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Assessment of nutritional and metabolic status of paraplegics

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Abstract—Nutritional and metabolic assessment using anthropometric, biochemical, immunological, and indirect calorimetric techniques was performed on 17 healthy paraplegic males with a mean age of 44.2 ± 14.6 years and mean duration of injury of 17.8 ± 12.3 years. Significant differences in energy expenditure were observed; only 29.4 percent were normometabolic [measured resting energy expenditure: (MREE) 90-110 percent of predicted resting energy expenditure (PREE)], 35.3 percent were hypermetabolic (MREE>110 percent of PREE) and 35.3 percent were hypometabolic (MREE<90 percent of PREE). Obesity (weight>110 percent ideal body weight) was maximum in hypometabolic patients (83.3 percent) due to the imbalance between caloric intake and energy expenditure ($p<0.05$). None of the patients had normal values for all four objective measurements of nutritional assessment (albumin, transferrin, total lymphocyte count, and cutaneous hypersensitivity). Mild malnutrition was evidenced in 47 percent of patients; 53 percent of patients demonstrated some index of moderate malnutrition. We conclude that nutritional therapy based on measurements of energy expenditure instead of predictive equations will benefit these patients. A larger long-term study is needed to determine the ideal predictive measurements of nutritional assessment with their optimal cutoff values applicable to the spinal cord–injured patient.

INTRODUCTION

Spinal cord injury results in various metabolic and endocrine disorders (8). Earlier studies (8, 10, 18, 32) demonstrated that spinal cord–injured patients have decreased body cell mass, increased proportion of body fat, and high incidence of malnutrition. In the early period after spinal cord injury, there is a decrease in the metabolic rate that is roughly proportional to the magnitude of the "spinal shock" (9, 31). Because little is known about the energy needs of the chronic spinal cord–injured patient, nutritional requirements are based on the assumption that such patients have decreased caloric needs. The absence of data concerning the nutritional and metabolic status of these patients may lead to overfeeding or underfeeding. We present data from anthropometric, biochemical, immunological and indirect calorimetric techniques in an attempt to define the nutritional and metabolic status of the spinal cord–injured patient.

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METHODS

Patient Population

Included in this study were 17 males with chronic paraplegia who ranged in age from 27 to 75 years (mean ± SD 42.8 ± 12.7 yr). Spinal cord lesions were at the level of T_4 to L_2, with a mean duration of injury of 17.8 ± 12.3 years (range 1.5 to 39 yr). All 17 patients had completed a rehabilitation program and were receiving a regular diet. Patients were free of severe cardiac, renal, or hepatic disease; exhibited no acute

TABLE 1
Parameters used for classification of malnutrition

Parameter	Level of Malnutrition			Positive Responses
	mild	moderate	severe	
Serum albumin, gm/dL	<3.5	<3.0	<2.1	
Serum transferrin, mg/dL	<175	<150	<100	
Total lymphocyte count, mm^3	<2,000	<1,200	<800	
Delayed hypersensitivity				
Normal				2 or more
Relatively anergic				1 or more
Anergic				none

distress; and were without large decubitus ulcers.

Assessment of Nutritional and Metabolic Status

An automated metabolic profile was used (1). Anthropometric evaluation included body weight, height, wrist circumference, and triceps skinfold. Triceps skinfold was taken as the mean of three readings at the back of the nondominant arm at a point midway between the acromial and olecranon processes. The percentage of ideal body weight and triceps skinfold was calculated from adult standards (20, 33). Measurements of serum albumin and serum transferrin (derived from total iron-binding capacity) were used as an indication of visceral protein status. Total lymphocyte count and delayed cutaneous hypersensitivity were used to assess immunocompetency. Twenty-four-hour urine was used to determine creatinine excretion and urinary creatinine excretion per kilogram and as an index of muscle mass. The prognostic nutritional index (PNI; in percent) was derived as (28)

$$PNI = 158 - 16.6A - 0.78TSF - 0.20TR - 5.8D$$

where A is serum albumin, TSF is triceps skinfold, TR is serum transferrin, and D is delayed cutaneous hypersensitivity.

