Short-Term Changes in Energy Metabolism After 1 Month of a Regular Oral Diet in Severely Malnourished Cirrhotic Patients

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Malnutrition in patients with liver cirrhosis is currently associated with abnormal fuel metabolism. The aim of this study was to evaluate changes in energy production and substrate oxidation rates in a group of 26 nonanorectic severely malnourished cirrhotic patients in stable clinical condition after 1 month of an oral diet. Child-Pugh score, nutritional status, energy expenditure, rates of nutrient oxidation, and plasma levels of intermediary metabolites in the postabsorptive phase were assessed before and after 1 month of oral nutrition. Upon entry onto the study, caloric and protein intakes were 40.1 ± 2.0 kcal/kg and 1.44 ± 0.8 g/kg, respectively. The Child-Pugh score did not change during the study, whereas nutritional status improved as shown by increased muscular midarm circumference, ([MMAC] $P < .02$), height-creatinine index ($P < .05$), triceps skinfold thickness ([TST] $P < .01$), and fat mass ($P < .001$). Inflammatory state improved during the study, as shown by the decrease of C-reactive protein ([CRP] $P < .01$) and orosomucoid ($P < .001$). The ratio of caloric intake to resting energy expenditure (REE) increased (1.53 ± 0.06 *v* 1.66 ± 0.07, $P < .05$), as well as the rates of glucose oxidation ([Gox] 73.6 ± 9.9 *v* 128.1 ± 10.3 mg/min, $P < .001$) and urine nitrogen excretion (6.69 ± 0.47 *v* 7.96 ± 0.48 g/d, $P < .02$). On the other hand, the rate of lipid oxidation (Lox) decreased (67.3 ± 3.9 *v* 47.3 ± 4.9 mg/min, $P < .001$) and was correlated with the decrease of free fatty acid (FFA) levels ($P < .05$). These results show that a regular oral diet may improve nutritional status in malnourished cirrhotic patients, provided caloric intake is well adapted to energy requirements. Improvement of nutritional status is related to the change in energy metabolism, showing a normalization of carbohydrate storage while mobilization of fat stores is reduced.

THE BENEFITS of nutritional therapy for patients with alcoholic liver diseases remain debatable. Many factors may contribute to malnutrition, which is a common finding in these patients and may lead to a poor prognosis.[1] Poor dietary intake may occur before, during, and after hospitalization. It must be emphasized that poor dietary intake is an extremely important factor in both hospitalized and ambulatory patients with liver diseases.[1] Other factors such as impaired nutrient digestion and absorption, increased energy requirements, accelerated protein breakdown, and inefficient protein synthesis are potential causes of malnutrition in liver diseases.[2] Several studies have shown that enteral nutrition may improve nutritional status in cirrhotic patients; however, it is unclear whether oral caloric intake may be sufficient to induce a nutritional benefit. Furthermore, the recommended caloric intake is not precisely known in these patients, since energy requirements may vary somewhat extensively—resting energy expenditure (REE) may differ from predicted values in 70% of the patients.[3] Therefore, it would appear that there is a need for precise evaluation of the optimal ratio of caloric intake to energy expenditure to promote a nutritional gain.

On the other hand, liver cirrhosis is associated with abnormal fuel metabolism: oxidative metabolism is unbalanced in patients with poor nutritional status, as shown by an increased rate of lipid oxidation (Lox) and a decreased rate of glucose oxidation (Gox).[4-6] This metabolic pattern characterizes accelerated starvation with early recruitment of alternative fuels and may contribute to malnutrition. One expected result of nutritional therapy would be to restore normal fuel metabolism, showing an improvement of storage of nutrients. However, it is not known whether refeeding may change oxidative metabolism and to what extent this change is related to an improvement of liver function.

This study was undertaken to evaluate short-term changes in energy expenditure, substrate oxidation rate, and nutritional status after 1 month of a regular oral diet in a group of 26 malnourished nonanorectic cirrhotic patients. It was a conventional diet supplying larger amounts of calories than the usual dietary recommendation. Finally, we searched for a relationship between changes in the metabolic pattern and in clinical condition, as well as the degree of liver failure.

