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Is Nutritional Intake Adequate in Chronic Heart Failure Patients?

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OBJECTIVES The goal of this study was to investigate the nutrition adequacy and energy availability for physical activity in free-living, clinically stable patients with chronic heart failure (CHF).

BACKGROUND Little information exists regarding the nutrition adequacy and alimentary habits of patients with clinically stable CHF. We hypothesized that CHF patients have an inadequate intake of calories and protein, leading to a negative calorie and nitrogen balance, an expression of increased tissue breakdown.

METHODS In 57 non-obese patients with CHF (52 males and 5 females; 52 ± 3 years; body mass index <25 kg/m^2) and in 49 healthy subjects (39 males and 10 females) matched for age, body mass index, and sedentary life style we evaluated total energy expenditure (TEE), calorie intake ($kcal_I$), and nitrogen intake (N_I) from a seven-day food diary, total nitrogen excretion (TNE), and energy availability (EA = $kcal_I$ − resting energy expenditure). A zero calorie balance (CB) occurred when $kcal_I$ = TEE; a nitrogen balance (NB) in equilibrium was set at NB (= N_I − TNE) 0 ± 1 g/day.

RESULTS In patients and controls $kcal_I$ and N_I were similar. However, in CHF patients the $kcal_I$ was <TEE with a consequent negative CB (−186 ± 305 kcal/day vs. + 104.2 ± 273 kcal/day of controls; $p < 0.01$). Nitrogen balance resulted negative in CHF (−1.7 ± 3.2 g/24 h vs. + 2.2 ± 3.6 g/24 h in controls; $p < 0.01$). Energy availability in CHF patients was 41% lower than in controls ($p < 0.05$).

CONCLUSIONS Non-obese, free-living patients with clinically stable CHF have an inadequate intake of calories and protein and reduced energy availability for physical activity. (J Am Coll Cardiol 2003;42:1218–23) © 2003 by the American College of Cardiology Foundation

Nutritional intake is adequate to body needs when it provides energy substrates for current body energy needs and restores catabolized endogenous proteins, thus increasing the low glycogen content in muscles. These priorities may be particularly relevant in patients with chronic heart failure (CHF) who can be hypermetabolic (1,2), hypercatabolic (3), and have low glycogen content (4).

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In clinical practice a useful method to assess nutritional adequacy is to calculate the calorie-nitrogen balance (CNB), that is the difference over a day, between calorie-nitrogen intake and total calorie expenditure-nitrogen excretion. Previous studies dealing with nutrition in CHF did not calculate CNB (5).

We hypothesized that the calorie and protein intakes of non-obese patients with CHF, although clinically stable and living freely at home, might be inadequate to meet body requirements, thus causing negative CNB, as in clinical practice, non-obese ambulatory CHF patients are not routinely provided a personalized nutritional prescription or advice despite their possible catabolic factors.

We, therefore, assessed nutritional adequacy and calculated energy availability for daily life physical activities in non-obese, clinically stable patients with CHF, living at home.

METHODS

Population. Fifty-seven non-obese patients (body mass index ≤25 kg/m^2) with CHF, admitted to the Heart Failure Unit of Montescano for assessment or reassessment of indications for cardiac transplantation, were enrolled for the study three days before their discharge from the unit. Patients with diabetes mellitus, liver, and renal insufficiency were excluded from the study.

Table 1 summarizes the demographic, clinical, functional, neurohormonal, and treatment characteristics of the patients recorded while they were clinically stable (no evidence of fluid retention—peripheral or pulmonary edema; jugular venous pressure not raised; no changes in medication for at least seven days).

No patient had clinical signs of intestinal malabsorption

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Abbreviations and Acronyms

AMA = arm muscle area
BW = body weight
CB = calorie balance
CHF = chronic heart failure
CHO = carbohydrates
CNB = calorie-nitrogen balance
EA = energy availability
HB = Harris-Benedict equation
$kcal_I$ = daily calorie intake
NB = nitrogen balance
N_I = daily nitrogen intake
REE = resting energy expenditure
TEE = total energy expenditure
TNL = total nitrogen loss
UNE = urine nitrogen excretion

such as steatorrhea, diarrhea. Twenty patients (35%) had digestive disturbances such as early satiety, abdominal discomfort and fullness after meals.

