6-1966

Influence of Caloric Distribution on Weight Loss

Georgia E. Hodgkin

LOMA LINDA UNIVERSITY

Graduate School

INFLUENCE OF CALORIC DISTRIBUTION ON WEIGHT LOSS

by

Georgia E. Hodgkin

A Thesis in Partial Fulfillment
of the Requirements for the Degree
Master of Science in the Field of Nutrition

June, 1966

Each person whose signature appears below certifies that he has read this thesis and that in his opinion it is adequate, in scope and quality, as a thesis for the degree of Master of Science.

Raymond Crawford, Chairman
Raymond Crawford, M. D.
Associate Professor
School of Medicine

Mary P. Byers
Mary Byers, M.S., Chairman
Department of Home Economics
La Sierra College

Martha Miller
Martha Miller, Ed.M.
Assistant Professor
School of Nutrition and Dietetics

Elizabeth Wenz
Elizabeth Wenz
Instructor
School of Nutrition and Dietetics

ACKNOWLEDGMENTS

The writer wishes to express her appreciation to:

Dr. John E. Peterson, Associate Dean of the School of Medicine, for his guidance in setting up the research problem and his financial assistance in making the study possible;

Dr. Raymond Crawford, Associate Professor of the School of Medicine, for his expert guidance in the writing of this paper;

Drs. Eckman and Wilson of the Riverside County Hospital and San Bernardino County Hospital respectively for their assistance in securing the subjects of the research;

The dietary and nursing staffs of the two county hospitals for their excellent cooperation in the details of the research;

Miss Martha Miller, Assistant Professor of the School of Nutrition and Dietetics, for her helpful suggestions in writing this paper;

Mrs. Freda Knauft for typing and editing the final form of this paper;

Dr. John E. Hodgkin, my husband, for his assistance in evaluating the findings of the research and encouragement in this project.

TABLE OF CONTENTS

LIST OF FIGURES

CHAPTER I

STATEMENT OF THE PROBLEM

Obesity is one of the nation's leading health problems (50,57). At least 30 per cent of the male and 40 per cent of the female adult population over 40 years of age are 20 per cent above their desirable weight (45).

With the widespread prevalence of obesity, questions arise concerning the mechanisms whereby adipose tissue stores are increased or decreased. The question that this study attempts to answer has to do with the timing of meals. Does it make a difference whether all the calories in a day's ration are consumed in three meals or six meals? Does it affect the rate of weight loss to eat three meals divided into two small and one large feeding or to eat six meals of equal caloric value, both patterns being isocaloric?

The rate of weight loss on each program will be the basic factor under consideration.

CHAPTER II

REVIEW OF THE LITERATURE

A review of the literature on obesity will briefly cover the diagnosis, etiology, complications, management and treatment, and prognosis of the obese.

DIAGNOSIS

Obesity is often most simply defined in relation to standard height-weight tables where the weight of the individual in question is compared to the standard (35,39). These tables may not always be satisfactory for a number of reasons. The figures have not been collected under standard conditions. "Normal" people exhibit a wide variation in body types which has resulted in the divisions of small, medium, and large frames. Many athletes would be rated obese by the tables. The increased muscle mass of some athletes results in a total body weight considerably above that given in the height-weight tables, yet this is not obesity as there is little adipose tissue present (39). The value listed on the table is the average for a population which does not necessarily mean normal or optimum; this can be illustrated by the increased weight with advancing years that may or may not be normal. The latest

height-weight tables have overcome some of these difficulties by listing a recommended weight rather than an average weight (39).

The problem still remains that increased weight may not be fatness. How then can excess adipose tissue or fatness be determined? Several methods can be used which include: 1. determine specific gravity of the whole body and calculate the amount of fat by formula; 2. estimate total body water and calculate the amount of fat by formula; 3. estimate body fat from potassium-40 content; 4. measure the thickness of certain skinfolds (2,21,30, 34,39,51).

Specific gravity is determined by weighing the subject without clothes totally submerged in water and correcting this weight to exclude the weight of air in the lungs. This method cannot be used with large groups of people and is limited to research work (34,39).

Body water is determined through a dilution principle. A test subject is placed in an air-tight chamber of known capacity, and indicator gas is injected into the chamber, a test sample of gas is then withdrawn and analyzed for its concentration. This test uses very sensitive equipment and is also limited to research work (34).

Body fat can be determined by measurement of the natural gamma ray emission of potassium-40 in the body. There is a correlation between body potassium, lean body

weight and fat. By the use of formula fat can be computed. This method is limited to research work (2).

Skinfold thickness is determined by pinching up the fat in several selected areas. This is then measured by a standard skinfold thickness caliper. The areas most commonly used are the umbilical, triceps and subscapular on the left side (21,39). This method is a simple measurement that can be completed readily with a high degree of validity (39).

There are many possible methods of diagnosing obesity. A review of the literature suggests that the most frequently used methods are inspection and palpation of the skinfold thickness (35,39).

In the diagnosis of obesity, the presence of the excess fat is quite obvious; and possibly a more important factor would be why excess adiposity exists in the given individual. An evaluation of the development of obesity over the years with a description of previous attempts at treatment and their results along with the patient's explanation of the causes of obesity will prove helpful in understanding the patient's psychological approach to obesity. A psychiatric evaluation may prove beneficial (1,35).

Laboratory tests often used are skull x-rays to eliminate pituitary disease, serum protein-bound iodine to eliminate thyroid problems, glucose tolerance tests to

evaluate hypo or hyperglycemia, water and salt balance records to record fluid retention, and hormone studies to point out imbalances that could contribute to obesity (35).

ETIOLOGY

The word "obese" merely means "fat", as distinct from "thin". The incidence of obesity varies considerably and in direct relation to the supply of food (39). Yet within a given area some individuals will become obese while others will remain thin. What then is the cause of obesity? Several theories have been advanced as probable explanations.

In essence, obesity is the result of a caloric intake in excess of caloric expenditure (4,15,39,41). The primary question with the etiology of obesity asks why there is a positive energy balance.

Hypothalamus

The olfactory, ocular and taste senses have considerable effect on the rate of food ingestion or appetite. Less apparent is the role of the hypothalamus in controlling appetite. Glucostatic mechanisms have been hypothesized to trigger the hunger sensation when blood glucose levels reach a certain low (38). Electrical stimulation of the lateral nuclei of the hypothalamus leads to overeating in animals (41). Bilateral lesions of the hypothalamus in normal animals will lead to obesity (15,54). Others have

postulated that metabolic disturbances may affect factors relating to the regulation of the hypothalamus (39). None of the above factors has been shown to be the cause of obesity in man (15).

Endocrine factors

Within the realm of endocrine factors postulated as contributing to obesity are malfunctions of the pituitary gland, the thyroid gland, the adrenal cortex, and other glands affected by these, such as the gonads (15,41,43).

Looking at the malfunctions per se, clinicians have observed inconsistencies regarding the status of total body weight. With the severest types of hypothyroidism, obesity may not develop. Some individuals will adjust their caloric intake to a lower level to correspond to their low energy output (15). Is not the obesity then a matter of energy balance again?

An excessive secreting of insulin by the pancreatic islets has been considered as a cause for obesity. An excessive supply of insulin results in hypoglycemia that increases appetite. The hypoglycemia that causes excessive ingestion of food must be prolonged and severe. Such a situation cannot be demonstrated in the majority of obese individuals (15).

A malfunction of the adrenal cortex known as Cushing's syndrome results in a characteristic type of distribution of body fat. Much adipose tissue will be

found about the face, neck, and shoulders. However the total weight of the individual may be below average (15). Thus it would appear that Cushing's syndrome has more to do with the distribution of body fat than the accumulation of it.

Heredity

Obesity itself cannot be inherited, but possibly a tendency toward it is inherited. Studies have shown that "fat people" tend to come from "fat families" (39). However, is this heredity or environment? The patterns of eating of families are deeply ingrained in their feelings of satisfaction or dissatisfaction regarding food, in the availability of food, and in the standards of beauty, hospitality, and cultural norms of the group in which they live (46).

Physical activity

Recent investigations with mice have shown that the obese mice are far less active than their non-obese litter mates (36). Possibly a low level of physical activity can be a cause of human obesity (8,17).