SUBJECTS AND METHODS

Patients

Twenty-six patients were entered onto the study, and all patients provided informed consent. There were 21 men and five women with a mean age of 51.3 ± 2.0 years (range, 31 to 77). The patients were enrolled a few days after admission to a rehabilitative unit for liver diseases, provided they were nonanorectic. Before admission to the unit, they had been hospitalized for severe complications of the disease for 4 to 5 weeks. The principal cause of hospitalization was ascites in 20 cases. Among these 20 patients, 13 had experienced other complications of the disease such as gastrointestinal bleeding, hepatic encephalopathy, spontaneous bacterial peritonitis, or other sepsis. The remaining six patients had been hospitalized for jaundice, sepsis, and hepatic encephalopathy. Liver cirrhosis was proven histologically in 13 patients, alcoholic hepatitis in six of them. When patients were enrolled onto the study, they were in stable clinical condition: they were not febrile or septic and were free of hepatic encephalopathy. Alcoholic intake had ceased for several weeks before hospitalization. None of these patients suffered from any other chronic disease.

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Medication

During the study, all patients received vitamins (vitamins B_1 and B_6 and folic acid). Twelve patients received diuretics (furosemide and/or spironolactone), six received propranolol, five received lactulose, two received ranitidine, and five ascitic patients received norfloxacine. Four patients with alcoholic hepatitis had received corticosteroids before admission onto the study. In all four cases, corticosteroids were stopped, on average, 1 week before admission.

Clinical Assessment

The degree of liver failure was assessed by the Child-Pugh score.[7] Nine patients were ascitic on the day of admission onto the study, and paracentesis was performed once per week on seven of them. Each paracentesis removed, on average, 4 to 6 L ascitic fluid and was followed by an intravenous infusion of one or two bottles of human albumin (Albumine humaine 20%, 100 mL; Biotransfusion, Paris, France). During the study, one patient had a bout of hepatic encephalopathy that rapidly improved. The diet was not modified over the following days.

Dietary Intake

Before admission to the unit, the patients' diet was not controlled. They received the usual diet given in French hospitals, supplying an average of 30 to 35 kcal/kg body weight. After admission onto the study, caloric intake was monitored by a dietician, who questioned the patients about food preferences and encouraged them to eat. The diet was conventional and was not enriched by any specific substrate. The hospital diet was unrestricted except for Na and fluid in patients with ascites. Intakes of energy, nitrogen, and essential nutrients were calculated from five consecutive 24-hour dietary recalls. Patients recorded food intake with help from the dietician. The composition of the diet was determined from tables.[8]

Fat and protein digestibility was calculated using the following equation: digestibility = intake − (fecal output × 100)/intake.

Assessment of Nutritional Status

Muscular midarm circumference (MMAC) and skinfold thicknesses at four areas, tricipital (TST), bicipital, subscapular, and suprailiac, were measured in all patients by the same operator throughout the study. Height-creatinine index was derived from patients' height and 24-hour urinary creatinine and expressed as a percentage of normal.[9] Fat mass was calculated according to predictive equations.[10]

