

The relationship between body mass index/body composition and survival in patients with heart failure

By: Shirley Shelby, [Leslie L. Davis](#), and Barbara Waag Carlson

Shirley, S., Davis, LL, & Carlson, B. (2008) The relationship between BMI/body composition and survival of heart failure patients. *Journal of the American Academy of Nurse Practitioners*, 20(6): 326-332.

Made available courtesy of Wiley: <http://dx.doi.org/10.1111/j.1745-7599.2008.00328.x>

***© American Association of Nurse Practitioners. Reprinted with permission. No further reproduction is authorized without written permission from Wiley. This version of the document is not the version of record. Figures and/or pictures may be missing from this format of the document. ***

This is the peer reviewed version of the following article: Shirley, S., Davis, LL, & Carlson, B. (2008) The relationship between BMI/body composition and survival of heart failure patients. *Journal of the American Academy of Nurse Practitioners*, 20(6): 326-332, which has been published in final form at <http://dx.doi.org/10.1111/j.1745-7599.2008.00328.x>. This article may be used for non-commercial purposes in accordance with Wiley Terms and Conditions for Self-Archiving.

Abstract:

Purpose: The purpose of this review was to summarize the literature on the relationship between obesity and survival in persons with heart failure (HF). In particular, the article examines the ways in which studies define body size/composition (body mass index [BMI], body composition, weight, cachexia, fluid retention, or albumin) and the relationship of BMI and survival after controlling for factors such as HF severity, etiology of the HF, gender, race, age, and/or time since HF diagnosis.

Data sources: The keywords heart failure and body mass index, heart failure and obesity, and heart failure and body composition were indexed in PubMed. Articles published from 1999 to 2006 that used multivariate analyses to examine the relationship between obesity and survival in persons with HF were included in the review.

Conclusions: BMI is the standard most often used for measuring body weight in patients with HF. Yet, BMI does not address other major components of body weight (fat, lean body mass, and fluid) that may factor into the mortality of patients with HF. Four of the six studies reviewed reported a positive relationship between obesity and improved survival. However, the studies are limited by design, with the majority being cross-sectional. Furthermore, most of the data were collected through secondary data analysis from patient records in the 1990s, before contemporary HF treatment was used.

Implications for practice: Until further research solidifies a clear association between higher BMIs and improved survival in patients with HF, nurse practitioners and others should continue to counsel their patients with HF who are overweight to lose weight. Assessing BMI alone as a predictor of survival for patients with HF may be misleading and should be performed in the context of other factors. Moreover, care should be taken in managing patients with HF who are cachexic because these patients have a worrisome prognosis.

Keywords: Heart failure | obesity | body mass index | systolic dysfunction | cachexia | body composition | advanced practice nurses

Article:

Introduction

The prevalence of heart failure (HF) in the United States is expected to increase from 5.2 million in 2004 (Rosamond et al., 2007) to 10 million by 2030 (Adams et al., 1999). This rise in HF has been attributed to the advancing age of the population; rising obesity; and the added stress of hypertension (HTN), smoking, obesity, high-fat/high-cholesterol diet, sedentary lifestyle, and diabetes mellitus (DM), all of which either cause myocardial damage or increase myocardial workload (Hall et al., 2005). Despite recent advances in the management of patients with HF, morbidity and mortality rates remain high, with an estimated 5-year mortality rate of 50% (Zevitz, 2005). In fact, HF is the only cardiovascular condition in which there has not been a substantial decline in both incidence and prevalence over the past 20 years (taking into account the progressive aging of the population) (Stewart, MacIntyre, Capewell, & McMurray, 2003). Thus, HF poses a great burden on the population and the healthcare system.

