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10 June 74

Dear Bob:

Attached is a draft of a memo that has been written to assist the Heart Association staff in answering the myriad of questions they receive from physicians about the various diets available for the treatment of hyperlipidemia. It seems that many physicians are confused about this.

Dr. Hurley asked me to have you review this manuscript and make changes and comments as you see fit.

I look forward to seeing you in Seattle.

Sincerely yours,

Mary Winston
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MW:jmc
Th/8

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Memorandum



To: All Heart Associations

MAT-74-517

From: Mrs. Mary Winston, Nutrition Consultant

Subject: Guide for Physicians in Use of Diet Therapy in Hyperlipidemia

Attached is an analysis of four different sets of diets now available to aid physicians in the treatment of hyperlipidemia. Three are AHA publications and the fourth is from the National Heart and Lung Institute. This analysis is intended to identify basic differences between these dietary recommendations, and to aid the physician in deciding which to prescribe for each of his patients.

Use it as an answer to questions you receive about the diets. We recommend also that you distribute it to hospitals, clinics, physicians and nutritionists in your area, especially those who have expressed an interest in diet as a therapeutic regimen.

The diets analyzed are:

The Way To A Man's Heart EM455

Planning Fat-Controlled Meals EM288 and EM288A

A Maximal Approach to the Dietary Treatment of the Hyperlipidemias

EM585, EM585A, EM585B, EM585C, EM585D

The Dietary Management of Hyperlipoproteinemia DHEW Pub. No. (NIH) 73-110-115

DRAFT

MAT-74-517

The Dietary Treatment of Hyperlipidemia -- A Note to the Physician

The decision as to the appropriate diet therapy for the hyperlipoproteinemias is sometimes difficult to make. To facilitate this decision the following guidelines are offered.

Hyperlipidemia, an abnormal elevation of serum cholesterol, triglyceride or both, may be primary or secondary. Secondary hyperlipidemia results from a specific disease state such as diabetes or hypothyroidism, and treatment is directed at the underlying disease. When it is controlled, the hyperlipidemia ^{is lessened and may disappear} disappears.

Primary hyperlipidemia may be a manifestation of a genetic abnormality or environmental factors. Whatever the cause, diet is the foundation of all treatment programs.

The majority of Americans who develop coronary heart disease have mild hypercholesteremia (200-280 mg. %) and no, or only slight elevation of serum triglycerides (100-200 mg. %). These individuals may be identified by simple measurement of plasma cholesterol and triglyceride. (?)
Fasting blood

THE WAY TO A MAN'S HEART EM455

For such individuals, the initial recommendation might be "THE WAY TO A MAN'S HEART" which is the American Heart Association's basic fat-controlled diet. This diet is low in saturated fat and cholesterol. It has been demonstrated that this diet will effect significant reductions in hypercholesteremia and hypertriglyceridemia. It is moderate

in total fat (30-35% of total calories), low in saturated fat (less than 10%), moderate in carbohydrate (40-45% of calories), low in sugar and moderate in alcohol. The caloric level should be adjusted to meet individual needs, but the percentage composition of the diet should always be maintained.

*in relation
to overall
nutritional diet*

If serum lipid response is inadequate with good dietary adherence to this regimen, the classification of the patient into one of five lipoprotein types according to the schema proposed by Fredrickson, Levy, and Lees may be helpful in assessing further nutritional modifications.

Type of Hyperlipoproteinemia: Refers to abnormal lipoprotein patterns.

- Type I: loss of enzymatic step in removal of chylomicrons.
- Type II: excess production or inadequate clearance of LDL (low density lipoproteins).
- Type III: block in metabolism of VLDL (very low density lipoproteins) to LDL, causing an abnormal "intermediate" form of lipoprotein to circulate in plasma.
- Type IV: excess production or inadequate clearance of VLDL.
- Type V: VLDL excess combined with poor chylomicron removal.

? of serum lipid levels

The dietary prescription for each type should be directed to the lipoprotein fraction or fractions which are elevated. Any one of several different dietary approaches may be used. The diet therapy selected should be tailored to the patient's physiological, psychological and socio-economic needs.

