Effects of a Nutritional Intervention on Body Composition, Clinical Status, and Quality of Life in Patients With Heart Failure

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OBJECTIVES: We assessed the effects of a nutritional intervention on clinical and nutritional status and quality of life in patients with heart failure.

METHODS: Sixty-five patients with heart failure were assigned to one of two groups: the intervention group (IG; $n = 30$) received a sodium-restricted diet (2000 to 2400 mg/d) with restriction of total fluids to 1.5 L/d, and the control group (CG; $n = 35$) received traditional medical treatment and general nutritional recommendations. Anthropometric, body composition, physical activity, dietary, metabolic, clinical, and quality of life assessments were performed in all patients at baseline and 6 mo later.

RESULTS: At the end of the study, kilocalories, macronutrients, and fluid intakes were significant lower in the IG than in the CG. Urinary excretion of sodium decreased significantly in the IG and increased in the CG (−7.9% versus 29.4%, $P < 0.05$). IG patients had significantly less frequent edema (37% versus 7.4%, $P = 0.008$) and fatigue (59.3% versus 25.9%, $P = 0.012$) at 6 mo than at baseline; in addition, functional class improved significantly, and no changes were observed in the CG. Extracellular water decreased -1.1 ± 3.7% in the IG and increased 1.4 ± 4.5% in the CG ($P = 0.03$). Physical activity increased $2.5\% \pm 7.4\%$ in the IG and decreased -3.1 ± 12.0 in the CG ($P < 0.05$). The IG had a greater increase in total quality of life compared with the CG (19.3% versus 3.2%, $P = 0.02$).

CONCLUSIONS: In this study, the beneficial effect of a supervised nutritional intervention was proved as part of a non-pharmacologic treatment of patients with heart failure, with improvements in clinical status and quality of life. *Nutrition* 2004;20:890–895. ©Elsevier Inc. 2004

KEY WORDS: heart failure, dietary recommendations, body composition, quality of life

INTRODUCTION

Dietary therapy goals in patients with heart failure (HF) are to provide an optimum nutrition with less cardiovascular stress, to accomplish and maintain a good nutritional status, and to decrease or avoid fluid retention for reasons in addition to correcting cardiac cachexia syndrome or limiting its severity. In this sense, the specific recommendations are focused on sodium and fluid restrictions and weight optimization (i.e., decreasing weight in obese patients and increasing weight in cachectic patients). Moreover, due to the high prevalence of coronary disease as a cause of HF, it is necessary to decrease intakes of saturated fats, cholesterol, and simple carbohydrates.[1–3]

Dietary salt restriction is considered essential in the management of symptomatic HF[4] because these patients are unable to normally excrete a sodium load and plasma atrial natriuretic factor levels do not increase in response.[5] Moreover, dietary transgression may be a precipitating factor in more than 20% of patients hospitalized for decompensate HF,[4] and diuretics become less effective.[2] However, the level of restriction is less clear. A diet with 2 g of sodium is unpalatable for most patients, and the cost of low-sodium foods can be a burden for economically disadvantaged patients. A diet with 3 g of sodium may be a realistic target for patients with mild to moderate HF.[1,6] Nevertheless, in patients with advanced HF, a sodium restriction to no more than 2 g/d is acceptable, particularly if large doses of diuretics are required.[1,2]

The guidelines for the evaluation and management of patients with HF from the American College of Cardiology and American Heart Association[7] and the European Society of Cardiology[8] recommend restricted intakes of sodium and fluid as basic in the non-pharmacologic management of HF and as cautious to prevent disease exacerbation. However, no studies have evaluated specific recommendations related to these restrictions and the clinical effects of these recommendations, so the current level of evidence is C. The purpose of the present study was to evaluate the effects of a dietary intervention focused on the improvement of clinical and nutrition statuses and quality of life and relief from symptoms.

MATERIALS AND METHODS

Patients

This 6-mo randomized controlled study included 65 male and female patients who attended the Heart Failure Clinic of the National Institute of Medical Science and Nutrition "Salvador Zubirán" (INCMNSZ) in Mexico City. Patients were recruited if they were 18 y or older and had a confirmed diagnosis of HF (defined as decreased systolic and/or diastolic function as determined by echocardiogram). Candidates were excluded if they already had a specific dietary regimen, renal failure, uncontrolled

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dysthyroidism, hepatic failure, unstable ischemic heart disease (unstable angina and/or myocardial infarction), recent myocardial revascularization procedures (arterial coronary angioplasty and/or surgical revascularization in the previous 3 mo), or severe arrhythmias.

The present study was approved by the institutional ethics committee of biomedical research in humans of the INCMNSZ, and all patients were informed regarding the purpose of the study and signed informed consent forms.