Nearly, a third of Americans are obese (Murphy et al., 2005). Obesity is a risk factor for HF and many other diseases, increasing the chance of developing other significant cardiovascular risk factors such as HTN, DM, and dyslipidemia. The Framingham Heart Study found that being overweight or obese was an independent predictor of HF (Kenchiah et al., 2002). In fact, overweight women have a 50% greater risk of developing HF. Further, in patients with established HF, obesity has been found to be associated with an increased risk of mortality (Eckel, 1997) and is consistently associated with left ventricular hypertrophy and dilatation (Kenchiah et al.). This prior evidence had led clinicians toward recommendations for weight loss for the obese in both the general population and those with HF (Adams et al., 2006). However, more recent research suggests that obesity may actually be associated with improved survival in persons already diagnosed with HF (Curtis et al., 2005; Davos et al., 2003; Gustafsson et al., 2005; Hall et al., 2005; Horwich et al., 2001; Lavie, Osman, Milani, & Mehra, 2003). This research suggests that clinicians may need to distinguish between weight management strategies for healthy individuals as opposed to those with HF (Curtis et al.).

This article reviews the current evidence regarding whether obesity is indeed protective in persons with HF. Specifically, the article examines the ways in which studies define body size/composition (by body mass index [BMI], body composition, weight, cachexia, fluid retention, or albumin) and the relationship of BMI and survival after controlling for factors such as (a) severity of HF (left ventricular ejection fraction [LVEF] and/or New York Heart

Association [NYHA] functional class; Table 1), (b) etiology of HF (ischemic vs. nonischemic), (c) age, (d) gender, (e) race, and (f) time since HF diagnosis.

Table 1. Criteria for NYHA functional classification in patients with HF

NYHA class	Definition
NYHA class I	No limitation in physical activity. Ordinary physical activity does not cause undue fatigue, palpitations, or dyspnea.
NYHA class II	Slight limitation of physical activity. Comfortable at rest, but ordinary physical activity results in fatigue, palpitations, or dyspnea.
NYHA class III	Marked limitation of physical activity. Comfortable at rest, but less than ordinary activity causes fatigue, palpitations, or dyspnea.
NYHA class IV	Unable to carry on any physical activity without discomfort. Symptoms of cardiac insufficiency present at rest. If any physical activity is undertaken, discomfort is increased.

Note. Adapted from HFSA 2006 comprehensive heart failure practice guideline (HFSA 2006).

Method

The keywords heart failure and body mass index, heart failure and obesity, and heart failure and body composition were indexed in PubMed. All published articles from 1999 to 2006 that examined a relationship between obesity and survival in persons with HF were examined. The abstracts of original articles reporting studies of HF, obesity, and the relationship of BMI to body composition were reviewed. Articles were included in the review if the study used multivariate analyses to examine the relationship between obesity and HF survival.

Review of the evidence: Link of BMI to survival

Six studies published between the years 1999 and 2006 met the criteria for the analysis (Table 2). Horwich et al. (2001) were the first to report the possible beneficial effects of obesity on the survival of patients with HF. They analyzed hospital records from 1203 patients with advanced systolic HF (NYHA classes III and IV = 93.9%) who were referred to a university medical clinic for heart transplant evaluation and were followed in a comprehensive HF management program. BMI was used as the primary index of obesity, with percent ideal body weight (PIBW) used as an alternative index of total body fat. To rule out edema as a confounder for accuracy of body size/composition, weights were measured upon conclusion of pulmonary artery catheter-guided therapy. This assured that the optimal hemodynamics and euvolemia had been achieved (Horwich et al.). The researchers expected that obese patients with HF would have worse mortality outcomes than those with recommended weight (20.7–27.7 kg/m² for the purposes of this study). On univariate analysis, however, low LVEF, low peak VO₂ (oxygen consumption), high pulmonary capillary wedge pressure, and low use of angiotensin-converting enzyme (ACE) inhibitors were predictors of mortality at 1 year. However, higher BMI and PIBW were not predictors of mortality. Furthermore, multivariate analysis determined that a higher BMI level was not associated with improved survival at 12 months ($p = .072$). Although a higher BMI was associated with a survival benefit at 2 years ($p = .016$), the benefit was not

sustained at 5 years ($p = .259$). When PIBW was used as an alternative to BMI, the same relationships held true (Horwich et al.).

