Impact of Obesity in Patients With Congestive Heart Failure

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Obesity is a known risk factor for developing cardiovascular disease, including heart failure. However, the impact of obesity on patients with heart failure is unclear. Weight reduction is a recommended method of prevention of cardiovascular disease. However, the phenomenon of the "obesity paradox" (or "reverse epidemiology") revealed that overweight and mild to moderate obesity are associated with better outcomes in patients with heart failure compared with patients at normal or ideal weight. Even more, increases of weight in cachectic heart failure patients might improve survival, although patients with heart failure who are overweight or mildly to moderately obese have better outcomes than patients with heart failure who are at ideal or normal weight. In heart failure patients, weight reduction through diet regulation, moderate exercise, and bariatric surgery can improve quality of life and New York Heart Association functional class, but it is yet unclear if these measures will improve survival.
[Rev Cardiovasc Med. *2009;10(3):142-151 doi: 10.3909/ricm0480*]

Key words: Heart failure • Obesity • Weight reduction • Reverse epidemiology

Obesity is an alarming health concern in the United States and worldwide. In 2001, the prevalence of obesity among US adults had risen to 20.9%, which amounts to 44.3 million people.[1] Furthermore, more than 50% of the US adult population is considered to be either overweight or obese.[2] At the same time, heart failure is the fastest growing cardiovascular disorder in the United States,[3] with more than 550,000 new diagnoses each year, for a total of more than 5 million cases, a number that has tripled in the last 3 decades.[4] The annual health-care expenditure for these 2 conditions combined

is approximately $80 billion.[3,4] Heart failure has been described by Garg and colleagues[5] as the "most important public health problem in cardiovascular medicine." With increasing attention focused on obesity, surgical interventions such as bariatric surgery (gastrointestinal weight-loss surgery) are becoming popular. Hence, the role of obesity in the cause and the course of heart failure and its impact on outcome and potential therapy in heart failure patients need to be studied carefully.

Obesity as a Risk Factor for Heart Failure

Preexisting obesity has been identified as a risk factor for heart failure.[6-8] In a study by Kenchaiah and colleagues[6] using Framingham data on more than 5800 patients followed over a period of 14 years, the authors found that with every increment of 1 kg/m^2 in body mass index (BMI) (Table 1), there was an added risk of heart failure of 5% in men and 7% in women. Obese individuals were twice as likely as normal-weight individuals to develop heart failure. The population-attributable risks due to obesity for the incidence of heart failure in men and women were 11% and 14%, respectively.

The same study also found that the risk of developing heart failure in obese individuals varied with the coexistence of other cardiac risk factors. The effect of obesity on the development of heart failure is less pronounced if other risk factors, such as hypertension or myocardial infarction, dominate. For example, the hazard ratio for heart failure in normal-weight, overweight, and obese individuals with hypertension was 1.3 (95% confidence interval [CI], 1.11-1.52), whereas the hazard ratio was significantly higher, at 1.66 (95% CI, 1.33-2.07), in subjects without hypertension across the 3 different weight categories.

He and coworkers[9] observed that obesity increases the risk of congestive heart failure (CHF), with a relative risk of 1.30 and a population-attributable risk of 8%, and that obesity alone is responsible for about 1 out of 10 cases of CHF in women and 1 out of 18 cases of CHF in men. Hubert and colleagues[8] found that obesity was an independent risk factor for cardiovascular disease in the Framingham data analysis. The authors divided subjects into 3 groups based on their Metropolitan Relative Weight (MRW) or percentage of desirable weight (desirable weight was derived from the 1959 Metropolitan Life Insurance Company tables)[10] and found a 2.5- to 3-fold increase in the incidence of CHF among the group with the highest weight (MRW $\geq$ 130%) compared with the group with the lowest weight (MRW $<$ 110%). Wilhelmsen and coworkers[11] investigated a random group of about 7500 men for a period of 27 years and found that the odds ratio of developing heart failure was 1.5 times higher for men with a BMI of 27 kg/m^2 or more as compared with men below that BMI. In a cohort of healthy individuals older than 65 years, Chen and colleagues[7] reported that CHF was 1.6 times more likely to occur in those subjects with a BMI exceeding 28 kg/m^2 as compared with those whose BMI was less than 24 kg/m^2.

