LETTERS TO THE EDITOR

Obesity, Weight Reduction and Survival in Heart Failure

We read with great interest the study by Horwich et al. (1), and while we generally agree with their intriguing findings alluding to better survival with higher body mass index (BMI) among patients with severe systolic heart failure (HF), we strongly object to their conclusions suggesting that promotion of weight loss “may even be potentially harmful.”

In a much smaller cohort of patients with moderate systolic HF who were followed for an average of 19 months (2), we noted that 83 obese patients (BMI >30 kg/m²) had fewer major events (cardiovascular death or urgent transplantation) than 142 nonobese patients (10% events vs. 17% events), although this did not quite meet statistical significance (p = 0.10). Event-free survivors had significantly greater percent body fat (26.4 ± 7.6% vs. 22.6 ± 6.7%; p = 0.02) than those with a major event. When we divided patients into quartiles of percent body fat, the lowest quartile had an annual event rate of 11% compared with 5% in those in the highest quartile (p < 0.05), again implying that body composition may predict prognosis in chronic HF (3), as is also supported by data from others (4). However, whether this relationship is merely an association or is causal is subject to debate, and we strongly suspect the former relationship. It is likely that lower body weight (e.g., lower BMI or lower % body fat) is associated with a heightened metabolic state, associated with higher levels of tumor necrosis factor and other cytokines in the “cardiac cachexia” of severe HF. Clearly, cachexia and wasting appears to be an independent risk factor for mortality in HF (5). In this regard, involuntary weight loss would likely be associated with a bad prognosis, whereas we doubt that this would also apply to purposeful weight reduction.

Clearly, obesity has been associated with numerous adverse effects on hemodynamics and cardiac structure and function, including eccentric left ventricular hypertrophy (LVH) and systolic and diastolic abnormalities, as well as a propensity for more ventricular arrhythmias and sudden cardiac death (6–10). In a study of 74 morbidly obese patients, Alpert et al. (11) demonstrated that nearly one-third had clinical evidence of HF, and the probability of HF increased with increasing duration of morbid obesity (at 20 and 25 years of obesity duration, the probability of congestive heart failure was 66% and 93%, respectively). They demonstrated significant improvements in New York Heart Association (NYHA) functional class in nearly 90% of patients who achieved marked weight reduction; these patients also had significant improvements in systolic and diastolic ventricular function. Likewise, weight reduction has been shown to significantly reduce LVH in obese hypertensives (6,7,10) and to be associated with marked improvements in exercise capacity and coronary risk factors in patients with coronary artery disease (12).

Finally, we urge caution that a “risk marker” should not be confused with a “risk factor” and sweeping conclusions not be entertained. Thus, whereas low serum cholesterol may mark a heightened metabolic state, associated with higher levels of tumor necrosis factor and other cytokines in the “cardiac cachexia” of severe HF, we strongly suspect the former relationship. It is likely that lower body weight (e.g., lower BMI or lower % body fat) is associated with a heightened metabolic state, associated with higher levels of tumor necrosis factor and other cytokines in the “cardiac cachexia” of severe HF. Clearly, cachexia and wasting appears to be an independent risk factor for mortality in HF (5). In this regard, involuntary weight loss would likely be associated with a bad prognosis, whereas we doubt that this would also apply to purposeful weight reduction.

If proven otherwise, we believe that purposeful weight reduction remains a viable therapy in the prevention and treatment of most cardiovascular disorders, including HF.

Carl J. Lavié, MD, FACC
Section of Cardiology
Ochsner Heart and Vascular Institute
1514 Jefferson Highway
New Orleans, Louisiana 70121

Richard V. Milani, MD, FACC
Mandeep R. Mehra, MD, FACC
Hector O. Ventura, MD, FACC
Franz H. Messerli, MD, FACC

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REPLY

We appreciate the interest of Lavié et al. with regard to our recent analysis of obesity’s role in the prognosis of advanced heart failure (HF) (1) and the supporting data they have provided from their own institution (2). Our study has found that obese HF patients...
have similar to improved prognosis compared with normal or underweight HF patients, and we maintain that weight reduction as a therapeutic goal needs to be more closely examined in the population of advanced HF patients.