A study was done to compare the physical activity and attitudes toward physical activity of obese and non-obese adults. It was shown that obese women were less active than non-obese women, but a significant difference was not shown between men. Obese women were more passive than non-obese women, but men did not show this difference. It was concluded that decreased activity may play a part

in the obesity of women, but it is much less important in men (8).

In the last analysis, what then is the cause of obesity? It is accepted that an overall intake of energy in excess of body output of energy results in obesity. However, Conn and Neuburgh concluded no one factor can be pointed to as the cause of this excessive ingestion of food (15,43).

COMPLICATIONS

Obesity is associated with an increased mortality rate and decreased life expectancy. With persons at the age of 45 to 50 years, the death rate is 8 per cent above the average with 10 pounds overweight; 18 per cent with 20 pounds; 28 per cent with 30 pounds; 56 per cent with 50 pounds. Cardiovascular disease is fatal 60 per cent more frequently with obese persons than with those of normal weight (35).

There is an increased incidence among the obese to such diseases as gout, diabetes mellitus, hypertension, degenerative arthritis of the back and knees, orthopedic disorders, postoperative thromboembolism, venous varicosities and leg ulcers. There is a higher death rate among the obese with such diseases as diabetes, nephritis, pneumonia, cirrhosis, biliary disease, appendicitis, and postoperative complications. There even seems to be a higher death rate from accidents with the obese. (19,35,50,57)

The likelihood of complications with pregnancy, and childbearing are increased with obesity (16,35). Menstrual disorders may occur with obesity (35).

The psychologic effect of the pressures of society on the obese child should not be underestimated. This is particularly true with the obese girl (37).

Considering the above, obesity becomes a serious health hazard.

MANAGEMENT AND TREATMENT

The treatment of obesity must be considered a long-term project. As there are variations in the cause of each person's obesity, so no one treatment will be suitable to all. During the course of treatment, plateaus will be reached at which time there will be no apparent weight loss or even a weight gain may result (18,29,35).

The primary objective in the treatment of obesity is to reduce the caloric intake below the requirement of the individual (7,18,29,35,50). A caloric deficit forces the body to metabolize endogenous materials for the energy to maintain vital functions. At first the glycogen reserves are used, followed by small amounts of protein from the body's metabolic pool and of fat from the fat depots (18). To determine the approximate daily weight loss of the individual, the following formula is used (7):

$$\frac{\text{Approximate Daily Caloric Requirement} - \text{Number of Calories in Diet}}{4000} = \text{Weight loss in pounds per day}$$

Although exercise results in an increased caloric expenditure, it requires extreme physical activity to effect a significant weight loss. A deficit of 3500 Calories is required to lose one pound of body fat (50). Playing 18 holes of golf, for instance, will raise the caloric requirement by only 100 to 150 Calories (7). Walking one mile uses 92 Calories which can be replaced by only one slice of bread or 1.5 ounces of ice cream (18). Exercise plays an important role in the weight reduction program in that it improves muscle tone and circulation, strengthens bone and improves digestion and intestinal elimination (18,28).

In order for the reducing diet to be effective, it must not only be below the individual's caloric requirement, it must also be such that the patient can follow it. It should include familiar foods that are readily available. It should be composed of a variety of foods so that it will not become monotonous and with a few additions can continue to be followed after the weight reduction has been accomplished. It should also include foods that will check the appetite and provide satiety (50).

The number of calories per day must be prescribed by a physician and is based on the patient's age, occupation, temperament, and the urgency of the need for weight reduction (7). The usual diets range from 800 to 1600 Calories (7,18,35,50).

Drugs are widely prescribed for weight reduction with variable degrees of effectiveness (57). The principal drugs used are amphetamines, diethylproprions, and thyroid preparations (7,16,28,57). The amphetamines and diethylproprions function to suppress the appetite and give a sense of well being.

Thyroid preparations are said to speed up metabolism. However, the low basal metabolic rate of the obese is due to the fact that it is a measure of oxygen consumption in terms of body surface area. The apparently low basal metabolic rate results then from the increased surface area and the large amount of relatively poor oxygen using tissue, namely adipose tissue (7). Thus the obese patient is in actuality rarely hypothyroid. The prolonged administration of thyroid may depress the patient's normal thyroid secretion (7). Goodman and Gilman point out that the symptoms of hyperthyroidism may result from the use of exogenous thyroid (25). Therefore, the thyroid preparations should be prescribed with discretion (7,25).

An important phase in any weight reduction regimen is an understanding of the mechanisms leading to the patient's obesity. Overeating may satisfy many drives in the individual. They may eat when they have nothing to do, when they are under a strain, or feel that they are not appreciated (7,16,57). Treatment should be discontinued if the effects of the treatment are worse than the obesity.

Actual personality changes can be seen with some patients whose preponderance has been their front with which to face the world (1,57). Psychotherapy may be necessary before a successful program of weight reduction can be accomplished (18).

Motivation is the key to any successful weight reduction program (27). Many various methods have been tried in an attempt to enhance cooperation from patients. Halpern has found even candy-coated vitamin and mineral pills have proved effective, probably because of the placebo effect (28).

Manipulation of the caloric increments of diets have been suggested by various investigators for the correction of obesity. Gordon uses a diet of 100 grams protein, 80 grams fat, and 50 grams carbohydrate divided into six meals per day in the treatment of obesity. He suggests six feedings minimizes lipogenesis and decreases the efficiency of food utilization. Six feedings also decrease the feeling of hunger (26).

Gold also suggests a high protein diet for weight control. He begins his program of weight reduction with two weeks of a low calorie liquid diet. To this are added small protein feedings resulting in a low fat, low carbohydrate diet. The calories are not counted, but the diet is low calorie if only the prescribed foods are eaten (24).

Dr. Taller's "Calories Don't Count" theory has become famous in certain circles. The basis of his diet is

the ingestion of as much food as is desired provided a minimum of highly unsaturated safflower oil is consumed (51). A high fat diet for reducing may be beneficial from the standpoint that it is a diet from which carbohydrate has been nearly eliminated, thus reducing total caloric intake (31). There is an absence of hunger accompanying a high fat diet (31). However, Nutrition Reviews points out the lack of evidence for Taller's theory as a sound diet for weight reduction (31). The Pennington diet allows the patient to eat as much as he desires also provided he consumes fat meat three times per day and adds fat if the cut of meat is too lean (44). As with Taller's diet, the patient is so soon satisfied by the fats and oils that the resultant calorie intake is small. Pennington also prohibits the use of sodium which encourages a decrease in caloric intake (44).

A diet of fat (60 per cent of the total calories) and protein (40 per cent of the total calories) totaling 1500 Calories was fed two men and two women for three days. This diet is similar to the metabolic mixture furnished by starvation. The same phenomena as seen in starvation were noted with this diet--weight loss, electrolyte diuresis, nitrogen loss and fatigue. The authors conclude that the ketosis of starvation represents carbohydrate deficiency. They also conclude that protein used as an energy source

is satisfactory at a "rate limited" level, and in order to achieve nitrogen balance one must feed either carbohydrate with adequate protein or excessively large quantities of protein alone (48).

The use of thyroglobulin in conjunction with a low calorie diet resulted in a greater decrease of lean body mass than the decrease in loss of body fat. The low calorie diet alone leads to a smaller weight loss where the ratio of fat is one-third greater than the loss of lean body mass. When the regimen included physical exercise, practically no loss of lean body mass occurred and the loss of adipose tissue was thirty per cent greater than the loss of body fat attained by the low-calorie diet alone (47).

Protein cannot be stored dry, but retains with it three or more parts of water, while fat can be stored in an almost pure state. Thus the loss of one pound of body protein will result in a loss of four pounds of body weight. Total starvation brings about a loss of protein tissue to supply carbohydrate by way of gluconeogenesis. This explains the drastic weight loss of totally fasted individuals. It also explains the rapid weight gain on refeeding of even a moderate calorie diet (56).

A study was done in which subjects were maintained for many weeks on a constant calorie intake. The composition of the diets was varied at intervals. The fat content varied from 12 to 83 per cent, protein from 14 to 36 per cent,

and carbohydrate from 3 to 64 per cent of the total calories. The rate of weight loss in any one patient was essentially constant throughout the entire study. The significant factor responsible for weight loss is the reduction of calories, regardless of the composition of the diet (33).