Obesity increases the risk of heart failure by multiple mechanisms. By augmenting the risk for hypertension,[12-14] diabetes mellitus,[14-16] and hyperlipidemia,[17] obesity increases the risk of coronary heart disease,[14] which could then lead to CHF. Hypertension and hyperlipidemia increase the risk of myocardial infarction and are independent risk factors for heart failure.[6,7,9,11] Obesity is associated with increased risk of hypertension,[12,14] which was evident in a study that screened more than 1 million Americans through the nationwide Community Hypertension Evaluation Clinic.[13] Prevalence of hypertension in the overweight group was 50% to 300% higher compared with normal-weight and underweight groups.[13] In 2 independent studies in men and women, obesity and weight gain were demonstrated risk factors for clinical diabetes.[15,16] Men with a BMI at or above 35 kg/m^2 had a multivariate relative risk for developing diabetes of 42.1 compared with men with a

Table 1
Classification of Body Weight According to BMI

BMI (kg/m^2)	WHO Classification	NHLBI Classification	Popular Terminology
$<$ 18.5	Underweight	Underweight	Thin
18.5-24.9	Normal range	Normal	Ideal/healthy/normal
25.0-29.9	Pre-obese	Overweight	Overweight
30.0-34.9	Obese class 1	Obesity class I	Obesity
35.0-39.9	Obese class 2	Obesity class II	Obesity
$\geq$ 40.0	Obese class 3	Obesity class III	Morbid obesity

BMI, body mass index; WHO, World Health Organization; NHLBI, National Heart, Lung, and Blood Institute.

BMI under 23 kg/m^2. Also, waist circumference was a better predictor of diabetes in the top 20% of the cohort.[15] Among women, the relative risk for diabetes mellitus was 1.9 with a weight gain of 5 kg to 7.9 kg and 2.7 for a weight gain of 8 kg to 11 kg compared with women who maintained a stable weight (gain or loss of $\leq$ 5 kg). In addition, the risk of diabetes was reduced by 50% or more in women who lost more than 5 kg.[16] Obesity is associated with a risk of hyperlipidemia because it increases plasma triglyceride levels.[17] Obese men have triglyceride levels that are approximately 100 mg/dL higher than normal-weight men[18]; this difference is approximately 60 mg/dL in women.[19] The Metabolic Syndrome is a known risk factor for coronary heart disease and cardiovascular disease, and it is associated with increased overall mortality. Obesity is associated with the Metabolic Syndrome and potentiates the risk of heart failure.[14] In morbidly obese individuals, an elevated hemodynamic load can also cause altered left ventricular (LV) remodeling.[14,20] Increased peripheral vascular resistance,[21] loss of aortic wall elasticity,[22] elevated preload, and hypoxic pulmonary vasoconstriction from obstructive sleep apnea may possibly contribute to heart failure.[14] Obesity is also associated with increased oxidative stress and neurohormonal activation, which play a role in cardiac remodeling and progression of heart failure. In obesity, increased metabolic demand necessitates high circulating blood volume and cardiac output.[23] Kasper and colleagues[24] found that obese CHF patients had significantly higher cardiac output compared with nonobese CHF patients (5.62 $\pm$ 1.48 vs 4.42 $\pm$ 1.38 L/min; $P < .05$), even though heart rate was not different. Increased cardiac output can lead to ventricular dilation with higher ventricular wall stress that might result in eccentric ventricular hypertrophy and subsequent diastolic heart failure. The presence of systemic hypertension further aggravates eccentric LV hypertrophy.[25] Systolic dysfunction may result, along with increased blood volume, which predisposes to left atrial enlargement and a subsequent increase in the risk of atrial fibrillation.[23]