Table 2. Selected results from studies reviewed

Study, sample size, and year of data collection	Overall result	NYHA class =n(%)	Severity of HF (LVEF)	Etiology of HF	Age (years)	Gender	Race
Horwich et al. (2001), 1203 subjects, and between 1983 and 1999	BMI and PIBW were not predictors of survival at 1 year ($p = .07$) and 5 years ($p = .259$); BMI and PIBW were predictors of survival at 2 years ($p = .016$)	NYHA II = 66 (5.5); NYHA III = 401 (33.3); NYHA IV = 729 (60.6)	Mean LVEF = 22%; LVEF > 40% were excluded	Ischemic = 48%	Average age for each category of BMI: underweight = 48.4 ± 15.4 ; normal weight = 53.2 ± 11.4 ; overweight = 52.5 ± 10.0 ; and obese = 48.5 ± 11.5	Males = 76.6%	Not reported
Lavie et al. (2003), 209 subjects, and between 1996 and 1998	Higher percent body fat and BMI were independent predictors of event-free survival; events included cardiovascular death (13) or urgent transplantation (15)	NYHA I = 21 (10.1); NYHA II = 86 (41.1); NYHA III = 102 (48.8)	LVEF = 19% for event group; LVEF = 24 ± 13 for event-free group	Ischemic = 42%	Major clinical events—with events: age = 53 ± 13 ; event-free group: age = 54 ± 12	Male = 81%	Not reported
Davos et al. (2003), 525 subjects, and between 1991 and 2000	Improved survival in noncachectic group; in the noncachectic group, quintile 4 (mean BMI: 29.2 kg/m^2) had the best 12- and 36-month survival	Noncachectic patients: NYHA I = 101 (19.2); NYHA II = 230 (43.8); NYHA III = 152 (28.95); NYHA IV = 42 (8)	Noncachectic patients: LVEF = 31.6 ± 14.8	Noncachectic patients: ischemic = 57%	Noncachectic patients: average age = 61.0 ± 12.4	Noncachectic patients: male = 83%	Not reported

Study, sample size, and year of data collection	Overall result	NYHA class =n(%)	Severity of HF (LVEF)	Etiology of HF	Age (years)	Gender	Race
Curtis et al. (2005), 7767 subjects, and between 1991 and 1993	Improved survival in overweight and obese group; possible plateau effect, with BMI > 35 kg/m ²	NYHA I = 1087 (14); NYHA II = 4272 (55); NYHA III/IV = 2408 (31)	LVEF = 31.9 ± 12.5	Ischemic = 68.9%	Age = 63.9 ± 10.9	Male = 75.4%	African American = 14.4%
Hall et al. (2005), 2707 subjects, and from 1995 to unknown	Improved survival with higher BMI groups	Not reported	LVEF ≤ 40%	Not reported	Not reported	Not reported	Not reported
Gustafsson et al. (2005), 4700 subjects, and between 1993 and 2002	Improved survival with higher BMI groups—only in those with preserved LVEF and without COPD	NYHA III and IV only	LVEF ≤ 0.35	Ischemic = 58%	Age = 72.4	Male = 61%; more males in the overweight and obese groups	Not reported

Lavie et al. (2003), who retrospectively studied 209 ambulatory patients with mild-to-moderate HF (NYHA classes I–III), examined the impact of obesity and various body composition parameters on clinical outcomes in patients with chronic systolic HF. Five body composition parameters were assessed: body surface area, BMI, percent body fat, total fat, and lean body weight. Using established methods, body surface area was estimated from the patient’s height and body weight and reported in square meters. Percent body fat was determined using the average of three skinfold measures to estimate total body fat (body weight in kilogram multiplied by percent body fat) and lean body weight (body weight in kilogram minus total body fat).