Protein in the diet provides satiety in that the emptying time of the stomach is decreased as compared to the rate of carbohydrate (16). However, it is doubtful that the protein in a sub-calorie diet of 900 Calories or less will be used for tissue building or the specific functions of protein. The energy yield of a 900 Calorie diet is too low to permit protein to be used for functions other than energy (57).

The number of meals per day has been reported to have varying effects. Fabry found that overweight, hypercholesterolemia, and diminished glucose tolerance tend to increase as the frequency of the meals decreases (22). On the other hand Seaton reports greater success with sub-calorie diets when only two meals per day are given (49). Both of these studies were done with out-patients enhancing the probability of less adherence to the diet than if hospitalization were maintained.

The frequency of food ingestion and its effects have been investigated in experimental animals. Cohn and associates compared various parameters in rats allowed to eat ad libitum and those that were force fed. The force

fed rats were fed by a stomach tube two times each day an amount of food equal to that consumed by the ad libitum group. After three weeks, the animals fed by stomach tube (meal eaters) contained more body fat and less protein and water than did their ad libitum (nibbling) counterparts although their total body weights were essentially the same; there was one gram difference in the mean weights of the two groups. These results led the authors to conclude that the animal consuming its calories in meals makes a more economical use of those calories than the animal that eats ad libitum or nibbles. This economy of calorie expenditure results in a greater deposition of fat (11).

In another set of experiments Cohn and his associates have studied the effect of meal eating on the production of atherosclerosis in chickens. The results showed a seven times greater incidence of coronary artery disease and double the level of serum cholesterol in meal eating chickens over those allowed to eat ad libitum (nibble)(13).

The rate of clearing of the atherosclerotic lesions was subsequently examined in another group of chickens. It was found that the meal eaters showed a decreased ability to clear the arteries of the atherosclerotic lesions as compared to the nibblers, and the meal eaters serum cholesterol levels remained elevated (14).

The severity of experimental diabetes mellitus is increased by meal eating in the rat (10). The authors hypothesize that meal eating may result in changes in intermediary enzymatic pathways of metabolism, as evidence by decreased thyroid function in the meal eaters and increased activity in glucose-6-phosphate dehydrogenase (12).

Cohn and associates conclude from their numerous experiments that meal eating in the experimental animal may be associated with obesity, diabetes mellitus, elevated serum cholesterol levels and decreased clearing of atherosclerotic lesions. On this basis they suggest man should return to the nibbling pattern of food ingestion that he manifests at birth (9).

Hollifield studied adipose tissue and liver glycogen in rats which were allowed to eat as much food as they were able to ingest in 2 hours out of each 24 for periods of 1 to 7 days. There was increased lipid synthesis in the rats allowed the short daily feeding period (32).

Bortz et al studied the effect of feeding frequency on weight loss in 6 obese females. A 600 Calorie liquid formula diet was followed by these patients in a metabolic ward for 7 to 8 weeks. During this time the formula was divided into one, three, or nine feedings per day for intervals. The rate of weight loss was seen to be the same regardless of the number of feedings per day. They concluded that weight loss is effected by caloric restriction without regard for the pattern of ingestion of those calories (6).

Formula diets as a means of weight reduction are of recent popularity. Some 80 to 90 formula mixtures are on the market. These have not proven to be the panacea for obesity (42).

A review of the literature indicates the cure for obesity will require re-education, understanding, guidance, support, persistence, and careful tailoring of programs to meet the multiple problems each overweight individual presents (42).

PROGNOSIS

In no malady is the prescription so simple and the possibility of cure so high as in obesity (29). The prescription is simply that of maintaining a caloric deficit in each day's meals. However, it will be noted that this is a very unpleasant circumstance for the human organism. It requires courage and self-discipline of the highest order to remain on a program of caloric restriction for any length of time (29).

When the criterion for reporting success in weight reduction is listed as a loss of 20 to 40 pounds per patient, the reports of the last 30 years show remarkably similar results. The subjects of the reports were grossly overweight, yet only 25 per cent were able to lose 20 pounds and only 5 per cent were able to lose 40 pounds (52). The maintenance of weight loss is no more successful than

the initial weight loss. The majority of patients regain the majority of pounds lost (52,58).

A higher percentage of success might be obtained in the treatment of obesity by greater attention to the underlying factors of the individual patient's obesity. If overeating has provided essential emotional and psychological satisfactions, these must be satisfied by other sources before the dietary program can be successful. The physician's patience and understanding are important determining factors in the success of the program (23,29).

CHAPTER III

METHODS USED

Five hospitalized obese adults were placed on a reducing diet of 1200 Calories. The patients were chosen from the long term care units of two nearby county hospitals, San Bernardino County Hospital and Riverside County Hospital. The bases on which the patients were chosen are the following:

1. Obesity. The degree of overweight was not a prime factor in choice.
2. Expected hospital stay of eight weeks.
3. Probability of cooperation. Each patient was contacted to explain the program to him and to get his verbal consent to participate in the study.

For about a four-week period the day's ration was divided into three meals of 200 Calories each for breakfast and lunch and 800 Calories for the evening meal. Such a program simulates the typical working American's dietary pattern of little or no breakfast, a light lunch, and a large evening meal (3,20).

For another approximate four-week period the 1200 Calories were divided into six isocaloric meals. This represents the "nibbling" pattern which is characterized by small frequent feedings (9). The licensed vocational nurse in charge of each patient recorded the amount of

food that the patient left on the tray following each meal. A visual estimation of the amount of each serving left on the tray was recorded on a form left at the patient's bedside.

Laboratory analyses of serum cholesterol, two-hour post prandial blood sugar, blood urea nitrogen, hemoglobin and hematocrit were made at weekly intervals. The two-hour post prandial blood sugar was not preceeded by the customary 100 gram carbohydrate meal, but rather the blood sample was taken at 10:00 a.m. following the 8:00 a.m. breakfast allowed on the reducing regimen. Total body weight was recorded at regular intervals.

Routine hospital fare for reducing regimens was used for these diets. The exchange system was followed to determine the portion size of the foods used. The carbohydrate, protein, fat ratio were essentially the same with both patterns. On three meals, the diet contained 138 grams carbohydrate, 57 grams protein and 45 grams fat. With six meals per day, there were 159 grams carbohydrate, 60 grams protein and 40 grams fat.

The following are sample menus for each dietary pattern:

3 Meals Per Day

Breakfast--1/2 cup wheat hearts
1 cup non-fat milk
2 tablespoons light cream

Dinner------Fat-free broth
1/2 cup mashed potato
1/2 cup fat-free beets
Celery sticks
1 teaspoon margarine
1 cup non-fat milk

Supper------3 ounces meat balls with tomato sauce
1 cup fat-free noodles
Tossed green salad with zero dressing
1 slice whole wheat bread
2 teaspoons margarine
1 large apple (160 grams)
1 cup whole milk

6 Meals Per Day

Breakfast---1/2 cup farina
2 medium water pack apricots
1/2 cup non-fat milk
2 tablespoons light cream

10:00 a.m.--2 graham crackers
1 teaspoon margarine
1 cup non-fat milk

Dinner------Fat-free broth
2 ounces baked chicken
1/2 cup mashed potato
Fat-free baked banana squash
Tossed green salad with zero dressing
Tea

3:00 p.m.---1 banana
1 slice whole wheat bread
1 teaspoon margarine

Supper------2 ounces roast beef
Fat-free green beans
Sliced tomato
1/2 slice whole wheat bread
80 grams water pack pineapple
Coffee

Bedtime-----3 soda crackers
1 teaspoon margarine
1 cup non-fat milk

CHAPTER IV

RESULTS

The letters A, R, L, Z, and P will be assigned to the patients as a means of identifying each of them.

Patient R began the program on six meals per day and lost 5.5 pounds during the four weeks. On three meals per day she lost 3.5 pounds. Her cholesterol dropped from 272 milligrams per cent to 170 milligrams per cent on six meals per day. On three meals it showed a slight elevation from 167 milligrams per cent to 176 milligrams per cent. The mean Caloric intake on six meals was 1182.1 Calories with a standard deviation of 44.7; on three meals the mean was 1198 with a standard deviation of 38.98.

Patient A started the program on three meals per day and lost 12.0 pounds during the first four weeks. The second four weeks he consumed six meals per day and lost 10.0 pounds. The cholesterol ranged from 163 milligrams per cent to 153 milligrams per cent on three meals per day and 153 milligrams per cent to 169 milligrams per cent on six meals per day. The mean caloric intake on three meals per day was 1155.8 Calories with a standard deviation of 81.7; on six meals the mean was 1159.7 with a standard deviation of 51.8.

