Nutrition Self-care in Heart Failure

State of the Science

Terry A. Lennie, PhD, RN

The purposes of this article were to describe the state of science in nutrition self-care in heart failure and make recommendations for future research. Four areas of nutrition self-care were reviewed: sodium restriction recommendations, nutrition recommendations, body weight recommendations, and self-care strategies. A review of the available evidence demonstrated that the current state of knowledge in each of these 4 areas is insufficient for clinicians to provide evidence-based interventions to improve nutrition self-care. Subsequently, research priorities in each area were identified. Although there is a growing recognition of the important role that nutrition may play in heart failure outcomes, advancements in our knowledge base are needed before nutrition self-care can evolve into an essential component of heart failure self-care.

KEY WORDS: heart failure, nutrition

Nutrition self-care differs from some other self-care behaviors such as medication self-care in that the patient is not required to engage in a new activity but rather to modify existing behaviors. In one respect, this is advantageous in that it does not require those with limited physical reserve to engage additional behaviors. In another respect, it is more challenging because it involves changes in habitual behaviors that are frequently imbedded in culture or have social consequences. Accordingly, effective nutrition self-care may necessitate the patient making a conscious decision to change long-standing behaviors. Four characteristics have been identified as being important for making decisions to engage in self-care: knowledge, experience, skills, and compatibility with values. Once the decision has been made, these characteristics are also needed for the patient to engage in self-care behaviors. To assist patients with nutrition self-care, clinicians need evidenced-based knowledge for the development of effective interventions. The state of the science in nutrition self-care, however, is currently insufficient to provide clinicians with the comprehensive evidence base needed to adequately assist patients. The purposes of this article are to describe the state of science regarding nutrition self-care in heart failure and to identify key areas where additional research is needed.

Overview of the State of the Science in Nutrition Research

The state of the science in nutrition-related research in heart failure is nearly 60 years behind that of research on medical management. This point can be illustrated by the description of Conraads et al of 4 models of heart failure that characterize the evolution in modern heart failure management. In the initial cardiorenal model, the focus was on relief of symptoms caused by excess accumulation of sodium and fluid. In addition to digitalis and diuretics, the primary treatment was strict dietary restriction of sodium and fluid. In the 1960s, the focus shifted to a hemodynamic model of management in which treatment was geared toward controlling hemodynamic changes associated with heart failure and optimizing the heart as a pump. Vasodilators and inotropic drugs were added with limited success. The subsequent neurohormonal model of heart failure management was aimed at controlling the physiological responses to heart failure. Angiotensin-converting enzyme inhibitors and β-adrenergic antagonists were introduced with considerable success. An appreciation of the role that inflammation plays in heart failure leads to the most recent inflammation model, in which treatments to control the inflammatory response are being explored.

Each of these models marks an era in which advancements in heart failure management were driven by clinical trials that identified new strategies for
medical management of patients with heart failure. In contrast, the only nutrition-related strategy identified over the same time period was sodium restriction. Although this strategy was introduced more than 60 years ago, no randomized controlled clinical trial has ever been conducted to determine the optimal level of sodium intake for patients with heart failure. Clearly, the enthusiasm and resources devoted to research on the medical management of heart failure have not been duplicated in nutrition-related research. Consequently, little is known about eating habits, nutritional needs, and nutritional adequacy of the diets of patients with heart failure. Furthermore, little is known about the effects of nutrition interventions on heart failure outcomes.

Although nutrition-related research has been limited, some advances in our understanding of nutrition self-care have been made over the past 60 years. In the following sections, the state of science regarding sodium restriction, nutritional recommendations, body weight recommendations, and self-care strategies are described, and direction for future research is identified.

**Sodium**

Restriction of dietary sodium remains an area of interest for self-care research in heart failure. A number of researchers have documented excess sodium intake as a precipitating factor for hospital admission due to decompensated heart failure. Among the most commonly cited studies, nonadherence to a sodium-restricted diet was estimated to be a primary precipitating factor in as few as 6% to as many as 55% of admissions. Although these studies provide the best evidence to date regarding to role of sodium intake in heart failure exacerbations, there are limits to the conclusions that can be drawn.