Patients with the lowest percentage of body surface area (2.0 m²), BMI (27.7 kg/m²), body fat (22.5%), total fat (19.7 kg), and lean body weight (65.5 kg) had the highest percentage of major clinical events (cardiovascular death and urgent transplantation). A higher percentage of body fat was the strongest independent predictor of event-free survival ($p = .002$). In fact, as percent body fat increased by 1%, the likelihood of clinical events decreased by more than 13%. When percent body fat was replaced by BMI and total fat, the relationships held true. Other independent

predictors of event-free survival based on multivariate analysis were a nonischemic etiology and higher peak oxygen consumption. It is important to note that more than half (15) of the 28 major clinical events were urgent transplantation. Furthermore, the obese patients in this study were significantly younger than the lean patients ($p < .02$); yet, age, LVEF, NYHA class, and gender were not predictors of event-free survival in multivariate analysis.

Cachexia has been recognized as a risk factor for mortality, leading Davos et al. (2003) to prospectively study 589 patients with chronic HF in order to determine whether BMI had a differential effect on survival in persons with and without cardiac cachexia. Cachexia was defined as a nonedematous and unintentional weight loss of more than 7.5% over the past 6 months. The researchers hypothesized that because chronic HF appears to be an inherently catabolic state, a higher BMI might prove protective even in the absence of cachexia.

To test this hypothesis, Davos et al. (2003) compared survival rates in patients with cachexia ($n = 64$) to those without cachexia ($n = 525$). Patients with primary cachexic states (infection, malignant disorder, thyroid disorder, severe liver disease, or acquired immunodeficiency syndrome) were excluded from the study. Noncachexic patients were categorized into quintiles according to their BMI (quintile means and standard deviations were as follows—BMI at quintile 1: $22.2 \text{ kg/m}^2 \pm 1.5$; at quintile 2: $24.9 \text{ kg/m}^2 \pm 0.6$; at quintile 3: $26.9 \text{ kg/m}^2 \pm 0.6$; at quintile 4: $29.2 \text{ kg/m}^2 \pm 0.8$; and at quintile 5: $34.1 \text{ kg/m}^2 \pm 2.8$).

Improved 12-month survival was found in the noncachexic group compared to the cachexic group (OR = 2.71 [95% confidence interval (CI): 1.94–3.79]; Davos et al., 2003). In other words, cachexic subjects were almost three times more likely to die than the noncachexic subjects. On average, the cachexic group was 4 years older and had a lower LVEF (by 5.3%), worse exercise capacity, and lower systolic blood pressure (by 14.9 mmHg), compared to the noncachexic group.

In the noncachexic group, subjects who were moderately obese, those with BMI values in the fourth quintile (mean \pm SD BMI: $29.2 \text{ kg/m}^2 \pm 0.8$), had the best survival at 1 year ($p = .01$) and 3 years ($p < .0001$). When examining survival rates, the relative risk of death compared to the improved survival for the fourth quintile was 2.3 for quintile 1 ($p = .001$), 1.7 for quintile 2 ($p = .027$), 1.8 for quintile 3 ($p = .016$), and 1.5 for quintile 5 ($p = .14$). Multivariate analysis revealed that an increased BMI, a higher LVEF, and better exercise capacity (peak oxygen consumption) were independent predictors of survival, even after correcting for age, presence of ischemic heart disease (IHD) as an etiology, NYHA class, and duration of disease. It is important to note that short-term survival rates (first 2 years) and the lowest BMI quartile of noncachexic subjects were similar to those of cachexic subjects. Furthermore, authors considered the possibility of whether a U-shaped relationship existed between BMI and survival in HF, in which the very thin and the very obese had worse survival. However, the number of subjects in the extreme BMI categories did not permit for testing for a curvilinear relationship.

Curtis et al. (2005) also reported improved survival in patients with an increased BMI. The authors retrospectively studied the association between BMI and outcomes in 7767 stable outpatients with HF between the years 1991 and 1993 using the Digitalis Investigation Group trial database. Subjects were grouped according to BMI into four categories as defined by The