PLANNING FAT-CONTROLLED MEALS EM288 and EM288A

The diets recommended for treating each of the hyperlipidemias vary somewhat but they have a similar food pattern.

The presence of chylomicrons (Types I and V) requires a low fat diet.

The presence of high pre-beta lipoprotein fraction (Types IIb, III,

and IV) calls for the ^{diets high in} ~~(unsaturated fat~~ ^{control} diets with 30-35% of calories

as fat, and 40-50% calories as carbohydrate for best results. A

further limitation of concentrated sweets and alcohol may also be

required. ^{for hypoglycemia} These diets are outlined in the American Heart Association

booklets "PLANNING FAT-CONTROLLED MEALS" EM288 and EM288A.

For beta lipoprotein reduction in Type IIa, the kind of fat rather

than total fat is of utmost importance -- the fat must be poly-

unsaturated ^{in significant proportion} and low in cholesterol. "PLANNING FAT-CONTROLLED MEALS"

is an effective guide for this diet.

NATIONAL HEART AND LUNG INSTITUTE DIETS

The National Heart and Lung Institute has developed a series of six booklets.

The Dietary Management of Hyperlipoproteinemia, a Handbook for
Physicians and Dietitians -- DHEW Pub. 73-110

Diet 1 (Type I Hyperlipoproteinemia) DHEW Pub. 73-111

Diet 2 (Type IIa Hyperlipoproteinemia) DHEW Pub. 73-112

Diet 3 (Type IIb or Type III Hyperlipoproteinemia) DHEW Pub. 73-113

Diet 4 (Type IV Hyperlipoproteinemia) DHEW Pub. 73-114

Diet 5 (Type V Hyperlipoproteinemia) DHEW Pub. 73-115

The diets have resulted from the experience of National Heart and Lung

Institute investigators and others. NHLI has concentrated largely on studies of familial forms of hyperlipoproteinemias. These diets are therapeutic, and not designed for the general population.

The numbers of the diets correspond to the five types of hyperlipoproteinemia with one exception. It is recommended that persons with type IIb begin with diet number 3. If the result is a lessening of triglyceride excess, but continued elevation of beta lipoproteins, the stricter control of diet number 2 may be indicated.

A MAXIMAL APPROACH TO THE DIETARY TREATMENT OF THE HYPERLIPIDEMIAS

A manual for the physician and a set of four diets entitled "A MAXIMAL APPROACH TO THE DIETARY TREATMENT OF THE HYPERLIPIDEMIAS" have been prepared by Dr. William Connor for the American Heart Association.

A MAXIMAL APPROACH TO THE DIETARY TREATMENT OF THE HYPERLIPIDEMIAS

A Manual of Dietary Modifications and Instructions for Patients
with Hyperlipidemia as a Prescription from Their Physicians
Physician's Handbook EM585

Diet A: The Low Cholesterol (100 MG) Moderately Low Fat Diet EM585A

Diet B: The Low Cholesterol (200 MG) Moderately Low Fat Diet EM585B

Diet C: The Low Cholesterol, High Polyunsaturated Fat Diet EM585C

Diet D: The Extremely Low Fat Diet EM585D

These are designed for use in the treatment of various hyperlipidemias and aimed at a maximal lipid lowering response. Each diet is based on a system of food exchanges and involves a restrictive modification of

usual food habits. They may be most suitable for highly motivated patients.

The attached chart outlines the characteristics of the various diets and gives the physician an opportunity to evaluate the differences between them.

Key to Diet Books

▲ NHLI (I,II,III,IV,V)
 ○ AHA - Planning Fat-Controlled Meals
 * A Maximal Approach to the Dietary Treatment of the Hyperlipidemias (A,B,C,D)