Assessment of Metabolic Pattern

REE was measured by indirect calorimetry after an overnight fast and a half-hour in recumbency. A ventilated hood was placed over the head and shoulders of the patients. To avoid air loss, an airtight jacket was attached to the hood and fastened around the upper arms and chest of the patients. The hood was continuously ventilated by air pumped at a flow rate of 45 to 50 L/min for a half-hour. Flow rate was measured at the inlet of the hood with a flow meter. O_2 and CO_2 concentrations were measured at the outlet, respectively, using O_2 paramagnetic and CO_2 infrared analyzers (oxygen 500D and FM2 CO_2 analyzers; Morgan, Rainham-Gillingham, UK). Outflowing air was continuously analyzed during the test. O_2 consumption ($\dot{V}O_2$) and CO_2 production ($\dot{V}CO_2$) were calculated at standard temperature and pressure–dry (0°C and 760 mm Hg) from the differences in O_2 and CO_2 levels between fresh incoming and outflowing air. Analyzers were calibrated before each measurement with a gas mixture containing 1% CO_2 and 20% O_2. Respiratory quotient (RQ) and nonprotein RQ were calculated (RQ = $\dot{V}CO_2/\dot{V}O_2$ and nonprotein RQ = nonprotein $\dot{V}O_2$/nonprotein $\dot{V}CO_2$). Known amounts of absolute alcohol were supplied via a syringe pump and burned inside the hood to calibrate the system. Measurement of $\dot{V}O_2$ was verified from the weight of burned alcohol and the stoichiometric equation of alcoholic oxidation. $\dot{V}O_2$ observed was 3.8% less than the theoretical value. $\dot{V}O_2$ was measured with a coefficient of variation of 2.7%. The observed RQ was 0.66 and was measured with a coefficient of variation of 2.3%. Moreover, reproducibility of the measurements was tested in five patients: REE was measured over 3 consecutive days. $\dot{V}O_2$ and RQ varied with a coefficient of variation, respectively, of 4.5% and 2.9%. REE and rates of Gox and Lox were calculated according to Ferrannini's equations from $\dot{V}O_2$, $\dot{V}CO_2$, and the rate of urine nitrogen excretion.[11] Measured REE was compared with predicted REE according to the equations reported by Harris-Benedict[12] and Owen et al.[13-14] Predicted REE according to the Harris-Benedict equation was as follows: REE (kcal/d) = (66 + 5H + 13.7W) − 6.8A for men and (655 + 1.9H + 9.6W) − 4.7A for women. Predicted REE according to the equation of Owen et al was as follows: REE (kcal/d) = 879 + 10.2W for men and 795 + 7.2W for women. In each case, H is height in centimeters, W is weight in kilograms, and A is age in years.

Biological Parameters

Plasma glucose, lactate, free fatty acids (FFA), glycerol, ketone bodies, creatinine, bilirubin, albumin, transthyretin, C-reactive protein (CRP), and orosomucoid levels were measured on a Monarch analyzer (Instrumentation Laboratory, Lexington, MA) using available commercial kits. Total urine was collected over 3 days, and urine creatinine level was measured on a Monarch analyzer and urine nitrogen was assayed by the Kjeldahl method. Stools were collected from all patients over a period of 3 days; these were weighed, and then nitrogen and fat were assayed, respectively, by the Kjeldahl and Van De Kammer methods.[15]

Study Design

REE was measured at day 0, and total urine and stools were collected over a 3-day period (days −1 to +1). Caloric intake was calculated over 5 days (days −1 to +3). At day 0, anthropometric measurements were performed, the Child-Pugh score was assessed, and a blood sample was collected to assay biological parameters. One month later (day 30), the same procedure was performed; however, stools were not collected. Patients stayed in the metabolic ward and their diet was monitored throughout the study.

Statistical Analysis

Results are expressed as the mean ± SEM. Student's t test for paired data was used to compare two means between days 0 and 30. Regressions were calculated according to the least-square analysis. The significance threshold was retained for P less than .05.

RESULTS

Characteristics of Patients

Usual biological parameters such as total bilirubin and prothrombin time did not change throughout the study. Serum albumin increased significantly in ascitic patients who underwent paracentesis and were infused with albumin (25.2 ± 1.4 *v* 27.3 ± 1.4 g/L, $P < .05$), but it did not change significantly in patients with no ascites (27.4 ± 0.8 *v* 28.6 ± 1.0 g/L). Transthyretin, which has a shorter half-life than albumin, did not change throughout the study. Regard-

Table 1. Characteristics of Patients at the Start and End of the Study

	Bilirubin (μmol/L)	Albumin (g/L)†	Transthyretin (g/L)	Orosomucoid (g/L)†	CRP (mg/L)*	Prothrombin Time (%)	Creatinine (μmol/L)	Child-Pugh Score
Day 0								
Mean	41	26.7	0.09	0.57	10	46	70	9.1
SEM	7	0.7	0.01	0.07	2	3	4	0.5
Day 30								
Mean	36	28.2	0.09	0.44	6	48	69	8.6
SEM	6	0.8	0.01	0.05	1	3	4	0.5
Normal value	<20	35-47	0.20-0.37	0.40-1.00	<6	80-100	55-105	

NOTE. Ascites was present in 9 of 26 patients at day 0 and in 8 of 26 at day 30. Comparison of data was performed using a *t* test.
*$P < .01$.
†$P < .001$.

ing the inflammatory state, CRP (normal value, <6 mg/L) and orosomucoid (normal value, 0.40 to 1.00 g/L) decreased significantly at the end of the study. Mean plasma creatinine was normal and did not change during the study. At the start of the study, six patients were in Child-Pugh group A, nine in group B, and 11 in group C. The score tended to decrease by the end of the study, but insignificantly. It improved in 12 patients, remained unchanged in 10, and worsened in four (Table 1).

