

# Dietary treatment of severe malnutrition in adults<sup>1,2</sup>

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**ABSTRACT** The aim of this study was to compare the effects of two diets, differing primarily in protein content, on the nutritional rehabilitation of severely malnourished adults. The study took place in the Concern Worldwide Adult Therapeutic Feeding Centre in Baidoa, the town at the epicenter of the 1992 famine in Somalia. The response to treatment in 573 patients admitted to the center between November 1992 and March 1993 was studied. Mortality, appetite, rates of edema loss, and weight gain in 2 groups of patients receiving either a higher-protein (16.4% of energy from protein) or lower-protein (8.5% of energy from protein) diet were compared. Among edematous patients, the use of the lower-protein diet during the initial phase of treatment was associated with a threefold decrease in mortality ( $P < 0.05$ ) and accelerated resolution of edema ( $P < 0.05$ ). Among marasmic patients, no differences in mortality or rate of weight gain were observed. The large reduction in mortality associated with the use of the lower-protein diet in edematous patients appeared to be due to the lower amount of dietary protein. However, differences in the 2 diets other than or in addition to the protein content may have contributed. Notwithstanding, the data obtained suggest strongly that severely malnourished adults, particularly those with edema, recover more successfully with a diet of lower protein content than usually recommended. The lower-protein diet used in this study was much cheaper and more easily obtained than the conventional higher-protein diets in Baidoa. *Am J Clin Nutr* 1998;68:193–9.

**KEY WORDS** Malnutrition, refeeding, protein-energy malnutrition, PEM, edematous malnutrition, dietary protein, Somalia, marasmus, famine

## INTRODUCTION

Severely malnourished adults are encountered frequently during emergency famine relief programs (1). Because adult energy requirements are proportionately less than those of children, the peak incidence of severe malnutrition and death in adults generally occurs later than in children (2). Often, by the time an emergency relief operation is up and running, many of the children have already died (3) and malnourished adults constitute a large proportion of the nutritional problem (4, 5). The difficulties of feeding severely malnourished adults, especially those with edematous malnutrition, have long been recognized (6). During the first half of this century, these problems received considerable

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attention from the scientific community and, generally, diets containing relatively high amounts of protein were recommended (7–12). In the early 1950s, scientific attention shifted toward malnourished children, in whom the quantity and quality of dietary protein required for successful rehabilitation became the focus of much research (13, 14). In particular, the importance of liver pathology in kwashiorkor was recognized (15). Recently, it was shown in children with kwashiorkor that mortality was minimal when diets containing only maintenance levels of protein ( $< 1 \text{ g} \cdot \text{kg}^{-1} \cdot \text{d}^{-1}$ ) were given (16, 17). Up to the time of the Somali famine in 1992, however, these diets had been tested only in specialized hospital units and no field research had been performed during an emergency relief program.

The difficult circumstances that exist during famines generally prevent the execution of classical, scientifically rigorous research. This has resulted in an absence of field research since the late 1940s when the concentration camps in Europe and the Far East were liberated. In the absence of field research, results from studies performed in less severely malnourished subjects, usually in hospital settings, have been extrapolated to the very different circumstances found during war and famine. Thus, it was shown that adult subjects with body mass indexes (BMIs, in  $\text{kg}/\text{m}^2$ ) of 17–18 respond well to diets with protein-to-energy ratios (P:Es)  $> 19\%$  (18). As a result, such high-protein diets have continued to be recommended for the management of severe adult malnutrition. Such diets were used in all Concern Worldwide therapeutic feeding centers (TFCs) during the Somali relief operation in 1992–1993. However, the adults admitted to these TFCs generally had BMIs of 10–13, far less than those that had been studied. They also often had edema (19). It was observed that many of these patients, particularly those with edema, were refusing the high-protein diets. Thus, the hypothesis was advanced that the high-protein diets were deleterious during the initial phase of rehabilitation. Therefore, the aim of this study was to compare the immediate and short-term effects of a lower-protein diet with

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those of the conventional higher-protein diet during nutritional rehabilitation of severely malnourished adults admitted to a TFC during the emergency relief program in Baidoa.