In animal models, obesity has been associated with steatosis and lipo-apoptosis in the myocardium that might play a causative role in progressive heart failure.[14] Obesity-induced sleep apnea is also associated with events that can potentiate the risk of heart failure, such as hypoxia-induced pulmonary vasoconstriction, increased sympathetic activity, and elevated blood pressure. Furthermore, in animal models of obstructive sleep apnea, acute pulmonary edema has been demonstrated.[14] It has also been suggested that obesity increases proteinuria and renal insufficiency, which can increase the risk of comorbidities that might determine the outcome in patients with heart failure.[14] Along with the BMI, the distribution of body fat is also a major determinant of risk; abdominal visceral fat distribution has a higher cardiovascular risk compared with gluteofemoral fat distribution.[26-28]

Significance of Obesity in Preexisting CHF and the Obesity Paradox

Although obesity has been linked to an increased risk of heart failure, the exact mechanisms remain ambiguous. In obese patients who do not have CHF, it has been found that intentional weight loss can possibly prevent the development of CHF by favorably modifying risk factors for development of coronary heart disease. However, the impact of obesity on patients who have preexisting heart failure is different from that in the general population (Table 2). Davos and colleagues[29] observed a U-shaped survival curve in relation to BMI in CHF patients. CHF patients with extremely high or low BMI had worse survival compared with moderately obese CHF patients.

The authors grouped their noncachectic subjects into 5 groups of increasing BMI (Q1 to Q5) and found that the best survival was in the fourth quintile (BMI of 29.2 $\pm$ 0.8 kg/m^2). Noncachectic patients with the lowest BMI had twice the mortality rate compared with subjects belonging in the fourth quintile; the relative risk for Q1 (BMI of 22.2 $\pm$ 1.5 kg/m^2) and Q4 were 2.3 and 1.0, respectively.

The above-mentioned pattern of survival has been described as the "obesity paradox" (or "reverse epidemiology").[30-32] This phenomenon has been previously described in patients with end-stage renal disease undergoing dialysis.[33] Curtis and colleagues[30] studied 8000 patients with stable heart failure and described a difference in all-cause mortality between 45% among underweight patients and 28% among overweight patients. Using multivariate analysis, the authors demonstrated the hazard ratio for underweight, overweight, and obese patients to be 1.21, 0.88, and 0.81, respectively. Horwich and coworkers[34] categorized 1200 patients with advanced heart failure into different groups based on BMI. Higher BMI offered better survival on multivariate analysis at 1 and 2 years. The authors described obesity as an independent predictor for better survival, even when underweight subjects were excluded from the analysis. Among the 5 body composition

Table 2
Studies on Impact of Obesity in Patients With Congestive Heart Failure

Study/Author	Period of Follow-Up	Body Composition Parameter Used	Division of Study Population Depending on Body Weight	Result		Outcome	Comments
Davos et al[29]	53.0 ± 25.2 months	BMI (kg/m^2)		RR		All-cause mortality	U-shaped relation of survival with BMI
			C (21.5 ± 2.5)	2.71			
			Q1 (22.2 ± 1.5)	2.3			
			Q2 (24.9 ± 0.6)	1.7			
			Q3 (26.9 ± 0.6)	1.8			
			Q4 (29.2 ± 0.8)	1.0			
			Q5 (34.1 ± 2.8)	1.5			
Curtis et al[30]	24-48 months	BMI		All-cause mortality	HR	All-cause mortality	Higher BMIs were associated with lower mortality risk: the "obesity paradox"
			Underweight	45.0	1.21		
			Healthy weight	37.8	1.00		
			Overweight	32.4	0.88		
			Obese	28.4	0.81		
Horwich et al[34]	5 years	BMI	Underweight Recommended weight Overweight Obese	Patients with higher BMIs had better survival at 1 and 2 years but no difference at 5 years		Sudden death/ cardiovascular death/urgent transplantation	Higher BMI was associated with improved survival
Lavie et al[35]	2 years	Body surface area BMI Percent body fat Total fat Lean body fat	Percent body fat Q1 (16.4 ± 2.5) Q2 (21.3 ± 1.1) Q3 (24.9 ± 0.8) Q4 (28.9 ± 1.6) Q5 (37.7 ± 4.5)	Events 22% 18% 9% 15% 5%		Cardiovascular death/urgent cardiac transplantation	For every 1% absolute increase in percent body fat there is >13% reduction in major events
Gustafsson et al[36]	5-8 years	BMI		WMI ≤ 1.2	WMI >1.2	All-cause mortality	Patients with normal systolic function had a linear survival curve, whereas patients with systolic dysfunction had a U-shaped survival curve
			Underweight	1.11	1.66		
			Normal weight	1.0	1.0		
			Overweight	1.07	1.01		
			Obesity	1.21	0.92		