Patient L began the program on six meals per day and showed a net gain of 0.5 pounds. On three meals she lost 13.5 pounds. The cholesterol dropped from 220 milligrams per cent to 190 milligrams per cent on six meals, while on three meals it dropped from 190 milligrams per cent to 105 milligrams per cent. The mean caloric intake on six meals was 1195 Calories with a standard deviation of 43.07; on three meals the mean was 1200 with a standard deviation of 0.

Patient Z began the program on three meals per day and showed a net gain of one pound during the first four weeks. On three meals per day her cholesterol rose from 280 milligrams per cent to 300 milligrams per cent. On six meals per day the cholesterol dropped from 290 milligrams per cent to 220 milligrams per cent. The mean caloric intake on three meals per day was 1093 Calories with a standard deviation of 134.2; on six meals per day the mean was 1144 Calories with a standard deviation of 143.2.

Patient P started the program on three meals per day and showed a loss of 10.0 pounds. On six meals she lost 4.0 pounds. The cholesterol was 196 milligrams per cent at the beginning of the four weeks and 195 milligrams per cent at the end of the four weeks on three meals per day. On six meals per day it dropped from 191 milligrams per cent to 170 milligrams per cent. The mean caloric intake on three meals per day was 941.9 Calories with a standard

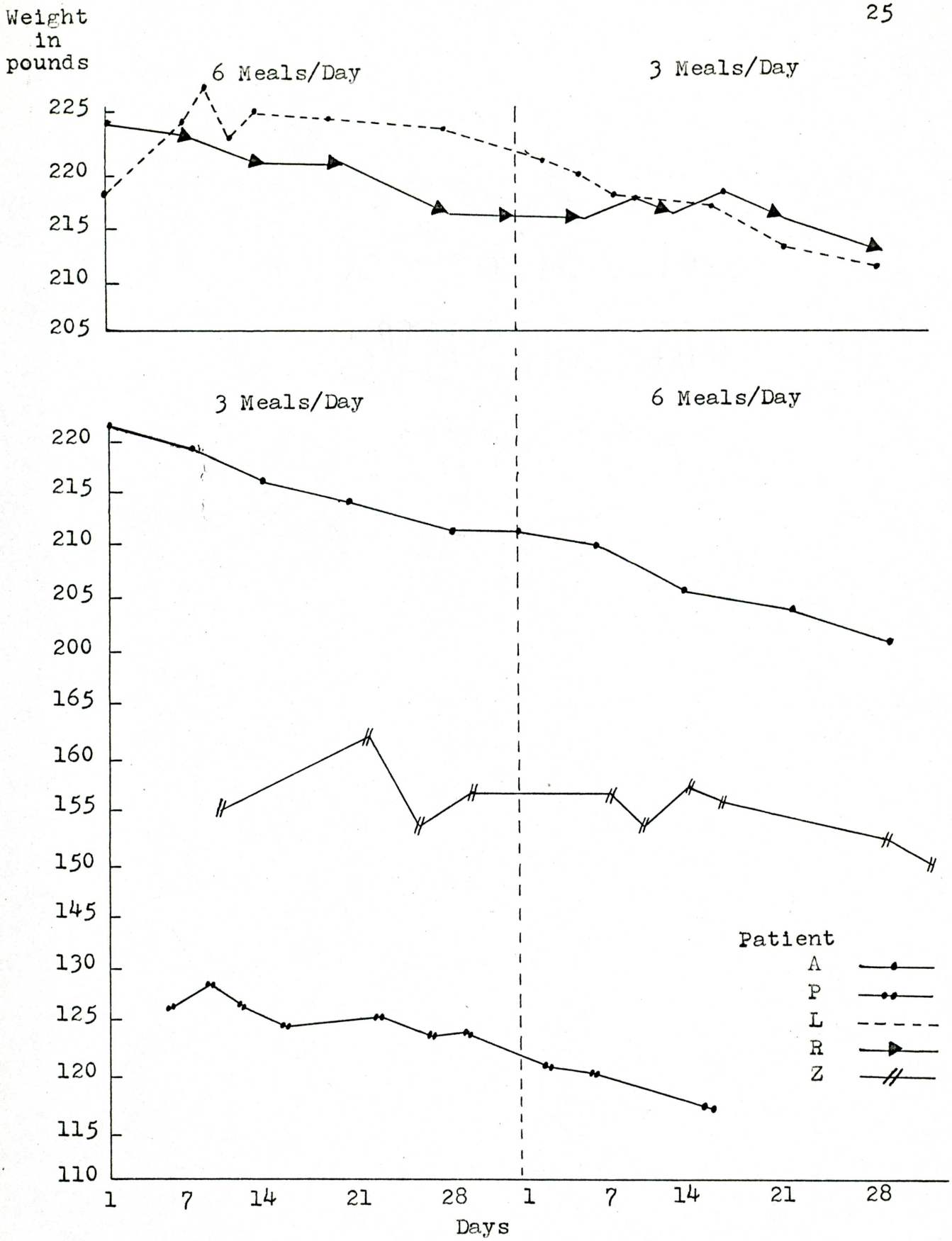

FIGURE 1

WEIGHT

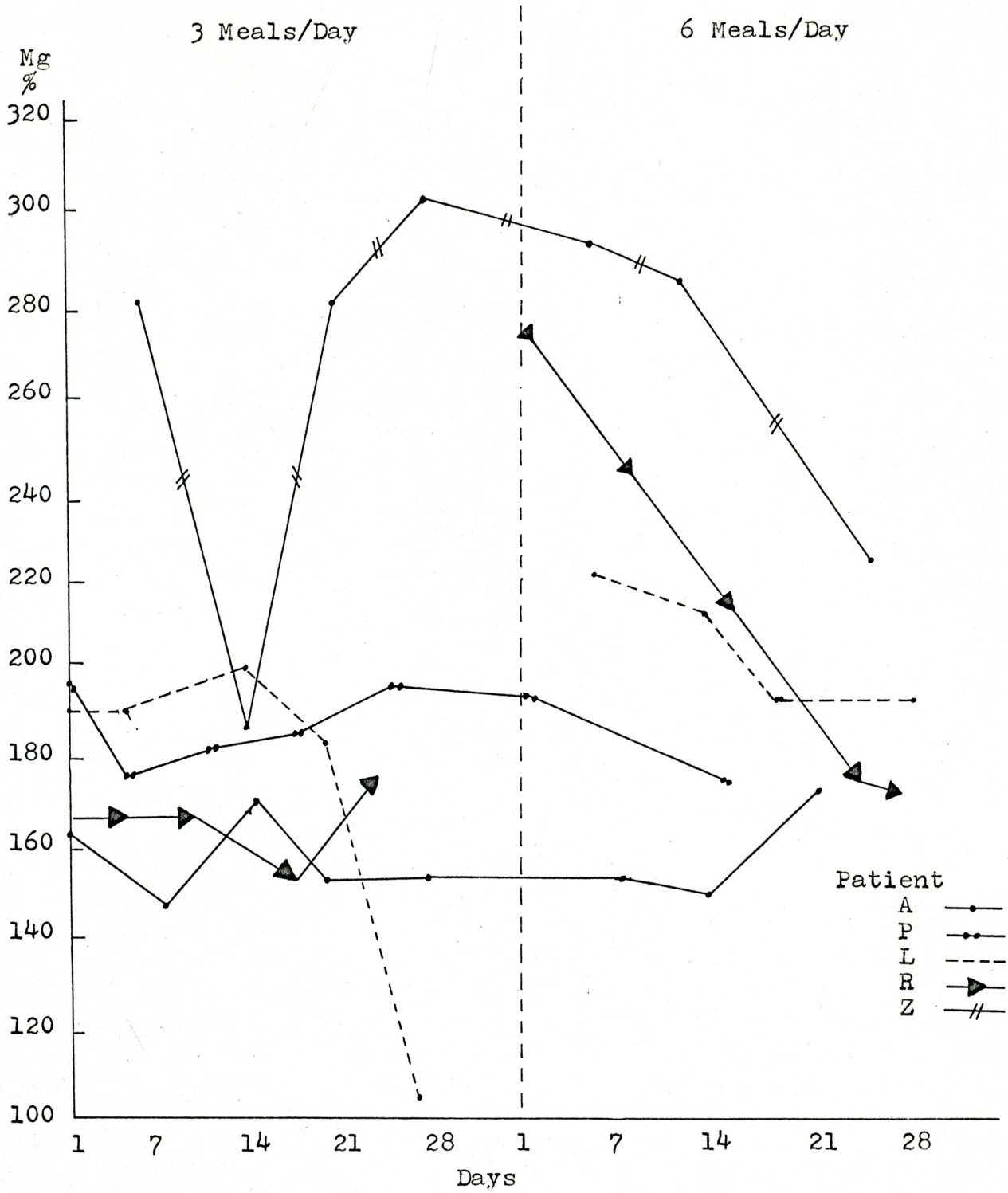

FIGURE 2

SERUM CHOLESTEROL

deviation of 197.5; on six meals per day the mean was 1076 Calories with a standard deviation of 84.38.