## SUBJECTS AND METHODS

All adults admitted to the Concern Worldwide Adult Therapeutic Feeding Centre in Baidoa between 25 October 1992 and 30 March 1993 were studied. The center was established according to internationally accepted guidelines and operated according to the principles of best clinical practice given the prevailing circumstances. The setting and the conditions, that is, an emergency feeding center in a war zone, did not allow us to carry out a formally designed study and the number of observations made and the degree of control possible were constrained by the circumstances. The criteria for admission varied depending on the space available in the center and the existence of other medical facilities in Baidoa. In general, only patients with a BMI < 13.5, those assessed as very severely malnourished by the clinician (SC), those too ill to be weighed and measured on admission, or those with edematous malnutrition were admitted to the center. Less severely malnourished persons were referred to supplementary kitchens. During October and November 1992, there were no alternative medical centers in Baidoa and 16 patients with BMIs > 13.5 and medical rather than nutritional problems were admitted. This practice stopped in mid-December 1992 when a medical inpatient unit was opened in Baidoa.

Five hundred seventy-three patients were admitted to the center. They were aged between 15 and 80 y (median: 30 y) and 46% were male. The age distributions of male and female patients were similar. On admission, patients were registered, were weighed, and had their height measured by trained local assistants, supervised by a specially trained nurse or the clinician (SC). A rapid clinical screen, assessing degree of pitting edema, ascites, hydration, dysentery, diarrhea, anemia, signs of chest infection, and ability to stand was performed by either the clinician (SC) or the nurse. The weight or clinical condition or both of each patient were monitored daily during rehabilitation and outcome was recorded. Lack of trained staff and an extremely high patient load prevented detailed recording of individual food intake. Pitting edema was graded by using the classification of Beattie et al (12) as grade 0 (absent), 1 (minimal edema on the foot or ankle, demonstrable but not visible), 2 (visible on foot or ankle), 3 (demonstrable up to knee), 4 (demonstrable up to inguinal ligament), and 5 (anasarca). Pitting edema that was recorded as present but unquantified was assumed to be moderate (grade 3); unquantified edema was recorded in 4.3% of patients. Periorbital edema was separately graded as 0 (absent), 1 (mild), 2 (moderate), and 3 (severe), with present but unquantified edema assumed to be grade 2. Ascites was similarly graded. Edematous malnutrition was defined as either pitting edema greater than grade 2, periorbital edema greater than grade 1, or any ascites. Missing data values were coded as absent and excluded from further analysis. Data were analyzed by using EPI INFO (20), LOGISTIC (21), and Microsoft EXCEL (22).

### The center, the staff, and the equipment

The center consisted of nine stick shelters with roofs made of plastic sheeting and mud floors. Separate shelters were designated for new admissions, those with edematous malnutrition, those with dysentery, and those suspected of having pulmonary

tuberculosis. The center was staffed by 1–3 expatriate staff members, 3 local nursing staff members, and ≈30 other helpers and cooks. These staff members distributed the food and oral rehydration solution (ORS), encouraged patients to eat and drink, and helped feed those who were unable to feed themselves. High levels of supervision, with one staff member for less than 9 patients, were maintained during the daytime, particularly during the first few days after admission. At night, frequent gun battles and attempts at hostage-taking meant that supervision by expatriate workers was impossible.

A set of standard spring scales and a locally constructed height board were used to weigh and measure patients. The scales were calibrated in London and Baidoa with known weights. The results were similar at both sites. In the useful range of 20–50 kg, there was a linear increase in error ( $r = 0.99$ ) with the true weight being expressed by the following equation: true weight =  $(1.013987 \times \text{scale weight}) + 0.707$ . The correction factor derived from this equation was used to correct each weight measurement.

### Medical treatment in the center

Oral antibiotics were given to most patients at admission, with many continuing to receive an antibiotic throughout their stay. Penicillin V, ampicillin, cotrimoxazole, and metronidazole were first-line antibiotics and chloramphenicol was the second-line antibiotic. Discharge criteria were predominantly clinical. Absence of clinical evidence of infection, a good appetite, constant weight gain for a minimum of 3 d, and the ability to walk and care for oneself were all necessary conditions for discharge. The presence of minimal pedal edema did not preclude discharge.

