

Dietary management of obesity: evaluation of the time-energy displacement diet in terms of its efficacy and nutritional adequacy for long-term weight control

BY ROLAND L. WEINSIER, MARJORIE H. JOHNSTON,
DANIEL M. DOLEYS AND JANE A. BACON

*Department of Nutrition Sciences, The University of Alabama in Birmingham,
Birmingham, Alabama 35294, USA*

(Received 10 June 1981 – Accepted 19 November 1981)

1. An unsupplemented 4200 kJ (1000 kcal) diet emphasizing large quantities of relatively unrefined complex carbohydrates was evaluated among sixty obese adults for its effectiveness and nutritional adequacy in a long-term weight-control programme. Patients were followed individually as outpatients by a physician and dietician – an average of thirteen visits over 26 weeks. Assessment of health indices included anthropometric measurements, blood pressure, lipid levels and assays for seven vitamins, β -carotene and iron.

2. Weight loss averaged 8.2 kg or 24% of excess weight during the 6 months of active treatment. Over an average of 17 months of post-treatment follow-up, 44% of patients continued to lose weight and 92% remained below pretreatment levels.

3. Average skinfold thickness fell 7 mm ($P < 0.001$) whereas muscle mass was maintained (arm muscle circumference + 10 mm, not significant; creatinine-height index + 3% of standard (Bistrain *et al.* 1975; not significant). Systolic and diastolic blood pressure fell 7 and 5 mmHg respectively ($P < 0.01$). Total serum cholesterol and triglycerides fell 200 and 660 mg/l respectively ($P < 0.01$), while high-density-lipoprotein-cholesterol remained statistically unchanged. Mean serum levels of retinol, β -carotene, folate, vitamin B₁₂, ascorbic acid, Fe and transferrin saturation, and activity coefficients for thiamin, riboflavin and pyridoxine were within normal limits after periods of treatment ranging from 5 to 84 weeks.

4. An earlier age of onset of obesity tended to be associated with greater weight loss during treatment and lesser weight rebound during follow-up.

5. The results indicate that the experimental diet, without supplementation, was nutritionally adequate as well as effective for long-term weight control.

The central therapeutic modality for controlling obesity continues to be energy restriction, whether from the standpoint of various dietary manipulations, behavioural modification or anorectic drugs. The role of reduced energy intake is evident even when therapy is directed towards increasing energy losses through exercise (Leon *et al.* 1979) or surgically induced malabsorption (Bray & Benfield, 1977). Thus, it is reasonable that the diet modification should not be left to the patient's own discretion, but be designed to meet acceptable criteria such as those outlined by Young (1973). Specifically, it should: (1) satisfy all nutrient needs except energy, (2) be adaptable to meet individual tastes and habits, (3) minimize hunger and fatigue, (4) be readily obtainable and socially acceptable, (5) favour establishment of a lasting pattern of eating. In addition, since most obese persons will require extended periods of time to achieve their weight goals, it is vitally important that the imposed diet pattern be conducive to maintenance, if not improvement, of over-all health. Accordingly, it was the intent of this study to design and then evaluate an energy-restricted diet in terms of its efficacy for long-term weight control and its adequacy for maintaining normal clinical and laboratory criteria of nutritional status.

MATERIALS AND METHODS

Patient sample

All adult obese patients referred to the Nutrition Clinic of the University of Alabama in Birmingham Hospitals were considered candidates for the study. Each patient was seen in

Table 1. *Characteristics of patient population*
(Mean values and standard deviations)

Characteristics	All patients	Men	Women
No. of patients	60	18	42
Age (years)	36 ± 11	36 ± 11	36 ± 11
Percentage juvenile-onset	50	67	43
Initial weight (kg)	102 ± 26	128 ± 24	91 ± 18
Percentage ideal body-weight*	166 ± 36	176 ± 36	162 ± 36
Education (years)	15 ± 3	16 ± 2	15 ± 3

* From values adapted from Metropolitan Life Insurance Company (1959): build and blood pressures study.

a preliminary interview in order to explain the basic treatment approach and clinic charges. Patients were charged for services according to the usual university outpatient fee schedule; however, the fee was reduced or eliminated for those less able to meet these charges. The only criteria for rejection of a patient wishing to join the programme were food intolerances which would have precluded adherence to the recommended diet pattern and inability to read and understand the basic teaching materials presented. There were few patients who were excluded for these reasons. By contrast, a totally blind patient was accepted although special teaching materials had to be prepared. Values from two patients were not included in the results when, after failure to lose weight over 20 weeks, they were hospitalized in an attempt to explain marked discrepancies between intake records and weight response, at which time intentional falsification of records was confirmed. With this exception, however, the results are based on the response of sixty consecutive patients joining the programme regardless of the weight loss, or lack thereof, or whether they dropped out early or stayed beyond the usual 20-week treatment phase. The characteristics of the study population are presented in Table 1.