United States Preventive Services Task Force (2003). More than half the participants were overweight (BMI 25.0–29.9 kg/m²) or obese (BMI ≥ 30.0 kg/m²). A smaller proportion of overweight (32.4%) and obese (28.4%) patients died during follow-up than did underweight (45%) or normal weight (37.8%) patients. Thus, patients who were overweight or obese were less likely to die, with adjusted hazard ratios of 0.88 (95% CI: 0.80–0.96) and 0.81 (95% CI: 0.72–0.92), respectively, compared to those with a normal BMI (18.5–24.9 kg/m²). Whereas, the risk of death for those underweight was greater than for those with a normal BMI, with an adjusted hazard ratio of 1.21 (95% CI: 0.95–1.53). Rates of all-cause hospitalization and hospitalization because of worsening HF were similar across BMI groups (underweight = 70.6%, normal weight = 64.7%, overweight = 65.6%, and obese = 67.2%). The relationship between BMI and HF outcomes (survival and hospitalization rates) held when subjects were stratified by LVEF, HF etiology, gender, and duration of HF. Authors, however, noted a possible plateau relationship between BMI and survival among the very obese patients (BMI > 35 kg/m²) as this subgroup had an increased risk of death.

Hall et al. (2005) examined long-term survival (6 years) of hospitalized patients with HF based on BMI, controlling for age, gender, and severity of illness. In this retrospective review of patients in a 20-hospital integrated healthcare delivery system, all 2707 patients had a primary diagnosis of HF identified by ICD-9 coding or by identification of an LVEF ≤ 40%. BMI was used as the measure of body weight and was calculated using the first measured heights/weights at hospital admission. Quartiles for this study were defined as follows—BMI at quartile 1: <24.3 kg/m², at quartile 2: 24.4–28.5 kg/m², at quartile 3: 28.6–34.1 kg/m², and at quartile 4: ≥34.2 kg/m². Three-year survival rates improved with increasing BMI quartiles (quartile 1 = 57%, quartile 2 = 61%, quartile 3 = 69%, and quartile 4 = 74%). After adjusting for age, gender, and severity of HF (not defined), survival rates were better for the three highest BMI quartiles, with adjusted hazard ratios of 0.77 (95% CI: 0.63–0.94) for quartile 1, 0.70 (95% CI: 0.56–0.86) for quartile 2, and 0.77 (95% CI: 0.59–0.99) for quartile 3, compared to those in quartile 1, with a BMI < 24.3 kg/m².

Like Hall et al. (2005), Gustafsson et al. (2005) evaluated the influence of BMI and LVEF on mortality in subjects hospitalized with HF. They examined a total of 4700 subjects with new or worsening HF from the Danish Investigations of Arrhythmia and Mortality on Dofetilide HF registry. Patients were categorized into four groups based on their BMI (underweight: <18.5 kg/m², normal weight: 18.5–24.9 kg/m², overweight: 25–30 kg/m², and obese: >30 kg/m²). These subgroups are very similar to those used by Curtis et al. (2005), as recommended by The United States Preventive Services Task Force (2003).

As in the previous studies, the risk of death decreased steadily with increasing BMI from the underweight to the obese. At the end of follow-up (ranging from 5 to 8 years), after adjusting for age and gender, the risk ratios (RRs) for death were 1.56 (95% CI: 1.33–1.84) for those underweight, 0.90 (95% CI: 0.83–0.97) for those overweight, and 0.77 (95% CI: 0.70–0.86) for the obese group. However, when a subgroup analysis was performed, the overweight or obese subjects with a decreased LVEF had an excessive risk of mortality. In fact, for those with systolic dysfunction, the obese subjects had an RR of 1.21 (95% CI: 1.01–1.45) compared to those subjects of normal weight. In other words, of the overweight and the obese, only those with preserved left ventricular function (normal or high LVEF) had improved survival. Furthermore,

the authors also found that the effect of BMI on survival depended on the presence or absence of coexisting chronic obstructive pulmonary disease (COPD). Thus, this study hints that the protective effect of higher BMI status on HF survival is dependent on the presence of preserved systolic function and the absence of coexisting COPD (Gustafsson et al., 2005).