Suggested Diet Rx for Hyperlipidemias

TYPE	LIPOPROTEIN	ELEVATED LIPID	DIET PRESCRIPTION	CALORIES	MGS. CHOL.	% of TOTAL CAL. CHO.	% of TOTAL CAL. PRO.	% of TOTAL CAL. FAT	% of TOTAL CAL. SATURATED FAT	% of TOTAL CAL. POLY. FAT	ALCOHOL
I	Chylomicrons	Tri-Glyceride	▲ (1) * (D) OR (A)	Not restricted Achieve and maintain ideal weight Achieve and maintain ideal weight	Not restricted 60-78 100	Not limited 68-71 65	Not limited 15-20 15-20	20 12 20	Kind of fat not important Kind of fat not specified 5.3	Kind of fat not important Kind of fat not specified 8.1	Not recommended At M.D.'s discretion. Not recommended for weight control At M.D.'s discretion. Not recommended for weight control
V	Chylomicrons	Tri-Glyceride	▲ (5) * (D) OR (A)	Achieve and maintain ideal weight Achieve and maintain ideal weight Achieve and maintain ideal weight	300-500 60-78 100	45-53 Controlled - conc. sweets restricted 68-71 65	21-24 15-20 15-20	25-30 Restricted 12 20	Not specified Kind of fat not specified 5.3	Not specified Polys. recommended Kind of fat not specified 8.1	Not recommended At M.D.'s discretion. Not recommended for weight control At M.D.'s discretion. Not recommended for weight control
IV	Pre-Beta	Tri-Glyceride Cholesterol (often)	▲ (4) * (A) OR (B) OR (C) ○	Achieve and maintain ideal weight Achieve and maintain ideal weight Achieve and maintain ideal weight Achieve and maintain ideal weight Achieve and maintain ideal weight	300-500 100 200 200 300	44-46 Controlled - conc. sweets restricted 65 60 48 40-50 Conc. sweets restricted	Not limited except for wt. control 15-20 15-20 15-20 20-26	Not limited except for wt. control 20 25 35 30-35	Not specified 5.3 6.1 7.6 4-5	Not specified. Polys. recommended 8.1 8.5 12.6 11-13	Limited to 2 svgs./day (substitute for carbohydrates) At M.D.'s discretion. Not recommended for weight control At M.D.'s discretion At M.D.'s discretion Not recommended
IIb	Pre-beta Beta	Cholesterol Tri-Glyceride	▲ (3) * (A) OR (B) ○	Achieve and maintain ideal weight Achieve and maintain ideal weight Achieve and maintain ideal weight Achieve and maintain ideal weight	< 300 100 200 300	40-45 Controlled - conc. sweets restricted 65 60 40-50 Conc. sweets restricted	18-21 15-20 15-20 20-26	Controlled to 40 20 25 30-35	Not specified 5.3 6.1 4-5	Not specified Polys. recommended 8.1 8.5 11-13	Limited to 2 svgs./day (substitute for carbohydrates) At M.D.'s discretion. Not recommended for weight control At M.D.'s discretion Not recommended
III	Beta "broad" Band Low Density	Tri-Glyceride Cholesterol	▲ (3) * (A) OR (B) OR (C) ○	Achieve and maintain ideal weight Achieve and maintain ideal weight Achieve and maintain ideal weight Achieve and maintain ideal weight Achieve and maintain ideal weight	< 300 100 200 200 300	40-45 Controlled conc. sweets restricted 65 60 48 40-50 Conc. sweets restricted	18-21 15-20 15-20 15-20 20-26	Controlled to 40 20 25 35 30-35	Not specified 5.3 6.1 7.6 4-5	Not specified Polys. recommended 8.1 8.5 12.6 11-13	Limited to 2 svgs./day (substitute for carbohydrates) At M.D.'s discretion. Not recommended for weight control At M.D.'s discretion At M.D.'s discretion Not recommended
IIa	Beta	Cholesterol	▲ (2) * (A) OR (B) OR (C) ○	Not restricted Achieve and maintain ideal weight Achieve and maintain ideal weight Achieve and maintain ideal weight Achieve and maintain ideal weight	< 300 100 200 200 300	Not limited 65 60 48 40-50 Concentrated sweets restr	Not limited 15-20 15-20 15-20 20-26	Not specified 20 25 35 30-35	Restricted 5.3 6.1 7.6 4-5	Increased to achieve P/S Ratio of 2:1 8.1 8.5 12.6 11-13	Use with discretion At M.D.'s discretion. Not recommended for weight control At M.D.'s discretion At M.D.'s discretion At M.D.'s discretion

NUTRITIONAL THERAPY OF THE PATIENT WITH
ACUTE MYOCARDIAL INFARCTION

George Christakis, M.D.
and
Mary Winston, R.D.