Management Protocol

Subjects were randomized to the intervention group ($n = 30$) or the control group ($n = 35$). In addition to the traditional medical management of HF, a salt-restricted diet was prescribed to the patients in the intervention group (2000 to 2400 mg/d of sodium),[9,10] and its composition, in total calories, was 50% to 55% of carbohydrates (with a preference for complex carbohydrates and fiber), approximately 15% of protein, 30% to 35% of total lipids (<10% saturated, 10% polyunsaturated, 15% monounsaturated, and <300 mg/d of cholesterol).[11,12] Total fluids were limited to 1.5 L/d, including the water in food.[2] These patients received written and oral instructions from the dietitian and were advised to avoid sodium-rich food, limit the intake of food with moderate amounts of sodium, and eat no more than one-fourth a teaspoon of salt per day. Adherence to the diet was evaluated by a 24-h food recall collected every 3 mo, and suggestions and corrections were provided, when necessary.

Patients in the control group received traditional management of HF, including common dietary advisories regarding decreased sodium and fluid intakes.

Anthropometric variables, body composition, physical activity, dietary intake, metabolic, clinical, and quality of life evaluations were performed at baseline and at 6 mo.

Anthropometry and Body Composition

Weight and height were measured in accordance to the reference manual of anthropometric standardization,[13] and all subjects wore little clothing and were barefoot.

Body mass index was calculated by dividing total body weight (kilograms) by height squared (square meters). Body composition was evaluated with tetrapolar and multiple-frequencies equipment (BodyStat QuadScan 4000).

Physical Activity and Dietary Intake

Daily physical activity was evaluated by a 3-d questionnaire (2 weekdays and 1 weekend and using the average of the 3 d) validated in a Mexican population,[14] which was filled out at home by each patient. Data on dietary intake and physical activity were obtained by a 3-d food record. Nutrients obtained from a 3-d average of food records were analyzed and entered into a computerized nutrition database (SCVAN 1.0), which contains the nutritional values of Mexican food, for analysis.

Biochemical Analysis

Fasting blood samples were analyzed for plasma concentrations of glucose, albumin, hemoglobin, hematocrit, triacylglycerol, total cholesterol, high-density lipoprotein cholesterol, and low-density lipoprotein cholesterol. Plasma glucose was enzymatically determined with the glucose oxidase technique, and plasma concentrations of total cholesterol and triacylglycerol were measured with an automated system (Synchron CX5, Beckman Instruments). High-density lipoprotein cholesterol was precipitated with phosphotungstic acid and Mg^{2+}, and low-density lipoprotein cholesterol was calculated as follows: total cholesterol − (high-density lipoprotein cholesterol + triacylglycerol/5).

We defined dyslipidemia as a total cholesterol level above 200 mg/dL, a low-density lipoprotein cholesterol level above 100 mg/dL, a high-density lipoprotein cholesterol level above 40 mg/dL, and/or a triacylglycerol level above 150 mg/dL.

Twenty-four–hour urine samples were assessed for sodium excretion. To evaluate sodium intake, we assumed that 24-h urinary sodium excretion is equivalent to approximately 86% of the daily sodium intake, as reported by Korhonen et al.[15]

Clinical Data

Symptoms such as fatigue, dyspnea, edema, congestion (dyspnea, edema, weight gain, decubitus intolerance, and effortful coughing), and functional class according the New York Heart Association (NYHA)[16] were evaluated during the medical interview. A baseline echocardiogram was performed for every patient to determine cardiac function. All evaluations were performed by a cardiologist blinded to the study groups.

Quality of Life

Quality of life evaluation was carried out by a questionnaire adapted from the Kansas City Cardiomyopathy Questionnaire[17] and the Minnesota Living With Heart Failure Questionnaire.[18,19] This questionnaire identified the following domains: physical limitations, social interference, symptoms, food intake, emotional and economic aspects, sleep disturbances, medication side effects, and comprehension of medical indications; overall quality of life was the sum of these domains. For the test–retest reliability of this instrument, we obtained a κ value higher than 7.0 ($P < 0.05$) for each item. To facilitate interpretability, the score for each domain was transformed to a 0 to 100 range by subtracting the lowest possible score, dividing by the range of the scale, and multiplying by 100.[17,20] Thus, results near 100 would indicate better quality of life.

Statistic Analysis

All analyses were performed with commercially available software (SPSS 10.0 for Windows, SPSS, Inc., Chicago, IL, USA). Continuous variables are expressed as mean ± standard deviation, and categorical variables are presented as absolute and relative frequencies. Unpaired *t* test was used to compare the two groups at baseline and to compare changes from baseline to 6 mo (end of study) between groups if the variables were continuous. To compare the changes from baseline to 6 mo between groups for categorical variables, McNemar's test was used for dichotomy variables and Friedman's test was used for variables of more than two categories. $P < 0.05$ was considered statistically significant.

RESULTS

Of the 65 patients included in the present study, three were excluded from the intervention group due to non-adherence to the diet. In the control group, one patient was excluded from the clinic and three were lost to follow-up; thus, 58 patients completed the study, with 27 in the intervention group and 31 in the control group.

