où l'inoculation a été faite, et repiquer un autre animal. On arrivera ainsi à obtenir finalement une culture du *Pl. tetani.*

On pourra suivre dans les trois dernières illustrations, l'évolution de ce deuxième cas. Ce jeune homme, lui aussi, est maintenant guérit.

Il ne fait toutefois aucun doute, que l'issue heureuse du traitement dans ces deux cas est attribuable aux méthodes thérapeutiques bien établies comme le débridement des plaies, les antibiotiques, les anti-toxines, les soins, la trachéotomie et le maintient adéquat de la nutrition, de l'hydratation et de la ventilation pulmonaire. Néanmoins, nous croyons maintenant que le méthocarbamol est une drogue sûre et efficace dans le traitement spécifique du spasme tétanique.

RÉSUMÉ

Le méthocarbamol administré à des lapins tétanisés semble plus sur et aussi efficace que le curare. Deux cas de tétanos, prouvés bactériologiquement, sont présentés où le médicament a réussi à juguler les crises tétaniques. Il est évident qu'on doit associer cette médication aux méthodes thérapeutiques bien établies.

La façon la plus sûre et la plus rapide d'identifier l'agent du tétanos consiste à inoculer deux ou trois cobayes avec une bonne quantité du matériel présumé tétanigène.

Nous tenons à remercier les Docteurs Claude Marchand et André Gilbert pour leur étroite collaboration dans la préparation de ce travail.

SUMMARY

The Use of Methocarbamol in Tetanus: Experimental and Clinical Study.

There is some evidence that the toxin of tetanus selectively depresses the normal presynaptic inhibition of neurons with multisynaptic reflexes, thus permitting presynaptic stimulation to take place. Pharmacologically, methocarbamol depresses the synaptic transmission at the level of these neurons and diminishes pre-existing stimuli, thus cutting down the spasm of tetanus.

In experimentally tetanized rabbits, methocarbamol (Robaxin) was found to be more effective and more predictable than curare. Two patients with severe tetanus, bacteriologically proved, were completely cured after the administration of this drug. Methocarbamol was given intravenously and by the intragastric route up to a high dose of 16 g. without ill effects. In particular, respiratory depression was not noted.

The authors stress that besides this new drug the established therapeutic measures, such as careful debridement, administration of antibiotics and antitoxins, and maintenance of respiration remain very important.

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REVIEW ARTICLE

DIET AND DEGENERATION*

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THE SO-CALLED degenerative diseases are now the major cause of death in our population, and there is growing evidence to suggest that malnutrition plays an important part in the etiology of several of these disorders.

Fifty years ago the average life expectancy at birth was only 48 years.[1] This was partly owing to the high mortality from infectious diseases such as pneumonia and tuberculosis. At that time, the science of nutrition was concerned with the diet required for maximum growth and for the prevention of deficiency and infectious diseases. In other words, a diet for optimal health was provided for a young population. Today, deficiency and infectious diseases have largely been controlled and the average life expectancy at birth has increased to 67 years. People now live to the age where they suffer and die from degenerative diseases such as atherosclerosis, diabetes and hypertension. Even though the degenerative diseases usually become apparent in later life, some, such as atherosclerosis, have their beginning in youth. The attitude that degenerative diseases are the inevitable accompaniment of ageing, and need not arouse concern, is not justified. The fact is that they exist, and perhaps they can be delayed or even prevented. It is possible that the dietary standards of the past, based on maximum growth and development in the young, are conducive to the early development of degenerative diseases and are not compatible with optimum longevity and health in adult life.

McCay, a nutritionist who has studied longevity,[2] has written as follows:

". . . . almost all attention has been devoted to the study of growing animals to the neglect of the adult. . . . . the reason is due to the ease of studying young animals. The diseases that appear in old age may have started but are unobserved in the young. . . . the growing body is a seat of very rapid chemical changes. As a result the effects of different diets become evident

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very quickly. . . . in the adult, it requires long protracted efforts to produce dietary effects. . . . many of the current concepts concerning the nutrition of mature animals are questionable because they are derived from reasoning by analogy from young immature animals. Hence the decisions regarding the diets of adults usually represent compromises between food habits established by long usage and modern evidence based upon experiments with developing animals."