THE NUTRITION PLAN

Each patient sustaining an acute myocardial infarction evolves a unique metabolic response which mitigates against the development of a general formula for nutritional care. The preceding information points to a rational approach to nutritional therapy.

The nutritional plan should be regarded as part of total patient care to be implemented from the time of the patient's admission to the Coronary Care Unit, through full recovery, discharge to home and subsequent follow-up. Careful consideration should be given to the patient's immediate clinical status, his associated condition, e.g. diabetes or hyperlipidemia, the extent of his myocardial damage and the drug therapy being administered.^(29,30,31) Coordination of nutritional therapy in the acute, sub-acute and rehabilitative phases of the patient's illness allows for a step-by-step progression in his nutritional regimen.

I. Acute Phase:

Opinions vary as to the preferred diet to be given during this phase of the patient's illness. One extensive review of the literature from 1935 to 1962 revealed that for the initial two to three days after myocardial infarction, patients were given a rigidly restrictive diet consisting of liquids. A 500 to 800-calorie diet for the remainder of the acute phase was commonly used. Physicians favor some form of the liquid diet, with a daily volume of 1000 to 1500 ml for the first few days.^(30,31,33) Such a practice may reduce the risk of cardiac arrest by avoidance of gagging on food boluses which may induce vasovagal responses with arrhythmic effects. Food aspiration is partially avoided with a liquid diet.

Usually two to three days is sufficient time for the patient to remain on the liquid diet; however, when arrhythmias persist, the liquid diet should be continued for several more days. It is important to vary the peroral or parenteral aspects of the diet according to the severity of the patient's condition.

Examples of fluids which may be given include fruit juice, skim milk, clear soup, broth, tea, gingerale and water. Extremes in temperature of liquids are to be avoided, e.g. very hot or cold soups or beverages and ice, in order to avert possible precipitation of arrhythmias via neural mechanisms.^(34,35) It is common practice to eliminate coffee and tea since they contain stimulants

which may increase the heart rate. Non-caffeine and decaffeinated beverages may be served as substitutes. Every precaution should be taken to avoid abdominal distension in patients who cannot tolerate milk, excessive amounts of fruit juices and carbonated beverages.^(30,36) Physicians may find that 200 cc of low-sodium milk, 3 to 4 times a day, may be well tolerated.⁽³⁴⁾

Acutely ill patients may be unwilling to consume liquids, thereby necessitating parenteral feeding. It is imperative that the physician have knowledge of the patient's daily intake and output of all fluids.⁽³⁷⁾

Adjustment in the sodium and potassium content of the diet may be necessary, depending on the patient's clinical status, associated illness and medication administered.⁽³⁸⁾ Sodium must be restricted if there is evidence of congestive heart failure. Some physicians routinely restrict sodium.^(29,31,35) However, others warn of the potential danger of sodium restriction after an acute myocardial infarction. Severe sodium restriction may precipitate or aggravate shock by complementing the extensive loss of salt occurring as a result of profuse diaphoresis. Sodium loss in urine also occurs with myocardial infarction.^(39,40)

If vomiting persists during the first few days after the acute myocardial infarction, drug therapy should be reviewed to determine its relationship to the vomiting.⁽³⁰⁾

Small amounts (50 to 100 ml) of a hypertonic solution of dextrose (10 to 20%) may be given intravenously via a micro-drip at regular intervals during the day, or by very slow continuous drips. This will provide adequate fluid and carbohydrate.^(30,31,37) As soon as the patient can take fluids orally, the IV should be discontinued. The reduced efficiency of a damaged myocardium and the hazard of pulmonary edema contra-indicates the use of unrestricted intravenous infusions.⁽³¹⁾

Current practice includes the oral feeding of a patient during the acute stages of his illness. This has been done rather routinely without

evaluating the effects of the activity. The report of a small recent study involving seventeen patients with acute myocardial infarction indicated that being fed or feeding oneself had little effect on blood pressure or pulse rate. Parameters such as cardiac output and oxygen consumption were not measured. In the opinion of the investigators, patients, especially males, prefer to be independent and to feed themselves.⁽⁴¹⁾