Dietary approach

The diet was designed with the following points in mind: (1) it was to consist of relatively large volumes of low-energy complex carbohydrates that would require more than average time in ingestion, with the intent to displace more-energetically-dense items (thus, the designation time-energy displacement), and (2) it was to be nutritionally sound and conducive to a lifelong pattern of healthful eating by both the patient and family. Fig. 1 depicts the dietary approach with the relative energy densities of the major food groups. For prescription purposes the reference food chart deletes the sweets group and adds a dairy products group and, under each food group, gives a detailed list of preferred and occasional food items. Preferred foods, in general, were those that require more time for ingestion and provide more fibre and less energy. For example, raw and cooked vegetables would be preferred to vegetable juices; fresh fruit preferred to canned fruit and fruit juice; unrefined cooked starches (e.g. potatoes, maize) preferred to refined dry starches (e.g. crackers, many cereals). The patient then selected a prescribed number of daily serving equivalents from each food group. A maximum number of fat servings was given but no minimum required; a minimum and maximum range was given for the meat, dairy and starch groups to ensure nutritional adequacy but not excess energy; and a minimum with no upper limit was allowed for fruits and vegetables in order to emphasize and encourage the least-energy-dense items. The energy prescription was approximately 4200 kJ (1000 kcal) for both men and women, regardless of starting weight. Vitamin and mineral supplements were not recommended in order to assess nutritional adequacy of the diet. The calculated nutrient content of the diet

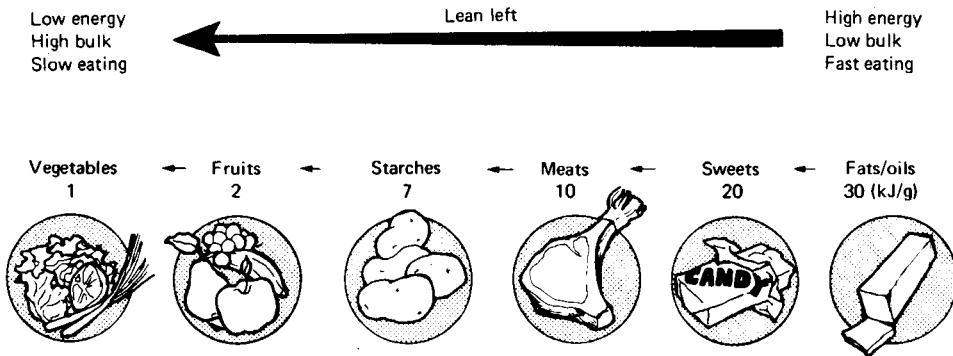


Fig. 1. Spectrum of relative energy densities of various food groups. During energy restriction, emphasis is on those food groups to the left in order to maximize the volume of food ingested and to minimize the chances of nutritional deficiencies.

as compared to the US 'Dietary Goals' (Select Committee on Nutrition and Human Needs, United States Senate, 1977) and recommended allowances (US Food and Nutrition Board, 1980) is shown in Table 2. Sodium intake was routinely restricted to minimize fluctuations in water retention.

Daily intake records were kept by all patients throughout the period of treatment except in cases where close dietary adherence had been well documented over at least 20 weeks. Thereafter records were kept only if weight failed to decline or when nutritional status was being evaluated.

Treatment protocol

All patients were followed as outpatients and treated individually by a physician and a dietician. Clinic visits averaged 30 min, during which time dietary records were reviewed, problems discussed and recommendations made. Family members participated in these sessions when appropriate. Increased physical activity was generally discussed and supported although no standardized programme of exercise was prescribed. Relevant emotional and behavioural problems were discussed. Behavioural changes were elicited through record-keeping, and maladaptive patterns were addressed individually by the physician, dietician and, in many instances, a behavioural psychologist.

Clinic visits were scheduled weekly for the first 6 weeks and then bi-weekly for a total of thirteen visits over 20 weeks. Some patients dropped out before completion of the 20-week treatment programme whereas others continued in treatment for longer periods of time. Post-treatment follow-up was defined as the interval from the patient's last clinic visit until subsequent contact by telephone. Since all previously treated patients were called at approximately the same time, length of post-treatment follow-up varied considerably.

Assessment of health criteria

Anthropometric measurements were obtained initially and then at approximately 10-week intervals throughout therapy. Triceps, subscapular, and abdominal skinfold thickness and mid-arm circumference were measured by accepted methods. Creatinine-height index was expressed as a percentage of the standard (Bistrian *et al.* 1975) on the basis of the 24 h urinary creatinine excretion and the patient's height. Blood pressure was taken at each clinic visit. Serum total and high-density-lipoprotein (HDL)-cholesterol and triglycerides were measured on a fasting sample by the Dupont autoanalyser method (Automatic Clinical

Table 2. Nutrient content of time-energy displacement dietary model and comparison with US 'Dietary Goals' (Select Committee on Nutrition and Human Needs, United States Senate, 1977) and recommended allowances (US Food and Nutrition Board, 1980). (Calculations are based on a sample menu for 1 week, averaging 4452 kJ (1060 kcal)/d)

	Percentage of energy as:												
	Complex CHO	Simple CHO	Fat	Protein	Cholesterol (mg)	Vitamin A (RE)	Thiamin (mg)	Riboflavin (mg)	Niacin (mg)	Ascorbic acid (mg)	Calcium (mg)	Iron (mg)	Sodium (g)
TED model diet	51 (138 g)	6 (15 g)	19 (22 g)	24 (63 g)	201	1177	1.3	1.3	13.7	208	729	11	1.1
US dietary goals	40-50	15	30	10-15	300	—	—	—	—	—	—	—	1.2
Recommended dietary allowances													
♂				56 g	—	1000	0.5*	0.6*	6.6*	60	800	10	1.1-3.3
♀				44 g	—	800	0.5*	0.6*	6.6*	60	800	18	1.1-3.3

CHO, carbohydrate; RE, retinol equivalents.

