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Reply

We appreciate the comments from Drs. Bode and Simpson regarding our report on simvastatin's effect on skeletal muscle.

We agree that a crossover design would be a more optimal approach. However, we did find that the case patients and control individuals were appropriately and close to optimally matched when the present design was applied.

The inherent "clinical" differences between case and control individuals as Drs. Bode and Simpson suggested are possible but in our minds unlikely. In support for their contention, they cited a paper for finding alterations in coenzyme Q10 concentrations in patients with diabetes, but unfortunately the essential information of whether these patients were being treated with statins is lacking (1).

We do not claim to have proven that a decrease in coenzyme Q10 content is instrumental in the myalgia. Rather, we wrote that the impaired oxidative phosphorylation capacity was a plausible mechanistic explanation for the muscle pain.

A study conducted in mice showed that treatment with a lipophilic statin (atorvastatin) impaired mitochondrial activity (NADH oxidase activity) as well as ubiquinone content without an effect on mitochondrial density, compared with control mice (2). This was reversed with coenzyme Q10 supplementation (2). This study confirmed that the statin treatment itself had the negative effect on skeletal muscle (2), as we reported in our study (3). However, overall reports on the effects of coenzyme Q10 supplementation on myalgia are ambiguous (4,5), but none of these studies investigated coenzyme Q10 content in human skeletal muscle.

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Issues Needing Clarification Regarding Population Characteristics in the Analysis From the National Cardiovascular Data Registry

We read with interest the paper by Baklanov et al. (1), but we think that some issues need to be clarified regarding the population characteristics of the Prevalence and Outcomes of Transradial Percutaneous Coronary Intervention for ST-Segment Elevation Myocardial Infarction: Analysis From the National Cardiovascular Data Registry (2007 to 2011).

First, there is no information about sheath sizes used in the study groups. One of the endpoints was bleeding, and overt access site bleeding and retroperitoneal hemorrhage within 72 h of percutaneous coronary intervention (PCI) were included in the definition of bleeding. Doyle et al. (2) reported that using sheath sizes >6-F was identified as a strong independent predictor of major femoral bleeding. Trimarchi et al. (3) reported that adopting a sheath size of 8-F or larger was independently related to independent predictors of retroperitoneal hematoma after PCI.

Second, the incidence of high-risk C-type coronary lesions was significantly greater in the femoral access group compared with the radial access group. Barbash et al. (4) concluded that a type-C coronary lesion was an angiographic predictor of PCI failure for ST-segment elevation myocardial infarction. In addition, Wilensky et al. (5) reported that PCI for complex lesions was associated with increased in-hospital mortality. When considering that PCI success and mortality were defined as primary endpoints of the study, results may be affected because of this difference between groups.