Discussion

Four studies included in this analysis found a significant positive association between obesity and HF survival; however, two of the four studies suggested that a nonlinear relationship may exist. Four of the six studies used body weight and height recorded during initial screening, and then categorized participants into BMI groups. Lavie et al. (2003) used body composition parameters (body surface area, percent body fat, total fat, and lean body weight) in addition to BMI. Horwich et al. (2001) used both BMI and PIBW while making sure that subjects were euvoletic prior to using those numbers for analysis. All studies divided their sample into BMI quartiles or quintiles that were roughly similar, allowing for comparison across studies. However, cross-comparison is challenging because ranges of data were not reported in all studies (only mean values).

Most of the studies used BMI measured at one point in time as the sole determinant of body composition. While BMI is a simple, commonly used measure for rating obesity level, it is misleading, given that BMI ignores the major components of body weight: fat, lean body mass, and fluid (Heyward & Wagner, 2004). Specifically, BMI does not take into account differences in the proportion of fat to lean body mass or the distribution of fat, a factor that previous research suggests is important for predicting HF survival (Heyward & Wagner). Furthermore, BMI overestimates body fat in people who are very muscular or who have edema. For example, it overestimates fat in young adult males compared to young adult females because of the lesser percent body fat in males. BMI also seems to be overestimated in Caucasians, when age- and gender- matched with other ethnic groups (Heyward & Wagner). Conversely, BMI underestimates body fat in people who have lost muscle mass, such as the elderly (Brigham and Women's Hospital, 2003). BMI also has limitations for individuals or populations with extremes of heights or who have very long or short limb lengths in relation to trunk measurement (World Health Organization, 1995).

Davos et al. (2003) provided the best insight into the problem because the authors specifically classified subjects in terms of cachexic states. In HF, the wasting process affects not only muscle but also fat, bone, and the heart (Anker et al., 2003). Lean body mass is usually within normal limits at the time of diagnosis, unless the patient has an underlying morbidity that causes muscle or protein loss. Depletion of lean body mass contributes to complications and exacerbations of HF. As cardiac cachexia develops near the time of death, lean body mass is nearly depleted (Poirier et al., 2006). In the study by Davos, survival of patients in the lowest BMI quintile was similar to that of cachexic patients for the first 2 years of follow-up, but long-term survival did not differ between any of the groups (Davos et al.). Thus, further studies are needed to examine changes in BMI over time and evaluate the effects of cardiac cachexia.

It is also possible that patients' BMI reflects changes in other prognostic indicators. For example, patients with sarcopenia or decreased muscle mass are known to have restricted exercise capacity

and reduced mobility, both of which are associated with increased mortality (Roubenoff, 2000). Moreover, an increase in BMI, even in the obese, may not be related to elevations in body fat but may reflect preserved or increased lean mass. In order to assess regional adiposity and fat distribution, anthropometric measurements should be used, including waist circumference or sagittal abdominal diameter (Heyward & Wagner, 2004). Clearly, further research on the topic of HF and body weight should include other measurements of the distribution of body weight.

Perhaps, the greatest limitation of the research to date when examining the relationship between obesity and HF survival is identifying potential variables that demonstrate an interaction between body size/composition and survival. In the studies reviewed, six variables (severity of HF, etiology of HF, gender, race, age, and time since HF diagnosis) were examined to determine if relationships existed between BMI and survival for persons with HF.

Patients with systolic dysfunction (associated with a lower LVEF) have a higher mortality rate (10%–15% annually) than those with diastolic HF (associated with preserved LV function, 5%–8% annually; Franklin & Aurigemma, 2005). However, for patients with HF older than 70 years, mortality rates for diastolic HF approach those for systolic HF. For patients with systolic HF, mortality rates are directly related to age and the presence of an ischemic etiology (Franklin & Aurigemma). In the six studies reviewed, all considered LVEF as a variable in their models for analysis.

Croft et al. (1999) found that patients with IHD as an etiology of HF had less favorable 6-year survival than those with HTN or combined HTN and IHD. IHD was the most common comorbid condition among white patients with HF, whereas HTN was the most common comorbid condition among black patients. It is possible that increased use of ACE inhibitors and blockers following myocardial infarction or as part of HTN management has reduced disease severity among HF patients with these conditions (Croft et al.).

