EDITORIAL COMMENT

Effects of Obesity and Weight Changes on Cardiac and Vascular Structure and Function

Does the Clinical Impact Carry Any Weight?*

Carl J. Lavie, MD,† Richard V. Milani, MD,‡ Hector O. Ventura, MD

Overweight and obesity have increased in the United States and most of the Westernized world over recent decades, reaching a prevalence in the United States of well over 70% (1). Clearly, overweight and obese patients have increased cardiovascular (CV) risk factors, including hypertension, dyslipidemia, glucose abnormalities (impaired fasting glucose, metabolic syndrome, type II diabetes), and inflammation (1,2). Although a lack of physical activity is the primary cause of obesity (3–5), once obesity is manifest, physical activity and cardiorespiratory fitness progressively decline with weight gain. Therefore, it is not surprising that obesity is associated with a marked increased risk for most CV diseases.

It is well known that obesity increases the risk for hypertension and coronary heart disease (CHD), which are leading causes of heart failure (HF) (2). Not surprisingly, the prevalence of HF is markedly increased in the setting of obesity, with progressive increases in HF prevalence correlating with increasing severity of obesity (2,6–7). Other related CV diseases (e.g., atrial fibrillation) are also more common in the setting of obesity (1,2).

Recently, it was demonstrated that “metabolically healthy obesity” did not increase the risk for CHD and first myocardial infarction, but even metabolically healthy obesity was associated with a significantly increased risk for HF (8,9). This is not surprising considering the effects of obesity on cardiac structure and function (Figure 1) (2). Obesity has adverse effects on preload and afterload and on both the left and right chambers of the heart, markedly increasing overall CV risk, particularly for HF (2,10).

In this issue of JACC: Heart Failure, 2 papers utilizing state-of-the-art Doppler echocardiographic methodology demonstrate the adverse effects of obesity on cardiac structure and systolic and diastolic function, as well as on ventricular-arterial aging or stiffening (11,12). In the study by Kishi et al. (11) of data from the CARDIA (Coronary Artery Risk Development in Young Adults) study, increases in body mass index (BMI) over time, even when adjusted for other CV risk factors, was associated with adverse effects on both systolic and diastolic function over 25 years, from young adulthood to middle age. In a study from the Mayo Clinic utilizing the Olmsted County, Minnesota, study cohort, effective arterial elastance improved with control of blood pressure and worsened with greater weight gain (12). Moreover, weight gain was associated with end-diastolic elastance in both sexes, and central obesity was associated with greater age-related increases in left ventricular end-systolic elastance in women, a group particularly susceptible to HF with preserved ejection fraction (12). These studies support the

*Editorials published in JACC: Heart Failure reflect the views of the authors and do not necessarily represent the views of JACC: Heart Failure or the American College of Cardiology.

From the †Department of Cardiovascular Diseases, John Ochsner Heart and Vascular Institute, Ochsner Clinical School-University of Queensland School of Medicine, New Orleans, Louisiana; and the ‡Department of Preventive Medicine, Pennington Biomedical Research Center, Louisiana State University System, Baton Rouge, Louisiana. The authors have reported that they have no relationships relevant to the contents of this paper to disclose.
adverse effects of obesity on early cardiac and vascular structure and function, and provide ammunition for the potential of weight loss in preventing CV diseases, including HF.

Although purposeful weight loss appears especially promising for patients with more severe degrees of obesity, as we recently discussed elsewhere (2,10–12), despite the theoretical benefits of weight reduction (2,10–14), including the data reviewed in this issue (11,12), there is a paucity of data to support weight loss in overweight patients or those with mild degrees of obesity. As a result, the more recent HF management guidelines do not provide any firm recommendations for weight loss in HF, whereas increased physical activity and exercise training are recommended (15). Conversely, there are considerable data to support the crucial role of cardiorespiratory fitness in improving prognosis, suggesting that cardiorespiratory fitness is a more important predictor of prognosis than is fatness for CV-related and all-cause mortality (11,16,17). Clearly, cardiorespiratory fitness is an important predictor of prognosis in CV disease, including CHD (18), as well as in HF (19). We have demonstrated, however, that despite an obesity paradox in CV patients (CHD and HF) with low levels of cardiorespiratory fitness, in which unfit, lean patients with CHD or HF have a worse prognosis than do heavier, unfit patients, in those with preserved cardiorespiratory fitness, prognosis is considerably better regardless of weight. These data demonstrate that cardiorespiratory fitness markedly alters the relationship between adiposity and prognosis in patients with CV diseases, including HF.

Thus, the lack of data on cardiorespiratory fitness was a significant limitation of both of these papers (11,12) (although the CARDIA study provided data on physical activity). Nonetheless, in conclusion, we applaud both groups of authors for the intriguing observations suggesting the importance of obesity and changes in weight in improving cardiac and vascular structure and function. Long-term studies, particularly including information on physical activity and/or cardiorespiratory fitness, are needed for assessing purposeful weight reduction with diet, increased physical activity/exercise training, pharmacologic agents, and bariatric surgery on the long-term prevention and treatment of many CV diseases, including HF. Only then will we know whether the promising theoretical benefits noted in the studies from CARDIA (11) and the Mayo Clinic (12) carry any “weight” regarding clinical impact. In the meantime, efforts to promote physical activity, which improves cardiorespiratory fitness, throughout the health care system are needed (20).

**FIGURE 1** Pathophysiology of Obesity Cardiomyopathy

Central hemodynamic, cardiac structural abnormalities, and alterations in ventricular function that may occur in severely obese patients and predispose to heart failure. Left ventricular (LV) hypertrophy in severe obesity may be eccentric or concentric. In uncomplicated (normotensive) severe obesity, eccentric LV hypertrophy predominates. In severely obese patients with long-standing systemic hypertension, concentric LV hypertrophy is frequently observed and may occur more commonly than eccentric LV hypertrophy. Whether and to what extent metabolic disturbances such as lipotoxicity, insulin resistance, leptin resistance, and alterations of the renin-angiotensin-aldosterone system contribute to obesity cardiomyopathy in humans is uncertain. RV = right ventricular. Adapted with permission from Lavie et al. (2).
REFERENCES

7. Alpert MA, Terry BE, Mulekar M, et al. Cardiac morphology and left ventricular function in morbidly obese patients with and without conge

KEY WORDS fitness, LV function, obesity, weight loss