Vinson et al were among the first to document that excess sodium intake was associated with hospitalization for heart failure. The sample of 140 patients was restricted to those 70 years and older admitted with heart failure during a 6-month period in 1987. Nonadherence to sodium restriction was identified by chart review and patient/family interview. Bennett et al collected data by chart review of 585 patients admitted to 2 hospitals in 1992. The major proximate cause of readmission was determined to be sodium retention leading to volume overload. As defined, sodium retention in this study could be attributed to both nonadherence to dietary sodium restriction and nonadherence to medication regimen (eg, diuretics) that led to sodium retention. Chin et al reviewed the charts of 436 patients admitted for heart failure in 1993 and 1994. Dietary nonadherence to sodium restriction was defined as a specific mention of nonadherence in the chart. In the more recent prospective study by Tsuyuki et al, proximate causes of exacerbation of heart failure were determined in patients participating in the Randomized Evaluation of Strategies for Left Ventricular Dysfunction-Pilot study. Of the 768 participants, 180 were hospitalized during a 43-week follow-up. Individual investigators were asked to indicate the single factor most responsible for exacerbation. No standardized criteria were provided for the determination of factors responsible for exacerbation.

It should be noted that most of these studies were conducted 15 to 20 years ago and may not reflect patient responses to sodium intake under current management guidelines. Furthermore, identification of dietary nonadherence was dependent on patient self-report or documentation in the medical record by clinicians based on their own judgment. It is likely that clinicians used different criteria to define nonadherence. Most importantly, actual sodium intake was not measured in any of the studies. Therefore, the level of sodium intake associated with exacerbations of heart failure and whether this level varies according to the clinical characteristics of patients have not been determined.

The limited available evidence may be responsible, in part, for the lack of consensus regarding sodium intake among the guidelines published by the Heart Failure Society of America, the American College of Cardiology/American Heart Association (AHA), and the European Society of Cardiology. Recommendations vary from 2 to 3 g/d, to 3 to 4 g/d, to no specific recommendation other than a statement that sodium restriction may be beneficial in some cases of severe heart failure (Table 1). Interestingly, both the US Department of Agriculture (USDA) and the Nutrition Committee of the AHA recommend a 2.5-g sodium diet for healthy adults, which is more restrictive than all of the heart failure guidelines.

In sum, close examination of the research to support a specific level of sodium restriction demonstrates that the evidence is not sufficient. As was the case in previously published guidelines, the current guidelines cite expert clinical opinion to support recommendations regarding sodium intake. One possible option to resolve this issue is to adopt the USDA and AHA recommendation of 2.5 g of sodium intake per day as the standard guideline. The evidence to support this recommendation, however, is based on research for the prevention or treatment of hypertension. The wisdom of using this evidence to support recommendations for patients with heart failure treated with agents aimed at blocking renin-angiotensin-aldosterone and β-adrenergic responses is uncertain. The alternative is to conduct controlled clinical trials to determine the appropriate level of
dietary sodium intake. Additional patient outcomes would need to be included, such as quality of life, diuretic dose requirements, hospitalizations, mortality, nutritional adequacy of the diet, and indicators of patient ability to incorporate the diet into self-care regimen. This option would provide the evidence needed to support dietary sodium recommendations.

**Nutrient Recommendations**

Our current understanding of the roles that various nutrients play in health indicates that they may be important for improving outcomes in patients with heart failure.\(^{19,20}\) The available nutrition-related research in heart failure is not sufficient to provide specific nutrient recommendations. Several investigators who documented altered nutritional status of patients with heart failure did not quantify nutritional intake\(^{21,22}\) or included only a small number of patients.\(^{23}\) Other investigators who quantified food intake did not assess nutritional status.\(^{24}\)

The study by Aquilani et al\(^{25}\) is notable because it is among the first to quantify energy intake, energy expenditure, and nitrogen balance in community-dwelling patients and healthy adults. A total of 57 patients with heart failure (52 men and 5 women) and 49 similarly aged healthy adults (39 men and 10 women) provided 7-day food diaries. Nitrogen balance and energy expenditure were also measured. Energy intake was similar in the 2 groups, but patients with heart failure had higher energy expenditure, resulting in 70% of patients having a negative energy balance. Although protein intake was also similar in the groups, 59% of patients had a negative nitrogen balance, indicating that they had higher protein metabolism. Thus, although the nutritional intake of patients with heart failure was not less than that of healthy adults, nutrient utilization was greater. Other investigators have also documented increased protein turnover in patients with increased energy expenditure.\(^{26}\) These results suggest that published nutritional recommendations for healthy adults\(^{27}\) may not be sufficient to meet the needs of patients with heart failure.