### Diets

During their stay in the center, patients received 6–8 meals each day. For the first month of operation, the standard higher-protein diet (HP diet) used in all of the other TFCs in Baidoa was the only diet available. This diet consisted of recovery milk [King's Food (Ermelo, Holland), a blend of dried skim milk, vegetable oil, vitamins, and minerals], UNIMIX (a blend of soya flour, oil, and sugar), rice, beans, and BP5 biscuits (Compact, Bergen, Norway) (Table 1). The recovery milk and BP5 biscuits were premixed fortified foods, designed especially for famine relief and marketed in Europe; the UNIMIX was a premixed, fortified food made up for UNICEF in various factories in Africa. Patients receiving this diet were offered, on average, 158 g protein/d and 16.2 MJ/d (P:E, 16.4%), including 70 g oil and 137 g lactose. The cost of this diet was ≈US\$1.90 per person daily.

**TABLE 1**  
The higher-protein diet<sup>1</sup>

Ingredient	Quantity	Protein	Energy	Sodium	Potassium
	g/d	g/d	kJ/d	mmol/d	mmol/d
Recovery milk	252	55	4276	42	71
UNIMIX	100	12	1680	0	14
Rice	200	12	2940	0	8
Beans	130	33	1966	1	46
BP5 biscuits	275	46	5290	2	18
Total	—	158	16151	45	156

<sup>1</sup> The higher-protein diet contained 16.4% of energy from protein. UNIMIX was made up for UNICEF in various factories in Africa. BP5 biscuits were manufactured by Compact (Bergen, Norway).

**TABLE 2**  
The lower-protein diet<sup>1</sup>

Ingredient	Quantity	Protein	Energy	Sodium	Potassium
	g/d	g/d	kJ/d	mmol/d	mmol/d
Dried skim milk	180	65	2646	44	73
Oil	150	0	5733	0	0
Sugar	160	0	2688	0	0
Rice	200	12	2940	0	8
Bananas	500	5	2100	0	51
Total	—	82	16107	44	132

<sup>1</sup> The lower-protein diet contained 8.5% of energy from protein.

During the first few days after admission, the milk component of this diet was diluted to half strength with the World Health Organization (WHO) formula ORS (WHO, Geneva), containing 3.5 g NaCl/L, 2.9 g trisodium citrate/L, 1.5 g KCl/L, and 20 g glucose/L. This reduced the total daily average offered to 129 g protein and 14 MJ (P:E, 15.5%).

From 5 December 1992, a lower-protein diet (LP diet) mixed on site from basic commodities was also available. The LP diet consisted of "high-energy milk," a blend of dried skim milk, vegetable oil, and sugar, together with bananas, white rice, and sweet tea (**Table 2**). Patients receiving this diet were offered, on average, 82 g protein and 16.1 MJ/d (P:E, 8.5%), including 152 g oil and 95 g lactose. The cost of this diet was ≈US\$1.00 per person daily. During the first few days after admission, the milk component of this diet was diluted to one-third strength with the WHO formula ORS. This reduced the total daily average offered to 35 g protein and 10.9 MJ (P:E, 5.0%). Initially, the potassium and sodium contents of the two diets were similar. However, during January 1993 mineral supplements became available and patients receiving the LP diet received (per kg body wt·d<sup>-1</sup>) 1 mmol KCl, 1 mmol tripotassium citrate, 0.4–0.8 mmol MgSO<sub>4</sub>, 0.031 mmol zinc acetate, and 0.003 mmol CuCl<sub>2</sub>, raising the amount of potassium they received by 2 mmol·kg body wt<sup>-1</sup>·d<sup>-1</sup>. For those patients who required rehydration, usually only during the initial phase of treatment, the WHO formula ORS was used. It was not feasible to quantify the amount of ORS and, therefore, the additional sodium and potassium consumed during these periods.