Sodi-Pallares advocates what has become a controversial nutritional regimen during the acute phase of a myocardial infarction. This is the "polarizing treatment" designed to maintain metabolic and electrolyte balance. The treatment consists of a low-sodium, high-potassium diet, a polarizing solution consisting of one liter of glucose solution with variable glucose concentration (10 to 50%), according to the severity of the infarct, adequate regular insulin to cover the concentration of glucose, and potassium depending on the level of serum potassium.⁽⁴²⁾

Arnott et al report that no significant benefit was derived in a clinical trial of potassium, glucose and insulin solution given in the treatment of patients who sustained a myocardial infarction, although Sodi-Pallares noted that the nutritional components of the solution were not comparable to those used in his study.⁽⁴³⁾

Maroko et al found that glucose-insulin-potassium infusion in the normal dog prevents some myocardial cells from undergoing ischemic necrosis following occlusion. Their results suggest that further trial of this treatment in patients with acute myocardial infarction is warranted.⁽⁴⁴⁾

II. Sub-Acute Phase:

Once the acute phase has passed, the physician may put the patient on a diet consisting of more solid food. It is reasonable to allow a daily diet of 1000 to 1200 calories, composed of approximately 20% protein, 45%

carbohydrate and 30 to 35% fat, with a cholesterol limit of 300 mgs a day. The therapeutic goal is to provide sufficient calories for basal metabolic requirements and optimal levels of essential micronutrients for the maintenance of good nutritional status without inducing an increase in oxygen uptake. It is also important to maintain a positive nitrogen balance.

Foods which are easily digestible, free of gastric irritants, soft and low in roughage, are preferred. (30,31,32,37,44) They should be chosen so as to avoid flatus and constipation. Such foods include skim milk, tender lean cuts of meat, fish and poultry; tender cooked or canned vegetables and fruits; plain breads, cooked cereals; simple puddings and gelatin desserts. Egg yolks are limited. Coffee and tea may be permitted in moderation provided they do not increase restlessness or sleeplessness. As an alternate, non-caffeine and decaffeinated beverages may be served. Again, extremes in temperature are to be avoided. "Planning Fat-Controlled Meals" or the "Moderate Sodium-Restricted Fat-Controlled Diet" may be used as a guide during this period. Unless contra-indicated because of clinical complications, the patient will welcome a return to normal eating patterns which should enhance his psychological status.

Frequent small feedings may be desirable and may also contribute to a serum cholesterol-lowering effect. They are especially ideal for those patients who experience dyspnea or anginal pain at rest or with effort after meals. (31)

III. Rehabilitative Phase:

As a patient resumes moderate activity, a wider variety of foods may be introduced into his daily diet. However, he must still adhere to the principles of the fat-controlled diet, with sodium restriction when necessary. Evidence is available which strongly suggests that lowering the serum cholesterol level through dietary means may lower the recurrence rate of secondary

coronary events.^(44,45,46,47,48)

It is during the rehabilitative period that caloric adjustments should be made to maintain optimal weight or begin to reduce weight, if necessary. It is not currently known whether the metabolic changes that occur during weight reduction will influence the course of the myocardial infarction positively. However, weight reduction may impose a physical and psychological burden which the patient may not tolerate. In adjusting calories, it is well to remember that the patient may be inactive for two to three months.

Effective dietary recommendations can best be made after serum cholesterol, triglyceride and lipoprotein electrophoresis have been determined. Diets relating to each pattern have been proposed by the National Heart and Lung Institute.⁽⁴⁹⁾ Various adjustments, such as caloric modification and change in amounts and kinds of carbohydrate and amounts and kinds of fat, may have to be made in the patient's diet over a period of time. As emphasized above, the metabolic condition of the patient may not stabilize for an extended period of time. Physicians are cautioned about the inherent fallacy in making an immediate diagnosis of hyperlipoproteinemia after myocardial infarction, since the type of hyperlipoproteinemia may change. On the contrary, it is reasonable to expect that firm diagnostic inferences can more likely be made after lipid analysis at six months and one year after a myocardial infarction.⁽⁵⁰⁾

The hospital stay provides a unique opportunity for helping the patient and his family understand the principles of his particular diet. The hospital tray is a valuable educational tool which serves to demonstrate to the patient the various ways different foods can be combined to make tasty attractive meals. Support and encouragement from the hospital staff

is paramount to the patient's acceptance of a dietary regimen which, if it is to be effective, must become a way of life in the months and years ahead.