Baseline

Table I lists baseline characteristics. Distribution by sex was significantly different between groups. It is important to mention that there were no significant differences in symptoms, comorbidities, and diuretic use between groups, and that, according to the NYHA classification, both groups had the same degree of compromise in functional capacity. There was a high prevalence of dyslipidemia, hypertension, and ischemic heart disease in both groups. Mean nutrient intake at baseline was similar between groups (Table II).

TABLE I.

BASELINE CHARACTERISTICS OF PATIENTS

Variables	Intervention (n = 27)	Control (n = 31)	P
Men	33.3% (9)	61.3% (12)	0.03
Women	66.7% (18)	38.7% (19)	
Age (y)	64.2 ± 16.2	59.9 ± 16.4	NS
Anthropometric			
BMI (kg/m^2)	27.5 ± 5.4	27.3 ± 4.9	NS
Weight (kg)	63.9 ± 15.4	67.6 ± 13.0	NS
Height (m)	1.52 ± 0.1	1.58 ± 0.07	0.03
Metabolism			
Albumin (g/dL)	3.9 ± .4	3.7 ± .5	0.005
Hemoglobin (mg/dL)	13.6 ± 1.4	14.4 ± 2.4	NS
Hematrocrit (%)	40.9 ± 4.1	43.2 ± 6.7	NS
Total cholesterol (mg/dL)	182.6 ± 51.7	194.3 ± 40.8	NS
LDL cholesterol (mg/dL)	110.6 ± 39.5	118.9 ± 31.2	NS
HDL cholesterol (mg/dL)	42.1 ± 12.3	41.7 ± 10.7	NS
Triacylglycerol (mg/dL)	137.1 ± 71.5	143.8 ± 67.9	NS
Cardiologic			
LVEF (%)	40.0 ± 15.6	42.3 ± 15.5	NS
LVEDd (mm)	51.3 ± 9.5	52.1 ± 8.3	NS
LVESd (mm)	38.1 ± 11.6	37.2 ± 11.7	NS
IVS (mm)	11.7 ± 3.3	10.6 ± 2.2	NS
PW (mm)	10.6 ± 2.3	9.8 ± 1.6	NS
RVDd (mm)	39.8 ± 11.8	45.7 ± 10.1	NS
NYHA class			
I	59.3% (16)	56.7% (17)	
II	22.2% (6)	30.0% (9)	NS
III	18.5% (5)	13.3% (4)	
Symptoms			
Congestion	7.4% (2)	16.1 (5)	NS
Edema	37.0% (10)	38.7% (12)	NS
Fatigue	59.3% (16)	38.7% (12)	NS
Comorbidities			
Dyslipidemia	74.1% (20)	76.9% (20)	NS
Hypertension	66.7% (18)	69.0% (20)	NS
IC	50.0% (13)	50.0% (13)	NS
Diabetes	29.6% (8)	51.7% (15)	NS
Rheumatoid arthritis	22.2% (6)	23.1% (6)	NS
Dysthyroidism	22.2% (6)	20.7% (6)	NS
Drugs			
Diuretics	54.5%	56.0%	NS

BMI, body mass index; HDL, high-density lipoprotein; IC, ischemic cardiomyopathy; IVS, interventricular septum; LDL, low-density lipoprotein; LVEDd, left ventricular end-diastolic diameter; LVEF, left ventricular ejection fraction; LVESd, left ventricular end-systolic diameter; NS, not significant; NYHA, New York Heart Association; PW, posterior wall; RVDd, right ventricular diastolic diameter.

TABLE II.

MEAN NUTRIENT INTAKES OF PATIENTS AT ENTRY

	Intervention (n = 27)	Control (n = 31)	P
Fiber (g)	8.1 ± 5.6	8.1 ± 4.5	NS
Energy (kcal)	1635.3 ± 662.2	1654.4 ± 570.7	NS
Carbohydrate (g)	209.4 ± 76.7	221.1 ± 83.0	NS
Kilocalories from carbohydrate (%)	52.6 ± 9.8	53.5 ± 6.8	NS
Sugar (%)	16.3 ± 8.8	16.6 ± 8.3	NS
Protein (g)	64.4 ± 36.4	64.5 ± 30.2	NS
Kilocalories from protein (%)	15.4 ± 2.4	15.5 ± 3.6	NS
Total fat (g)	62.5 ± 40.7	58.3 ± 22.2	NS
Kilocalories from total fat (%)	33.5 ± 8.4	31.8 ± 5.4	NS
Cholesterol (mg)	252.3 ± 282.0	219.9 ± 184.6	NS
Saturated fat (g)	16.6 ± 14.7	13.4 ± 6.1	NS
Kilocalories from saturated fat (%)	8.7 ± 3.4	7.5 ± 2.9	NS
Sodium (mg)*	691.3 ± 504.8	676.4 ± 574.1	NS
Total fluids	1616.0 ± 713.9	1560.1 ± 536.7	NS

* Excluding salt used in cooking.
NS, not significant

Follow-up

ANTHROPOMETRY AND BODY COMPOSITION. After 6 mo of follow-up, anthropometric variables showed no significant differences between baseline and final measurements in the two groups. Body composition evaluation showed significant changes between groups only with respect to extracellular water (liters; Table III), which decreased in the intervention group and increased in the control group (−1.1 ± 3.7 versus 1.4 ± 4.5, P = 0.03), and in the impedance value to a frequency of 5 kHz, which is used to determine the water content in this body compartment.

