trials has shown significant improvements in ejection fraction (an increase of 3.7%; 95% confidence interval [CI], 1.59 to 5.77) and cardiac output (an increase of 0.28 liter per minute; 95% CI, 0.03 to 0.53) in patients with systolic heart failure,[3] and the randomized, controlled Q-SYMBIO study,[4] which addresses symptoms, biochemical markers, and clinical outcomes, is ongoing. Moreover, we have recently shown that coenzyme Q10 levels, but not statin therapy, are an independent predictor of total mortality in an observational study of 236 patients with heart failure.[5] Future trials incorporating a group treated with coenzyme Q10 supplementation together with a statin may be postulated to result in the improved clinical outcomes that CORONA did not show.

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THE AUTHORS REPLY: The reports on coenzyme Q10 and possible interactions with heart failure and statins are intriguing. As part of our analysis plan, we are currently analyzing coenzyme Q10 in a subgroup of the CORONA population, at baseline and 3 months after inclusion.

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