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.

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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).

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PII S0002-9149(02)01807-7

PII S0002-9149(02)01801-6
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Is Early Invasive Treatment of Unstable Coronary Artery Disease Equally Effective for Both Women and Men?

Lagerqvist et al. (1) are to be congratulated for their recent elaboration of the findings of FRISC II. I am sure we are all very interested in reiteration of the fact that women do not benefit as much as men from an early invasive strategy. I must take issue, however, with one suggestion that they make. They speculate that women might have done less well (in fact, women appear to have done non-significantly worse) with an invasive strategy because of more advanced age, more frequent diabetes and more frequent previous myocardial infarctions (MIs). If this were true, then these groups themselves would have been expected to do less well with an invasive strategy. In fact, all these groups—over age 65 (risk ratio [RR] 0.59), diabetics (RR 0.62) and previous MIs (RR 0.86)—appeared to benefit from an invasive strategy (2). The investigators will surely have to look elsewhere for an explanation of their tantalizing findings.

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