PHYSICAL ACTIVITY AND DIETARY INTAKE. Physical activity evaluated by questionnaire increased 2.5 ± 7.4% in the intervention group after 6 mo of follow-up and decreased −3.1 ± 12.0% in the control group ($P < 0.05$).

In the intervention group, dietary changes decreased levels of biochemical markers from baseline to the end of the study, except protein, expressed as a percentage of total energy intake, which was significantly higher than in the control group. Total fluid intake at the end of the study was lower in the intervention group than in the control group (Table IV). Mean sodium intake at baseline and at the end of the study in both groups was less than 1000 mg. However, these data should not be considered as the mean total intake because it did not account for the salt used in cooking, which represents a major component of salt intake in this population. For that reason, we preferred urinary sodium excretion determination (see below).

METABOLIC OUTCOMES. At the end of the study, there were no significant differences between groups with respect to metabolic profiles, except for 24-h urinary sodium excretion, which decreased significantly in the intervention group compared with the control group (Figure 1). At baseline, 24-h urinary sodium excretion levels were 2070.3 ± 726.8 mg/L in the intervention group and 1769.3 ± 661.0 mg/L in the control group. And after 6 mo of follow-up, 24-h urinary sodium excretion levels were 1670.6 ± 487.3 mg/L in the intervention group and 2180.3 ± 870.9 mg/L in the control group. However, the average urinary sodium excretion has been evaluated to be 86% of total sodium intake.[15] On that basis, the urinary sodium excretion levels at the end of the study corresponded to an average sodium intake of 1942.7 ± 566.7 mg/d in the intervention group and 2535.2 ± 1012.3 mg/d in the control group.

CLINICAL DATA. At the end of the study, frequency of the main signs and symptoms of HF decreased in the intervention group, with significant differences for edema (37% versus 7.4%, P = 0.008) and fatigue (59.3% versus 25.9%, P = 0.012). No significant changes were found in the control group.

Patients in the intervention group had significant improvements in functional class, as shown by fewer patients in classes II and III and more patients in class I (Figure 2). In the control group, a

TABLE III.

MEAN ANTHROPOMETRIC VARIABLES AND MAGNITUDE OF CHANGE FROM BASELINE TO END OF STUDY

	Baseline		End		% Change		
Variables	Intervention (n = 27)	Control (n = 31)	Intervention (n = 27)	Control (n = 31)	Intervention (n = 27)	Control (n = 31)	P for % change
BMI	27.5 ± 5.4	27.3 ± 4.9	27.1 ± 5.5	27.2 ± 4.9	−1.0 ± 4.3	−0.2 ± 4.9	NS
Weight (kg)	63.9 ± 15.4	67.6 ± 13.0	63.4 ± 15.9	67.5 ± 12.6	−1.0 ± 4.3	−0.2 ± 4.9	NS
FM (%)	41.3 ± 9.5	34.3 ± 8.5	41.4 ± 9.8	35.0 ± 8.4	2.1 ± 4.8	3.3 ± 15.2	NS
FM (kg)	26.1 ± 8.2	23.3 ± 8.1	25.9 ± 8.2	23.8 ± 8.4	1.6 ± 5.7	3.7 ± 17.7	NS
LM (%)	58.7 ± 9.5	65.7 ± 9.0	58.6 ± 9.8	65.0 ± 8.4	−1.5 ± 3.6	−0.8 ± 5.5	NS
LM (kg)	37.8 ± 12.1	44.4 ± 9.7	37.6 ± 12.6	43.6 ± 9.1	−2.0 ± 5.4	−0.9 ± 6.3	NS
DLW (kg)	6.4 ± 4.6	9.7 ± 4.4	6.4 ± 4.8	8.7 ± 4.3	−3.8 ± 12.3	−9.2 ± 27.9	NS
TBW (%)	50.4 ± 6.7	52.2 ± 5.8	50.7 ± 6.3	52.4 ± 6.8	−0.4 ± 2.9	0.3 ± 6.4	NS
TBW (lt)	31.7 ± 7.4	35.0 ± 6.3	31.7 ± 7.5	34.9 ± 5.8	−1.0 ± 4.0	0.4 ± 6.1	NS
ECW (%)	23.3 ± 2.7	23.3 ± 2.3	23.4 ± 2.7	23.6 ± 2.7	−0.5 ± 2.8	1.3 ± 4.4	NS
ECW (lt)	14.6 ± 2.8	15.5 ± 2.1	14.5 ± 2.8	15.7 ± 2.2	−1.1 ± 3.7	1.4 ± 4.5	0.03
ICW (%)	27.1 ± 3.2	29.3 ± 4.	27.2 ± 2.9	28.9 ± 3.6	−0.1 ± 1.9	−1.0 ± 7.5	NS
ICW (lt)	17.3 ± 4.9	19.8 ± 4.8	17.3 ± 5.0	19.4 ± 3.7	−0.7 ± 4.1	−0.8 ± 7.4	NS
BCM (kg)	24.7 ± 7.0	28.3 ± 6.8	24.7 ± 7.1	27.7 ± 5.3	−0.6 ± 3.9	−0.8 ± 7.4	NS
TSW (lt)	0.2 ± 1.7	−0.3 ± 1.0	−0.1 ± 1.1	−0.1 ± 1.2	11.2 ± 96.7	−1.6 ± 119.0	NS
Impedance index	0.8 ± 0.0	0.8 ± 0.1	0.8 ± 0.0	0.8 ± 0.0	−0.3 ± 1.6	3.5 ± 20.6	NS
5 kHz*	664.5 ± 140.4	636.3 ± 112.5	671.2 ± 138.9	621.7 ± 114.3	2.3 ± 7.7	−2.5 ± 7.9	0.03
50 kHz*	602.9 ± 136.1	569.4 ± 110.4	608.8 ± 135.5	562.2 ± 104.5	2.0 ± 7.2	−1.1 ± 10.2	NS
100 kHz*	576.0 ± 133.4	544.8 ± 103.3	581.5 ± 132.4	536.5 ± 100.4	2.0 ± 7.1	−1.7 ± 8.5	NS
200 kHz*	550.5 ± 128.8	517.3 ± 106.78	556.5 ± 132.4	511.8 ± 96.7	1.9 ± 6.9	−0.4 ± 13.1	NS