* Based on recommended allowance per 4200 kJ (1000 kcal).

Table 3. *Weight change during treatment and post-treatment follow-up in obese subjects undergoing a dietary programme of time-energy displacement**

	All patients		Men		Women	
	Mean	SD	Mean	SD	Mean	SD
Response during treatment:						
No. of patients		60		18		42
Weight loss (kg)	8.2	6.8	12.9	7.9	6.2	5.3
Patients losing:						
≥ 9 kg (20 lb) (%)		35		72		19
≥ 18 kg (40 lb) (%)		7		17		2
Rate of weight loss (kg/week)†	0.4	0.4	0.6	0.6	0.3	0.4
% of weight loss of excess	24	20	28	16	23	22
Length of treatment (weeks)	26	23	24	15	26	26
No. of visits	13	8	13	7	13	8
Response post-treatment:						
No. of patients		48		14		34
Weight change (kg)	-0.3	6.3	2.3	5.6	-1.3	6.0
Length of follow-up (months)	17	12	13	10	19	13
Over-all response:						
Over-all weight loss (kg)	8.9	8.4	10.9	6.1	8.1	9.0
Over-all percentage weight loss of excess	25	24	22	15	27	27

* For details, see p. 368.

† Calculated as the mean of individuals' rates of weight loss.

Analyzer, 1981) at approximately 10-week intervals. Significance of the change in each of these criteria between initial and final assessment was determined by paired *t* testing. Not all values were available for comparison in every patient. If a patient failed to complete the initial 20-week phase of therapy or was continued in therapy beyond this period, the values used were those from the last assessment.

Vitamin and Fe nutriture was assessed in a subgroup of twenty-six patients who had been on the diet for periods ranging from 5 to 84 weeks. Adherence to the prescribed dietary guidelines in these patients was determined by their dietary intake records and ongoing weight loss. Average energy intakes were calculated on the basis of 7 d intake records 1 week before laboratory assessment. None of these patients was taking vitamin or Fe supplements. On a fasting sample, serum vitamin A was determined by the method of Garry *et al.* (1970), β -carotene by the method of Baker & Frank (1968), folates by microbiological assay as described by Herbert (1966), vitamin B₁₂ by radioisotope-dilution assay with the procedure of Lau *et al.* (1965), and vitamin C by the method of Zannoni *et al.* (1974). Thiamin, riboflavin and pyridoxine were assessed indirectly, determining the *in vitro* activation of erythrocyte transketolase (EC 2.2.1.1) glutathione reductase (EC 1.6.4.2) and glutamic:oxaloacetic transaminase (EC 2.6.1.1), respectively, by the addition of appropriate cofactors as described by Bayoumi & Rosalki (1976). Serum Fe and percentage transferrin saturation were determined by the Dupont autoanalyser method (Automatic Clinical Analyzer, 1981).

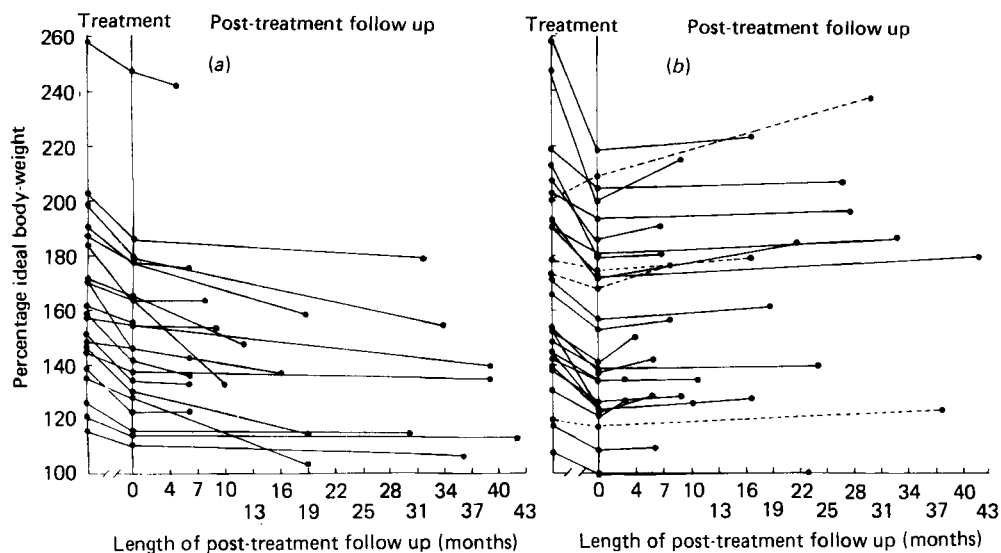


Fig. 2. Changes in percentage ideal body-weight from onset to end of treatment and at follow-up of the forty-eight subjects who could be contacted post-treatment. (a) Patterns of subjects who remained at or below their post-treatment weights (44%, 21 subjects). (b) Patterns of subjects who regained part (solid lines, 48%, 23 subjects) or all (dashed lines, 8%, 4 subjects) of their treatment weight losses.