The mean weight loss of all patients on six meals per day was 5.0 pounds. The mean weight loss of all patients on three meals per day was 7.8 pounds. A t test at the 5 per cent significance level showed no significant difference in the two methods of treatment on the rate of weight loss.

The mean drop in cholesterol of all patients on six meals per day was 41 milligrams per cent. The mean drop in cholesterol of all patients on three meals per day was 13 milligrams per cent. A t test at the 5 per cent level of significance showed no significant difference in the slopes of the regression analyses of the two dietary patterns effecting the drop in serum cholesterol.

The total change in the two-hour post prandial blood sugar levels during the eight weeks ranged from a gain of 8 milligrams per cent to a decrease of 10 milligrams per cent with a mean of .25 milligrams per cent. One patient was diabetic at the onset of the experimental period with a blood sugar of 456 milligrams per cent. This dropped to 173 milligrams per cent after two weeks on the reducing diet, rose to a high of 214 milligrams per cent and fell to 154 milligrams per cent by the end of the eight weeks. This person was not included in computing the mean change of the two-hour post prandial blood sugar levels.

The change in the hematocrit during the eight week

experimental period ranged from plus seven to minus six per cent with a mean of one. The hemoglobin changes ranged from plus 2.2 to minus 2.3 grams per cent with a mean change of 0.4 grams per cent. The blood urea nitrogen changes ranged from minus three to plus two milligrams per cent with a mean change of minus 1.4 milligrams per cent.

The inclusion of an individual with elevated blood glucose levels in a study concerned primarily with changes in total body weight might be questioned. Diabetic patients are known to display symptoms of polyuria with blood glucose levels above normal (5). Excretion of excessive volumes of urine would decrease body weight. Thus 24 hour urine collections were kept with total volume, acetone, and glucose recorded daily on the diabetic patient. During the eight week experimental period, the urine volume ranged from 425 cubic centimeters to 1600 cubic centimeters which is well within the range of 600 to 2,500 milliliters encountered in a representative population (55).