Dietary Intake

Caloric and protein intakes improved during the course of study. However, both increases were no longer significant when related to body weight. The percentage of calories derived from carbohydrates, lipids, and proteins remained unchanged throughout the study (Table 2).

Fat and Nitrogen Content in Stools

Fat and nitrogen contents were 5.1 ± 0.6 and 1.9 ± 2.3 g/d. In seven patients, fat content was greater than 6 g/d (9.2 ± 0.9) and nitrogen content was enhanced (2.6 ± 0.2 g/d). Fat and protein digestibilities were, respectively, 73.2% ± 4.4% and 70.3% ± 4.4%.

Anthropometric Data

MMAC was less than the 5th percentile of a reference population in all patients and correlated positively with urine creatinine ($r = .39$, $P < .05$). The height-creatinine index was less than 80% of the normal value in 23 patients. TST was less than the 5th percentile in 12 patients, the 10th percentile in one patient, and the 25th percentile in eight patients, whereas it was normal in the remaining patients.[16] Urine creatinine and the height-creatinine index increased significantly by the end of the study. MMAC, TST, and particularly fat mass improved, whereas body weight did not change significantly. However, nonascitic patients showed a significant increase in body weight (60.8 ± 1.4 *v* 63.6 ± 1.3 kg, $P < .001$). Patients with ascites showed a tendency to lose weight, but weight loss did not reach the significance threshold (55.4 ± 2.4 *v* 53.3 ± 2.8 kg) (Table 3).

Energy Expenditure and Substrate Oxidation

Measured REE (1,533 ± 53 kcal/d) was greater than predicted REE according to the equations of Harris-Benedict and Owen et al (respectively, 1,360 ± 29 kcal/d, $P < .001$, and 1,430 ± 26, $P < .05$). Measured REE did not change throughout the study. It correlated positively with body weight ($r = .50$ and $P < .01$ initially and $r = .64$ and $P < .001$ at day 30) and with caloric intake ($r = .41$ and $P < .05$ at day 0). The ratio of caloric intake to REE improved to such an extent that by the end of the study our patients consumed 1.66 ± 0.07 times their expenditure.

The similar increase in RQ and nonprotein RQ was highly significant and resulted from a clear decrease in Lox and an increase in Gox. Accordingly, the amount of REE derived from carbohydrates increased and that derived from lipids decreased. The rate of urinary nitrogen excretion increased significantly, showing an enhanced rate of protein oxidation (Table 4).

Table 2. Protein-Calorie Consumption and Composition of the Meals at Entry Onto the Study and 1 Month Later

	Calorie Intake		Protein Intake		Calorie Source (%)		
	kcal/d*	kcal/kg/d	g/d*	g/kg/d	CHO	Lipid	Protein
Day 0							
Mean	2,314	40.1	82.8	1.44	53.9	31.5	14.6
SEM	95	2.0	3.4	0.08	0.9	0.7	0.4
Day 30							
Mean	2,526	42.8	90.2	1.54	55.0	30.5	14.5
SEM	94	1.9	3.0	0.07	0.9	0.8	0.4

NOTE. Data were compared using a paired *t* test.
Abbreviation: CHO, carbohydrate.
*$P < .01$.

Table 3. Changes in Parameters of Nutritional Status

	Weight (kg)	Urinary Creatinine (mmol/d)*	Height-Creatinine Index(%)*	MMAC (cm)†	TST (mm)‡	Fat Mass (%)§
Day 0						
Mean	58.9	7.65	61.9	21.1	7.3	16.4
SEM	1.6	0.44	3.4	0.4	0.7	1.2
Day 30						
Mean	60.0	8.51	68.7	21.6	8.8	18.6
SEM	1.6	0.39	2.7	0.4	0.8	1.3

NOTE. Data were compared using a *t* test.
*$P < .05$.
†$P < 0.02$.
‡$P < 0.01$.
§$P < .001$.