Most of the literature describing micronutrient requirements in patients with heart failure is based on data extrapolated from other populations.\(^{19,28-33}\) This is because the micronutrient intake of patients with heart failure has been examined only in a few studies, many of which focused on a single nutrient. For example, one group of investigators focused on whether vitamin B deficiencies were related to the homocysteine and severity of heart failure.\(^{34}\) No relationship was found. It was not possible to determine the incidence of vitamin B deficiency or the level of vitamin B requirements of patients with heart failure from this study. Others examined magnesium.\(^{35,36}\) In a study in which deficiencies were examined, 12% of 404 hospitalized patients had magnesium deficiency, which was associated with shorter survival.\(^{36}\) Gorenlik et al\(^{24}\) provided a more comprehensive assessment of dietary intake. In their study, food intake was estimated by 6-month diet recall in older (>60 years) patients hospitalized with heart failure and in similar patients hospitalized without heart failure. Both groups of patients had a comparably high number of dietary deficiencies in energy, calcium, folic acid, manganese, magnesium, riboflavin, thiamin, and zinc. It is important to note that these data were obtained using 6-month diet recall in patients who required hospitalization. They may not reflect nutritional intake in stable patients. However, we have also identified dietary deficiencies in calcium, magnesium, iron, and vitamin D using 4-day weighed food diaries in a high number of stable, community-dwelling patients.\(^{37,38}\) This suggests that dietary deficiencies may be a common problem in patients with heart failure.

There is insufficient evidence to make specific recommendations regarding nutritional supplementation because only a few studies have been published. Witte et al\(^{39}\) conducted a small, randomized controlled trial of a 9-month, high-dose supplementation of 9 vitamins, 5 minerals, and coenzyme Q10. The size of the left ventricle was reduced by 13% in the supplementation group compared with only 4% in the placebo group. Left ventricular ejection fraction increased by 5% in the intervention group but was unchanged in the placebo group. Quality of life also improved in the intervention group and declined slightly in the placebo group. There were no
differences in exercise capacity or levels of inflammatory cytokines between groups. This study provides some evidence of the value of adequate micronutrient intake. Similarly, studies of vitamin C, vitamin D, thiamin, and magnesium supplementation provide limited evidence that supplementation of these nutrients may be beneficial. Studies of vitamin E supplementation have produced conflicting results but, in sum, suggest that supplementation may not be beneficial.

There is no consensus among the guidelines regarding recommendations for vitamin and mineral supplementation (Table 1). Specific recommendations for macronutrient intake are also lacking. Again, this is because of insufficient evidence to support a standard recommendation. Data from a small study suggest that interventions to lower saturated fat and increase omega-3 fatty acids in the diet can decrease the magnitude of inflammation in patients with heart failure and increase longevity. These data provide initial evidence that diet does indeed affect heart failure outcomes.

Additional large-scale studies that include a diverse population of patients with heart failure are needed to adequately assess nutrient intake, nutritional status, and their relationships to clinical outcomes in patients with heart failure. Future research should address the following key issues that have not been captured by previous research. First, patients should be followed over time to determine the effects of nutritional intake on outcomes. Second, females need to be equally represented so potential sex differences can be examined. Third, potential differences in nutritional requirements among underweight, normal weight, overweight, and obese patients need to be examined. This is of particular importance given the high number of patients with heart failure who are overweight or obese (see “Body Weight” section). Fourth, more information is needed regarding micronutrient intake and requirements. Lastly, the interplay among length of time since diagnosis, severity of heart failure, and nutrition should be considered. Once completed, these data should provide guidance for randomized clinical trials to test whether appropriate nutritional interventions improve outcomes.

Body Weight

Cachexia

The development of cardiac cachexia is associated with poor clinical outcomes, making it an attractive target for nutritional intervention. There is currently no consensus regarding the definition of cardiac cachexia. Estimates of the prevalence of cachexia based on below-normal body mass index (BMI) indicate that patients who are cachetic represent 4% to 13% of the current heart failure population. The introduction of β-adrenergic antagonists may be responsible, in part, for the small number of patients with abnormally low BMI. Initiation of β-adrenergic antagonist therapy has been shown to be associated with an increase in body fat. BMI, however, may not be the best indicator of cachexia. Unintentional weight loss of 7.5% or more of previous normal body weight in a 6-month period was demonstrated to be an independent predictor of mortality. This definition of cachexia is clinically relevant and can identify patients at high nutritional risk before they become severely depleted. This definition also produces a higher incidence of cachexia, with 23% of outpatients being considered cachexic in one study.

