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Editorial Comment

# Atrial Fibrillation and Obesity

## An Association of Increasing Importance*

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Atrial fibrillation (AF) is the most common human arrhythmia. Based on the incidence trends of the past decades, projections for the future forecast a further significant increase in the prevalence of the disease (1). AF causes considerable morbidity and mortality, and its management is complex. The only available curative treatment is catheter ablation, but this technique has a moderate success rate and some potentially severe complications. Importantly, the disease also poses a large economic burden on health care systems (2). For these reasons, preventive strategies are urgently needed.

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Obesity represents another major health care problem with increasing prevalence and negative effects on the population's health (3). Obesity is associated with increased left atrial size and impaired left ventricular diastolic function and could thus theoretically lead to an increased AF risk (4). Indeed, several previous investigations identified obesity as a risk factor for the development of AF (5,6). It is noteworthy that to stress the importance of this association, investigators have emphasized the potentially modifiable nature of this risk factor. However, and despite this emphasis, data on the effect of dynamic weight *changes* on the associated AF risk have so far been scarce.

In this issue of the *Journal*, Tedrow et al. (7) present findings from the Women's Health Study concerning the effect of overweight and obesity on the incidence of AF. The Women's Health Study was a randomized trial of aspirin and vitamin E use in female health care professionals without previous cardiovascular disease. The authors provide data on the impact, not only of baseline body mass index (BMI), but more importantly of short-term BMI changes on AF risk based on updated measures during a long-term follow-up of approximately 13 years in a large population of >34,000 women.

The study corroborates the role of obesity as a risk factor for AF, showing a linear association between BMI and AF risk. Furthermore, the authors provide important novel findings. First, they convincingly demonstrate an association between short-term BMI changes and short-term changes in AF risk. As comprehensively shown, women in whom obesity developed during follow-up showed a significant 41% increase in AF risk compared with women who maintained a BMI <30 kg/m$^2$. Remarkably, this increased risk even exceeded the risk of women who were obese at baseline and remained obese. In contrast, the AF risk of women who were obese at baseline but lost weight and reached a BMI <30 kg/m$^2$ during follow-up did not differ from the risk of women who were nonobese at baseline and remained nonobese during follow-up. Although the strength of the latter analysis is somewhat limited by the relatively small number of subjects (n = 599) who lost weight during follow-up, these findings are essential because they imply a potential reversibility of the obesity-associated AF risk increase. Second, the authors calculated the proportion of the observed AF cases that were attributable to short-term BMI increases and found that this proportion was significant (18%). These results emphasize the role of weight control as one of the strategies for AF prevention and thus as a way to battle the rising AF epidemic.

As the authors correctly point out in the Discussion section, the study has both strengths and limitations. The main limitation is related to the follow-up method applied for the detection of AF episodes. At several follow-up time points, the study participants were asked to report diagnoses of AF. Those who reported an AF event were sent a questionnaire with additional questions and the respective medical records were reviewed. Numerous previous publications have shown that asymptomatic AF episodes are very frequent in AF patients (8,9). To detect at least some of them, systematic and scheduled electrocardiographic recordings are needed. Regular Holter recordings or repetitive transtelephonic short-time electrocardiographic recordings are recommended for studies with rhythm control as an outcome parameter (10). Whether such follow-up methods are necessary for the detection of incident AF in populations without previously observed AF episodes is conjecture.

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Nevertheless, we must assume that the follow-up applied by Tedrow et al. (7) was not rigorous enough for the reliable detection of asymptomatic AF episodes. A better design would have been, for instance, to obtain regular Holter electrocardiograms from all participants. However, one must acknowledge that intense rhythm follow-up methods in such huge patient populations would be associated with logistical problems and an enormous work load and that this drawback of the study is, at least to some important degree, counterbalanced by its main strengths, namely, the very large population and the long follow-up.

Another point that needs to be addressed is the selective female study population. Sex-related differences in presentation, treatment, and outcome have been reported in various cardiovascular diseases and particularly in AF (11,12). Therefore, as the authors correctly mention, the generalizability of the findings to male populations is limited. However, as discussed in the article, there are recent data from a Swedish male population that are consistent with the presented results (13).

In conclusion, Tedrow et al. (7) demonstrate that changes in body weight have a significant influence on the associated AF risk. Considering the predictions for the future development of both obesity and AF prevalence, weight control will probably become an increasingly important strategy, not only for prevention of the well-known adverse health effects associated with obesity, but also for prevention of AF.

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