Table 4. Energy Expenditure and Rates of Nutrient Oxidation at Entry Onto the Study and 1 Month Later

	REE (kcal/d)	CI/REE*	RQ‡	npRQ‡	Gox (mg/min)‡	Lox (mg/min)‡	UN (g/d)†	REE Source (%)		
								CHO‡	Lipid‡	Protein
Day 0										
Mean	1,533	1.53	0.79	0.79	73.6	67.3	6.69	27.2	57.0	15.7
SEM	53	0.06	0.01	0.01	9.9	3.9	0.47	3.5	2.3	1.4
Day 30										
Mean	1,558	1.66	0.85	0.86	128.1	47.3	7.96	48.2	38.3	13.5
SEM	61	0.07	0.01	0.01	10.3	4.9	0.48	3.4	3.0	1.3

NOTE. Data were compared using a paired *t* test.

*$P < .05$.

†$P < .02$.

‡$P < .001$.

Abbreviations: CI, caloric intake; RQ, respiratory quotient; npRQ, nonprotein RQ; UN, urinary nitrogen excretion; CHO, carbohydrate.

Blood Intermediary Metabolites

Glucose values remained unchanged for the duration of the study. Lactate, FFA, glycerol, and ketone bodies showed trends toward decreased values at the end of the study, but the differences were not statistically significant. Differences in FFA levels between days 30 and 0 strongly paralleled those in glycerol ($r = .630$, $P = .001$) and in β-hydroxybutyrate ($r = .728$, $P < .001$) (Table 5). They correlated positively to a lesser extent with changes in Lox ($r = .424$, $P < .05$) but negatively with changes in RQ and nonprotein RQ (respectively, $r = -.413$ and $P < .05$ and $r = -.401$ and $P < .05$) (Fig 1).

DISCUSSION

In this study, we showed that nutritional status might improve in severely malnourished nonanorectic cirrhotic patients with an oral caloric intake of 40 kcal/kg body weight. The diet given to the patients was a conventional one not enriched by any specific component and had a composition similar to that of the diet usually recommended to healthy people, with a proportion of carbohydrate, lipid, and protein, respectively, of 55%, 30%, and 15%. It is noteworthy that the amount of protein was large, averaging 1.4 g/kg body weight at the time of admission onto the study, and was well tolerated by the patients. All patients enrolled onto the study had no chronic encephalopathy, which may have accounted for the lack of side effects with this protein intake. It is well known that a reduced dietary intake is a main factor of malnutrition in these patients; however, our results showed that impaired nutrient absorption was probably involved, since 26% of the patients had an increased rate of nitrogen and fat excretion in their stools. As reported by several others, enteral nutrition has beneficial effects on nutritional status and clinical outcome of cirrhotic patients.[17-20] Improvement of nutritional status is probably dependent on the caloric load of the enteral diet. Cabre et al[19] have shown that MMAC and TST did not change within 1 month with a caloric intake of 2,115 kcal/d. Therefore, it seems important to ensure that the caloric intake is adapted to energy requirements whether patients are fed by enteral tube or by an oral diet when nonanorectic. Many studies have focused on energy expenditure in liver cirrhosis, showing that REE in cirrhotic patients is in the same range as in healthy control subjects.[3-6,21] However, REE appears to increase in cirrhotic patients when related to lean body mass.[22,23] In a study including a large number of cirrhotic patients, Müller et al[3] have shown that REE differed from predicted values in most patients whether they appeared hypermetabolic or hypometabolic. In our study, the equations of Harris-Benedict and Owen et al equations underestimated REE, respectively, by 11% and 6%. The level of REE is affected by several factors, including caloric intake. Overfeeding induces an increase in REE, whereas malnutrition has an opposite effect.[24] The high caloric intake tended to increase REE in our patients, as shown by the significant correlation between caloric intake and REE. The ratio of caloric intake to REE initially averaged 1.5 and increased to 1.7 by the end of the study. Since muscular activity was reduced in this hospitalized patient population, this ratio shows that caloric intake was probably adapted to energy requirements. All anthropometric parameters increased during the study. The change in nutritional status is in line with that in acute-phase reactants, showing improvement of the inflammatory state. The increase in MMAC was slight but significant, showing an increase in lean body mass concordant with the increase in urine creatinine. On the other hand, fat mass increased to a greater extent than lean body mass. Malnutrition does not affect the different body compartments to the same extent in cirrhotic patients: lean body mass is severely depleted, whereas fat mass may be preserved.[25] Fat preservation may be related to hyperinsulinemia, which is a common feature in liver cirrhosis. Hyperinsulinemia promotes fat storage, and we have shown that after meal intake, cirrhotic patients have an increased