The patients were adequately informed about the aims and methods of the study and gave written consent for their participation. The study was approved by our institution's ethical scientific committee.

Table 1. Patients' Demographic, Clinical, Functional, Neurohormonal, and Treatment Characteristics

Number (n): 57 pts
Age (yrs): 52 ± 3
Men/women: 52/5
Etiology
  Coronary artery disease 31 pts (= 54.3%)
  Idiopathic cardiomyopathy 20 pts (= 35%)
  Valvular heart disease 6 pts (= 10.5%)
Duration of disease (months)
  <18 23 pts
  ≥18 34 pts
NYHA class
  I 5 pts (= 8.7%)
  II 28 pts (= 49%)
  III 21 pts (= 37%)
  IV 3 pts (= 5.3%)
Left ventricular ejection fraction 20.3 ± 5.6%
Peak VO_2 (ml O_2/kg/min)
  <14 (11.8 ± 2.5) 26 pts
  >14 (18.5 ± 4.7) 31 pts
Anaerobic threshold (ml O_2/kg/min)
  peak VO_2 < 14 10 ± 2.1
  peak VO_2 > 14 14.8 ± 3.7
Respiratory exchange ratio
  baseline 0.84 ± 0.1
  peak VO_2 1.1 ± 0.1
Neurovegetative status evaluation
  norepinephrine (pg × ml^{-1}) 330.0 ± 19
    (reference values from our laboratory: 278 ± 75)
Medication
  ACE inhibition 45 pts (79%)
  Digoxin 16 pts (28%)
  Diuretics 54 pts (95%)
  Warfarin 17 pts (29.8%)
  Calcium antagonists 8 pts (14%)
  Oral nitrates 14 pts (24.6%)

ACE = angiotensin-converting enzyme; NYHA = New York Heart Association; pts = patients.

Materials. CHF NUTRITIONAL-HORMONAL EVALUATIONS. Triceps skinfold thickness and midarm muscle circumference were measured in all patients in order to calculate arm muscle area (AMA) (6). Body weight (BW) was recorded and compared with the usual BW in the last six to 12 months before hospital admission.

A non-intentional loss of >10% of BW in the preceding year or >7.5% in the last six months was considered as a marker of energy depletion (7). Severe muscle protein malnutrition and, hence, loss of lean body mass was diagnosed when the AMA was below the 5th percentile (6). The co-presence of BW loss and a reduced AMA was diagnostic of combined calorie and protein malnutrition.

Resting energy expenditure (REE) was measured by indirect calorimetry (8) and expressed in kcal/day, kcal/kg BW, and kcal/m^2 body surface.

A normal REE value lies between 90% to 110% of the value predicted by the Harris-Benedict equation (HB) (9).

At 8 AM peripheral venous blood samples were drawn for assays of cortisol (nmol/l) and insulin (μUI/ml) levels (commercial kits Cord-CT Radioimmunoassay Kit CIS France, Coat A Count Insuline, D.P.C., Los Angeles, California).

The patients were extensively informed on how to keep a seven-day food diary at home and asked to collect three-day 24-h urine samples for determination of urea nitrogen excretion (UNE) (in g/24 h) (10). After receiving the patients' food diaries and UNE results, we used computerized nutritional analysis to calculate the macronutrient (carbohydrates [CHO], proteins, lipids) and calorie intakes ($kcal_I$).

Total energy expenditure (TEE) (kcal/day) was estimated as REE × 1.3 where 1.3 is a correction factor for physical activity (1). Energy availability (EA) (kcal/day) for daily physical activity was calculated as $kcal_I$ − REE.

CRITERIA FOR NUTRITION ADEQUACY TO BODY NEEDS.

1) Adequate calorie intake: a neutral or positive calorie balance (CB), where CB (±kcal/day) = $kcal_I$ − TEE.
2) Adequate protein intake: a neutral or positive nitrogen balance (NB), where NB (g/24 h) = daily nitrogen intake (N_I) − total nitrogen loss (TNL). N_I = nitrogen intake with food (= protein intake: 6.25); TNL = total nitrogen loss (g/24 h) = UNE (g/24 h) + 2g (11).

If NB = 0 ± 1 g/24 h, body anabolic and catabolic processes were in equilibrium; a negative NB (>−1 g/24 h) denoted an excess of endogenous protein consumption (catabolic process) while a positive NB (>+1 g/24 h) indicated a predominance of anabolic processes.