Regardless of how cachexia is defined, nutritional self-care is not likely to be a feasible goal for these patients. Patients with cachexia are highly vulnerable and likely to require close monitoring, frequent intervention, and intensive nutritional support. Therefore, although research is needed to identify ways that clinicians can provide appropriate nutritional support to improve outcomes, research on promoting nutrition self-care would be better directed toward groups more likely to be able to engage in this form of self-care.

Overweight and Obesity

Patients who are overweight or obese now represent a majority of patients with heart failure. Based on studies in which BMI was reported, approximately 36% to 40% of patients with heart failure are overweight and 17% to 25% are obese. Combined, overweight and obese patients comprise up to 65% of the heart failure patient population. A number of studies collectively involving more than 18,000 outpatients with heart failure have demonstrated a positive relationship between higher BMI and survival. Most of these studies have been reviewed elsewhere. Data from the most recent study involving patients from the Candesartan in Heart Failure: Assessment of Reduction in Morbidity and Mortality trial suggest that the positive relationship may plateau at BMIs above 35 kg/m². Thus, there may be an upper limit above which additional body fat does not convey positive survival value. This observation merits further investigation.

Evidence from the Acute Decompensate Heart Failure National Registry shows that the same relationship is found in patients hospitalized for acute heart failure. The mortality rate observed in 108,927 admissions in the registry was highest in patients (6.3%) with BMIs less than 18.5 kg/m² and lowest in patients (2.4%) with BMIs greater than 30 kg/m².
Patients with BMIs between 16 and 23 kg/m\(^2\) had a 46% higher risk of dying than did patients with a BMIs above 33 kg/m\(^2\).

Combined, these results question the wisdom of recommending weight loss as a component of nutrition self-care for overweight patients and, possibly, for patients at obesity class I (BMI <35 kg/m\(^2\)). Currently, there is no consensus among the major guidelines regarding body weight recommendations for overweight and obese patients (Table 1). It is of concern that no definitive recommendations are available for what now constitutes most patients with heart failure. Clearly, additional research is needed to clarify the role of body weight/body fat on patient outcomes and whether the relationships vary within subgroups of patients. Factors in addition to hospitalization and mortality, such as functional status and quality of life, need to be included in future studies. The potential mechanisms responsible for these observations also need to be identified.

**Studies to Improve Nutrition Self-care**

Not surprisingly, the limited research found on nutrition self-care in heart failure was related to improving adherence to sodium-restricted diets. Several investigators have identified potential barriers to dietary sodium adherence. These barriers included lack of awareness of need to limit sodium in diet; lack of knowledge or ability to identify both low- and high-sodium foods, particularly when eating in restaurants; interference with ability to participate in social (eg, church) events serving food; lack of food selection; general difficulty following the diet; no perceived benefit to following the diet; and friends or family not following the same diet or serving low-salt foods when visiting.

Researchers testing interventions to improve adherence to low-sodium diet have used education and skill building to increase awareness of need to limit sodium intake, provide low-salt cooking strategies, improve ability to read sodium content of food labels, and identify low-salt foods in restaurants. One group of investigators extended their educational intervention to other components of the diet, including dietary fat, protein, and carbohydrate intake. Education by a diettian improved recommended intake of these components of the diet as well. The results of these studies demonstrate that education and skill building are effective at improving nutrition self-care, particularly when counseling or oral teaching sessions are provided in addition to written instructions.

Other factors are also important to self-care. In particular, patients identified family and friends as having an impact on nutrition self-care. Concor-

stantly, data from Chung et al show that the presence of a spouse, at least in men, impacts adherence to low-sodium diet. Dunbar et al provided an educational intervention with follow-up telephone calls plus 2 additional sessions that focused on developing a family-patient partnership to improve adherence to low-sodium diet. The intervention included helping family members to improve supportive communication techniques, handle difficult situations when the patient was either nonadherent or at risk of being nonadherent (eg, restaurant or social gathering), enhance patient self-confidence, and identify additional ways they could become involved in helping the patient with adherence. Including the family enhanced patient adherence to low-sodium diet.