Table 5. Blood Intermediary Metabolite Concentrations (mmol/L) at Entry Onto the Study and 1 Month Later

	Glucose	Lactate	FFA	Glycerol	BHB	AC	Total Ketone Bodies
Day 0							
Mean	5.29	1.69	0.55	0.085	0.056	0.028	0.087
SEM	0.28	0.12	0.06	0.007	0.007	0.005	0.010
Day 30							
Mean	5.23	1.58	0.48	0.079	0.051	0.023	0.076
SEM	0.23	0.09	0.06	0.007	0.008	0.003	0.010

Abbreviations: BHB, β-hydroxybutyrate; AC, acetoacetate.

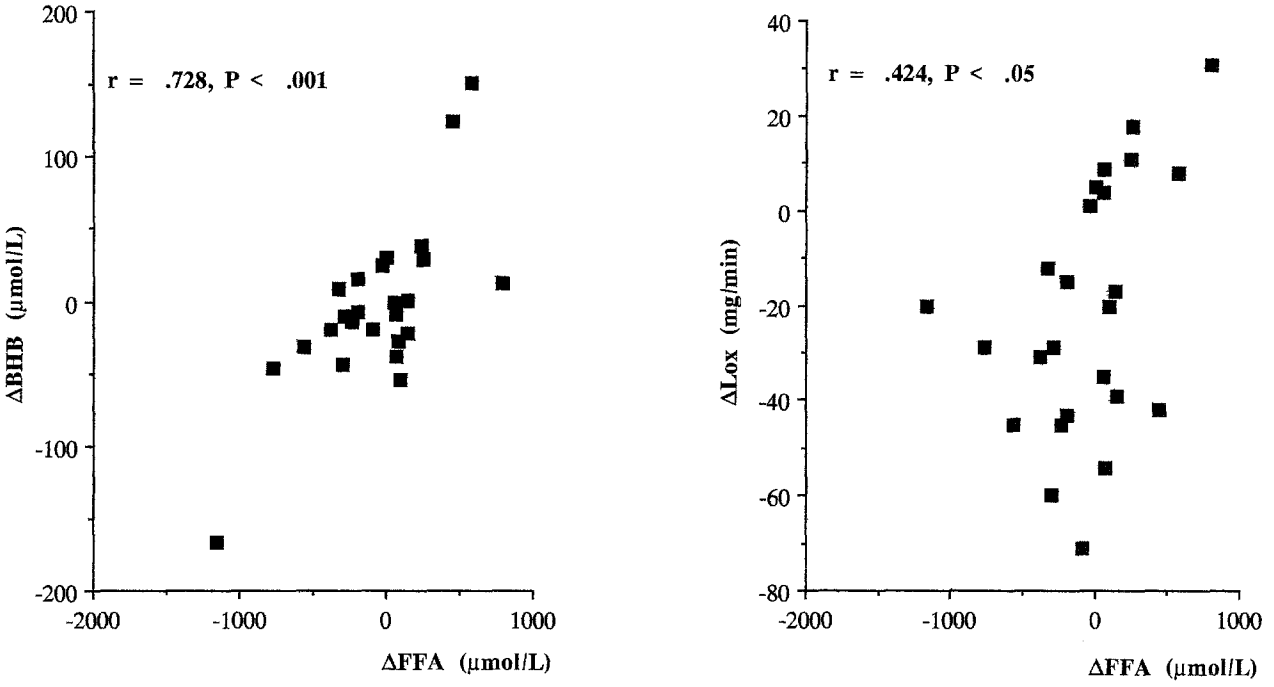

Fig 1. Correlation of changes in FFA between days 30 and 0 with changes in β-hydroxybutyrate (BHB) and in rate of Lox.

rate of lipid storage, whereas glucose storage is reduced, as compared with normal subjects.[26] This greater ability to store fat may account for the large increase in fat mass observed after 1 month of oral diet.