CONTROL GROUP ENROLLMENT. A control group of 49 healthy subjects (39 males and 10 females) was selected to meet the following criteria: 1) comparable age (44 ± 16 years; p = NS); 2) a very sedentary lifestyle (12); 3) no

Table 2. Anthropometric Measures, Resting Energy Expenditure for Basal Homeostasis, and Calculated Total Daily-Energy Expenditure in Patients and Controls of the Study

	All-CHF (n = 57)	All-C (n = 49)	Normo-CHF (n = 26)	Normo-C (n = 33)	Maln-CHF (n = 31)	Under-C (n = 16)
BMI kg/m^2	22.4 ± 3	23 ± 6.2	25 ± 1.5	25.1 ± 1.8†	21.5 ± 2.9¶	18 ± 1.7‡
BW						
kg	64.9 ± 9.9	62.5 ± 18.2	74.2 ± 7.6	77.9 ± 15.9	60.2 ± 9.2¶	49.7 ± 5.3
% Usual	96 ± 9	98 ± 3	101 ± 5	102 ± 4	90 ± 7	85.4 ± 8.8
AMA cm^2	47 ± 9.8 (46)*	—	55.7 ± 4.9	—	41.8 ± 9¶	—
REE						
kcal/day	1,499 ± 228	1,309 ± 315#	1,609 ± 256	1,494 ± 312	1,461 ± 192	1,156 ± 242§
kcal/kg	22.9 ± 2.4	21.4 ± 3.4	21.9 ± 2.4	19.3 ± 2.8	23.9 ± 2.3	23 ± 2.9
kcal/m^2	870 ± 98.7	777 ± 113**	856.7 ± 92	812 ± 103	872 ± 94	749 ± 123§
% Predicted	103.2 ± 11	92 ± 13**	102.8 ± 8.8	94.8 ± 8.5	105 ± 12	92 ± 13§
TEE = REE × 1.3						
kcal/day	1,949 ± 296	1,702 ± 409#	2,092 ± 333	1,942 ± 406	1,898 ± 250	1,503 ± 315§
kcal/kg	29.8 ± 3.1	27.8 ± 4.4	28.5 ± 3.1	25.1 ± 3.6	31.1 ± 3	30 ± 3.8

Data are given as mean ± SD. *In brackets: value under the fifth percentile of normal values. Statistical analysis: *t* test. Statistical significance was set at $p < 0.05$. Normo-CHF vs. Normo-C: †$p < 0.05$; Maln-CHF vs. Under-C: ‡$p < 0.05$; §$p < 0.01$; Normo-CHF vs. Maln-CHF: ¶$p < 0.01$; All CHF vs All-C: #$p < 0.05$; **$p < 0.01$. All-CHF = all patients with CHF; All-C = all healthy controls; Normo-CHF = normally nourished CHF patients; Normo-C = normal weight controls; Maln-CHF = malnourished CHF patients; Under-C = underweight controls.

AMA = arm muscle area; BMI = body mass index; BW = body weight; CHF = chronic heart failure; REE = resting energy expenditure; TEE = total energy expenditure.

symptoms or signs of heart disease or diabetes; 4) weight stability (± 1 kg) during the three months before their enrollment; 5) comparable body mass index. To achieve this last match we selected people with a body mass index ranging from 17 kg/m^2 (underweight individuals) to 25 kg/m^2 (normal weight individuals). The underweight subjects were people who deliberately, and without advice from a nutritionist, had chosen to reduce their food intake in the last year.

The control group underwent the same experimental protocol as CHF patients.

Statistical analysis. Results are presented as the mean value ± SD. Comparisons between groups were performed with *t* tests. Differences were considered statistically significant at $p < 0.05$.

Furthermore, simple linear regression analysis was used to show possible correlations between plasma cortisol levels, plasma insulin levels, cortisol/insulin ratio, and anthropometric parameters and resting energy expenditure.

RESULTS

Patients' nutritional status and resting energy expenditure. The study showed that 31/57 patients (54.4%) were malnourished: nine had combined protein-calorie malnutrition (15.8% population), 22 (38.5%) had protein malnutrition with a normal BW (90% to 110% desirable BW) (Table 2).