BMI, body mass index; RR, relative risk; C, cachectic; Q, quartile; HR, hazard ratio; WMI, wall motion index.

parameters, such as body surface area, BMI, percent body fat, lean body weight, and total fat, the best predictor of event-free survival in patients with systolic heart failure was percent body fat. With every 1% absolute increase in percent body fat, there was a reduction of 13% in major clinical events.[35] Gustafsson and colleagues[36] examined the effects of LV systolic function on the obesity paradox. When LV systolic function was normal or near normal, then higher BMI offered better survival. In contrast, in patients with systolic dysfunction, survival followed a U-shaped curve, with better survival in patients with normal BMI but poorer survival in patients with below-normal or above-normal BMI.

Several mechanisms have been suggested to explain why obesity may possibly be protective in heart failure patients.[34] Overweight and obese patients tend to have higher blood pressures than normal-weight patients and better tolerate after-load

reducing drugs, such as angiotensin-converting enzyme (ACE) inhibitors. Therefore, most of these patients are on long-term therapy with ACE inhibitors that are well known to improve survival in patients with heart failure.[34,37] Moreover, according to the study by Horwich and colleagues,[34] there seems to be a positive correlation between increased cholesterol levels and improved survival in contrast to the well-established risk for morbidity and mortality associated with hypercholesterolemia in the general population.[38,39] Increased levels of lipoproteins seem to be beneficial in patients with heart failure through downregulation of inflammatory cytokines.[38,40] The tumor necrosis factor-α (TNF-α) system is altered in obese patients, with increased production of both type I and type II soluble TNF-α receptors. In obese patients with heart failure, elevated levels of TNF-α receptors prevent cardiac injury through prevention of negative inotropic and proapoptotic effects of TNF-α.[41,42] Changes in the sympathetic nervous system and the renin-angiotensin system have been observed in obese patients. Weber and colleagues[43] reported that lean hypertensive patients had significantly higher levels of plasma epinephrine and renin during exercise compared with obese hypertensive patients, even though baseline levels were similar. Elevated levels of epinephrine and renin are associated with poor prognosis in heart failure, so decreased stress responses of the neuro-hormonal system might possibly explain the better prognosis seen among obese patients.[34,43,44] It has been hypothesized that moderately obese patients tolerate the metabolic stress of heart failure better than lean patients, likely secondary to an increased metabolic reserve.[14,45] Several studies have concluded that the levels of B-type natriuretic peptide (BNP) and amino-terminal cleavage fragment (NT-proBNP) are significantly lower in obese individuals[46] and thus cannot be reliably used as a noninvasive test for estimation of LV filling pressure in these patients. Although evidence is not conclusive, it has been suggested that the decreased levels of BNP and NT-proBNP are secondary to a decreased production of peptides and are not related to an increased clearance.[46]

Interestingly, most studies that used increased BMI as a predictor of improved survival in patients with heart failure did not include morbidly obese patients.[29,30,35,36,47] Some studies found better survival for obese patients with heart failure but might be corrupted by lead-time bias because obese patients might seek treatment sooner due to more pronounced functional impairment at an earlier stage.[34] In addition, some studies[34] that assessed event-free survival might be skewed because cardiac transplantation is much more common in patients with normal body weight, and morbid obesity is still considered a relative contraindication for cardiac transplantation due to worse postoperative outcomes and because of the shortage of large-sized donor hearts.[34] Another potential issue with some studies is that changes of body weight over time were not evaluated, and the data used relied upon single-point measurements of BMI. Also, CHF patients might be miscategorized as overweight or obese due to an increased BMI secondary to fluid overload during conditions of cardiac decompensation. In addition, rapid weight loss following excessive diuresis can also result in miscategorization of patients to underweight or cachexic groups.[14,48]