This study was performed to investigate whether metabolic changes might occur in cirrhotic patients who had received an oral regular diet for 1 month. Much information is available in the literature on the alteration of fuel homeostasis in the postabsorptive state in liver cirrhosis.[3-6] After an overnight fast, cirrhotic patients show a metabolic profile comparable to that of normal subjects who have undergone 72 hours of starvation. Lipolysis and Lox are enhanced, with the latter supplying, on average, 70% of REE, whereas the rate of Gox is reduced. The rate of urinary nitrogen excretion is usually decreased, supporting evidence of a decreased rate in protein oxidation. However, there are conflicting results from studies using stable isotopes of amino acids, and it is not clear whether protein breakdown and oxidation are diminished in liver cirrhosis.[27] Taken as a whole, these metabolic abnormalities indicate a catabolic state and are related to several factors, such as depleted hepatic glycogen stores, insulin resistance, defective glucose production, and to some extent an altered nutritional status; patients with a severely damaged nutritional status reportedly have the most obvious abnormalities of fuel homeostasis.[28,29] However, the influence of nutritional support on such an unbalanced metabolic pattern has not yet been investigated. RQ, which reflects the proportion of carbohydrate to fat oxidation, varies with diet composition. Upon entry onto the study, our patients showed a fasting RQ level close to that of healthy subjects who consume a balanced diet that meets energy requirements for the maintenance of body weight.[30] The high caloric intake, which provided a large carbohydrate load, in our patients may have contributed to the higher RQ value than that previously reported in malnourished cirrhotics. The most striking result from this study is a highly significant shift of fuel metabolism induced by refeeding toward an increase in Gox and protein oxidation on one hand and a decrease in Lox on the other hand, helping fat storage, as shown by increased fat mass. Meanwhile, the increased protein oxidation observed is in line with previous studies showing that lipid-derived substrates reduce amino acid oxidation in vivo.[31,32] Therefore, our data give evidence of changes in oxidative metabolism that result from dietary support, and are in keeping with an improvement in nutritional status. These changes did not seem to be associated with an improvement of liver function as evaluated by the Child-Pugh score.

Among alterations in plasma intermediary metabolites widely described in liver cirrhosis, there are increased fasting levels of FFA, glycerol, ketone bodies, and lactate. The high level of plasma FFA and glycerol is caused by enhanced lipolysis.[4-6,33] Tissue uptake of FFA is preserved and even enhanced, resulting in an increase in FFA oxidation, which is consistent with a moderate hyperketonemia, as well as in nonoxidative FFA disposal as reesterification.[34] In this study, a trend toward a decrease in these different substrates could be observed. Moreover, changes in FFA levels paralleled those in glycerol, β-hydroxybutyrate, and Lox, and correlated inversely with changes in RQ. These data suggest that in relation to the large amount of carbohydrate calories supplied by the hospital diet, glucose was preferentially oxidized while fat was spared. Consequently, the mobilization and availability of endogenous fat-derived substrates were decreased.

In conclusion, this study shows that a high caloric intake providing an average of 40 kcal/kg may achieve an improvement in nutritional status in severely malnourished cirrhotic patients. This nutritional support may be provided orally if the patients are not anorectic. The beneficial effect of such a caloric intake lies in the fact that it is well adapted to energy requirements, with a ratio of caloric intake to REE ≥ 1.5. Nutritional support induces changes in oxidative metabolism as shown by increased rates of carbohydrate and protein oxidation and a decreased rate of Lox. This metabolic pattern is a likely indicator of an anabolic state. Assessment of fuel metabolism provides useful information in the follow-up evaluation of patients when nutritional support is given.

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