Thus, 92% (44 out of 48) of subjects remained below their pre-treatment weights at time of follow-up. There is no evident trend towards greater weight rebound with longer periods of follow-up.

RESULTS

Weight response of total patient sample

As shown in Table 3, the average patient was seen in treatment thirteen times over 26 weeks and lost 8.2 kg, or 24% of the total excess weight. The rate of weight loss averaged 0.4 kg/week. Compared to women, men tended to lose more weight, a greater percentage of their excess, and at a faster rate over a comparable period of time.

Post-treatment follow-up information was obtained on fifty of the sixty patients. Two of these persons had undergone surgical bypass procedures for obesity and were excluded from this sample. The average length of follow-up since final visit in the programme was 17 months, during which time weight had declined an average of 0.3 kg. Over-all weight loss (during treatment and post-treatment follow-up) was 8.9 kg, representing an average loss of 25% of the initial excess weight.

Fig. 2 graphically presents the change in percentage ideal body-weight during treatment and the trend towards weight rebound or continued weight loss over the follow-up interval of 3–42 months. Of the patients 92% (forty-four of the forty-eight) maintained weights below their pretreatment levels; 44% (twenty-one of the forty-eight) continued to lose. There was no significant correlation between the absolute amount of weight lost during treatment and that regained during follow-up ($r = 0.12$, not significant).

Measurement of health criteria

Table 4 presents the changes in various health criteria measured during weight reduction. Loss of subcutaneous fat was demonstrated by the decline in mean skinfold thickness of 7 mm ($P < 0.001$), whereas arm muscle circumference and creatinine-height index remained statistically unchanged (+10 mm and +3% of standard value respectively).

Table 4. Health criteria measured during weight reduction in obese subjects undergoing a programme of time-energy displacement*

	No. of observations	Paired changes		Statistical significance of change†: <i>P</i>	
		Mean	SD		
Anthropometrics:					
Skinfold thickness (mm)‡	38	-7	6	< 0.001	
Arm muscle circumference (mm)	40	+1	3	NS	
Creatinine-height index (% of standard)§	15	+3	21	NS	
Blood pressure:					
Systolic (mmHg)	60	-7	18	< 0.01	
Diastolic (mmHg)	60	-5	12	< 0.01	
Serum lipids:					
Total cholesterol (mg/l)	36	-200	380	< 0.01	
HDL-cholesterol (mg/l)	16	-10	90	NS	
Triglycerides (mg/l)	32	-660	1310	< 0.01	
Vitamin-iron status: 					
		Mean	SD	Range	Normal values
Vitamin A ($\mu\text{g/l}$)	26	500	150	290-830	250-700
β -carotene ($\mu\text{g/l}$)	26	1440	660	600-3150	790-2330
Folate (ng/ml)	26	7.1	4.9	2.4-18.2	2-10
Vitamin B ₁₂ (pg/ml)	26	343	132	178-752	205-700
Vitamin C (mg/l)	21	10.0	5.0	2.0-20.0	5.0-25.0
Thiamin (activity coefficient)¶	26	1.09	0.08	1.00-1.25	1.00-1.23
Riboflavin (activity coefficient)¶	26	1.27	0.20	1.00-1.78	1.00-1.67
Pyridoxine (activity coefficient)¶	26	1.45	0.17	1.09-1.72	1.00-1.89
Serum Fe ($\mu\text{g/l}$)	19	840	340	190-1600	500-1500
Percentage transferrin saturation	19	25	11	6-59	20-55

NS, not significant; HDL, high-density lipoprotein.

* For details, see p. 368.

† Paired *t* test.

‡ Average of triceps, subscapular and abdominal skinfolds.

§ Bistrian *et al.* (1975).

|| No patient was receiving vitamin or mineral supplements.

¶ 'Activity coefficient' is the ratio of stimulated enzyme activity to unstimulated enzyme activity. Stimulation is by in vitro addition of the enzyme's cofactor.

Systolic blood pressure fell an average of 7 mmHg, representing a 4% drop from the baseline value. Diastolic pressure fell 5 mmHg, or 5% from the baseline value. Despite the statistically significant drop in blood pressure during the course of therapy, there was not a significant correlation between the amount of weight an individual lost and the fall in systolic or diastolic blood pressure ($r = 0.08$ and 0.03 respectively). Total serum cholesterol fell an average of 200 mg/l from a baseline of 2130 mg/l, an average fall of 9%, whereas the mean level of HDL-cholesterol did not change significantly. Serum triglycerides decreased 660 mg/l from a baseline value of 2040 mg/l, representing an average drop of 32%.