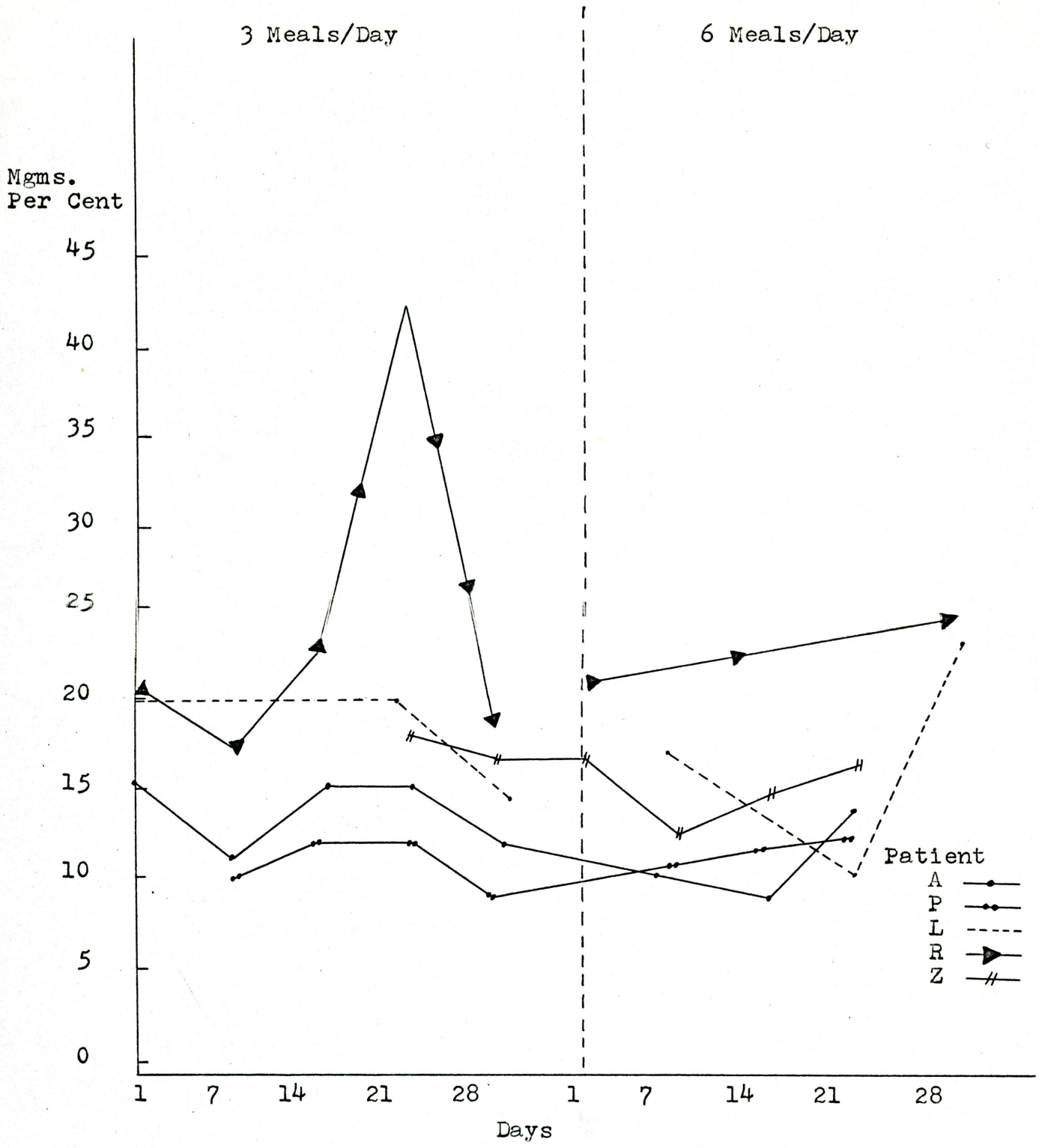

FIGURE 3

BLOOD UREA NITROGEN

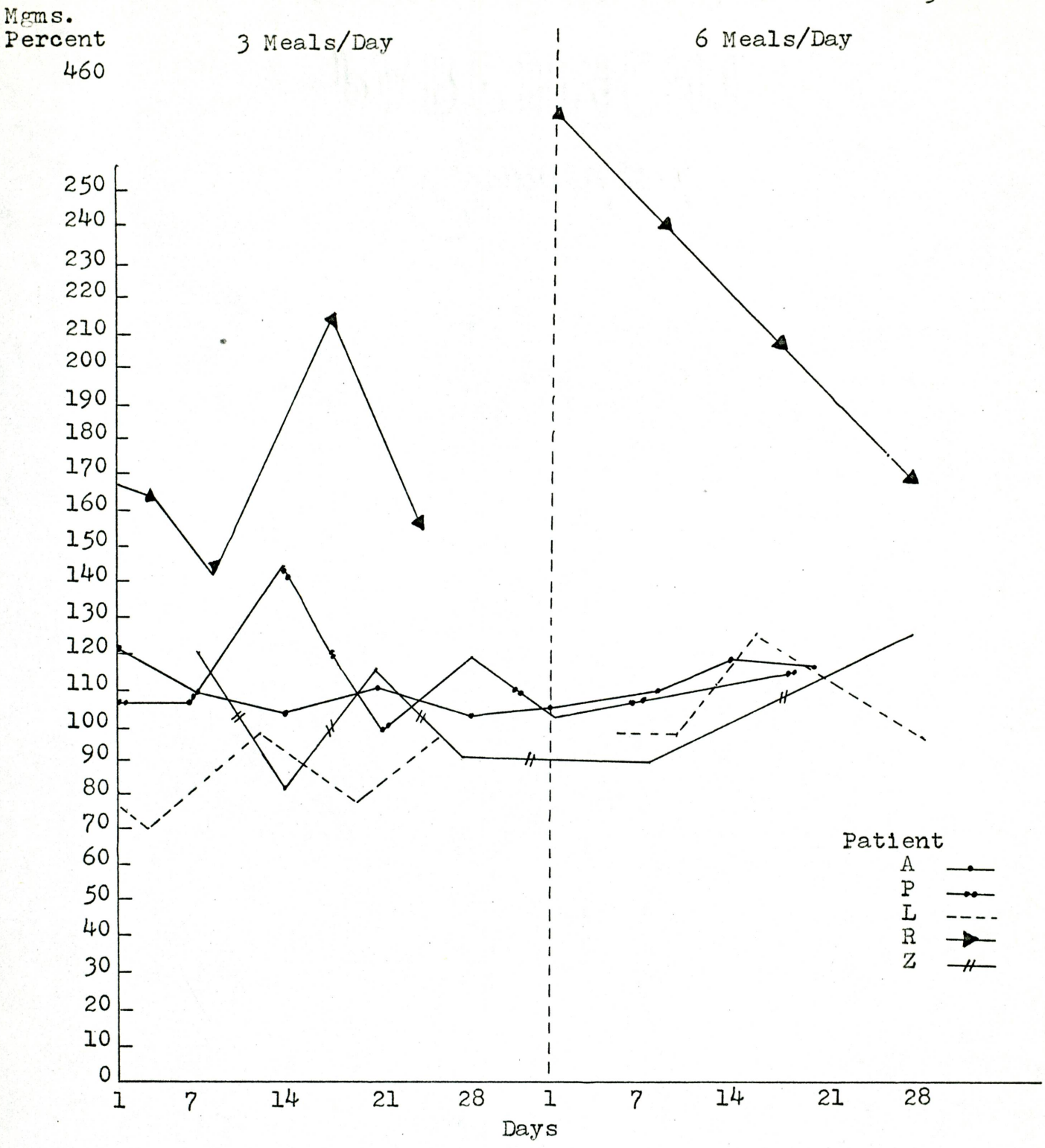

FIGURE 4

TWO HOUR POST PRANDIAL BLOOD GLUCOSE

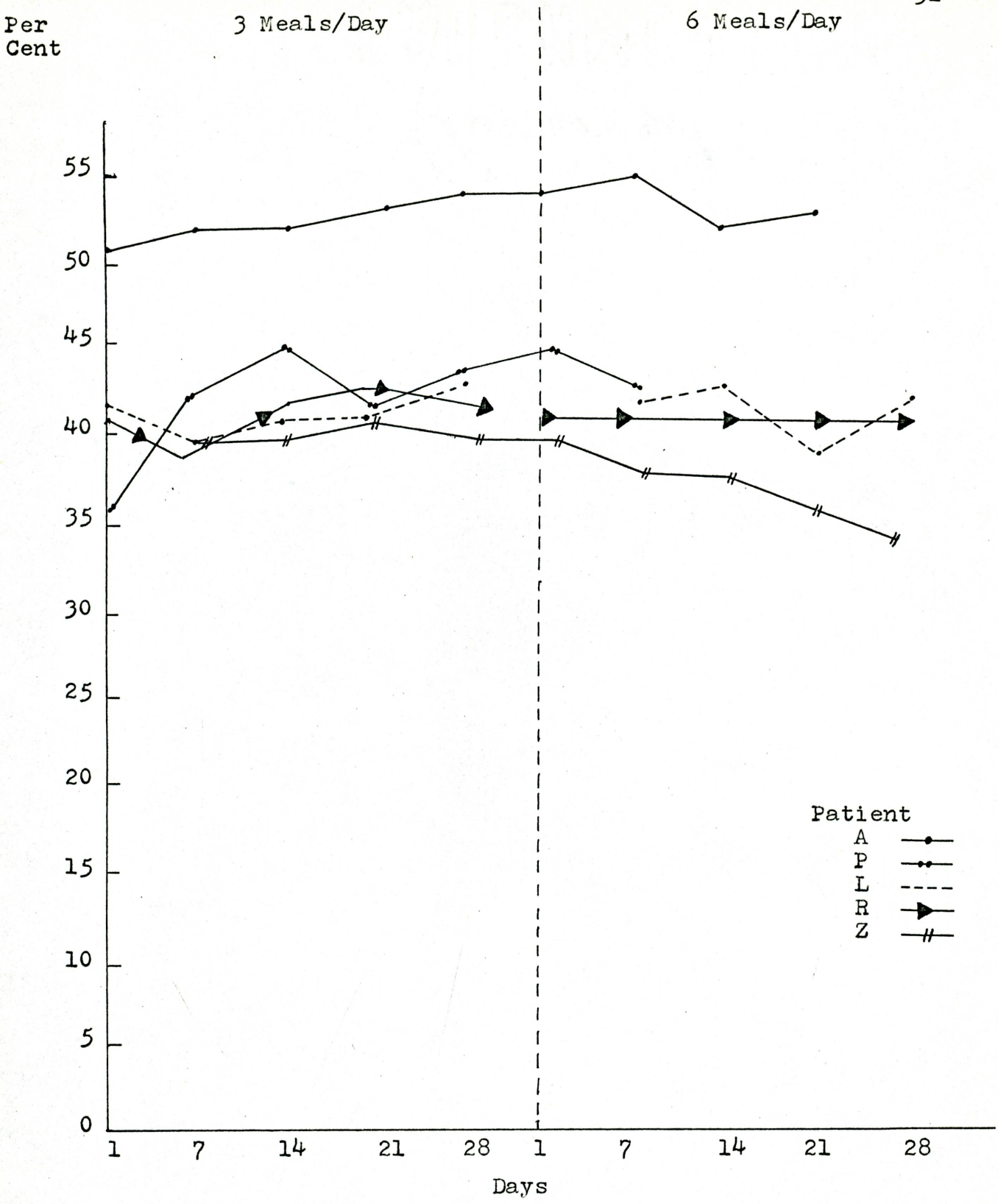

FIGURE 5

HEMATOCRIT

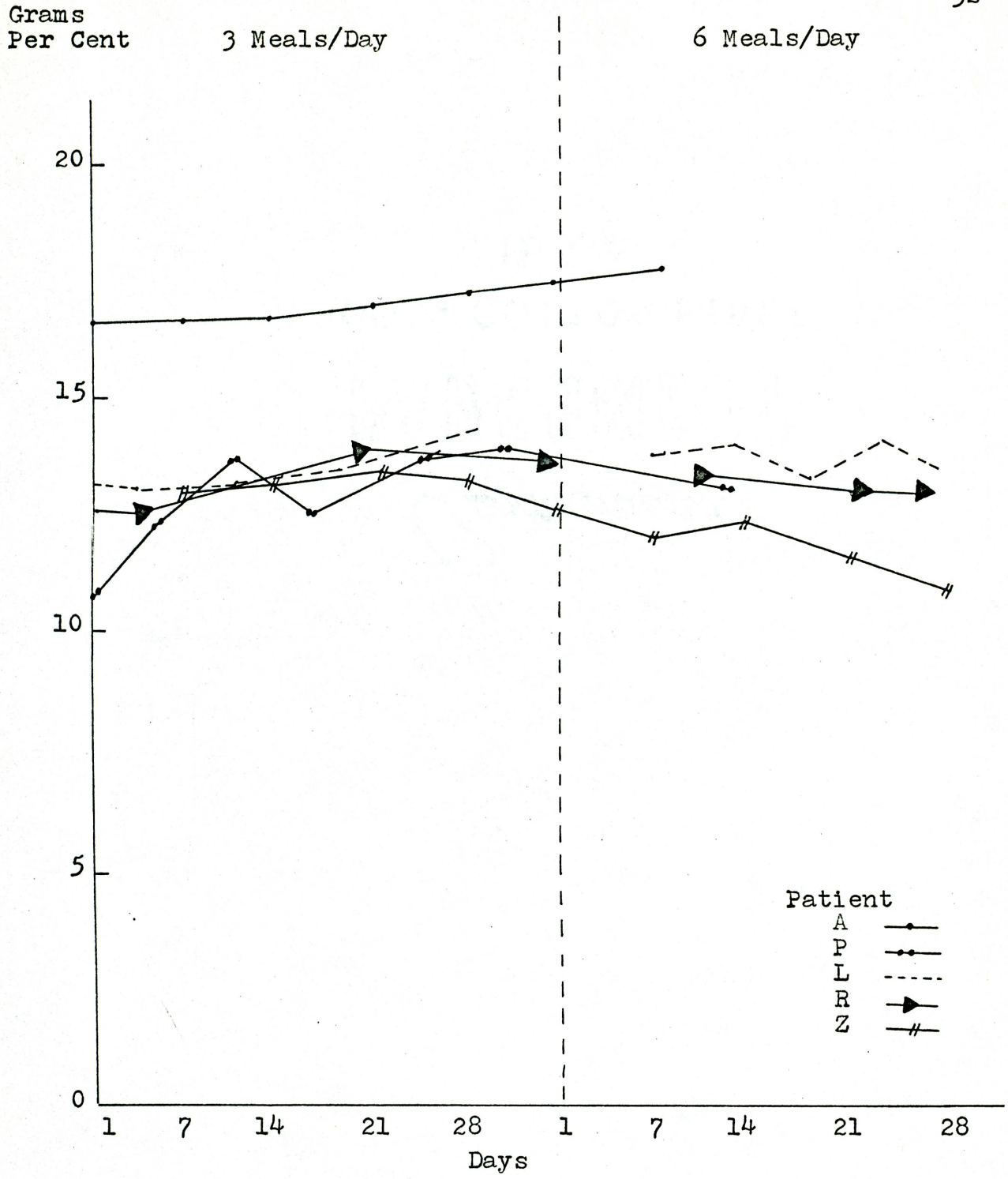

FIGURE 6

HEMOGLOBIN

CHAPTER V

DISCUSSION AND INTERPRETATION OF FINDINGS

All five patients showed a decrease in total body weight during the eight week experimental period on the 1200 Calorie reducing diet. One patient gained 0.5 pounds on the six meal per day pattern. This same patient lost 13.5 pounds on the three meal per day pattern. Another patient gained 1.0 pounds on the three meal per day pattern and subsequently lost 5.5 pounds on six meals per day.

The total body weight change ranged from plus 0.5 pounds to minus 10.0 pounds on six meals per day. The range on three meals per day was plus 1.0 pounds to 13.5 pounds.

The change in cholesterol per cent ranged from plus 16 milligrams per cent to minus 102 milligrams per cent on six meals per day. On three meals per day the range was from plus 20 milligrams per cent to minus 85 milligrams per cent.

These patients were hospitalized throughout the course of the experimental period. Their caloric intakes were rigidly controlled thereby prohibiting an intake of more than 1200 Calories per day. The amount of food left on the tray was recorded after each meal making it possible

to compute their actual caloric intake daily. Thus gross deviations from the 1200 Calories prescribed could be detected and related to the rate of weight loss.

Hospitalization also minimized the caloric expenditure which could result from climatic variations. The temperature in the two hospitals is regulated for all seasons. Exercise, another variable affecting caloric expenditure, remained fairly constant as the subjects were hospital patients who perform about the same tasks each day.

The changes in the two-hour post prandial blood sugar levels were not significant with the exception of the diabetic female whose diabetes was brought into control by diet and an oral antidiabetic agent. The hemoglobin and hematocrit changes were so slight as to be insignificant. The blood urea nitrogen remained essentially constant during the experimental period indicating there was no excessive tissue protein breakdown.

The results of our experiment concur with the findings of Bortz and associates who ran a concurrent study with almost identical objectives. Bortz and associates found there was no difference in the rate of weight loss whether the day's ration was divided into 1, 3, or 9 feedings per day. (6)

Personal verbal communication with the patients indicated 2 of the 5 preferred six meals per day as they experienced fewer hunger sensations. This has been reported

previously by Gordon (26). It was not possible to get satisfactory opinions from 2 of the patients as to which diet they preferred. One patient disliked 6 meals per day as this seemed to be too many meals per day. From this discussion it is evident there is no conclusion to be reached in this regard.

CHAPTER VI

SUMMARY AND CONCLUSIONS