Inasmuch as a substantial number of adults, particularly Black and Asians, may be sensitive to milk through lactase deficiency, the use of partially fermented milk as contained in yoghurt or low-fat cottage cheese are low-residue foods which reduce the chances for gastrointestinal distress. Some patients also exhibit gastrointestinal irritability when consuming citrus juices; this can be avoided by using apple juice or fruit nectars.

OUTLINE OF A SUGGESTED NUTRITIONAL PATTERN FOR A CORONARY CARE UNIT

This coronary care unit nutritional pattern is suggested for short-term use for the initial five to ten day-period following acute myocardial infarction.

1. NPO prior to evaluation by M.D. In most instances, intravenous solution started to facilitate administration of drugs that may be required if arrhythmias and shock ensue.
2. 500-800 calorie (1000-1500 ml) liquid diet for first 24 hours, with only small amounts of liquid taken at a time. (Examples of foods which may be offered are clear soups, broth, skim milk, fruit juices, tea, gingerale and water).
3. Re-evaluation of patient for dietetic progression after the first 24 hours.
4. Caloric level of 1000-1200 calories to meet patient's basal metabolic requirement. Nutrient content approximately 20% protein, 45% carbohydrate and 30-35% fat (low saturated fat--polyunsaturates, the primary source of dietary fat) with a cholesterol limit of 300 mg per day; sodium restriction if indicated by patient's condition.
5. Beverages and other liquids served at body temperatures. Non-caffeine and decaffeinated beverages are preferred. (Stimulants and extremes in temperatures to be avoided).
6. Small frequent meals consisting of foods which are easily digestible, free of gastric irritants, soft and low in roughage.

7. Foods to be included are tender, lean cuts of meat, fish and poultry; tender cooked or canned vegetables and fruits; plain breads; cooked cereals; simple puddings and gelatin desserts. Egg yolks are limited to three a week.
8. Nutritional plan individualized on basis of patient's clinical status, physiological and psychological needs. Areas usually requiring modification: carbohydrates, protein, fat, total calories, electrolytes and fluids.

CONCLUSION

The nutritional care of the patient who has sustained a myocardial infarction is best considered from the viewpoint of the metabolic effects of this clinical event, associated conditions the patient may have, and the particular diet pattern that was characteristic of his life style prior to infarction.

Guidelines for effective serum cholesterol and triglyceride-lowering diets are available. With nutritional counseling from the physician and the dietitian, the patient can be taught to translate these practical dietary recommendations into appetizing and satisfying meals.

BIBLIOGRAPHY

1. SMITH, R.B., KIDDLE, G.B. and PRIOR, I.A. Early and Late Observations after Acute Myocardial Ischemic Episodes with Particular Reference to Glucose Tolerance. New Zeal. Med. J., 67:486-492, 1968.
2. TZAGOURNIS, M., SEIDENSTICKER, J.F. and HAMWI, G.J. Serum Insulin, Carbohydrate and Lipid Abnormalities in Patients with Premature Coronary Heart Disease. Ann. Intern. Med. 67:42-47, 1967.
3. PETERS, N., and HALES, C.N. Plasma-Insulin Concentrations after Myocardial Infarction. Lancet, 1:1144-1145, 1965.
4. LEOVITZ, H.E., SHULTZ, K.T., MATTHEWS, M.E. and SCHEELE, R. Acute Metabolic Responses to Myocardial Infarction. Circulation, 39:171-181, 1969.
5. DYKES, J.R.W., SAXTON, C. and TAYLOR, S.H. Insulin Secretion in Cardiogenic Shock. Brit. Med.J., 2:490, 1969
6. OPIE, L.H., NORRIS, R.M., THOMAS, M. et al. Failure of High Concentrations of Circulating Free Fatty Acids to Provoke Arrhythmias in Experimental Myocardial Infarction. Lancet, 1:818-822, 1971.
7. SHORE, B., NICHOLS, A.V., and FREEMAN, N.K. Evidence for Lipolytic Action by Human Plasma Obtained after Intravenous Administration of Heparin. Proc. Soc. Exp. Biol. Med., 83:216-220, 1953.
8. NEAVERSON, M.A. Metabolic Acidosis in Acute Myocardial Infarction. Brit. Med.J., 2:383-385, 1966.
9. LOGAN, R.W., MURDOCH, W.R. Blood Levels of Hydrocortisone, Transaminases, and Cholesterol after Myocardial Infarction. Lancet, 2:521-524, 1966.
10. OLIVER, M.F., KURIEN, V.A. and GREENWOOD, T.W. Relation between Serum-Free-Fatty-Acids and Arrhythmias and Death after Acute Myocardial Infarction. Lancet, 1:710-714, 1968.