As shown in Table 4, mean values for all ten measured criteria of vitamin and Fe status were within normal limits. At the time of nutrition assessment these patients had been on the diet for an average of 21 ± 19 weeks (range 5-84 weeks), and had lost an average of 10 ± 6 kg body-weight. Average energy intake of this group, determined by 7 d intake records at the time of nutrition assessment, was 4528 ± 605 kJ (1078 ± 144 kcal)/d.

Table 5. *Weight responses as a function of age of onset of obesity in obese subjects undergoing a programme of time-energy displacement**

	Juvenile onset (≤ 15 years of age)		Adult onset (> 15 years of age)		Level of significance: <i>P</i>
	Mean	SD	Mean	SD	
Population characteristics:					
No. of patients	30		30		
Sex (% women)	63		77		NS
Age (years)	35	10	37	11	NS
Initial weight (kg)	109	30	93	24	NS
Percentage ideal body-weight	173	39	157	30	NS
Education (years)	15	3	14	2	NS
Response during treatment:					
Weight loss (kg)	10.2	7.9	6.2	4.5	< 0.02
Rate of weight loss (kg/week)	0.4	0.5	0.4	0.3	NS
Length of treatment (weeks)	27	22	26	24	NS
No. of visits	14	9	12	7	NS
Response post-treatment:					
No. of patients	26		22		
Weight change (kg)	-1.4	6.5	1.0	5.8	NS
Length of follow-up (months)	17	12	18	12	NS

NS, not significant.

* For details, see p. 368.

Age of onset of obesity

By chance the total patient population of sixty fell into two equal groups according to juvenile- or adult-onset of their obesity. The groups were statistically similar in their characteristics of sex, age, initial weight and education, in addition to length of treatment, number of clinic visits and length of post-treatment follow-up (Table 5). During treatment, average weight loss was greater among the juvenile-onset patients than among the adult-onset (10.2 v. 6.2 kg, $P < 0.02$). During the period of post-treatment follow-up, there was a tendency for juvenile-onset patients to continue to lose and adult-onset patients to regain, although the changes were not statistically different between the two groups.

Factors associated with weight loss

Table 6 lists the correlation coefficients of various factors associated with weight loss during treatment and weight rebound during post-treatment follow-up. Those factors predictive of greater weight loss during treatment included an earlier age of onset of obesity, a greater initial body-weight, a higher education level, and a longer period of treatment. The five variables examined account for 50% of the total outcome variance for weight loss during treatment and for 40% of the variance for post-treatment weight rebound.

DISCUSSION

The results of this study suggest that the proposed experimental diet approach met our criteria for success in terms of being an efficacious while safe means to achieve long-term weight control. The word success must be interpreted cautiously, however, in that no accepted standard exists for comparison. The average weight loss in this study population (6 kg for women, 13 kg for men) represents a loss of approximately one-fourth of the initial

Table 6. Factors associated with weight loss during treatment and with weight rebound post-treatment in obese subjects undergoing a programme of time-energy displacement*

	During treatment (absolute weight loss) (n = 60)		Post-treatment (percentage weight rebound) (n = 48)	
	r	p	r	p
Age	0.01	NS	0.07	NS
Age of onset of obesity	-0.24	NS	0.23	NS
Percentage ideal body-weight	0.32	< 0.05	-0.03	NS
Education	0.41	< 0.01	0.50	< 0.01
Length of treatment	0.41	< 0.01	0.31	< 0.05

NS, not significant.

* For details, see p. 368.

excess weight. The rate of loss of 0.4 kg/week over an average of 6 months is clearly acceptable if compared to studies of similar length wherein average rates range between gains of 0.1 kg/week and losses of 0.3 kg/week (Gray & Kallenbach, 1939; Adlersberg & Mayer, 1949; Ressler, 1959). Our findings that 35% of the patients lost ≥ 9 kg (20 lb) and 7% lost ≥ 18 kg (40 lb) compare favourably with other reports averaging 28% (range 12–53%) and 6% (range 1–13%) respectively (Fellows, 1931; Stunkard & McLaren-Hume, 1959; Stunkard *et al.* 1970; Penick *et al.* 1971; Currey *et al.* 1977). It is noteworthy that our treatment and follow-up results include the responses of all patients including drop-outs. This is in contrast to the tendency to report results of only select subjects who remain in treatment for extended periods of time (Feinstein, 1960). In our study post-treatment follow-up spanned a period from 3 to 42 months, during which time there was no evident trend towards weight rebound for the group as a whole. This finding also differs from the frequently reported patterns of an increasing failure rate over time (Fellows, 1931; Innes, *et al.* 1974; Stunkard & Penick, 1979; Bjorntorp, 1980; Stunkard *et al.* 1980). According to the findings of Stunkard & Albaum (1981), the fact that follow-up weights were obtained by telephone does not offset the validity of these results.