On 5 December 1992, the LP diet was first offered to 11 edematous patients who had not responded to the HP diet. The positive response in these patients to the change of diet was so dramatic that it was soon considered unethical to use the HP diet in the treatment of edematous patients. Thus, from 7 December 1992 on, patients with edema and those whom the clinician (SC) considered very ill or moribund were offered the LP diet during the initial phase of treatment. The HP diet continued to be used from admission in the less severe cases and for many patients during the recovery phase of rehabilitation. The onset of this recovery phase was defined clinically in edematous patients by a

return of appetite and a substantial loss of edema or ascites or both. Because loss of edema was accompanied by loss of weight, weight loss was accepted as a sign of a positive response to treatment (16). In marasmic patients, the recovery phase was defined by good appetite and steady weight gain for ≥3 d.

### Analysis of mortality

Of the 573 patients for whom admission data were collected, 16 were admitted primarily on medical grounds and we did not have sufficient dietary information for 70. These 86 patients were excluded from the analysis of mortality. For analysis, patients were assigned to either the HP group (*n* = 343) or the LP group (*n* = 144) according to the diet they received during their initial period of stay in the center. Thirteen patients were wrongly allocated to diets by junior staff on admission. These mistakes were rectified within 3 d by the clinician (SC) and the patients were assigned to the correct diet. For the analysis, the diet group of these patients was that of the corrected diet. Twenty-seven patients (all from the marasmic group) were lost to follow-up and were excluded from further analysis of mortality associated with the two diets, leaving 377 patients with marasmus or mild edema and 83 patients with moderate to severe edematous malnutrition. These data are presented in **Table 3**.

Two-by-two contingency tables and multiple logistic regression were used to assess the effect of the 2 diets and the mineral supplement on mortality. This allowed us to control for the potential confounding effects of morbidity (edema, lower respiratory tract infection, dysentery, dehydration, and anemia) and other variables (age, sex, and time since opening of the center) on mortality. The time variable was included in the analysis to control for the effect of any time bias in the data, as it was conceivable that patient care, independent of the introduction of the LP diet, improved over time. Variables not independently associated with mortality were excluded from models in a stepwise fashion using estimation techniques.

### Analysis of effects of HP and LP diets on rates of weight change and loss of edema during rehabilitation

The rate of weight change (g·kg<sup>-1</sup>·d<sup>-1</sup>) for each consecutive 3-d period was calculated for each patient. In those edematous patients who changed from the HP to the LP diet after they had been in the center for >3 d and for whom there were sufficient data (*n* = 7), a matched analysis using a paired *t* test of the rate of weight change during the first 15 d of treatment on each diet was performed.