A few studies have demonstrated benefits of weight reduction in heart failure patients. According to Alpert and colleagues,[49] weight reduction in normotensive morbidly obese patients with and without CHF resulted in changes in cardiac morphology and function. Morphologic changes observed in morbidly obese patients with CHF were higher LV cavity size, LV end-systolic wall stress, LV mass/height index, and impaired LV systolic function and diastolic filling compared with patients without heart failure. Substantial weight loss reduced the LV mass/height index and improved LV systolic function and diastolic filling in obese patients with and without heart failure. Also, the New York Heart Association (NYHA) functional classification improved in most patients with CHF as a result of weight loss.[49,50]

The duration of (morbid) obesity is considered a strong predictor for the development of heart failure. For every 1-year increment in duration of morbid obesity, the odds of CHF increase by 1.46. Grady and colleagues[51] observed that preoperative obesity is associated with increased morbidity and mortality following cardiac transplantation. It has also been reported that weight loss by dietary methods in a morbidly obese patient with heart failure reversed the patient's severe CHF.[52] In an animal model, Kanda and coworkers[53] showed that weight loss improved survival due to the cardioprotective effects of inducing the expression of adiponectin, which might protect against the development of heart failure. Other studies showed benefits of intentional weight loss in patients with CHF in terms of improved quality of life and cardiac function.[54] According to the American Heart Association, however, no prospective studies have convincingly demonstrated that intentional weight loss in patients with heart failure is associated with improved survival.[23]