11. KURIEN, V.A. and OLIVER, M.F. Serum-Free Fatty Acids after Acute Myocardial Infarction and Cerebral Vascular Occlusion. Lancet, 2:122-127, 1966.
12. KIRKEBY, K., HJERMANN, I., BJERDEDAL, I. The Fatty Acid Composition in Serum Following Myocardial Infarction. Acta Med. Scand., 183:149-159, 1968.
13. VALORI, C., THOMAS, M. and SHILLINGFORD, J.P. Urinary Excretion of Free Noradrenaline following Acute Myocardial Infarction. Lancet, 1:127-130, 1967.
14. WELIN, G. Serum Cholesterol in Cardiac Infarction. Nord. Med. 37:324-326, 1948.
15. LEREN, P. Variations in Serum Cholesterol Concentration in Myocardial Infarction. J. Oslo City Hosp., 10:55-59, 1960.
16. WATSON, W.C., BUCHANAN, K.D., and DICKSON, C. Serum Cholesterol Levels after Myocardial Infarction. Brit. Med. J., 2:709-712, 1963.
17. DATEY, K.K. and NANDA, N.C. Hyperglycemia after Acute Myocardial Infarction. New Eng. J. Med., 276:262-265, 1967.
18. KIRBY, B.J. and McNICOL, M.W. Acid-Base Status in Acute Myocardial Infarction. Lancet, 2:1054-1056, 1966.
19. LEDINGHAM, I.McA., and NORMAN, J.N. Acid-Base Studies in Experimental Circulatory Arrest. Lancet, 2:967-969, 1962.
20. ANDERSON, R., GARDNER, F.V. and HONEY, H. et al. Relation between Metabolic Acidosis and Cardiac Dysrhythmias in Acute Myocardial Infarction. Brit. Heart J., 30:493-496, 1968.
21. CAULFIELD, J., and KLIONSKY, B. Myocardial Ischaemia and Early Infarction: An Electron Microscopic Study. Amer. J. Path., 35:489-523, 1959.
22. WARTMAN, W.B., JENNINGS, R.B., YOKOYAMA, H.O. and CLABAUGH, G.F. Fatty Change of the Myocardium in Early Experimental Infarction. Arch. Path., 62:318-323, 1956.

23. EVANS, J.R. Importance of Fatty Acid in Myocardial Metabolism. Circulation Research, XV Suppl. II: 96-105, 1964.
24. OWEN, P., THOMAS, M. and OPIE, L. Relative Changes in Free Fatty Acid and Glucose Utilization by Ischaemic Myocardium after Coronary-Artery Occlusion. Lancet, 1:1187-1190, 1969.
25. SOLOFF, L.W., and SCHWARTZ, H. Relationship between Glucose and Fatty Acids in Myocardial Infarction. Lancet, 1:449-452, 1966.
26. RIFKIND, B.M. Plasma Free Fatty Acid Levels and Intravenous Glucose Tolerance after Myocardial Infarction. J. Atheroscler. Res. 6:26-35, 1966.
27. EDDY, J.D., O'BRIAN, E.T. and SINGH, S.P. Glucagon and Haemodynamics of Acute Myocardial Infarction. Brit. Med. J. 4:663-665, 1969.
28. DIAMOND, G., FORRESTER, J., DANZIG, R., PARMLEY, W.W. and SWAN, H.J.C. Acute Myocardial Infarction in Man: Comparative Hemodynamic Effects of Norepinephrine and Glucagon. Amer. J. Cardiol. 27:612-616, 1971.
29. KULA, J.J. and CROSS, E.B. Diet Therapy: Principles in the Management of Myocardial Infarction Patients. Proceedings of the Third Joint Meeting of the Clinical Society and Commissioned Officers Association of the U.S. Public Health Service, p. 61, 1968.
30. FRIEDBERG, C.K. Disease of the Heart, 3rd Ed., Philadelphia, Pa. W. B. Saunders Company, 894-895, 1966.
31. WOHL, M.G. and GOODHART, S. Modern Nutrition in Health and Disease, 4th Ed., Philadelphia, Pa. Lea & Feibiger, 875-876, 1968.
32. NITE, G. and WILLIS, F.N. The Coronary Patient: Hospital Care and Rehabilitation, New York, N.Y. MacMillan Company, 136, 1964.
33. HURST, J.W. and LOGUE, R.B. (Editors). The Heart, Arteries and Veins, 2nd Ed., New York, N.Y. McGraw-Hill Company, p. 1005, 1970.