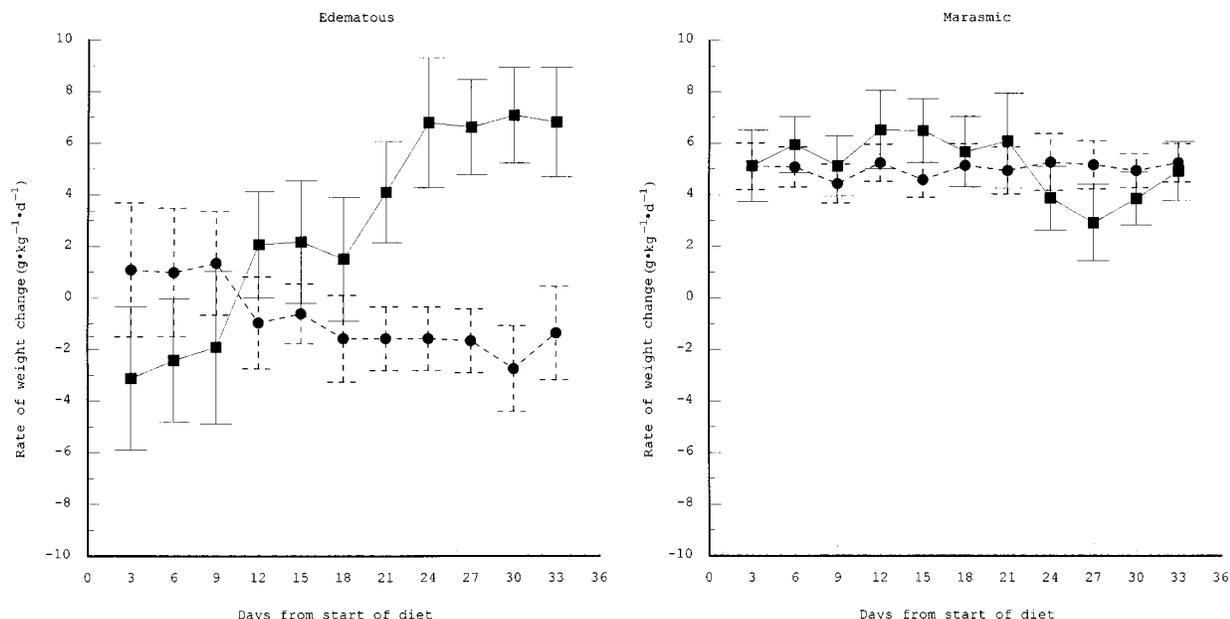
## RESULTS

### General patient data

The degree of emaciation and clinical condition of the 573 patients at the center were reported elsewhere (19). Mean BMI on admission

**TABLE 3**  
Outcome data associated with use of higher-protein (HP) and lower-protein (LP) diets

	All patients				Edematous patients			Marasmic patients		
	Total	Died	Survived	Lost to follow-up	Total	Died	Survived	Total	Died	Survived
HP diet	343	78	240	25	27	14	13	291	64	227
LP diet	144	25	117	2	56	14	42	86	11	75
Total	487	103	357	27	83	28	55	377	75	302



**FIGURE 1.** Mean ( $\pm 1$  SEM) rate of weight change in edematous ( $n = 45$ ) and marasmic ( $n = 219$ ) patients receiving the lower-protein (■) and higher-protein (●) diets during the first 33 d of treatment.

was 13.1 (95% CI: 12.9, 13.3); mean body weight was 35 kg. Four hundred thirteen (72%) of these patients survived, 122 (21%) died, and 38 (7%) were lost to follow-up. Edematous malnutrition was common and was associated with increased mortality. It was present in 16% of all admissions and 28% of those who subsequently died. Case fatality rates were 37% for edematous malnutrition and 20% for marasmus or mild edema [odds ratio (OR): 2.4; 95% CI: 1.4, 3.9; Yates's corrected chi-square: 11.7,  $P < 0.001$ ]. The prognosis in edematous malnutrition was worse for male than for female patients (male OR: 4.1, 95% CI: 1.9, 8.7; female OR: 1.4, 95% CI: 0.6, 2.9; Woolf's test for the heterogeneity of odds ratios: 4.59,  $P < 0.05$ ). Nineteen percent of deaths occurred within 48 h of admission, 48% within the first week, and 27% during the second week after admission. Median time to discharge for all patients was 28 d (35 d in edematous patients, 27 d in marasmic patients). Mean BMI at the time of discharge was 15.0.

#### Effect of HP and LP diets on mortality

Mortality was lower in patients with edematous malnutrition who received the LP diet during the initial phase of treatment (crude OR: 0.31; 95% CI: 0.10, 0.90; Yates's corrected chi-square: 4.74,  $P < 0.05$ ) than in those who received the HP diet. This effect remained after confounding variables were adjusted for by logistic regression (adjusted OR: 0.31; 95% CI: 0.12, 0.81; likelihood ratio statistic: 5.74,  $P < 0.05$ ). This is equivalent to a threefold reduction in mortality. No such difference was observed in marasmic patients (crude OR: 0.52; 95% CI: 0.24, 1.08; Yates's corrected chi-square: 2.97,  $P = 0.08$ , and adjusted OR: 0.87; 95% CI: 0.38, 1.97; likelihood ratio statistic: 0.10,  $P = 0.74$ ). Analysis of the effects of the mineral mix in patients receiving the LP diet did not uncover any significant differences in mortality before and after addition of the mineral supplement.