Patients and controls were designated as follows: all patients with CHF (All-CHF) (n = 57); normally nourished CHF patients (Normo-CHF) (n = 26); malnourished CHF (Maln-CHF) patients (n = 31); all healthy controls (All-C) (n = 49); normal weight controls (Normo-C) (n = 33); underweight controls (Under-C) (n = 16).

Compared with controls, CHF patients had similar REEs/kg (hence, TEEs) but different REEs/m^2. They lost metabolic adaptation to body tissue wasting (13), as their REEs percent predicted HB was higher than that in controls.

Nutritional intake, calorie and nitrogen balance, energy availability. Negative CB and NB occurred in 70.1% and 59.6% of CHF patients, respectively, despite them having similar calorie (kcal$_I$/kg) and nitrogen (N_I/kg) intakes as controls (Table 3). A combined negative CNB was found in 40.3% of the patients.

Patients with CHF had a lower energy availability (EA) for physical tasks of daily life than did controls (−41%: 292.5 kcal/day).

From diaries, patients with CHF ingested less lipids than controls but similar amounts of CHO. The contributions of CHOs and lipids to ingested calories (% kcal) were higher and lower, respectively, in CHF patients than in controls ($p < 0.01$). As regards alimentary habits, CHF concentrated 82 ± 8% ingested calories over a day in the two main meals (lunch and dinner).

Thirty-five percent of CHF patients confirmed, in their diaries, the persistence of digestive disturbances.

Plasma cortisol and insulin concentrations, cortisol/insulin ratio. Plasma cortisol levels, higher in CHF patients than in controls, although within the normal range of values, were positively correlated with REE/kg ($r = 0.38$, $p < 0.02$) (Table 4).

Plasma insulin concentrations, similar between patients and controls, were correlated with BW ($r = 0.6$, $p < 0.01$), triceps skinfold thickness ($r = 0.45$, $p < 0.01$), AMA ($r = 0.48$, $p < 0.01$), but not with REE (p = NS).

Thus, the calculated cortisol/insulin ratio was higher in CHF patients than in controls, although this was only significant for All-CHF versus All-C ($p < 0.01$). The ratio was negatively correlated with BW ($r = -0.52$, $p < 0.01$) and AMA ($r = -0.46$, $p < 0.02$).

Table 3. Nutritional Intakes, Nitrogen Excretion, Calorie and Nitrogen Balances, and Energy Availability for Physical Activity in Patients and Controls of the Study

	All-CHF (n = 57)	All-C (n = 49)	Normo-CHF (n = 26)	Normo-C (n = 33)	Maln-CHF (n = 31)	Under-C (n = 16)
Daily nutritional intake						
Calories						
$kcal_I$	1,751.6 ± 237.2	1,806.5 ± 492.9	1,791.4 ± 124.9	1986.8 ± 312.6	1782.8 ± 171.1	1656.3 ± 590.1
$kcal_I$/kg	28.2 ± 5.4	29.7 ± 8.4	24.3 ± 1.9	25.9 ± 4.4	29.5 ± 4.7#	32.8 ± 9.9
Proteins						
g/kg	1.1 ± 0.2	1.2 ± 0.4	1 ± 0.1	1.2 ± 0.4	1.1 ± 0.1	1.2 ± 0.4
$\%kcal_I$	15.9 ± 4.1	16.4 ± 3.6	15.3 ± 2.6	18.1 ± 3.6	16.7 ± 5.4	15 ± 3.1
Carbohydrates						
g/kg	3.8 ± 0.8	3.4 ± 1.1	3.3 ± 0.3	2.9 ± 0.4	4 ± 0.8	3.8 ± 1.4
$\%kcal_I$	50.8 ± 4.1	44.1 ± 7.2†	50.5 ± 2.3	42.8 ± 7.5§	50.9 ± 4.8	45.2 ± 7.4¶
Lipids						
g/kg	1 ± 0.3	1.3 ± 0.4†	0.8 ± 0.1	1 ± 0.4‡	1 ± 0.2	1.4 ± 0.4¶
$\%kcal_I$	31.3 ± 3.7	40 ± 5.1†	31.6 ± 2.3	40.6 ± 7.4§	31.1 ± 4.3	39.5 ± 2.9¶
Nitrogen (N_1), g	11.3 ± 1.3	11.8 ± 4.1	11.8 ± 0.9	14.2 ± 3.8	11 ± 1.4	9.8 ± 3.4
Daily nitrogen excretion, g	10.5 ± 3	7.6 ± 2.8†	11.3 ± 2.6	9.8 ± 2.2	10.9 ± 3.3	5.7 ± 1.6¶
Nitrogen balance, g/day	−1.7 ± 3.2	2.2 ± 3.6†	−1.5 ± 2.9	2.4 ± 4.2‡	−1.9 ± 3.3	2.1 ± 3.5¶
Calorie balance ($kcal_I$ − TEE), kcal/day	−186.3 ± 305	104.2 ± 273†	−299.6 ± 326.7	45.1 ± 144.5‡	−115.2 ± 300.7	153.5 ± 354.9
Energy availability for physical activity ($kcal_I$ − REE), kcal/day	292.5 ± 284.9	497.1 ± 289.1*	181.9 ± 257.6	493.2 ± 96.6‡	322.7 ± 255.3	500.3 ± 399.6