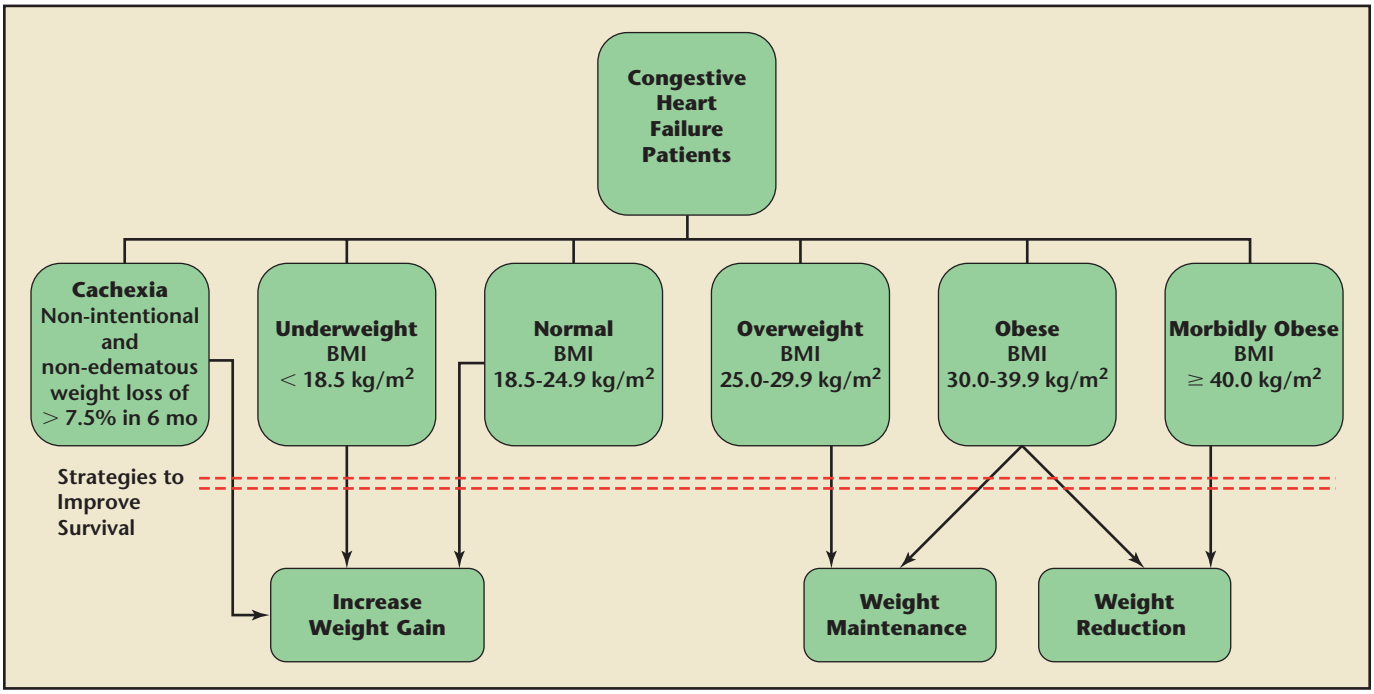

Figure 1. *Different strategies that can be used in heart failure patients in various weight categories. BMI, body mass index. Data from Alpert MA et al,[49] Kostis JB et al,[55] and McCloskey CA et al.[70]*

In general, weight reduction appears to be beneficial in morbidly obese and obese patients with heart failure. Cachectic and underweight patients with heart failure should try to gain weight to improve survival. For heart failure patients who are at normal weight or are overweight, no clear recommendations regarding potential weight loss can be made at this time (Figure 1).

Methods of Weight Reduction in CHF Patients

Kostis and colleagues[55] randomized 20 CHF patients into 3 treatment groups, each receiving 1 of the following interventions: a) combined nonpharmacological intervention, b) digoxin therapy, or c) double-blind placebo therapy. The nonpharmacological treatment included graduated exercise training 3 to 5 times per week, structured cognitive and stress therapy, and, in overweight patients, dietary intervention focused on salt reduction and weight reduction. The group that received nonpharmacological intervention achieved the most weight loss, significantly better quality of life indices, improved exercise tolerance, and elevated mood states (Table 3). As part of the dietary modification, the subjects had weekly counseling with a dietitian, and daily saturated fat and sodium chloride intake was reduced. Although digoxin improved the ejection fraction, there was no corresponding improvement in quality-of-life indices or exercise tolerance. Increased exercise tolerance, however, is associated with improved prognosis in patients with CHF.[56,57]

On the other hand, side effects of weight reduction have been reported in patients with heart failure. Weight-reducing drugs, such as sibutramine, can be harmful in patients with heart failure if they raise heart rate and blood pressure,[58] and a severe calorie restriction diet may worsen cardiomyocyte and overall cardiac muscle function in advanced heart failure.[59] The weight-reducing drugs fenfluramine and dexfenfluramine have been withdrawn from the market due to the development of pulmonary hypertension[60] and valvular heart disease.[61,62] In contrast, Beck-da-Silva and colleagues[63] demonstrated that diet counseling and therapy with orlistat promoted significant weight loss and relief of symptoms and was safe and well tolerated in patients with CHF. Only few data are available on optimal nutritional intakes and ideal food patterns for CHF patients.[64] In view of the current lack of data, nutritional management should be tailored to the individual patient's clinical conditions, needs, and progress[64] (Figure 1).

Exercise training in heart failure patients was formerly considered harmful, leading to a sedentary lifestyle that further promoted poorer exercise tolerance and worsening of symptoms, cardiac function,

Table 3
Studies Showing Benefits of Weight Reduction in Patients With Congestive Heart Failure