To increase the ability of clinicians to promote nutrition self-care, large randomized clinical trails are needed to strengthen the evidence provided by previous studies, identify confounding factors and new areas requiring investigation, and determine potential differences in patient subgroups. Research is also needed to test interventions that include additional factors that affect nutrition self-care, particularly attitudes, perceived benefits, and barriers related to nutrition self-care. Comorbidities such as diabetes mellitus, hyperlipidemia, and renal insufficiency that impact nutrition self-care also need to be addressed. These create an additional layer of complexity with respect to designing interventions to improve self-care. To date, no studies have included nutritional management of comorbidities as a component of the intervention. Furthermore, none of the previous investigators examined the effects of interventions beyond 3 months. Thus, it is not known if current interventions convey long-term effects and, if not, what type of follow-up intervention is beneficial. Finally, future intervention studies should include long-term outcomes such as quality of life, nutritional status, hospitalizations, morbidity, and mortality.

**Conclusion and Recommendations**

There is a growing recognition of the need for additional attention to be devoted nutrition-related research in heart failure and that the state of the science in nutrition self-care must encompass more than dietary sodium. The high-priority areas in need of additional research and the outcomes that were not fully addressed in previous research are outlined in Table 2. In addition to specific outcomes listed in the table, studies should address quality of life. Whenever possible, studies should include other long-term outcomes such as all-cause and cardiac-related hospitalization and mortality. As our knowledge increases, nutrition self-care is likely to emerge as a fundamental component of heart failure self-care.
TABLE 2 Research Priorities for Nutrition

<table>
<thead>
<tr>
<th>Key Areas</th>
<th>Specific Outcomes</th>
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</thead>
<tbody>
<tr>
<td>Sodium intake recommendations</td>
<td>Nutritional adequacy of the diet</td>
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<tr>
<td>• Determine optimal level of dietary sodium intake</td>
<td>Diuretic dose</td>
</tr>
<tr>
<td>○ Differences in recommended level in subpopulations</td>
<td>Ability to incorporate the diet into self-care regimen</td>
</tr>
<tr>
<td>Nutrient recommendations</td>
<td>Functional status</td>
</tr>
<tr>
<td>• Determine optimal amount of protein, fat, and carbohydrate intake</td>
<td>Symptom status</td>
</tr>
<tr>
<td>○ Effects of dietary fat on inflammation and outcomes</td>
<td>Nutritional status</td>
</tr>
<tr>
<td>• Determine recommended levels of micronutrients</td>
<td>Physiological status (eg, inflammation, neuroendocrine)</td>
</tr>
<tr>
<td>○ Calcium, iron, magnesium, thiamin, vitamin D, vitamin E, and zinc</td>
<td></td>
</tr>
<tr>
<td>Body weight recommendations</td>
<td>Functional status</td>
</tr>
<tr>
<td>• Evidence for body weight recommendations for overweight and class I obese patients</td>
<td>Symptom status</td>
</tr>
<tr>
<td>○ Upper limit of body fat that conveys positive outcomes.</td>
<td>Nutritional status</td>
</tr>
<tr>
<td>○ Effects of body fat distribution on outcomes</td>
<td>Emotional status</td>
</tr>
<tr>
<td>Interventions to improve self-care</td>
<td>Knowledge Skills</td>
</tr>
<tr>
<td>• Address attitudes, barriers, and benefits</td>
<td>Family interaction/family support</td>
</tr>
<tr>
<td>• Inclusion of friends and family</td>
<td>Nutritional adequacy of the diet</td>
</tr>
<tr>
<td>• Address diet changes required by comorbidities</td>
<td>Long-term maintenance of nutrition self-care</td>
</tr>
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one that is finally considered to be among those essential to controlling symptoms, improving quality of life, and increasing longevity.

REFERENCES

22. Schwengel RH, Gottlieb SS, Fisher ML. Protein-energy...
malnutrition in patients with ischemic and nonischemic
dilated cardiomyopathy and congestive heart failure. Am J

23. Broqivist M, Arnpivist H, Dahlstrom U, Larsson J,
Nylander E, Permutt J. Nutritional assessment and muscle
energy metabolism in severe chronic congestive heart
failure—effects of long-term dietary supplementation.

intake of various nutrients in older patients with conges-

adequate in chronic heart failure patients? J Am Coll
Cardiol. 2003;42(7):1218–1223.