Data are given as mean ± SD. Statistical analysis: *t* test. Statistical significance was set at $p < 0.05$. All-CHF vs. All-C: *$p < 0.05$; †$p < 0.01$; Normo-CHF vs. Normo-C: ‡$p < 0.05$; §$p < 0.01$; Maln-CHF vs. Under-C: ¶$p < 0.01$; Normo-CHF vs. Maln-CHF: #$p < 0.05$.
Abbreviations as in Table 2.

DISCUSSION

The investigation shows that non-obese patients with clinically stable CHF, living at home, do not have adequate daily calorie and protein intakes to meet their metabolic needs and have reduced energy availability for daily life physical activities.

Although strictly interrelated, calorie and nitrogen balances will be discussed here separately for greater clarity.

Negative CB and EA for physical activity. The negative CB can be attributed to a daily calorie intake not commensurate to daily total energy requirements. Some adaptive responses in patients' calorie intake to body energy requirements probably occur over time because depleted CHF patients have higher calorie intakes associated with tendentially higher REEs than non-depleted patients.

Digestive disturbances, alimentary habits and altered macronutrient ingestion are the main factors explaining inadequate calorie intake in patients with CHF.

Digestive disturbances, mainly early satiety (14), induce patients to stop their meals earlier. Early satiety can be caused/accentuated in CHF by the particular habit of distributing most of the day's calories in only two meals (15) or gastric compression by hepatomegaly (14). In this study, we did not quantify the patients' appetite, but a reduced appetite cannot be excluded considering the digestive disturbances, the anorectic action of tumor necrosis factor-alpha and angiotensin II (elevated in CHF) (3), digoxin, altered taste, and depression.

Altered macronutrient ingestion is another factor reducing calorie intake in CHF patients, as the lower lipid intake, the nutrient with the highest energy density, was not compensated for by a proportional increase in carbohydrate intake. Congestive heart failure patients might deliberately restrict fat intake to avoid or limit digestive symptoms (16).

Factors conditioning calorie intake in CHF deserve appropriate investigations because nutritional manipulation can adapt the alimentary pattern to the patient's current digestive status with a consequent calorie intake more adequate to body needs.

The low EA in CHF may be a consequence of loss of metabolic adaptation to body wasting. In fact, part of the food calories (11% in CHF as an entire group, 15% in

Table 4. Plasma Cortisol and Insulin Levels, and Cortisol/Insulin Ratio in Patients and Controls of the Study

	All-CHF (n = 57)	All-C (n = 49)	Normo-CHF (n = 26)	Normo-C (n = 33)	Maln-CHF (n = 31)	Under-C (n = 16)
Cortisol, nmol/l	631.2 ± 182.3	308.6 ± 94.2*	722 ± 181.2	268.1 ± 74.2†	551.2 ± 153.5	341 ± 103.2‡
Insulin, μUI/ml	13.2 ± 7.6	10.7 ± 4	19 ± 13.3	12 ± 4.6	11.5 ± 3.8	9.8 ± 3.7
Cortisol/insulin ratio	62.5 ± 36.6	31.9 ± 12.6*	56 ± 37.4	25 ± 9.2	58 ± 34.7	37.3 ± 13.1

Data are given as mean ± SD. Statistical analysis: *t* test. Statistical significance was set at $p < 0.05$. All-CHF vs. All-C: *$p < 0.01$; Normo-CHF vs. Normo-C: †$p < 0.01$; Maln-CHF vs. Under-C: ‡$p < 0.01$.
CHF = chronic heart failure. Other abbreviations as in Table 2.

depleted CHF, 8% in non-depleted ones) are not available for physical activity but serve to meet the not-reduced amount of REE as normally expected as an adaptive response to BW and lean body mass wasting (13).