Study/Author	Study Population Group	Method of Weight Reduction				Results		Comments
Alpert et al[49]	Morbidly obese With CHF Without CHF	Vertical band gastroplasty				Before weight loss	After weight loss	Symptomatic improvement with weight loss was observed
			LV internal dimension (cm)			6.5 ± 0.4	−0.8 ± 0.1	
			LV end systolic wall stress (g/cm^2)			214 ± 49	−50 ± 29	
			LV mass/height index (g/m)			223 ± 42	−31 ± 18	
			LV fractional shortening (%)			23 ± 5	5 ± 5	
			E/A ratio			0.79 ± 0.25	0.25 ± 0.12	
			NYHA class					
			III			7	0	
			II			7	6	
			I			0	8	
McCloskey et al[70]	Morbidly obese with severe cardiomyopathy	Roux-en-Y gastric bypass Sleeve gastrectomy Laparoscopic adjustable gastric banding				Before surgery	6 months after surgery	Two patients who were initially denied cardiac transplant due to morbid obesity underwent successful transplant following weight loss with bariatric surgery
			Mean BMI (kg/m^2)			50.8 ± 2.04	36.8 ± 1.72	
			Mean LV ejection fraction (%)			23 ± 2	32 ± 4	
			NYHA class					
			IV			2	0	
			III			6	2	
			II			6	12	
Kostis et al[55]	CHF patient groups Combined nonpharmacological intervention	Structured exercise and dietary modification	BMI (kg/m^2) Baseline: 30.2 ± 5.2 Change: −5%	6 min walk (m) Baseline: 400.5 ± 17.8 Change: +20%	Exercise time (s) Baseline: 496 ± 113 Change: +37%	Ejection fraction (%) Baseline: 35 ± 6 Change: −9%	Better quality of life indices and improved mood state were also observed in the nonpharmacology group compared with the other 2 groups
	Digoxin		Baseline: 28.6 ± 6.5 Change: +0.7%	Baseline: 399.9 ± 101 Change: −16%	Baseline: 492 ± 228 Change: +2.6%	Baseline: 30 ± 6 Change: +15%	
	Double-blind placebo		Baseline: 28.4 ± 5.3 Change: −1.4%	Baseline: 342 ± 3 Change: +1%	Baseline: 422 ± 120 Change: +22%	Baseline: 37 ± 9 Change: −3%	

CHF, congestive heart failure; LV, left ventricular; E/A ratio, early/atrial ratio; NYHA, New York Heart Association; BMI, body mass index.

and quality of life.[65] Several studies showed that exercise training in CHF patients is more beneficial than harmful if performed carefully under controlled (supervised) protocols.[4,45,66-68] The protocol for exercise therapy, including intensity and duration of exercise, should be tailored according to the needs and capacity of the individual patient.[65] In the Kostis study,[55] dietary modification was supplemented by exercise training as well. Intensity of the exercise training was at a moderate level at the beginning, in which the patients exercised for periods of 2 to 6 minutes followed by 1 to 2 minutes of rest over a period of 3 weeks. Intensity and duration of exercise was gradually increased to 1 hour, 3 to 5 times per week, with a heart rate

corresponding to 40% to 60% of functional capacity. Various methods of exercise included walking, cycling, rowing, and stair-climbing. Another randomized controlled trial by Belardinelli and colleagues[69] demonstrated that long-term moderate exercise training in patients with CHF can result in favorable outcomes with improved functional capacity and quality of life.

In a retrospective study of 14 morbidly obese patients with a mean BMI of 50 kg/m^2 and severe cardiomyopathy undergoing bariatric surgery, McCloskey and coworkers[70] reported that at 6 months after surgery, mean weight loss was 50%, with a decrease in mean BMI to 37 kg/m^2. Mean ejection fraction improved from 23% to 32%, with a corresponding improvement in NYHA functional class. In the same study, 2 patients who were previously denied cardiac transplantation due to morbid obesity subsequently underwent successful transplantation after achieving weight loss following bariatric surgery. In another study by Alpert and colleagues,[49] vertical band gastroplasty was used in 14 patients with CHF and 39 patients without CHF to study the effect of weight loss in morbidly obese patients with and without CHF. Weight loss produced changes in cardiac morphology and improvement of NYHA functional class.