* Resistance flow of the current at 5, 50, 100, and 200 kHz.

BCM, body cell mass; BMI body mass index; DLW, dry lean weight; ECW, extracellular water; FM, fat mass; ICW, intracellular water; LM, lean mass; NS, not significant; TBW, total body water; TSW, third space water

larger proportion of patients in classes I and III were observed in comparison with baseline measurements; nevertheless, none of these changes had statistical significance (Figure 3).

It is important to emphasize that the uses and doses of diuretics were similar at baseline and at the end of the study in both groups.

QUALITY OF LIFE. In the intervention group, quality of life showed a significant improvement in the domains of emotional aspects and sleeping disturbances, in comparison with the control group, and overall quality of life. Domains of symptoms and economical aspects showed a similar tendency (Table V).

TABLE IV.

MEAN NUTRIENT INTAKE AND MAGNITUDE OF CHANGE FROM BASELINE TO END OF STUDY

	Baseline		End		% Change		
Variables	Intervention (n = 27)	Control (n = 31)	Intervention (n = 27)	Control (n = 31)	Intervention (n = 27)	Control (n = 31)	P for % change
Fiber (g)	8.1 ± 5.6	8.1 ± 4.5	9.4 ± 4.7	7.9 ± 4.0	37.0 ± 76.0	17.2 ± 80.7	NS
Energy (kcal)	1635.3 ± 662.2	1654.4 ± 570.7	1289.9 ± 362.3	1749.3 ± 611.7	−16.8 ± 24.7	10.7 ± 30.6	0.001
Carbohydrate (g)	209.4 ± 76.7	221.1 ± 83.0	184.7 ± 64.7	234.1 ± 91.8	−9.7 ± 31.4	10.0 ± 30.7	0.02
Kilocalories from carbohydrate (%)	52.6 ± 9.8	53.5 ± 6.8	56.9 ± 5.8	53.0 ± 7.6	11.6 ± 26.4	−0.2 ± 14.3	NS
Sugar (%)	16.3 ± 8.8	16.6 ± 8.3	11.4 ± 5.4	15.3 ± 6.0	−1.3 ± 83.2	7.1 ± 54.8	NS
Protein (g)	64.4 ± 36.4	64.5 ± 30.2	52.9 ± 15.5	63.9 ± 20.8	−8.7 ± 29.4	7.5 ± 31.5	NS
Kilocalories from protein (%)	15.4 ± 2.4	15.5 ± 3.6	16.7 ± 3.0	14.9 ± 2.3	10.9 ± 27.0	−2.2 ± 18.2	<0.05
Total fat (g)	62.5 ± 40.7	58.3 ± 22.2	38.6 ± 12.0	63.0 ± 24.4	−26.4 ± 32.9	16.2 ± 40.6	<0.0001
Kilocalories from total fat (%)	33.5 ± 8.4	31.8 ± 5.4	27.2 ± 4.4	32.7 ± 6.1	−13.3 ± 25.1	4.4 ± 19.6	0.01
Cholesterol (g)	252.3 ± 282.0	219.9 ± 184.6	99.8 ± 65.8	253.0 ± 224.9	−17.6 ± 88.1	86.4 ± 266.6	NS
Saturated fat (g)	16.6 ± 14.7	13.4 ± 6.1	7.0 ± 4.1	16.5 ± 7.9	−43.0 ± 42.1	35.4 ± 65.1	<0.0001
Kilocalories from saturated fat (%)	8.7 ± 3.4	7.5 ± 2.9	4.8 ± 2.2	8.6 ± 3.0	−36.6 ± 31.2	28.3 ± 70.0	<0.0001
Sodium (mg)*	691.3 ± 504.8	676.4 ± 574.1	398.4 ± 235.0	881.7 ± 656.8	−50.5 ± 34.9	48.1 ± 164.7	0.012
Total fluids	1616.0 ± 713.9	1560.1 ± 536.7	1405.5 ± 359.1	1840.5 ± 579.2	−4.1 ± 36.9	25.3 ± 44.4	0.01

* Excluding salt for cooking.