Malnutrition was classified as mild, moderate, or severe based on serum albumin and transferrin levels, total lymphocyte count, and delayed hypersensitivity (Table 1). Indirect calorimetric techniques using the metabolic measurement cart (30) were used to determine resting energy expenditure. Measurements were done more than 2 hours after a morning or afternoon meal using a nonrebreathing valve mouthpiece and nose clip. The abbreviated Weir formula (36) was used to calculate the resting energy expenditure from a mean of four or five 1-minute measurements obtained after steady-state conditions. The predicted resting energy expenditure (PREE) was calculated using the formula of Harris and Benedict (19) and used to convert the measured resting energy expenditure (MREE) to percent PREE. The metabolic status of the patients was classified as hypometabolic (MREE <90 percent PREE), normometabolic (MREE >90 percent <110 percent PREE) or hypermetabolic (MREE >110 percent PREE) as previously reported (7). Data for MREE and PREE were corrected for kilogram of body weight, metabolic body size ($kg^{0.75}$) according to Kleiber (21); data for body surface area (BSA in m^2) were corrected according to DuBois (13).

A registered dietician determined the total daily caloric and protein intake, and a 24-hour record of diet was maintained.

Statistical analysis. A one-way analysis of variance was used and differences between sample groups were established using the f test. Differences were considered significant at a level of $p<0.05$.

RESULTS

Table 2 summarizes patient characteristics and the results of the nutritional and metabolic status of the 17 patients as a whole and according to metabolic group (based on percent PREE). The mean body weight of the 17 patients (73.9 kg) represents a wide range from 69.1 to 199.6 percent of ideal body weight. Of these 17 patients, 8 weighed over 110 percent and 2 weighed less than 90 percent of ideal body weight. According to metabolic group, five (83.3 percent) hypometabolic, three (60 percent) normometabolic and no hypermetabolic patients had a body weight greater than 110 percent of ideal (Fig. 1). Both patients with body weight less than 90 percent of ideal were hypermetabolic.

Triceps skinfold measurements ranged from 5

TABLE 2
Nutritional status of total patient population and each metabolic group*

	Metabolic Group†			
	hypo, <90%PREE	normo, 90–110% PREE	hyper, >110% PREE	Total
Patients	$N = 6$	$N = 5$	$N = 6$	$N = 17$
Age	44.2 ± 14.6	41.0 ± 5.2	48.3 ± 15.5	42.8 ± 12.7
Duration of injury, yr	15.6 ± 9.3	19.3 ± 14.3	18.6 ± 12.0	17.8 ± 12.3
Height, cm	178.0 ± 8.9	175.3 ± 4.5	180.3 ± 5.9	177.8 ± 6.0
Weight, kg	81.4 ± 12.7§	77.1 ± 18.5	63.7 ± 8.9	73.9 ± 15.7
Ideal body weight, %	122.6 ± 13.3§	126.5 ± 38.2	94.4 ± 14.1	113.8 ± 27.8
Body surface area, m^2	2.0 ± 0.2	1.9 ± 0.1	1.8 ± 0.1	1.9 ± 0.1
Triceps skin fold, mm	12.8 ± 9.2	13.8 ± 9.6	9.0 ± 3.1	11.5 ± 7.8
Ideal triceps skin fold, %	114.1 ± 84.5	121.2 ± 88.8	77.8 ± 28.2	101.5 ± 72.0
Serum albumin, gm/dL	3.7 ± 0.2	4.0 ± 0.3	4.0 ± 0.5	3.9 ± 0.4
Serum transferrin	189.4 ± 43.5	151.4 ± 32.1	194.0 ± 49.1	179.9 ± 46.5
Total lymphocyte count × $10^3/mm^3$	15.5 ± 2.8§	22.6 ± 13.1	24.0 ± 7.3	20.6 ± 9.3
24-Hr urinary creatinine, mg/kg	17.3 ± 4.4	13.1 ± 5.2	19.8 ± 5.4	16.9 ± 5.8
Delayed hypersensitivity‡				
Normal	1	1	1	3
Relatively anergic	2	3	2	7
anergic	3	1	3	7

All values are mean ± SD. * One percent predicted resting energy expenditure (PREE). † One-way analysis of variance showed no statistically significant differences among metabolic groups. ‡ Normal = 2 or more positive responses; relatively anergic = 1 positive response; anergic = no positive response. § Difference between hypometabolic and hypermetabolic significant at $p<0.05$.

to 13 mm (45 to 272 percent; 50th percentile of ideal TSF). No significant statistical differences were observed among the groups due to this wide range. In general, however, hypermetabolic patients had lower TSF measurement than hypometabolic and normometabolic patients.