Mortality as a result of HF has been shown to rise in parallel with age. The incidence of HF increases dramatically with age in both sexes, approximately doubling with each advancing decade (Kenchaiah et al., 2002). Comorbid conditions also influence survival. Gustafsson et al. (2005) found that the effect of BMI on mortality in HF appeared to depend on whether COPD and other diseases like renal insufficiency, stroke, and diabetes were present.

Gender and race have also been shown to be prognostic indicators for survival of patients with HF. Women with HF lived longer than men, with a median survival after the diagnosis of HF of 3.2 vs. 1.7 years (Kannel, Ho, & Thom, 1995). Black patients with HF had lower mortality than white patients during follow-up and slightly higher survival during each year of follow-up. Six-year survival was 19% among black men, 16% among white men, 25% among black women, and 23% among white women. After taking into account differences in age, white men had 10% greater mortality than black men (age-adjusted RR: 1.10 [95% CI: 1.07–1.13]; Croft et al., 1999). Yet, as shown in Table 2, the majority of the studies examined in our review did not report race and thus did not factor race into multivariate analyses. Additionally, males were overrepresented in the five studies that reported descriptive statistics for gender.

Conclusions

HF is the only cardiovascular condition *not* experiencing a substantial decline in both incidence and prevalence over the past 20 years (Stewart, MacIntyre, Capewell, & McMurray, 2003). Accordingly, continued research focusing on prevention and management of HF is needed. This review of the literature suggests an association between obesity and improved survival in patients with HF. However, until a mechanism is identified to explain this obesity paradox, causality cannot be satisfactorily explained. Given the limitations of using BMI alone, there need to be other ways to understand the relationship between weight and mortality as a result of HF. Further research is necessary to prospectively study within the context of contemporary HF treatment the prognostic implications of BMI and aspects of body composition, taking into consideration other complex factors such as natriuretic peptides, plasma norepinephrine, nutritional status, and caloric intake that relate to HF survival. With such studies, definitive weight loss recommendations for patients with HF will be forthcoming.

Acknowledgment

The authors thank Elizabeth Tornquist for her thoughtful editing in the preparation of this article.

Conflict of interest disclosure

No relationship exists between the authors and any commercial entry or product that might represent a conflict of interest. No incentives have been offered by any commercial entity to submit the article for publication.

References

- Adams, K. F., Baughman, K., Dec, W., Elkayam, U., Forker, A., Gheorghide, M., et al. (1999). Heart Failure Society of America: HFSA guidelines for management of patients with heart failure caused by left ventricular systolic dysfunction—Pharmacological approaches. *Journal of Cardiac Failure*, 5, 357–382.
- Adams, K. F., Lindenfeld, J., Arnold, J. M. O., Baker, D. W., Barnard, D. H., Baughman, K. L., et al. (2006). Executive summary: HFSA 2006 comprehensive heart failure practice guideline. *Journal of Cardiac Failure*, 12, 10–38.
- Anker, S., Negassa, A., Coats, A., Afzal, R., Poole-Wilson, P., Cohn, J., et al. (2003). Prognostic importance of weight loss in chronic heart failure and the effect of treatment with angiotensin-converting-enzyme inhibitors: An observational study. *Lancet*, 361, 1077–1083.
- Brigham and Women's Hospital. (2003). Obesity in women: A guide to assessment and management. Boston, MA: Author. Retrieved November 17, 2005, from www.guidelines.gov
- Croft, J., Giles, W., Pollard, R., Keenan, N., Casper, M., & Anda, R. (1999). Heart failure survival among older adults in the United States: A poor prognosis for an emerging epidemic in the Medicare population. *Archives of Internal Medicine*, 159, 505–510.

Curtis, J., Selter, J., Wang, Y., Rathore, S., Jovin, I., Jadbabaie, F., et al. (2005). The obesity paradox: Body mass index and outcomes in patients with heart failure. *Archives of Internal Medicine*, 165, 55–61.