The concept behind the experimental dietary approach of time-energy displacement was that an energetically restricted diet comprising large quantities of high-bulk complex carbohydrates would result in prolonged eating time, a greater sense of satiety, decreased energy absorption, and a large enough volume to displace intake of more-energy-dense items. Although evidence is accumulating that this concept may be valid (Macrae *et al.* 1942; Shearer, 1976; Haber *et al.* 1977; Alpert, 1979; Jenkins *et al.* 1979), and certainly there is a great deal of speculation to that effect (Heaton, 1973; Southgate, 1973; Trowell, 1976; Spiller & Kay, 1979), there is still room for scepticism (Bierman, 1979). However, it was not our intention to study the relative advantages of this diet compared to others, but to evaluate its efficacy for weight control and adequacy for health maintenance. Repeated evaluation using a detailed written questionnaire for subjective responses of our patients revealed that discomfort due to hunger and deterioration in physical or mental well-being were exceedingly rare despite long-term energy restriction. Such a sense of well-being is not always found during weight loss (Fellows, 1931; Mullins, 1958; Feinstein, 1960; Halmi *et al.* 1980), especially if carbohydrate intake is severely limited (American Medical Association Council on Foods and Nutrition, 1974; Rickman *et al.* 1974).

Anthropometric measurements taken before and after treatment suggested that a significant loss of adipose tissue had occurred with little or no change in muscle mass (as reflected by maintenance of the calculated arm muscle circumference and creatinine-height index). Previous studies have shown a progressive fall in creatinine excretion over 2–3 month periods suggesting loss of muscle mass, although the diets were generally more severely restricted in energy than ours (Buskirk *et al.* 1963; Jourdan *et al.* 1974). Blood pressure fell during weight loss, as has been found repeatedly (Chiang *et al.* 1969), but there was no correlation between the fall in blood pressure and the amount of weight loss. This raises again the unresolved issue whether the hypotensive effect of weight reduction is due to weight loss per se or simply to dietary change such as a decreased salt intake (Dahl, 1972; Weinsier *et al.* 1976). A significant fall in the mean total serum cholesterol level, as noted in our patients, does not invariably accompany weight loss since levels may remain unchanged despite massive weight reduction (Kempner *et al.* 1975) or even rise when carbohydrate-restricted diets are used (Young *et al.* 1953; Krehl *et al.* 1967; Rickman *et al.* 1974). Our finding that HDL-cholesterol levels changed variably with no significant change in the mean is in contrast to previous reports that levels tend to rise (Hulley *et al.* 1977) or to fall (Jeffery *et al.* 1978) with weight reduction.

Although Young *et al.* (1953) and Ritt *et al.* (1979) have calculated the intake of various vitamins and minerals among patients on energy-restricted diets, to our knowledge no one has assayed serum levels as a more direct and objective measure of nutrition status during long-term weight reduction. Assays of seven vitamins, β -carotene, and Fe, obtained among our patients who had been actively losing weight according to the prescribed dietary guidelines for up to 84 weeks, revealed that mean values were maintained well within normal limits. There were several instances in which abnormally low values were found, two of which were felt to be potentially significant. In one case a severely low vitamin C level was found after 16 weeks on the diet. This was thought to be a spurious value, however, since (1) the patient had no clinical manifestation of a deficiency state, (2) it could not be explained by the concomitant 7 d intake records, (3) it followed a high normal vitamin C level found 8 weeks previously. In a second instance serum Fe and transferrin saturation were subnormal in a woman after 8 weeks of dieting; however, the values were essentially unchanged from her baseline pretreatment levels. Despite energy restriction, there was a general trend among all the patients towards a rise in β -carotene and vitamin C levels, presumably reflecting their increased intake of fruits and vegetables. Contrary to the suggestion that 4200 kJ (1000 kcal)/d diets should be supplemented with vitamins (US Food and Nutrition Board, 1980), these values indicate that with careful attention to nutritional adequacy supplementation may not be necessary.

An unexpected finding of our study was that patients with juvenile-onset obesity lost more weight during treatment than patients with adult-onset obesity, and fared equally well during post-treatment follow-up. Age, sex, education, type of treatment, and length of treatment and follow-up of the two groups were similar, supporting the validity of these findings. However, the slightly greater starting weight of the juvenile-onset patients could have favoured greater weight loss for this group. Although not reaching statistical significance at $P < 0.05$, the correlation coefficients between age of onset of obesity and weight change also indicated that an early age of onset tended to be associated with greater weight loss both during and after treatment. These results are in direct conflict with previous reports showing that juvenile-onset obese patients are more recalcitrant to treatment (Young *et al.* 1955; Johnson & Drenick, 1977) or, at best, are equally as responsive as adult-onset patients (Ashwell, 1975; Stunkard, 1978). Of interest is the experience of Mullins (1958), who found early-onset obese persons to be traditionally difficult to treat except when they were 'highly intelligent'. To our knowledge, the educational level of these two groups has not been previously examined to ensure comparability of the juvenile- and adult-onset types.

It is possible that the relatively high, but similar, levels of education of our two populations make them respond in a fashion different from previous studies. Limited information from the literature regarding post-treatment weight maintenance of the two groups shows a similar (Currey *et al.* 1977) or poorer prognosis (Johnson & Drenick, 1977) for the juvenile-onset obese patient. Again, our findings would give a slight advantage to the early-onset group.