#### Effect of HP and LP diets on loss of edema and gastrointestinal function

In the 7 patients for whom matched data for rate of weight change were available, the rate of loss of edema, reflected as

weight loss, accelerated after transfer to the LP diet [mean ( $\pm$  SD) rate of weight change:  $6.3 \pm 12.1$  and  $-7.2 \pm 18.5$   $\text{g} \cdot \text{kg}^{-1} \cdot \text{d}^{-1}$  with the HP and LP diets, respectively; difference = 13.5; paired  $t = 3.18$ ,  $P < 0.05$ ]. The appetite of many of the edematous patients appeared to be poor with the HP diet. When these patients were switched to the LP diet, an increase in appetite was observed in many, including those who had suffered from persistent edema lasting several weeks while they received the HP diet. Associated with the increased appetite were episodes of watery diarrhea. These were occasionally severe, resulting in the rapid appearance of intravascular hypovolemia. Patients with watery diarrhea responded to dilution of the milk element of the diet with the WHO formula ORS during the first few days of treatment. The number of days and the degree to which the milk was diluted were tailored for each patient according to the severity of the diarrhea, clinical signs of intravascular hypovolemia (heart rate, peripheral perfusion, jugular venous pressure, and hydration of mucous membranes), and the response to previous dilutions. On occasion, it was necessary to dilute the milk to one-ninth strength for several days to reduce diarrhea and maintain the appropriate intravascular volume. Once the diarrhea resolved, the milk concentration was gradually increased to full strength over 1 wk.

#### Weight change during rehabilitation

The mean rates of weight change for edematous and marasmic patients during the initial month of treatment on each diet are shown in **Figure 1**. During the first 9 d, edematous patients receiving the LP diet tended to lose weight as they lost edema. By contrast, edematous patients receiving the HP diet tended to gain weight. After this period the situation reversed and those receiving the LP diet started to gain weight whereas those receiving the HP diet stayed at the same weight or gradually lost weight. Marasmic adults gained weight similarly with both diets during this initial period.

During the recovery phase (16–60 d after the start of treatment), mean ( $\pm$  SD) rates of weight change were similar in both

marasmic and formerly edematous patients (rate of weight change:  $6.1 \pm 5.3$  and  $5.1 \pm 4.2$  g · kg<sup>-1</sup> · d<sup>-1</sup>, respectively) and for the HP and LP diets (rate of weight change:  $5.9 \pm 5.6$  and  $5.6 \pm 4.8$  g · kg<sup>-1</sup> · d<sup>-1</sup>, respectively).

## DISCUSSION

### Importance of lower-protein diets during the initial treatment of severe malnutrition in adults

In this study, mortality was threefold higher in edematous patients receiving the HP diet than in those receiving the LP diet. Many of these patients also experienced prolonged anorexia and persistent edema. The LP diet was associated with lower mortality and accelerated loss of edema. These differences appear likely to have been due to the different amounts of dietary protein offered during the initial phase of therapy.

Other potential explanations, however, must also be examined. The study compared groups of patients differing primarily according to the diet they received. However, allocation of patients to the diets could not be randomized and it is possible that the differences in outcome ascribed to differences in diet were instead due to differences in time since the center opened or to differences in the subject groups assigned to each diet. In the analysis of mortality, the use of multiple logistic regression allowed us to control for differences in time. With respect to subject differences, during the first 6 wk of the study, all patients received the HP diet; during the next 16 wk, those patients assigned to the LP diet were edematous or otherwise very ill or moribund. On clinical grounds, these patients were expected to have a higher mortality than those assigned to or already recovering satisfactorily on the HP diet. However, the study showed that patients receiving the LP diet had a threefold lower rate of mortality and a more rapid loss of edema than patients receiving the HP diet. Thus, it is possible that had we randomly assigned patients the difference in outcome would have been in the same direction but even greater. This is compelling evidence that the difference in outcome was due to the difference in diet.