We concur that the associations observed in our study and in the other studies in this area prevent making any firm conclusions regarding lack of benefit or increased risk of weight loss in overweight and obese patients with HF. We also noted that even if obesity is not associated with worse HF survival, weight loss may be desirable if it results in improved functional capacity and reduced symptoms. Furthermore, we noted that preoperative obesity may increase morbidity and mortality with heart transplantation, as well as make it more difficult to find a suitable donor.

With regard to mortality, based on the associations observed in our study and in the absence of clinical trials demonstrating benefit, we do not believe raising the possibility that weight loss in patients with established HF may be an ineffective or even potentially harmful treatment represents a "sweeping" conclusion. Because there is potential risk to weight-loss therapies, it does not seem unreasonable to raise the issue regarding what degree of benefit, if any, could be expected. Severe calorie restriction in patients with advanced HF has the potential to worsen cardiac muscle function (3). In addition, fad diets and pharmacologic weight-loss agents are associated with risk; for example, the pro-adrenergic weight-loss agent sibutramine may raise heart rate and blood pressure (4). Although weight loss may be helpful in the prevention of coronary artery disease and HF, the data to support a mortality benefit with weight loss in advanced HF patients is limited. No randomized controlled trials of weight reduction in HF powered for clinical end points have been conducted to date.

There is clear and compelling evidence from multiple clinical trials that medical therapies, including beta-blockers, angiotensin-converting enzyme inhibitors and aldosterone antagonists, improve symptoms and prolong survival in patients with severe, chronic HF (5). We maintain that promotion and implementation of therapies proven to be life-prolonging take precedence over therapies for which clinical trial evidence is lacking. Based on the available evidence we disagree with Lavie et al. that weight reduction is a treatment for HF until proven otherwise. Clinical trials are clearly needed to provide definitive guidance. Given the public health implications, we hope the issues we and Lavie and colleagues have raised will encourage well-designed clinical trials to address this important issue. In the absence of definite clinical trial evidence, physicians should carefully weigh both the potential risks and potential benefits of weight loss in their overweight and obese HF patients.

REFERENCES


Ischemic Cardiomyopathy: A Historical Note

I enjoyed reading the recent article on a standardized definition of ischemic cardiomyopathy by Felker et al. (1). However, I was somewhat surprised by the fact that the investigators did not make any reference to the origin of the term "ischemic cardiomyopathy." It was George Burch and associates (2) who in 1970 coined the term "ischemic cardiomyopathy," which was quite provocative as well as controversial at the time.

Actually, the concept of ischemic cardiomyopathy dated back one year earlier when Raftery et al. (3) in England suggested a cause-and-effect relationship between coronary artery disease and congestive cardiomyopathy. But the term "ischemic cardiomyopathy" was introduced and popularized a year later by Burch et al. in New Orleans (2). They insisted that "The entity, therefore, should be accepted and recognized in view of the vast similarities to the other cardiomyopathies" (2). They emphasized that "ischemic cardiomyopathy is a true cardiomyopathy" (2).

There have been many systems of classification of cardiomyopathy ever since its original description by Brigden in 1957 (4). The World Health Organization (5) in 1981 classified cardiomyopathy into hypertrophic, dilated and restrictive types. But from a practical point of view a more useful classification is an etiologic one to distinguish between ischemic and non-ischemic varieties, because the prognosis is quite different. As Felker and associates (1) mentioned, angiographically diagnosed ischemic cardiomyopathy with heart failure is associated with a shorter survival than is non-ischemic cardiomyopathy with heart failure.

We are indebted to Burch et al. (2) for their foresight in introducing and conceptualizing the term "ischemic cardiomyopathy" 32 years before its validation by the Duke Databank (1). As Burch and associates so aptly stated in 1970, "Early recognition of coronary artery disease is important so that vigorous and prompt introduction of all measures available can be instituted to improve the coronary circulation and prevent the development of muscle damage or ischemic cardiomyopathy" (2).

Gregg C. Fonarow, MD, FACC
Ahmanson–UCLA Cardiomyopathy Center
47-123 CHS 10833 LeConte Avenue
Los Angeles, California 90095
E-mail: gfonarow@mednet.ucla.edu

Tamara B. Horwich, MD
Michele A. Hamilton, MD, FACC
W. Robb MacLellan, MD, FACC
Jan H. Tillisch, MD

Tsung O. Cheng, MD
Department of Medicine
The George Washington University Medical Center
2150 Pennsylvania Avenue, NW
Washington, DC 20037

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