Several factors were examined as to their predictive value for weight loss and maintenance. Age of the patient bore no significant relationship to either, which concurs with previous reports (Cormier, 1972; Johnson & Drenick, 1977). A higher initial weight did predict a greater weight loss during treatment, as has generally been found (Gray & Kallenbach, 1939; Cormier, 1972; Murray, 1975) and, once lost, the reduced weight was as likely to be maintained as for those losing less weight. Educational level has been reported to be a useful prognostic indicator by one therapist (Mullins, 1958) but not useful by another (Cormier, 1972). Under the circumstances of the present study, education was highly correlated with greater success during the period of treatment but, interestingly, was also closely correlated with a greater tendency towards weight rebound. This paradoxical finding is potentially very important in terms of therapeutic approach, but its explanation is purely conjectural. Similarly, it was evident that patients who remained in treatment longer lost more weight, but they were also more likely to regain upon long-term follow-up, implying a dependence on the established therapeutic relationship. The five variables examined in this study (age, age of onset of obesity, initial weight, education and length of treatment) explain only 50% of the total outcome variance for weight loss during treatment and only 40% for post-treatment weight rebound. This supports the theory that a multiplicity of factors must impinge on the successful outcome of weight reduction, no less than on long-term weight control.

COMMENT

Weight loss can be achieved with any energy-restricted diet, regardless of its composition, and can thus be promoted as an effective dietary regimen. The end result, however, does not necessarily justify the means. Weight control should be considered as only one way in which a diet can improve health; i.e. the diet should be directed at over-all good health, one aspect of which is weight control. The study diet, averaging approximately 4200 kJ (1000 kcal), enabled patients to lose weight at an acceptable rate for relatively prolonged yet tolerable periods of time. During that time, various measures of health were maintained or improved while patients gradually developed favourable eating patterns. The follow-up results showing weight maintenance suggest that this was the case. The fact that most patients did not reach their ideal weight even over relatively long periods of time emphasizes the importance of adopting lifelong habits conducive to weight control rather than temporary measures for acute weight loss. Although the described dietary programme of time-energy displacement may not be the ideal approach for all obese patients, used in the setting of this study for relatively well-educated patients, it appears to be not only effective but safe for long-term use.

The authors gratefully thank Ms Susan Wood, Ms Deborah Sahn, Ms René Hyatt, Ms Colleen Mitchell and Ms Ruby Aldridge for their help in patient care and data collection, and Ms Janet Hails, Dr Carlos Krumdieck and Dr C. E. Butterworth, Jr for their assistance in preparation of the manuscript. They are also appreciative of the support of Dr Phillip Cornwell and his associates of the Nutrition Support Laboratory for running the vitamin assays. This study was supported in part by the National Institutes of Health, Core Clinical Nutrition Research Center Grant P01 CA 28103.