NS, not significant

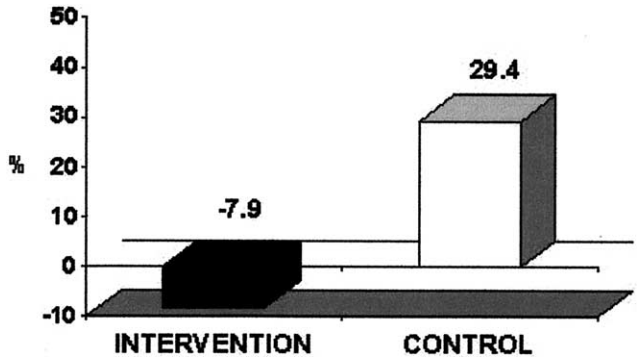

FIG. 1. Change in urinary excretion of sodium from baseline to the end of the study ($P < 0.05$).

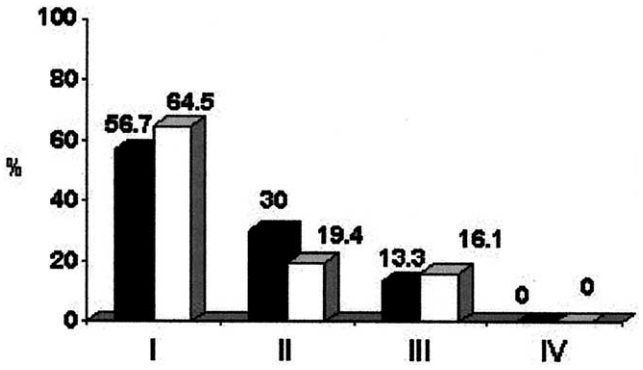

FIG. 3. Functional class at baseline (solid bars) and at the end of the study (open bars) in the control group (P not significant).

Despite differences in sex distribution between groups, there were no significant differences in the results (percentage of changes) when the two groups were stratified by sex according to a general lineal model.

DISCUSSION

This study reports the effects on body composition, clinical status, and quality of life of teaching free-living individuals with HF how to incorporate food choices with low contents of sodium, saturated fat, sugars, and fluids. The result of this non-pharmacologic intervention was that patients changed their selection of food but not the amount, and this was reflected in clinical status (signs and symptoms), NYHA functional class, and enhanced quality of life, but no significant changes in weight and body mass index before and after intervention. Therefore, weight loss as a main objective of nutritional invention has to be reconsidered in obese HF patients because the observed improvement in the intervention group occurred without significant decreases in weight and body mass index and because of the high risk of developing cardiac cachexia in these patients, which is a prognostic variable of higher morbidity and mortality rates at 18 mo.[21] Long-term studies are required to determine the prognosis of patients who have HF and remain obese in comparison with those who lose weight induced by nutritional treatment.

Our nutritional intervention resulted in lower levels of total and saturated lipids in the interventional group; nevertheless, these measurements were not reflected in weight or in changes in blood lipid profile. These results are consistent with those of other studies. De Lorgeril et al.[22] found that plasma lipids, body weight, and blood pressure did not change significantly after decreasing levels of dietary cholesterol and total and saturated fats but that the frequency of infarction and death decreased in the intervention group. Tang et al.[23] found in a meta-analysis that total cholesterol decreased 5.3% after a lipid-restricted diet of 6 mo and 7% with a very strict diet. However, in ambulatory patients seen in daily clinical practice, an adherence of 100% is very hard to achieve when prescribing strict diet regimens.

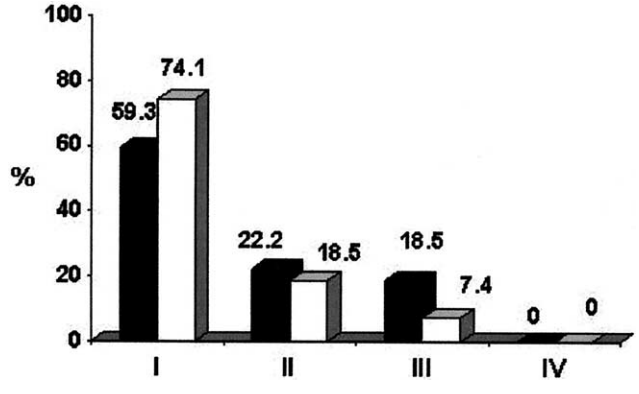

FIG. 2. Functional class at baseline (solid bars) and at the end of the study (open bars) in the intervention group ($P = 0.025$).

