prognostic value of peak oxygen consumption \( (\text{VO}_2) \) in heart failure (HF) patients (1). Lavie and colleagues present an interesting theory that the superior survival in female HF patients may be due to their higher body fat content. Although a plausible assumption at first glance, we controlled for several important variables in our study population to account for body fat composition. First, body mass index (BMI), a reliable surrogate for body fat content (2), was equivalent between genders in our study. Regardless of this, to address Lavie’s concerns, we reanalyzed our data to correct for body composition. As skin-fold measurements were not performed on our patients, we adjusted peak \( \text{VO}_2 \) values to “ideal body weight,” as calculated using the Wasserman formulae (3). We found that the gender difference in peak \( \text{VO}_2 \) persisted: peak \( \text{VO}_2/\text{ideal body weight} = 16.5 \pm 5.7 \text{ ml/kg/min in women and } 18.7 \pm 7.9 \text{ ml/kg/min in men (p < 0.002). Thus, higher percent body fat does not explain the lower peak \( \text{VO}_2 \) or improved survival in female HF patients. 

Although Dr. Lavie and colleagues speculate that the difference in body fat composition accounts for the gender differences in exercise capacity in HF patients, it is clear that the mechanisms for gender differences are much more complex. For example, myocardium responds differently between the genders to stresses such as increased afterload (4–6), and data suggest that this may be due to hormonal influences on regulation of vasculature and myocardial responses (6,7). Additionally, sudden cardiac death, which accounts for approximately 40% of HF deaths, occurs less frequently in women, potentially explaining their prolonged survival (8). Given the complex HF pathophysiology, multiple factors are likely responsible for the observed gender difference beyond just body fat composition. We hope our study will encourage additional research into the mechanism of gender differences in HF so that the best treatment decisions can be made and the best therapies can be applied to all patients.

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**Atrial Fibrillation and Clinical Events in Chronic Heart Failure**

The study by Olsson et al. (1) found that atrial fibrillation (AF) is associated with a worse outcome in patients with heart failure (HF) regardless of whether HF is associated with depressed or relatively preserved ventricular function, although those with preserved left ventricular (LV) function had better survival than did those with depressed LV function. In a series of 478 patients hospitalized with AF and HF we observed that those with preserved LV function had a similarly poor prognosis as did those with depressed LV function when adjusted for age, gender, coronary artery disease, and comorbid illness (2).

Differences in these findings might be illuminating. Whereas the CHARM (Candesartan in Heart Failure-Assessment of Reduction in mortality and morbidity) program enrolled stable HF patients, our study population were patients who had a deterioration necessitating hospitalization. We observed a steep increase in mortality shortly after the index hospitalization, suggesting that such an event is a potent marker for mortality warranting aggressive management. We also found an association between decreased mortality and statin therapy, intriguing in view of recent speculation that inflammation and fibrosis may be involved in promoting AF. It would be of interest to know whether intercurrent development of AF had a different prognosis as compared to pre-existing AF in CHARM and whether statin therapy disproportionately affects outcomes.

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**Reply**

Drs. Parkash and Stevenson relate that in their experiences the survival of patients with both atrial fibrillation (AF) and heart