The current literature consistently demonstrates that CCTA has significant prognostic usefulness; however, because of the highlighted limitations to the available published data, additional studies that include longer follow-up durations and detailed outcome reporting will further clarify the prognostic test parameters of CCTA.

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Alcohol Consumption and Risk of Atrial Fibrillation

We read with interest the meta-analysis on alcohol consumption and risk of atrial fibrillation (AF) by Kodama and coworkers (1). However, several characteristics of our prior publication that are mentioned in Table 1 of the meta-analysis are in need of clarification (2). First, the Women's Health Study is a prospective cohort study among female health professionals in the United States, not Switzerland (3). Second, the predominant AF pattern in the Women's Health Study is actually paroxysmal AF, not chronic AF, with about two-thirds of the participants with new-onset AF having paroxysmal AF. Third, AF diagnosis was not based on participants' reports, but all cases were confirmed by medical record review. Based on these corrections, the analyses stratified by these variables presented in Table 2 should be modified.

In addition, we would like to suggest an additional analysis for the authors' consideration. Although sex did not modify the relationship between alcohol and AF when the results comparing extremes of intake were combined in Table 2, the extreme levels of alcohol intake were generally lower among women as compared with men in these studies. Indeed, prior studies have suggested that the level of alcohol intake at which AF risk elevates may be substantially lower among women (about 2 drinks per day) as compared with men (about 4 to 5 drinks per day) (2,4,5). Therefore, it would be of interest to see the dose-response relationship between alcohol consumption and AF risk presented in Figure 4 stratified by sex.

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Reply

We appreciate Drs. Conen and Albert pointing out data that were mistakenly extracted from their study (1) in our meta-analysis of the relationship between alcohol consumption and risk of atrial fibrillation (AF) (2). We apologize. As a result, we carefully rechecked all data from all papers included in our meta-analysis. In addition, we found that, more precisely, AF was ascertained by electrocardiogram rather than from medical records in the study by Cohen et al. (3). Also, studies initially cited as conducted in Sweden by Ruigomez et al. (4,5) were performed in the United Kingdom.

After correcting these data, we reconducted the stratified analyses of pooled relative risk of AF for highest alcohol intake versus lowest alcohol intake according to study characteristics. Nonsignificant differences in AF risk were observed by geographic region (1.38 [95% confidence interval (CI): 1.22 to 1.56] in North America and 1.51 [95% CI: 1.23 to 1.86] in Europe; $p = 0.85$) or by the dominant AF type (1.42 [95% CI: 1.22 to 1.66] for chronic AF and 1.78 [95% CI: 1.43 to 2.22] for paroxysmal AF; $p = 0.11$). When AF was ascertained by medical records, the AF risk was larger (2.05 [95% CI: 1.20 to 3.53]) than by other methods (1.48 [95% CI: 1.29 to 1.70]), but was without statistical significance ($p = 0.34$). In total, the influences of the corrections were very slight.

We agree that it would be desirable to investigate the dose-response relationship between alcohol consumption and AF risk with stratification by sex, considering that the level of alcohol intake at which AF risk elevates may be substantially lower among women than among men (1). However, the number of eligible studies for a dose-response analysis is too few ($n = 9$) for such an investigation. This point might have been addressed as a study

limitation. Actually, we had performed multivariate regression analyses adjusting for sex of study participants but did not report the data. Significant relationships between alcohol intake and AF risk were found in both linear and spline dose-response curves ($p < 0.001$) but differences between the fits of the model were nonsignificant (R^2 0.47 and 0.48, respectively; $p = 0.93$). The coefficient for the linear term was $8.0 \pm 1.4 \times 10^{-3}$, meaning that the incremental increase in relative risk of AF per 10 g alcohol consumption per day was: $e^{10 \times 8.0 \pm 1.4 \times 10^{-3}} = 1.08$ (95% CI: 1.05 to 1.11). Therefore, we can conclude that the relationship between daily alcohol consumption with the risk of AF is explained as linear, independent of sex.

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