The loss of adaptation is probably caused by increased work of visceral organs and tissues, as indirectly suggested by REE/m^2 (2) and accentuated, in turn, by the increased cortisol levels as this hormone is correlated to REE. Of interest, simple elevations of cortisol seem sufficient to elevate the energy consumption for basal homeostasis.

Negative nitrogen balance. The finding of a negative nitrogen balance, indicating a hypercatabolic status, accords with results from Minotti's investigation documenting a negative protein balance across leg tissues in patients with cardiac cachexia (17).

Our investigation offers clear evidence that spontaneous nutrition of clinically stable CHF patients may not compensate for the various catabolic factors that can be present and that a normal protein intake (1.1 g/kg/day) is not synonymous with an adequate protein intake.

Inadequate calorie intake or hormonal derangements are probably responsible for the negative nitrogen balance. A negative nitrogen balance can be caused or exacerbated by inadequate calorie intake because even a partial restriction of normal calorie intake increases nitrogen excretion (18).

The hormonal derangements observed in this study confirm previous findings (7) and may play a role in inducing protein catabolism, given the catabolic action of cortisol and the negative correlation of the cortisol/insulin ratio with skeletal muscle protein stores and BW.

The normal insulin concentrations confirm that, over time, the patients' calorie intake was not in excess, thus explaining the discrepancy with results of previous investigations (7,19) and suggesting a lack of body anabolic response to the catabolic action of cortisol.

The coexistence of severe muscle protein malnutrition with normal BW was found in previous investigations (7) and may be explained by non-proportional rates of loss and apposition of adipose and muscular tissues during catabolic periods (hemodynamic instabilization) and anabolic periods (after resolution of acute decompensation).

Significance of an inadequate calorie protein intake in CHF. In clinically stable CHF patients, an inadequate calorie protein intake should be considered an additive catabolic insult contributing to 1) progressive deterioration of important cellular substrate concentrations such as muscle glycogen and amino acids (4), 2) exacerbation of muscle cell energy crisis (4), and 3) muscle protein breakdown.

In this way, increased wasting of lean mass occurs, which is critical for activities of daily life, and, hence, the subject's quality of life and survival (20).

In summary, inadequate calorie-protein intake in clinically stable, non-obese CHF patients assumes a greater metabolic relevance than would be suspected from its degree.

Clinical implications. The present study has some implications for clinical practice.

Daily intakes of 29.5 kcal/kg + 1.1 g protein/kg by free-living, clinically stable, depleted CHF patients and 24.3 kcal/kg + 1 g protein/kg by normally nourished ones may not be sufficient to ensure a neutral calorie nitrogen balance and tissue conservation.

Clinically stable, depleted CHF patients should have a daily intake of at least 31.8 kcal/kg + 1.37 g protein/kg and normally nourished ones a daily intake of at least 28.1 kcal/kg + 1.12 g protein/kg in order to preserve their actual body composition or limit the effects of hypercatabolism. This study indirectly suggests that great caution should be paid in prescribing a hypocaloric diet in overweight/obese CHF patients, as 24.3 kcal/kg, a calorie allowance higher than usually advised for dieting, is associated with increased body protein breakdown; this could be particularly important for patients with anabolic/catabolic hormone imbalance. The risk is that overweight CHF patients will lose more muscle than fat.

The study also highlights the need to calculate calorie nitrogen balance and not only macronutrient intakes in all patients with CHF in order to determine the patients' metabolic status.

Calculation of energy availability may be clinically useful as they can allow physician to prescribe or advise physical activity more commensurate to the patient's current metabolic possibility.

Study limitations. This investigation was not planned to provide any information about possible causality between insufficient nutrition and the patient's metabolic, tissue deterioration over time. This is a major limitation of the study. Proof of causality, if any, would require an appropriate longitudinal study.

Diary records of seven days may be too short a period to diagnose the presence of lasting inadequate alimentary habits in patients with CHF. We suggest that, in clinically stable, non-obese CHF, three periods of seven-days diary records (and calorie nitrogen balance calculations) over three months would be more representative of the patient's real alimentary habits.