An important result of this dietary intervention was decreased intakes of sodium and fluids. The changes in sodium intake presented in Table IV shows a decreased consumption of sodium-rich food but does not represent total sodium intake because we did not include the salt used in cooking in the performance of that analysis. For this reason, urinary sodium excretion was evaluated. Average urinary sodium excretion has been evaluated to be 86% of total sodium intake.[15] On that basis, the urinary sodium excretion levels at the end of the study corresponded to average sodium intakes of 1942.7 ± 566.7 mg/d in the intervention group and 2535.2 ± 1012.3 mg/d in the control group. These decreases in sodium and total fluid intakes to less than 1500 mL/d, observed in the intervention group after a 6-mo follow-up, were reflected in clinical improvements, mainly the absence of edema and fatigue.

Decreased frequency of edema in the intervention group was confirmed with decreased levels extracellular water as reflected in the percentage of change in this group at the end of the study. Because the use of diuretics was similar in the two groups (54.5% in the intervention group versus 56.0% in the control group, $P = 0.9$), one can assume that these drugs were not a factor in the different body water compositions between groups. Because of the relation between extracellular water and edema, bioelectric impedance must be considered a useful tool for the follow-up of extracellular water levels in patients with HF, to identify increased levels of body water and provide adequate treatments to avoid edema, which is the clinical expression of such alterations and more severe symptoms for the patient.

The differences in frequency of edema at baseline and at the end of study and the minimal changes in extracellular water and total body water are likely due to changes in HF, in which the distribution of extracellular water is preceded by edema; if edema depends on increased interstitial fluid, then even minimal changes in extracellular volume will have a clinical effect (in the presence of edema) when it returns to the vascular space, although without significant changes in total body water. A probe for this possibility is that patients with HF have dilutional anemia without edema before the edema appears, at least at the beginning, with a different fluid distribution in the body compartments. In addition, evaluation for edema was not modified by any knowledge of the group assignments of the patients because the cardiologist was blinded to this information.

TABLE V.

MEAN SCORES OF QUALITY OF LIFE AND MAGNITUDE OF CHANGE FROM BASELINE TO END OF STUDY

	Baseline		End		% Change		
Domains	Intervention (n = 27)	Control (n = 31)	Intervention (n = 27)	Control (n = 31)	Intervention (n = 27)	Control (n = 31)	P for % change
Physical limitation	61.4 ± 22.5	65.6 ± 23.0	67.8 ± 18.7	67.6 ± 19.9	19.8 ± 29.9	15.2 ± 72.5	NS
Social interference	34.8 ± 19.9	44.4 ± 27.1	39.2 ± 18.6	50.7 ± 28.5	35.4 ± 105.0	48.4 ± 171.9	NS
Symptoms	58.3 ± 17.0	63.2 ± 20.7	68.9 ± 20.0	62.8 ± 19.4	24.5 ± 33.6	9.1 ± 45.7	NS
Food intake	66.7 ± 25.2	74.2 ± 25.2	68.3 ± 27.0	71.3 ± 24.8	7.8 ± 35.3	2.9 ± 44.0	NS
Emotional aspects	47.2 ± 28.1	53.8 ± 25.6	62.5 ± 23.5	60.0 ± 25.0	98.3 ± 193.2	13.8 ± 48.2	0.03
Economic aspects	42.6 ± 36.6	47.6 ± 36.7	52.9 ± 30.3	52.50 ± 33.1	−24.0 ± 43.3	9.1 ± 77.3	NS
Sleep disturbances	74.1 ± 30.3	78.7 ± 31.0	87.7 ± 27.2	78.7 ± 28.7	39.5 ± 108.9	−5.1 ± 35.9	0.05
Medication side effects	81.5 ± 29.9	71.8 ± 32.1	86.5 ± 24.7	72.5 ± 35.0	1.0 ± 37.0	−4.8 ± 45.1	NS
Comprehension of medical indications	88.9 ± 25.3	87.1 ± 22.2	94.2 ± 16.3	83.3 ± 24.0	6.0 ± 30.0	6.7 ± 52.1	NS
Total quality of life	555.4 ± 117.0	586.3 ± 126.6	628.1 ± 99.4	599.3 ± 128.3	19.3 ± 31.7	3.2 ± 18.4	0.02

NS, not significant

Decreased edema and fatigue after nutritional intervention are likely explained by the improvement in sleeping (according to the quality of life questionnaire) and increased physical activity (according the 3-d questionnaire), and this was reflected in improvements in NYHA functional class and the scores for emotion and total quality of life.