Fifteen of seventeen patients (88 percent) had serum albumin levels greater than 3.5 gm/dL and only two patients had mildly decreased levels (3.3 gm/dL and 3.4 gm/dL, respectively). Six patients had serum transferrin values between 150 and 175 mg/dL, and three had moderately decreased levels of serum transferrin (between 100 and 150 mg/dL).

Immunocompetency, as determined by total lymphocyte count, revealed that only six patients (35 percent) had counts over $2000/mm^3$. Nine patients had counts between 1200 and $2000/mm^3$, and only two patients had counts between 800 and $1200/mm^3$ (i.e., moderate immunodeficiency). Similarly, there was an extremely variable response to skin test antigens. Only three patients (17.6 percent) had a normal response; seven patients (41.2 percent) were relatively anergic, whereas the remaining seven were anergic.

Nutritional status assessment using serum albumin (>3.5 gm/dL), serum transferrin (>175 mg/dL), total lymphocyte count ($>2,000/mm^3$) and skin test reactivity revealed that, although mean values (Table 1) were normal for serum albumin, serum transferrin and lymphocyte count, none of the patients were normal for all four parameters. Three patients (18 percent) had three normal values, eight patients (47 percent) had two normal values, and six patients (35 percent) had only one of four normal values. Therefore, eight patients (47 percent) had evidence of mild malnutrition and nine (53 percent) demonstrated a degree of moderate malnutrition. However, as shown in Table 2, there were no significant differences among the metabolic groups with respect to serum albumin, serum transferrin, total lymphocyte count, and delayed cutaneous hypersensitivity.

The mean MREE was similar to PREE, 102.7 percent of PREE, with no statistically significant differences. The mean values were also similar when energy-expenditure data were calculated for per kilogram body weight, body surface area, or metabolic body size. Individual

TABLE 3
Resting energy expenditure in different metabolic groups

Parameter	Metabolic Group* hypo, <90% PREE N = 6	normo, 90–110% PREE N = 5	hyper, >110% PREE N = 6	$p^{\dagger}$
VO_2, ml/min	224.5 ± 30.9	252.0 ± 40.6	256.0 ± 45.9	NS
VO_2, ml/min/kg	2.7 ± 0.2[bc]	3.3 ± 0.4[ac]	4.0 ± 0.3[ab]	< 0.0005
VCO_2, ml/min	187.8 ± 25.3	216.8 ± 41.2	218.2 ± 31.7	NS
VCO_2, ml/min/kg	2.3 ± 0.1[ab]	2.9 ± 0.5[a]	3.5 ± 1.0[b]	0.05
MREE, kcal/day	1,588 ± 209	1,757 ± 283	1,786 ± 255	NS
PREE, kcal/day	1,784 ± 267	1,722 ± 268	1,508 ± 207	NS
Calorie intake, kcal/day	2,116 ± 415‡	2,152 ± 709	2,005 ± 580	NS
MREE, % PREE	87.6 ± 2.6	102.1 ± 5.0	118.3 ± 3.0	NS
MREE, kcal/kg/day	19.3 ± 1.5[bc]	23.2 ± 2.7[ac]	28.2 ± 2.6[ab]	< 0.0005
PREE, kcal/kg/day	22.0 ± 1.4§	22.7 ± 2.3	23.8 ± 2.3§	NS
MREE, kcal/m^2/day	782.6 ± 42.7[ab]	917.8 ± 87.0[a]	986.6 ± 80.0[b]	< 0.005
PREE, kcal/m^2/day	895.1 ± 57.6§	899.6 ± 77.1	839.2 ± 74.6§	NS
MREE, kcal/$kg^{0.75}$/day	57.6 ± 3.8[bc]	68.2 ± 5.9[ac]	79.4 ± 6.7[ab]	< 0.0005
PREE, kcal/$kg^{0.75}$/day	65.8 ± 4.1§	66.8 ± 4.4	67.1 ± 5.7§	NS

* Values are mean ± SD † Results of one-way analysis of variance: a vs. a; b vs. b; c vs. c; $p<0.05$. ‡ Caloric intake vs. MREE. § $p<0.05$; MREE = measured resting energy expenditure; PREE = predicted resting energy expenditure.

patients, however, exhibited significant variations, with MREE ranging from 82 percent to 125 percent of PREE. Only 5 of the 17 patients (29.4 percent) were normometabolic. When the measured metabolic indices (oxygen consumption, carbon dioxide production, and MREE) were normalized to kilogram body weight, body surface area, or metabolic body size, the observed differences in the different metabolic groups became increasingly significant (Table 3).