Five obese adults were placed on a 1200 Calorie reducing diet. For about four weeks the 1200 Calories were divided into three meals of 200 Calories for breakfast, 200 Calories for lunch, and 800 Calories for dinner. For another approximate four week period these same patients were given six meals per day of 200 Calories each totaling 1200 Calories. The rate of weight loss on the two dietary patterns was compared. A t test performed on the slopes of the weight loss curves showed no statistical difference between the two dietary patterns on the rate of weight loss.

Laboratory analyses of blood parameters of cholesterol, hemoglobin, hematocrit, blood urea nitrogen, and two hour post prandial blood sugar showed no significant changes. The cholesterol level did drop on the sub-calorie diet, but there was no significant difference in the two dietary patterns affecting this drop. The hemoglobin, hematocrit, blood urea nitrogen remained essentially constant. The two hour post prandial blood sugar level remained the same with the exception of the diabetic patient whose diabetes was brought into control on the calculated diet and an oral antidiabetic agent.

The loss in total body weight can be attributed to caloric restriction. The timing of the meals did not effect the rate of weight loss.

BIBLIOGRAPHY

BIBLIOGRAPHY

1. Aldrich, C. K., M.D. "Mechanisms and Management of Obesity," Med. Clinics of N. Am., 47:77-84, January, 1963.

2. Anderson, E. C., and W. H. Langham. "An Estimation of Total Body Fat from Potassium-40 Content," Science, 133:1917-1918, June 16, 1961.

3. Beaudoin, R., and J. Mayer. "Food Intakes of Obese and Non-obese Women," J. Amer. Diet. Asso., 29:29, 1953.

4. Berryman, G. H., M.D. "Newer Concepts of Obesity," Ill. Med. J., Vol. 115, No. 2, February, 1959.

5. Bland, J. H., M.D. Clinical Metabolism of Body Water and Electrolytes, W. B. Saunders Company, Philadelphia, 1963, page 423.

6. Bortz, W. M., M.D., et al. "Weight Loss and Frequency of Feeding," N. Eng. J. Med., February 17, 1966.

7. Brainerd, H., S. Margen, and M. J. Chatton, Current Diagnosis and Treatment, Lange Medical Publications, Los Angeles, California, 1962, p. 515-516.

8. Chirico, A., M.D. and A. J. Stunkard, M.D. "Physical Activity and Human Obesity," New Eng. J. of Med., 263:935-940, November, 1960.

9. Cohn, C., M.D. "Meal-Eating, Nibbling, and Body Metabolism," J. Am. Dietetic Asso., 38:433, May, 1961.

10. Cohn, C., M.D. and D. Joseph. "Comparison of 'meal eating' and 'nibbling' on severity of alloxan diabetes," Proc. Soc. Exper. Biol. and Med., 105:575, 1960.

11. Cohn, C., M.D. and D. Joseph. "Effects on Metabolism Produced by the rate of Ingestion of the Diet, 'Meal Eating' vs. 'Nibbling'," Am. J. of Clin. Nutri., 8:682, 1960.

12. Cohn C., M.D., L. Oliner, and J. V. Morton. "Hypothyroidism produced by the forced feeding of normal rats," Endocrinology, 62:251, 1958.

13. Cohn C., M.D., R. Pick, and L. N. Katz. "Effect of Rate of Ingestion of the Diet ('meal eating' versus 'nibbling') on Atherogenesis in Chickens," Circulation, 20:969, 1959.

14. Cohn C., M.D., R. Pick, and L. N. Katz. "The Effect of Meal Eating Compared to Nibbling upon Atherosclerosis in the Chicken," Circulation Res., 9:139, 1961.