Unintentional Weight Loss or Cardiac Cachexia

Cardiac cachexia is defined as documented nonedematous and unintentional weight loss of more than 7.5% of the previous normal nonedematous weight over a period of at least 6 months.[71] In a study involving 171 patients with CHF, Anker and colleagues[71] observed that cachectic state was a strong independent risk factor for mortality. Cachexia has been previously described in other chronic illnesses, such as acquired immune deficiency syndrome (AIDS),[72] chronic renal failure,[73] chronic lung disease,[74] dementia,[75] and malignancies.[76] Several studies demonstrated that patients with cachexia are at higher risk for perioperative morbidity and mortality compared with noncachectic patients undergoing surgery.[77,78] The pathogenesis of cardiac cachexia is not completely clear, but weight loss has been linked to TNF-α–mediated inflammation. This chronic inflammation is postulated to be due to the bacterial or endotoxin translocation from bowel wall edema following severe heart failure, which in turn results in cytokine activation and protein energy malnutrition.[31,79]

Involuntary weight loss or cachexia is a poor prognostic indicator in heart failure. Underweight or cachectic patients with established heart failure should try to gain weight with adequate, balanced nutritional supplementation. In heart failure patients with normal BMI, whether dietary weight gain or weight loss should be recommended—and whether either could result in improved outcomes—is purely hypothetical. In morbidly obese individuals, however, the evidence is overwhelmingly in favor of weight reduction to improve survival, reduce comorbidities, and improve functional and cardiac capacity and quality of life.

For weight reduction, CHF patients can adopt nonsurgical interventions, such as dietary modification and long-term moderate exercise training. Specific dietary recommendations beyond reduced salt and fat intake, however, have not been convincingly studied among the heart failure population. Bariatric surgery as an invasive approach for weight loss has been shown to significantly reduce weight and improve NYHA functional class among few patients with stable heart failure. Risks of surgery, although below 1% to 2% among healthy individuals, must be considered in patients with heart failure, and preoperative optimization of the cardiac condition is imperative. The long-term efficacy, impact, and side effects of various weight-loss strategies in patients with CHF are yet to be evaluated. In view of the overall increasing weight among Americans as well as the increasing prevalence of heart failure, information on

Conclusion

Until proven otherwise, in healthy individuals, weight reduction is an optional method for prevention of most cardiovascular diseases, including heart failure. The risk of CHF progressively rises with every incremental increase in BMI across different categories of body weight. Clinical outcome of heart failure is impaired in the extremes of weight range (ie, among the underweight and morbidly obese patients). Best outcomes have been documented for overweight and moderately obese patient groups, whereas the worst outcomes are for cachexic, underweight, and morbidly obese groups. Mild to moderate obesity seems to be associated with better survival in heart failure patients as compared with lean and ideal-weight patients; whether this association is causal or not is yet to be studied.

The risk of CHF progressively rises with every incremental increase in BMI across different categories of body weight.

optimal weight control is of essential therapeutic and prognostic value to health care providers in all specialties. ■

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Main Points

- Obese individuals are twice as likely as normal-weight individuals to develop heart failure.
- Obesity increases the risk of heart failure by multiple mechanisms. By augmenting the risk for hypertension, diabetes mellitus, and hyperlipidemia, obesity increases the risk of coronary heart disease, which could then lead to congestive heart failure (CHF). Hypertension and hyperlipidemia increase the risk of myocardial infarction and are independent risk factors for heart failure.
- In obese patients who do not have CHF, it has been found that intentional weight loss can possibly prevent the development of CHF by favorably modifying risk factors for development of coronary heart disease.
- The phenomenon of the "obesity paradox" (or "reverse epidemiology") revealed that overweight and mild to moderate obesity are associated with better outcomes in patients with heart failure compared with patients at normal or ideal weight. Even more, increases of weight in cachectic heart failure patients might improve survival.
- In a weight-loss study of CHF patients who received combined nonpharmacological intervention, digoxin therapy, or placebo, the group that received nonpharmacological intervention achieved the most weight loss, significantly better quality of life indices, improved exercise tolerance, and elevated mood states.
- Several studies showed that exercise training in CHF patients is more beneficial than harmful if performed carefully under controlled (supervised) protocols.

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