34. Dietetic Policy in Levine Cardiac Unit, Personal Communication, Peter Bent Brigham Hospital, 1971.
35. Dietetic Policy in NIH Clinical Center, Personal Communication, National Heart and Lung Institute, Bethesda, Maryland, 1971.
36. SMITH, D.W. et al. Care of the Adult Patient, Philadelphia, Pa., J. B. Lippincott Co., p. 1059, 1971.
37. GRACE, W. and KEYLOUN, V. The Coronary Care Unit, New York, N.Y. Appleton-Century-Crofts, Inc., p. 193, 1970.
38. EARLES, V. Fluids, Electrolytes and Circulation. Cardiovasc. Nurs., 6:2, 1970.
39. LEVINE, S.A. Clinical Heart Disease, 5th Ed., Philadelphia, Pa., W. B. Saunders Company, p. 147, 1958.
40. GOLDBERG, E. (Ed.) Dangers of a Low-Sodium Diet in the Treatment of Myocardial Infarction. Amer. J. Cardiol. 8:300-301, 1961.
41. MERKEL, R. and BROWN, C. Evaluating Feeding Activities in the CCU. Amer. J. Nurs., 70:2348-2350, 1970.
42. SODI-PALLARES, D., BISTENI, A., MEDRANO, G.A. et al. The Polarizing Treatment for Myocardial Infarction. Amer. J. Cardiol. 24:607-608, 1969.
43. ARNOTT, W.M. Potassium-Glucose-Insulin. Amer. Heart J., 77:845-846, 1969.
44. MAROKO, P.R., LIBBY, B.A., SOBEL, B.E. et al. Effect of Glucose-Insulin-Potassium Infusion on Myocardial Infarction Following Experimental Coronary Artery Occlusion. Circulation, 45:1160-1175, 1972.
45. HOOD, B., SANNE, H., ORNDAHL, G. et al. Long-Term Prognosis in Essential Hypercholesterolemia: Effect of Strict Diet. Acta Med. Scand., Suppl. 446, 1966.
46. DAYTON, S., PEARCE, M.J., HASHIMOTO, S. et al. A Controlled Clinical Trial of a Diet High in Unsaturated Fat. Amer. Heart Assn. Monog. 25, 1969.

47. BIERENBAUM, M.L., GREEN, D.P., FLORIN, A. et al. Modified Fat Dietary Management of the Young Male with Coronary Disease. JAMA, 202:1119-1123, 1967.
48. LEREN, P. The Effect of Plasma Cholesterol-Lowering Diet in Male Survivors of Myocardial Infarction, a Controlled Clinical Trial. Acta Med. Scand., Suppl. 446, 1966.
49. NELSON, A.M. Treatment of Atherosclerosis by Diet. Northwest Med., 55:643-649; 792-795; 874-876, 1956.
50. National Heart and Lung Institute; The Dietary Management of Hyperlipoproteinemia, A Handbook for Physicians, Bethesda, Md. 1970.
51. STONE, N.J. and LEVY, R.I. Hyperlipoproteinemia and Coronary Heart Disease. Prog. Cardiovas. Disc. 14:341-359, 1972.