The diets, however, differed in more than their protein contents and it is also probable that intake of the two diets differed. Lower energy intake, particularly energy derived from carbohydrate, during the initial treatment with the LP diet is a possible explanation of the differences in outcome. Lower energy intake might be expected to lower mortality by reducing the incidence of refeeding syndrome (23). Although the aim was to offer diets of similar energy contents (Tables 1 and 2), the LP diet had a greater proportion of its energy derived from fat and was usually given in a more dilute form during the first few days of treatment. This dilution substantially reduced the energy being offered to patients during that time. Although the recovery milk element of the HP diet was also diluted during the first few days of treatment, this dilution caused less of a decrease in the energy content of the HP diet as a whole. These differences, however, are unlikely to explain the higher mortality associated with the HP diet because as a result of persistent anorexia most of the edematous patients receiving the HP diet consumed only a small fraction of the food offered. Their food intakes, although not formally measured, appeared to be very low, making refeeding syndrome unlikely. The marked increase in appetite, and thus presumed increase in food intake, observed in these patients when they were offered the LP diet is likely to have more than com-

pensated for the decreased energy content of the diluted diet. Indeed, it is likely that energy intake during the initial phase of treatment with the LP diet was higher than with the HP diet. This agrees with documented experiences in feeding severely malnourished children in Jamaica (24).

Immediately after the change to the LP diet, most of the edematous patients who had been in the center for some weeks developed watery diarrhea. This diarrhea appeared to be the refeeding diarrhea frequently described in new arrivals at feeding centers and generally regarded as the response of a starving and atrophic intestine to the initial reintroduction of food (25). The absence of refeeding diarrhea among the edematous patients receiving the HP diet and its development when these patients were transferred to the LP diet supports the observation that intake of the HP diet in these patients was low.

Other differences in composition of the two diets could also have contributed to the difference in mortality. The HP diet had more lactose, which could possibly have increased mortality. This is also unlikely, though, because the lower incidence of diarrhea among patients receiving the HP diet indicates that lactose intolerance was not a major problem.

In marasmic patients, the diet given during the initial phase of treatment did not affect mortality or initial rate of weight change. This suggests that the dietary differences interacted with metabolic differences between marasmus and edematous malnutrition. One of the main metabolic differences is in liver function. In this study, few of the marasmic but many of the edematous patients had clinical signs consistent with liver failure. These included petechial rash and jaundice as well as anorexia, edema, and ascites. Thus, it is possible that there was no difference in outcome associated with diet in marasmic patients because their liver function was better preserved. If correct, this again suggests that it was the protein content of the diet that was of importance.

The study failed to show any effect of the mineral supplements in patients given the LP diet. This may have been due to the study design, the aim of which was not to test the effect of these supplements. However, it is also possible that the mineral intake from the LP diet alone was adequate. This is likely to be true for potassium, dietary intake of which was usually  $\geq 2$  mmol · kg<sup>-1</sup> · d<sup>-1</sup>, but unlikely to be so for the other minerals.

Although recovery from severe adult malnutrition has been systematically studied only rarely, there have been reports of similar poor responses to HP diets wherein edema was slow to disappear and sometimes even appeared during treatment (26–28). Ex-inmates of Belsen concentration camp, who experienced degrees of weight loss comparable with those in patients in Baidoa (mean weight loss of 38%, corresponding to a BMI of 12–14) frequently suffered from famine edema and ascites (9). During rehabilitation with a diet containing 64.8 g protein and 3.4 MJ (P:E, 32.4%), famine edema often appeared or increased (10). In these patients, use of casein hydrolysates was associated with increased mortality (29). Similar accounts were reported from the Dutch famine of 1945, for which the recommended rehabilitation diet contained 300 g protein and 13.4 MJ (P:E, 37%) (8).