DISCUSSION

Studies on hospitalized patients (26, 28) show that both malnutrition and obesity contribute to an increased incidence of morbidity and mortality. Inadequate nutrition has been shown to be a significant factor in the development of acquired immune deficiencies (23), defective wound healing (11), decubitus ulcer formation (27), cardiac (35) and respiratory insufficiency (2), and infectious complications (29). Similarly, nutritional status may be related to the morbidity associated with spinal cord–injured patients. A majority of paraplegic patients have been noted to have some degree of nutritional risk and albumin and hematocrit levels at the lower limits of normal (3, 32). Although objective evidence of severe malnutrition was lacking in our study, all patients were abnormal in terms of the four nutritional assessment parameters; they exhibited mild to moderate malnutrition.

No single test used in the present study defined the nutritional status of a patient. Although serum albumin determinations are routinely used in most hospitals as an indicator of nutritional and visceral protein status, their usefulness has been shown to be limited (16). In Peiffer's study (32), although none of the paraplegics examined had serum albumin levels less than 3.0 gm/dL, only a small minority of their patients were normal when a series of parameters were evaluated. Greenway and coworkers (18) also found normal serum albumin levels in paraplegics. All our patients had serum albumin levels greater than 3.0 gm/dL with 88 percent having levels above 3.5 gm/dL. In contrast, serum transferrin levels, a more sensitive indicator, were greater than 175 mg/dL in only eight patients.

Results of the immunological assessment were variable. It has been shown that in many patients, the competence of the immune system is clearly related to the nutritional status (23); no studies have been done on the immunological competence of the spinal cord–injured patient. It has also been found that paraplegics and quadri-

plegics have a fivefold increase in healing complications of wounds below the level of injury (5). Systemic factors such as low serum albumin, anemia, and hypoxia did not appear to be causative agents, as healing complications occurred only below the level of spinal injury, and no patients were described as grossly malnourished. Similarly, none of our patients displayed evidence of gross malnutrition, although all were mildly to moderately malnourished and a large majority (82 percent) were immunodeficient. Therefore, immunocompetence may play a role in spinal cord-injured-patient morbidity. It remains to be determined, however, whether paralysis has an impact on the level of immunocompetence as well as the patient's nutritional status.

It is readily apparent that a single test is of minimal value in nutritional assessment. The PNI advocated by Muller and coworkers (28) has been shown to have a higher "predictive value" than individual measurements of serum albumin and transferrin levels, delayed cutaneous hypersensitivity, and anthropometry (12). Ten of the patients (58.8 percent) in our study had a PNI score greater than 40 (Detsky's high-low risk cutoff), which confirms the prevalence of malnutrition among spinal cord–injured patients.

Twenty-four-hour urinary creatinine excretion is a simple index of muscle mass, and although it was reduced in our patients, there were no significant differences among the three metabolic groups. Cordus and others (10) have also shown that compared with subjects without disabilities, urinary creatinine excretion is decreased in subjects with paraplegia. Although muscular paralysis does decrease muscle mass, it is not apparent whether muscle mass is influenced by nutritional status.