15. Conn, J. W. "II. Etiological Aspects," Phys. Rev. 24:31-44, January 1944.

16. Cooper, L. F., et al. Nutrition in Health and Disease, J. B. Lippincott Co., Philadelphia, Penn., 1958, p. 261-269.

17. Dabney, J. M., Ph.D. "Energy Balance and Obesity," Annals of Int. Med., 60:689-699, April, 1964.

18. Daft, F. S., Ph.D., et al. The Heinz Handbook of Nutrition, McGraw-Hill Book Co., Inc., New York, 1959, p. 193-203.

19. Dailey, M. J., M.D. "A Contrary Look at Obesity," Med. Times, 91:742-744, August, 1963.

20. Dole, V. P., et al. "Treatment of Obesity with a Low Protein Calorically Unrestricted Diet," Amer. J. Clin. Nutr., 2:381, 1954.

21. Edwards, K. D. G., and H. M. Whyte. "The Simple Measurement of Obesity," Clin. Science, 22:347-352, June, 1962.

22. Fabry, R., M.D. et al. "The Frequency of Meals, Its Relation to Overweight, Hypercholesterolemia and Decreased Glucose-Tolerance," Lancet, II:614-15, September 19, 1964.

23. Feinstein, A. R. M.D. "The Treatment of Obesity," J. of Chronic Diseases, 11:349-393, April, 1960.

24. Gold, A., "Do Calories Count?" Appl. Ther., 7:108-13, February, 1965.

25. Goodman, L.S., M.A., M.D. and A. Gilman, Ph.D. The Pharmacological Basis of Therapeutics, Macmillan Company, New York, 1955, page 1542.

26. Gordon, M. D., et al. "New Concept in Treatment of Obesity," J. A. M. A., 186:50-60, October, 1963.

27. Halpern, S. L., M.D., F.A.C.P. "Effective Weight Reduction," New York State J. of Med., 61:4205-12, December, 1961.

28. Halpern, S. L., M.D. "Methodology of Effective Weight Reduction," Med. Clinics of N. Am., 48:1335-45, September, 1964.

29. Harrison, T. R., M.D., et al. Principles of Internal Medicine, McGraw-Hill Book Co., Inc., New York, 1962, p. 191-197.

30. Heald, F. P., M.D. et al. "Measures of Body Fat and Hydration in Adolescent Boys," Pediatrics, 3:226-39, February, 1963.

31. "High Fat Diet for Weight Reduction," Nutr. Rev., 20:294-7, October, 1962.

32. Hollifield, G. and W. Parson. "Metabolic Adaptations to a 'Stuff and Starve' Feeding Program," J. of Clin. Invest., 41:245, 1962.

33. Kinsell, L. W., et al. "Calories Do Count," Metabolism, 13:195-204, March, 1964.

34. Lim, T. P., M.D., Ph.D., and U. C. Luft, M.D. "Body Density, Fat and Fat-Free Weight," Am. J. of Med., 30:825-831, June, 1961.

35. Lyght, C. E., M.D., et al. The Merck Manual of Diagnosis and Therapy, Merck Sharp and Dohme Research Laboratories, Rahway, New Jersey, 1961.

36. Mayer, J., et al. "Exercise, Food Intake and Body Weight in Normal Rats and Genetically Obese Adult Mice," Am. J. of Physiology, 177:544-548, June, 1954.

37. Mayer, J., Ph.D. "Obesity in Childhood and Adolescence," Med. Clin. of N. Am., 48:1347-1358, September, 1964.

38. Mayer, J. "Some Aspects of the Problem of Regulation of Food Intake and Obesity (To be Concluded)," N. Eng. J. Med., 274:662, March 24, 1966.

39. McCracken, B. H., M.D., M.R.C.P. "Etiological Aspects of Obesity," Am. J. of Med. Sciences, 243:99-111, 1962.

40. Miller, A. B., et al. Physicians' Desk Reference to Pharmaceutical Specialties and Biologicals, Medical Economics, Inc., Oradell, N. J., 1966, page 943.

41. Murphy, R., M.D. "The Complexities of the Problem of Obesity," Med. Clin. of N. Am., 44:439-444, March 1960.

42. Myers, M. L., M.S., and F. J. Stare, M.D., Ph.D. "Formulae, Fads, and Fatness," Food and Nutri. News, 33:1 and 4, November, 1961.

43. Newburgh, L. H. "Obesity, I. Energy Metabolism," Phys. Rev., 24:18-30, January, 1944.

44. Pennington, A. W. Delaware Med. J., 23:79-86, 1951.

45. Pollack, H., M.D. "Obesity," Food and Nutri. News, 35:1 and 4, May, 1964.

46. Powdermaker, H., Ph.D. "An Anthropological Approach to the Problem of Obesity," Bull. of the N. Y. Acad. of Med., 36:286-295, May, 1960.

47. Rath, R., et al. "Changes in Body Composition in the Obese Subjected to Different Types of Reducing Regimens, (Preliminary communication)," Nutr. Dieta (Basel), 6:273-8, 1964.

48. "The Role of Carbohydrates in the Diet," Nutr. Rev., 22:102-5, April, 1964.

49. Seaton, D. A., M.D., L. J. P. Duncan, M.D. "Treatment of Refractory Obesity with a Diet of Two Meals per Day," Lancet II:612-14, September 19, 1964.

50. Stefferud, A., et al. Food, the Yearbook of Agriculture 1959, U. S. Department of Agriculture, Washington, D. C., 1959, p. 104-108, 456.

51. Steinkamp, R. C., M.D. "Measure of Body Fat and Related Factors in Normal Adults -- I. Introduction and Methodology," J. Chron. Dis., 18:1279-1289, December, 1965.

52. Stunkard, A., M.D. and M. McLaren-Hume, M.S. "The Results of Treatment for Obesity," A.M.A. Arch. of Int. Med., 103:79-84, January, 1959.

53. Taller, H., M.D. Calories Don't Count, Simon and Schuster, New York, 1960.

54. Van Putten, L. M., D. W. Van Bekkum, and A. Querido, "Influence of Hypothalamic Lesions Producing Hyperphagia and of Feeding Regimens on Carcass Composition in the Rat," Metabolism, 4:68, 1955.

55. White, A., Ph.D., et al. Principles of Biochemistry, Second Edition, McGraw-Hill Book Company, Inc., New York, 1959, page 751.

56. Wishnofsky, M., M.D. "Weight Reduction. Letters to the Journal," J. A. M. A., 183:620, 1963.

57. Wohl, M. G., M.D. and R. S. Goodhart, M.D. Modern Nutrition in Health and Disease, Lea and Febiger, Philadelphia, Penn., 1960, p. 859-872.

58. Young, C. M. "The Prevention of Obesity," Med. Clin. of N. Am., 48:1317-1331, September, 1964.

LOMA LINDA UNIVERSITY

Graduate School

INFLUENCE OF CALORIC DISTRIBUTION ON WEIGHT LOSS

by

Georgia E. Hodgkin

An Abstract of a Thesis in Partial Fulfillment

of the Requirements for the Degree

Master of Science in the Field of Nutrition

June, 1966

ABSTRACT

The purpose of this study was to determine the effect timing of meals has on the rate of weight loss. Five hospitalized obese adults were placed on a 1200 Calorie reducing diet for approximately eight weeks.

For about a four-week period the 1200 Calories were divided into six meals per day of 200 Calories each. These same patients were given the 1200 Calories in three meals per day approximately another four-week period. With three meals per day, the calories were divided into 200 Calories for breakfast and lunch and 800 Calories for the evening meal. The former pattern represents ad libitum feedings or "nibbling," while the latter is characteristic of the typical working American's dietary pattern of little or no breakfast, a light lunch, and a large evening meal.

Routine hospital fare for reducing regimens was used for these diets. The exchange system was followed to determine the portion size. A visual estimation of the amount of each serving of food left on the tray was made by the licensed vocational nurse in charge of the patient and recorded following each meal. This provided an accurate count of the actual caloric intake daily.

Laboratory analyses of blood samples included

determinations of serum cholesterol, two-hour post prandial blood sugar, blood urea nitrogen, hematocrit, and hemoglobin. A t test showed no significant difference in the regression slope of the serum cholesterol on the two diets. The blood urea nitrogen, hematocrit and hemoglobin remained essentially constant throughout the experimental period. The two-hour post prandial blood sugar levels remained the same with the exception of the diabetic patient whose diabetes was brought into control on the calculated diet and an oral antidiabetic agent.

A t test performed on the regression slope of the weight loss showed no statistical difference between the three meal per day pattern and the six meal per day pattern. The loss in total body weight can be attributed to caloric restriction. The timing of the meals did not effect the rate of weight loss.