REFERENCES

- Adlersberg, D. & Mayer, M. E. (1949). *J. clin. Endocr.* **9**, 275. American Medical Association Council on Foods and Nutrition (1974). *Nutr. Rev.* **32**, Suppl. 15.
- Alpert, S. S. (1979). *Am. J. clin. Nutr.* **32**, 1710.
- American Medical Association Council on Foods and Nutrition (1974). *Nutr. Rev.* **32**, Suppl. 15.
- Ashwell, M. (1975). *Br. J. Nutr.* **34**, 201.
- Automatic Clinical Analyzer (1981). *Chemistry Instruction Manual*. Wilmington, Del.: Dupont Company.
- Baker, H. & Frank, O. (1968). *Clin. Vitaminology*, ch. 11. New York, N.Y.: Interscience.
- Bayoumi, R. A. & Rosalki, S. B. (1976). *Clin. Chem.* **22**, 327.
- Bierman, E. L. (1979). *Am. J. clin. Nutr.* **32**, 2712.
- Bistriani, B. R., Blackburn, G. L., Sherman, M. & Scrimshaw, N. S. (1975). *Surg. Gynec. Obstet.* **141**, 512.
- Bjorntorp, P. (1980). *Am. J. clin. Nutr.* **33**, 370.
- Bray, G. A. & Benfield, J. R. (1977). *Am. J. clin. Nutr.* **30**, 146.
- Build and Blood Pressure Study. Society of Actuaries, Chicago, 1959, Committee Report (1970). *Am. J. clin. Nutr.* **23**, 807.
- Buskirk, E. R., Thompson, R. H., Lutwak, L. & Whedon, G. D. (1963). *Ann. N.Y. Acad. Sci.* **110**, 918.
- Chiang, B. N., Perlman, L. V. & Epstein, F. H. (1969). *Circulation* **39**, 403.
- Cormier, A. (1972). *Can. J. Pub. Hlth* **63**, 327.
- Currey, H., Malcolm, R., Riddle, E. & Schachte, M. (1977). *J. Am. med. Ass.* **237**, 2829.
- Dahl, L. K. (1972). *Am. J. clin. Nutr.* **25**, 231.
- Feinstein, A. R. (1960). *J. Chron. Dis.* **11**, 349.
- Fellows, H. H. (1931). *Am. J. med. Sci.* **181**, 301.
- Garry, P. S., Pollock, J. D. & Owen, G. M. (1970). *Clin. Chem.* **16**, 766.
- Gray, H. & Kallenbach, D. E. (1939). *J. Am. Diet. Ass.* **15**, 239.
- Haber, G. B., Heaton, K. W., Murphy, D. & Burroughs, L. F. (1977). *Lancet* **ii**, 679.
- Halmi, K. A., Stunkard, A. J. & Mason, E. E. (1980). *Am. J. clin. Nutr.* **33**, 446.
- Heaton, K. W. (1973). *Lancet* **ii**, 1418.
- Herbert, V. (1966). *J. clin. Path.* **19**, 12.
- Hulley, S. B., Cohen, R. & Widdowson, G. (1977). *J. Am. med. Ass.* **21**, 2269.
- Innes, J. A., Campbell, I. W., Campbell, C. J., Needle, A. L. & Munro, J. F. (1974). *Br. med. J.* **11**, 356.
- Jeffery, R. W., Thompson, P. D. & Wing, R. R. (1978). *Behav. Res. Ther.* **16**, 363.
- Jenkins, D. J. A., Reynolds, D., Leeds, A. R., Waller, A. L. & Cummings, J. H. (1979). *Am. J. clin. Nutr.* **32**, 2430.
- Johnson, D. & Drenick, E. J. (1977). *Archs intern. Med.* **137**, 1381.
- Jourdan, M., Margen, S. & Bradfield, R. B. (1974). *Am. J. clin. Nutr.* **27**, 3.
- Kempner, W., Newborg, B. C., Peschel, R. L. & Skyler, J. S. (1975). *Archs intern. Med.* **135**, 1575.
- Krehl, W. A., Lopez-S, A., Good, E. I. & Hodges, R. E. (1967). *Am. J. clin. Nutr.* **20**, 139.
- Lau, K. S., Gottlieb, C., Wasserman, L. R. & Herbert, V. (1965). *Blood* **26**, 202.
- Leon, A. S., Conrad, J., Hunninghake, D. B. & Serfas, R. (1979). *Am. J. clin. Nutr.* **32**, 1776.
- Macrae, T. F., Hutchinson, J. C. D., Irwin, J. O., Bacon, J. S. D. & McDougall, E. I. (1942). *J. Hyg., Camb.* **42**, 423.
- Mullins, A. G. (1958). *Archs Dis. Childh.* **33**, 307.
- Murray, D. C. (1975). *Psychol. Rep.* **37**, 243.
- Penick, S. B., Filion, R., Fox, S. & Stunkard, A. J. (1971). *Psychosomatic Med.* **33**, 49.
- Ressler, C. (1959). *N.Y. State J. Med.* **59**, 615.
- Rickman, F., Mitchell, N., Dingman, J. & Dalen, J. E. (1974). *J. Am. med. Ass.* **228**, 54.
- Ritt, R. S., Jordan, H. A. & Levitz, L. S. (1979). *J. Am. diet. Ass.* **74**, 325.
- Select Committee on Nutrition and Human Needs, United States Senate (1977). *Dietary Goals for the United States*, 2nd ed. Washington, D.C.: US Government Printing Office.
- Shearer, R. S. (1976). *Curr. Ther. Res.* **19**, 433.
- Southgate, D. A. T. (1973). *Proc. Nutr. Soc.* **32**, 131.
- Spiller, G. A. & Kay, R. M. (1979). *Am. J. clin. Nutr.* **32**, 2102.
- Stunkard, A. J. (1978). *Int. J. Obesity* **2**, 237.
- Stunkard, A. J. & Albaum, J. M. (1981). *Am. J. clin. Nutr.* (In the Press.)
- Stunkard, A. J., Craighead, L. W. & O'Brien, R. (1980). *Lancet* **ii**, 1045.
- Stunkard, A., Levine, H. & Fox, S. (1970). *Archs intern. Med.* **125**, 1067.
- Stunkard, A. & McLaren-Hume, M. (1959). *Archs intern. Med.* **103**, 79.
- Stunkard, A. J. & Penick, S. B. (1979). *Archs gen. Psych.* **36**, 801.
- Trowell, H. (1976). *Am. J. clin. Nutr.* **29**, 417.
- (US) Food and Nutrition Board (1980). Recommended Dietary Allowances, 9th ed. Washington, D.C.: National Academy of Sciences—National Research Council.
- Weinsier, R. L., Fuchs, R. J., Kay, T. D., Triebwasser, J. H. & Lancaster, M. C. (1976). *Am. J. Med.* **61**, 815.

- Young, C. M. (1973). In *Obesity in Perspective*, p. 361 [G. A. Gray, editor]. Washington, D.C.: US Government Printing Office.
- Young, C. M., Moore, N. S., Berresford, K., Einset, B. M. & Waldner, B. G. (1955). *J. Am. Diet. Ass.* **31**, 1111.
- Young, C. M., Ringler, I. & Greer, B. J. (1953). *J. Am. diet. Ass.* **29**, 890.
- Zannoni, V., Lynch, M., Goldstein, S. & Sato, P. (1974). *Biochem. Med.* **11**, 41.