The present study indicates that individuals with paraplegia have a tendency toward increased body weight. Obesity and loss of weight are the result of an imbalance between caloric intake and energy output. Contrary to general belief, paraplegics do not have uniformly reduced energy needs; they exhibit significant differences in body weight. Although caloric intake of the three metabolic groups was identical, none of the patients in the hypermetabolic group was overweight since caloric intake was identical to measured energy expenditure. In contrast, the

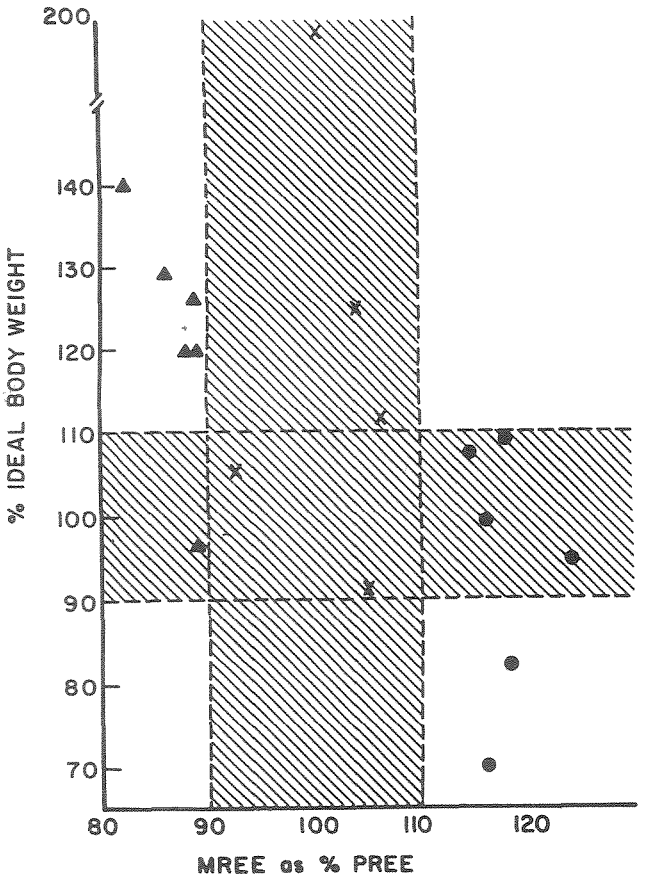

FIGURE 1
Relationship between weight and metabolic status in individuals with paraplegia. Note none of the hypermetabolic patients are overweight compared with normometabolic and hypometabolic patients; ($p<0.05$ by chi-square). MREE = measured resting energy expenditure; PREE = predicted resting energy expenditure. ▲ = hypometabolic; X = normometabolic; ● = hypermetabolic.

hypometabolic patients were consuming significantly more calories than were expended ($p<0.05$) and were therefore predisposed to obesity. After the initial weight loss that occurs in the first 3 to 4 months after spinal cord injury, there is very little change in the weight of paraplegics; weight changes seen in individual patients are correlated to changes in fat content (10). Similarly, if caloric intake is restricted in the hypermetabolic patients, these patients will be at high risk of becoming malnourished. Obesity further compromises the management of spinal cord–injured patients and causes an increased frequency of clinical diabetes, hypertension, arteriosclerosis, osteoarthritis, gall bladder disease, reduced left ventricular contractility, left ventricular dysfunction, ventilation/perfusion in-

equalities, postoperative complications, and mortality (25, 34). Excessive weight additionally discourages proper turning of the patient and an inability to fit them into armchairs.

The PREE of patients in our study was not significantly different between the various metabolic groups. In individuals without disabilities, the Harris-Benedict equation has been shown to precisely predict energy expenditure (6, 24). Our study is in agreement with the work of others (14, 15, 22), indicating that the Harris-Benedict equation cannot be accurately applied to individuals with abnormal body composition or in presence of pathology and cannot be used for estimating the caloric needs of individuals with paraplegia. Clinical studies in surgical patients have documented that when nutritional replacements are based on indirect calorimetry, positive nitrogen balance is easily achieved, weight loss is prevented, and patients generally do well (4, 17). Problems associated with overfeeding and underfeeding of spinal cord–injured patients can be prevented by a more precise definition of their caloric requirements.

CONCLUSION

Nutritional and metabolic assessment is primarily important both in identifying patients with clinical and subclinical malnutrition who are prone to nutrition-associated complications and in providing guidelines for nutritional intervention. Further long-term studies are needed in spinal cord–injured patients to determine which of the currently available parameters of nutritional and immunological assessment are useful in predicting clinical outcome. Additionally, standards established for subjects without disabilities may not apply to the spinal cord–injured patient. Optimal cutoff values for each of the nutritional assessment indices need to be determined; until then, results should be interpreted with caution.

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