Daily energy expenditure in free-living heart failure

27. Institute of Medicine. Dietary Reference Intakes: The
Essential Guide to Nutrient Requirements. Washington, DC:

28. Cohen N, Golik A. Zinc balance and medications com-
monly used in the management of heart failure. Heart Fail

29. de Lorgeril M, Salen P. Selenium and antioxidant defenses
as major mediators in the development of chronic heart

30. Witte KK, Clark AL. Nutritional abnormalities contributing
to cachexia in chronic illness. Int J Cardiol. 2002;85(1):
23–31.

31. Witte KK, Clark AL, Cleland JG. Chronic heart failure and

32. Sole MJ, Jeejeebhoy KN. Conditioned nutritional require-
ments and the pathogenesis and treatment of myocardial
417–424.

33. Allard ML, Jeejeebhoy KN, Sole MJ. The management of
conditioned nutritional requirements in heart failure.
Heart Fail Rev. 2006;11(1):75–82.

vitamins and their relation to the severity of chronic heart

35. Costello RB, Moser-Veillon PB, DiBianno R. Magnesium
supplementation in patients with congestive heart failure.

Serum magnesium aberrations in furosemide (frusemide)
patients with congestive heart failure receiving long-term
490–499.

37. Lennie TA, Moser DK, Habash DL. Low sodium diet:
nutritional adequacy and factors limiting adherence.

38. Lennie TA, Moser DK, Habash DL, Trupp R. Nutritional
adequacy of low sodium diets in patients with heart

39. Witte KK, Nikitin NP, Parker AC, et al. The effect of
micronutrient supplementation on quality-of-life and left
ventricular function in elderly patients with chronic heart

40. Hornig B, Arakawa N, Kohler C, Drexler H. Vitamin C
improves endothelial function of conduit arteries in patients

41. Ellis GR, Anderson RA, Lang D, et al. Neutrophil
superoxide anion—generating capacity, endothelial func-
tion and oxidative stress in chronic heart failure: effects of

42. Schleithoff SS, Zittermann A, Tenderich G, Berthold HK,
Stehele P, Koefer R. Vitamin D supplementation improves
cytokine profiles in patients with congestive heart failure:
a double-blind, randomized, placebo-controlled trial.

ventricular function after thiamine supplementation in
patients with congestive heart failure receiving long-term

44. Basmajian JV. Biofeedback: Principles and Practice for
Clinicians. 3rd ed. Baltimore, MD: Lippincott Williams &
Wilkins; 1989.

45. Fuentes JC, Salmon AA, Silver MA. Acute and chronic
oral magnesium supplementation: effects on endothelial
function, exercise capacity, and quality of life in patients
with symptomatic heart failure. Congest Heart Fail.

system in heart failure and therapeutic role of oral vitamin

clinical trial of vitamin E supplementation in patients with
219–224.

combined administration of low dose atorvastatin and
vitamin E on inflammatory markers and endothelial

49. Lennie TA, Chung ML, Habash DL, Moser DK. Dietary
fat intake and proinflammatory cytokine levels in patients

50. Anker SD, Coats AJ. Cardiac cachexia: a syndrome with
impaired survival and immune and neuroendocrine acti-

51. Anker SD, Sharma R. The syndrome of cardiac cachexia.

52. Horwich TB, Fonarow GC, Hamilton MA, MacLellan WR,
Woo MA, Tillisch JH. The relationship between obesity
and mortality in patients with heart failure. J Am Coll

and survival in patients with chronic heart failure without
cachexia: the importance of obesity. J Card Fail. 2003;

54. Gustafsson F, Kragelund CB, Torp-Pedersen C, et al. Effect
of obesity and being overweight on long-term mortality in
congestive heart failure: influence of left ventricular systolic

body mass index and outcomes in patients with heart

56. Lainscak M, Keber I, Anker SD. Body composition
changes in patients with systolic heart failure treated with
319–322.

57. Anker SD, Ponikowski P, Vansey N, et al. Wasting as
independent risk factor for mortality in chronic heart

failure in the general population: The Rotterdam Study.

and prognosis in patients with chronic heart failure: insights
from the Candesartan in Heart Failure: Assessment
of Reduction in Mortality and Morbidity (CHARM) program.


64. Dunbar SB, Clark PC, Deaton C. A family focused intervention is effective in reducing dietary sodium. *J Card Fail*. 2002;8(suppl 4):S5.