### Rates of weight change during the recovery phase of rehabilitation

The extreme levels of disruption and insecurity present in Baidoa during the time of this study made the operation of the center difficult. As a result, the mean rates of weight gain

(5–6 g · kg<sup>-1</sup> · d<sup>-1</sup>) during the recovery phase of rehabilitation probably represent the lower end of the spectrum of reasonable rates of weight gain in adults recovering from severe malnutrition. Severely malnourished ex-inmates of the Sanbostel concentration camp receiving 31.5 MJ and 297 g protein/d (P:E, 15.8%) gained ≈7 g · kg<sup>-1</sup> · d<sup>-1</sup> during the recovery phase of their treatment (30). In less severely malnourished patients, rates of weight gain appear to be lower. In the Minnesota experiment, 32 volunteers with mild to moderate malnutrition who received 10–14 MJ and 75–100 g protein/d (P:E, 10.6–14.4%) gained only 1.85 g · kg<sup>-1</sup> · d<sup>-1</sup> (31).

### Comparison with children

The patterns of presentation and recovery in severely malnourished adults are similar to those in children. In children, hypoalbuminemia is evidence of a poor prognosis (32). Although edema, in the absence of hypoalbuminemia, is not necessarily an indicator of a poor prognosis (14), in practice, the frequent coexistence of the two makes edema a useful prognostic marker. In Baidoa, edema in adults was associated with a much poorer prognosis (19). However, it is possible that this finding is not universal. In a Concern Worldwide TFC in Melange, Angola, during 1993 and 1994, 90% of adults admitted suffered from edema but this was not associated with such a poor prognosis (S Collins, unpublished observations, 1993). More work is needed on prognostic indicators in severe adult malnutrition.

The maximum rates of weight gain, typically 10–20 g · kg<sup>-1</sup> · d<sup>-1</sup>, recorded in children recovering from severe malnutrition (14, 33, 34) are considerably higher than the rates of weight gain reported here. However, the pattern of recovery is similar. Initially, with a low-protein, maintenance energy intake, edematous children often lose edema within 1 wk (16). Appetite returns and this, together with loss of edema, heralds the recovery phase. Among our edematous adult patients, the rates of edema loss were variable and often much slower. With the LP diet, some patients lost most of their visible edema and ascites within a few days, the rapid loss generally being accompanied by watery diarrhea. In these patients, care had to be taken to avoid intravascular hypovolemia. In the absence of guidelines for adults, we aimed at a loss of ≈0.25–0.5 L/d, equivalent to a weight loss of 0.25–0.5 kg/d. Regulation of the rate of edema loss was achieved by diluting the high-energy milk with ORS to an extent dictated by the severity of diarrhea. In other patients, particularly those receiving the HP diet, the rate of loss of edema was much slower and pedal edema or ascites persisted for weeks. This was accompanied by persistent anorexia and debility. These patients generally responded rapidly to introduction of the LP diet. The response involved marked increase in appetite and general well-being and loss of edema and ascites. However, minor grades of edema sometimes persisted for weeks, even after the patients had recovered much of their original body mass. When edema increased even with the LP diet, the patient usually died.

### Conclusions

Because of the extreme conditions in this TFC, which was operating in a war zone, the subjects in this study were not randomly assigned to the 2 diets, nor were their dietary intakes estimated. During most of the study, those with a worse prognosis on admission received the LP diet. Notwithstanding, with the LP diet, edematous adults suffered lower rates of mortality and lost edema more quickly than did those receiving a more conventional HP diet. In marasmic adults, there was no difference in

mortality between patients receiving LP and HP diets. During the recovery phase, there were no differences in rates of weight gain between the LP and HP diet groups.

Thus, it appears that, compared with the conventional HP diet, the LP diet was more effective in the treatment of edematous adults and as effective in the treatment of marasmic adults. The LP diet usually needed to be diluted during the initial phase of treatment. It was cheaper and more easily obtained than the specially manufactured famine relief foods of the HP diet. We suggest that such a diet, based on milk, oil, sugar, and locally available foods, with a relatively low P:E, should be offered to all severely malnourished adults in both the initial and recovery phases of rehabilitation. By using locally available produce, use of such diets may stimulate the local economy. Their use should also ease the organizational difficulties involved in the provision of food items during emergency feeding operations. More research on treatment protocols for use in severe adult malnutrition is needed. 

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