In conclusion, an isocaloric dietary intervention with restrictions of sodium, saturated fats, sugars, and fluids has favorable effects on the clinical state (signs and symptoms), NYHA functional class, and quality of life in patients with HF. Moreover, a restriction of sodium intake to less than 2400 mg/d and of total fluids to less than 1500 mL/d had a positive effect by decreasing the amount of extracellular fluid and, hence, the development of edema, elevating functional class, and enhancing quality of life by permitting engagement in ordinary activities that help integrate patients into their social environments, with the positive emotional effect that this implies.

REFERENCES

1. Dracup K, Barker DW, Dunbar SB, Dacey RA, et al. Management of heart failure: II. Counselling education, and lifestyle modification. JAMA 1994;272: 1442–1446
2. Uretsky BF, Pina I, Quigg RJ, Brill JV, MacInerney EJ, Mintzer R, et al. Beyond drug therapy: nonpharmacologic car of the patient with advanced heart failure. Am Heart J 1998;135:5264–84
3. Grady K, Dracup K, Kennedy G, Moser D, Piano M, Warner L, et al. Team management of patients with heart failure. A statement for healthcare professionals from the Cardiovascular Nursing Council of the American Heart Association. Circulation 2000;102:2443–56
4. Neily JB, Toto KH, Gardner EB, et al. Potential contributing factors to noncompliance with dietary sodium restriction in patients with heart failure. Am Heart J 2002;143:29–33
5. Volpe M, Tritto C, DeLuca N, Rubattu S, Rao M, Lamenza F, et al. Abnormalities of sodium handling and of cardiovascular adaptations during high salt diet in patients with mild heart failure. Circulation 1993;88(pt 1):1620–7
6. Cohn JN. The management of chronic heart failure. N Engl J Med 1996;335: 490–8
7. Hunt SA, Baker DW, Chin MH, Ciquegrani MP, et al. ACC/AHA guidelines for the evaluation and management of chronic heart failure in the adult: executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. Circulation 2001;104:2996–3007
8. Task Force for the Diagnosis and Treatment of Chronic Heart Failure, European Society of Cardiology. Guidelines for the diagnosis and treatment of chronic heart failure. Eur Heart J 2001;22:1527–60
9. National Institutes of Health, National Heart, Lung and Blood Institute. Available at: http. //dash.bwh.harvard.edu.
10. US Department of Health and Human Services. The DASH eating plan. Available at: www.americanheart.org.
11. Krauss RM, Deckelbaum RJ, Ernst N, Fisher E, Howard BV, Knopp RH, et al. Dietary guidelines for healthy American adults. A statement for health professionals from the Nutrition Committee, American Heart Association. Circulation 1996;94:1795–1800
12. Yu-Poth S, Zhao G, Etherton T, Naglak M, Jonnalagadda S, Kris-Etherton P. Effects of the National Cholesterol Education Program's Step I and Step II dietary intervention programs on cardiovascular disease risk factors: a meta-analysis. Am J Clin Nutr 1999;69:632–46
13. Lohman TG, Roche AF, Martorell R. Anthropometric standardization reference manual, Abridged edition. Champaign, IL: Human Kinetics Books, 1991
14. López A, Reyes S, Castillo L, Dávalos A, González J. Reproducibilidad y sensibilidad de un cuestionario de actividad física en población mexicana. Salud Publica Mex 2001;43:306–12
15. Korhonen MH, Järvinen R, Sarkkinen ES, Uusitupa M. Effects of a salt-restricted diet on the intake of other nutrients. Am J Clin Nutr 2000;72:414–20
16. New York Heart Association. Disease of the heart and blood vessels. Nomenclature and criteria for diagnosis, 6th ed. Boston: Little, Brown, 1964, p. 114.
17. Green CP, Porter CB, Bresnahan DR, Spertus JA. Development and evaluation of the Kansas City Cardiomyopathy Questionnaire: a new health status measure for heart failure. J Am Coll Cardiol 2000;35:1245–55
18. Rector TS, Cohn JN. Assessment of patients outcome with the Minnesota Living with Heart Failure questionnaire: Reliability and validity during a randomised, double-blind, placebo-controlled trial of pimobendan. Am Heart J 1992;124: 1017–25
19. Rector TS, Cohn JN. Patients' self-assessment of their congestive heart failure. Part 2: content, reliability and validity of a new measure, the Minnesota Living With Heart Failure questionnaire. Heart Failure 1987:198–209.
20. Fayers PM, Machin D. Quality of life. Assessment, analysis and interpretation. Chichester: John Wiley & Sons, 2000, p. 395.
21. Anker S, Ponikowski P, Susan V, Peng T, Clark A, Webb-Peploe K, et al. Wasting as independent risk factor for mortality in chronic heart failure. Lancet 1997;349:150–3
22. De Lorgeril M, Renaud S, Mamelle N, Salen P, Martin JL, Monjaud I, et al. Mediterranean alpha-linolenic acid-rich diet in secondary prevention of coronary heart disease. Lancet 1994;343:1454–9
23. Tang JL, Armitage JM, Lancaster T, Silagy CA, Fowler GH, Neil AW. Systemic review of dietary intervention trials to lower blood total cholesterol in free-living subjects. BMJ 1998;316:1213–20