Davos, C., Doehner, W., Rauchhaus, M., Cicoira, M., Francis, D., Coats, A., et al. (2003). Body mass and survival in patients with chronic heart failure without cachexia: The importance of obesity. *Journal of Cardiac Failure*, 9, 29–35.

Eckel, R. (1997). Obesity and heart disease: A statement for healthcare professionals from the Nutrition Committee, American Heart Association. *Circulation*, 96, 3248–3250.

Franklin, K., & Aurigemma, G. (2005). Prognosis in diastolic heart failure. *Progress in Cardiovascular Disease*, 47, 333–339.

Gustafsson, F., Kragelund, C., Torp-Pedersen, C., Seibaek, M., Burchardt, H., Akkan, D., et al. (2005). Effect of obesity and being overweight on long-term mortality in congestive heart failure: Influence on left ventricular systolic function. *European Heart Journal*, 26, 58–64.

Hall, J., French, T., Rasmusson, K., Vesty, J., Roberts, C., Rimmasch, H., et al. (2005). The paradox of obesity in patients with heart failure. *Journal of the American Academy of Nurse Practitioners*, 17, 542–546.

Heart Failure Society of America (2006). Executive summary: HFSA 2006 comprehensive heart failure practice guideline. *Journal of Cardiac Failure*, 12, 10–38.

Heyward, V., & Wagner, D. (2004). *Applied body composition assessment* (2nd ed.). Champaign, IL: Human Kinetics.

Horwich, T., Fonarow, G., Hamilton, M., MacLellan, W., Woo, M., & Tillisch, J. (2001). The relationship between obesity and mortality in patients with heart failure. *Journal of the American College of Cardiology*, 38, 789–795.

Kannel, W., Ho, K., & Thom, T. (1995). Changing epidemiological features of cardiac failure. *British Heart Journal*, 72, S3–S9.

Kenchiah, S., Evans, J., Levy, D., Wilson, P., Benjamin, E., Larson, M., et al. (2002). Obesity and the risk of heart failure. *New England Journal of Medicine*, 347, 305–313.

Lavie, C., Osman, A., Milani, R., & Mehra, M. (2003). Body composition and prognosis in chronic systolic heart failure: The obesity paradox. *American Journal of Cardiology*, 91, 891–894.

Murphy, N., Macintyre, K., Stewart, S., Hart, C. L., Hole, D., & McMurray, J. (2005). Long-term cardiovascular consequences of obesity: 20-year follow-up of more than 15 000 middle-aged men and women (the Renfrew-Paisley study). *European Heart Journal*, 27, 96–106.

Poirier, P., Giles, T., Bray, G., Hong, Y., Stern, J., Pi-Sunyer, F., et al. (2006). Obesity and cardiovascular disease: Pathophysiology, evaluation, and effect of weight loss: An update of the 1997 American Heart Association Scientific Statement on Obesity and Heart Disease from the Obesity Committee of the Council on Nutrition, Physical Activity, and Metabolism. *Circulation*, 113, 898–918.

Rosamond, W., Flegal, K., Friday, G., Furie, K., Go, A., Greenlund, K., et al.; American Heart Association Statistics Committee and Stroke Statistics Subcommittee. (2007). Heart disease and stroke statistics—2007 update: A report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. *Circulation*, 115, e69–e171.

Roubenoff, R. (2000). Sarcopenia and its implications for the elderly. *European Journal of Clinical Nutrition*, 54(Suppl. 3), 40–47.

Stewart, S., MacIntyre, K., Capewell, S., & McMurray, J. (2003). Heart failure and the aging population: An increasing burden in the 21st century? *Heart*, 89, 49–53.

The United States Preventive Services Task Force. (2003). Screening for obesity in adults: Summary of recommendations. Retrieved November 16, 2005, from <http://www.ahrq.gov>

World Health Organization. (1995). Physical status: The use and interpretation of anthropometry. Report of a WHO Expert Committee. Geneva, Switzerland: Author.

Zevitz, M. (2005). Heart failure. Retrieved November 17, 2005, from www.emedicine.com