In this study we deliberately avoided the term cachexia to define patients with involuntary reduction of total BW over the last six (7) or 12 months. Although useful in clinical settings, from a nutritional standpoint, cachexia should connote individuals with loss of >10% lean body mass, at which point impaired immune function can generally be documented (21).

Thus, in our study, the term cachexia would probably have been more pertinent for the non-obese patients with severe muscle protein malnutrition (54.4% population).

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REFERENCES

1. Toth MJ, Gottlieb SS, Fisher ML, Poehlman ET. Daily energy requirements in heart failure patients. Metabolism 1997;46:1294–8.
2. Opasich C, Pasini E, Aquilani R, et al. Skeletal mass function at low work level as a model for daily activities in patients with chronic heart failure. Eur Heart J 1997;18:1626–31.
3. Berry C, Clarck AL. Catabolism in chronic heart failure. Eur Heart J 2000;21:521–32.
4. Opasich C, Aquilani R, Dossena M, et al. Biochemical analysis of muscle biopsy in overnight fasting patients with severe chronic heart failure. Eur Heart J 1996;17:1686–93.
5. Mancini DM, Walter G, Reichek N, et al. Contribution of skeletal muscle atrophy to exercise intolerance and altered muscle metabolism in heart failure. Circulation 1992;85:1364–73.
6. Frisancho AR. New forms of upper limb fat and muscle areas for assessment of nutritional status. Am J Clin Nutr 1981;34:2540–5.
7. Anker SD, Chva TP, Ponikowski PP, et al. Hormonal changes and catabolic/anabolic imbalance in chronic heart failure and their importance for cardiac cachexia. Circulation 1997;96:526–34.
8. Frayn KN. Calculation of substrate oxidation rates in vivo from gaseous exchange. J Appl Physiol 1983;55:626–34.
9. Harris JS, Benedict FG. A Biometric Study of Basal Metabolism in Man (Carnegie Institution of Washington publication #279). Washington, DC: Carnegie Institute, 1919.
10. Aquilani R, Dossena M, Foppa P, et al. Low protein diet improves muscle energy metabolism in chronic renal failure. Nutr Clin Prac 1997;12:6.
11. Bistrian BR, Blackburn GL, Sherman M. Therapeutic index of nutritional depletion in hospitalised patients. Surg Gynecol Obstet 1975;141:512–6.
12. Nielsen DH, Amundsen LR. Exercise physiology: an overview with emphasis on aerobic capacity and energy cost. In: Amadsen LR, editor. Cardiac Rehabilitation. New York, NY: Churchill Livingstone, 1981: 11–28.
13. Golden MHN, Jackson AA. Chronic severe undernutrition. In: Olson RE, editor. Present Knowledge in Nutrition. 5th edition. Washington, DC: The Nutrition Foundation, 1984:57.
14. Carr JG, Stevenson LW, Walden JA, Heber D. Prevalence and hemodynamic correlates of malnutrition in severe congestive heart failure secondary to ischemic or idiopathic dilated cardiomyopathy. Am J Cardiol 1989;63:709–13.
15. Moore JG, Christian PE, Brown JA, et al. Influence of meal weight and caloric content on gastric emptying of meals in man. Dig Dis Sci 1984;29:513–9.
16. Hunt JN, Stubbs DE. The volume and energy content of meals as determinants of gastric emptying. J Physiol 1975;245:209–25.
17. Minotti J, Simonini A, Samarel AM. Skeletal muscle protein synthesis in congestive heart failure. Circulation 1993;88:1414.
18. Keys A, Brozek J, Henschel A. The Biology of Human Starvation. Minneapolis, MN: University of Minnesota Press, 1950;1:383.
19. Swan JW, Walton C, Godsland IF, Clark AL, Coats AJS, Oliver MF. Insulin resistance in chronic heart failure. Eur Heart J 1994;15: 1528–32.
20. Walesby RK, Goode AW, Spinks TJ, Herring A, Ranicar AS, Bentall HH. Nutritional status of patients requiring cardiac surgery. J Thorac Cardiovasc Surg 1979;77:570–6.
21. Freeman LM, Roubenoff R. The nutrition implications of cardiac cachexia. Nutr Rev 1994;52:340–7.