McCay fed rats a diet containing all the essential food elements but so restricted in calories as to retard normal growth and maturation. The life span of some of the rats was greatly prolonged because neoplasms and lung degeneration which usually terminate life in this species occurred less frequently. Unlike man, the rat does not die of degenerative cardiovascular disease. This work does not imply that restriction of calories and retardation of growth in man will necessarily promote better health and longevity, but it does indicate that nutrition influences degenerative diseases in one species and suggests that it might influence other degenerative diseases in other species.

There are several reasons why nutritional requirements should alter with age. Fewer calories are needed by mature and ageing animals because of cessation of growth, decreasing physical activity and lower metabolic rate. In Western civilization, the serum lipid concentrations increase at or after maturity,[3, 4] while the plasma lipemia clearing factor activity decreases with age.[5, 6] Older people have decreased fat tolerance as indicated by the greater rise and slower fall of the plasma chylomicron content after a fat meal.[7] These changes may signify an altered requirement for the amount and kind of dietary fats. The decreasing glucose tolerance[8a] and the increasing incidence of diabetes[8b] with age also suggest that older people have a slower metabolism with different nutritional requirements than younger people.

Therefore, it seems reasonable to re-evaluate present dietary standards in the light of the needs of our ageing population. The rational objective today should be the provision of a diet designed to promote maximum health throughout a long life. This would provide for optimum growth, prevention of deficiency disease and maximum freedom from infectious and degenerative diseases resulting in an optimum period of useful adult life. "Optimum growth" does not necessarily mean maximum growth since the latter may be incompatible with "maximum health throughout life".

There should be no careless recommendation of "restricted diets" which might compromise good health by increasing deficiency and infectious diseases. McCay recognized this danger in his longevity studies on animals.[2] (Lord) Boyd-Orr has provided evidence that vitamin and other dietary deficiencies were associated with less growth, more illness and increased mortality rate in humans.[9] It should be noted that his studies did not include the degenerative diseases in older decades. Stare[10] reported a high incidence of deficiency and infectious diseases among the Dutch who were subjected to multiple dietary deficiencies and starvation in 1944. It was noted that they also had a decreased incidence of atherosclerotic disease. This report has often been quoted as evidence of the role of nutrition in degenerative disease. It would seem more reasonable to infer that while nutritional factors may influence degenerative diseases, dietary regimens for the prevention of degenerative conditions must not produce deficiency disease and unusual morbidity and mortality from other causes.

Of the common degenerative diseases, diabetes mellitus, hypertension and atherosclerosis appear to be influenced by nutritional factors. It is certain that nutrition is not the only factor concerned in the cause or cure of all of these conditions. In diabetes there is a hereditary factor which predisposes certain individuals to the disease, but, in addition, among the causes listed by Joslin for the increasing incidence of diabetes are "the lessening need for physical work and the abundance of food consumed with resulting obesity".[8c] This opinion is supported by the fact that of adults who become diabetic, 80% are overweight.[8d] The therapeutic value of carbohydrate and calorie restriction in the treatment of diabetes is obvious. In the case of hypertension, the incidence of this disease among various population groups may be influenced by the salt content of the diet.[11, 12] Increasing the salt content of the diet of experimental animals aids in initiating hypertension. In human sufferers, salt restriction may help lower the blood pressure. With regard to atherosclerosis, there is strong evidence that nutritional factors are etiologically important. In 1908 Ignatovski first produced experimental atherosclerosis by feeding rabbits milk and eggs which are rich in lipids.[13] This observation has been confirmed by many other workers.[14] Atherosclerosis produced experimentally by dietary means resembled the human disease in many respects, but certainly not in all. Thrombosis, which is so commonly associated with human atherosclerosis, was absent in these early studies.

Later investigators considered that the cholesterol content of foods was the important atherogenic factor, and fed pure cholesterol in abnormally large amounts to rabbits, thereby raising their blood cholesterol to high levels and producing atheromatous lesions in their arteries. This may have been unfortunate, because subsequent workers concentrated almost exclusively on the study of the cholesterol component in food and blood. It is only recently that other foods and blood fats have been considered of equal or greater importance in atherogenesis. Atherosclerosis has been induced in several other species by feeding cholesterol and various other types of fat. Except by grossly abnormal measures,[15, 16] all animal experiments failed to produce an associated thrombosis which is so common in human atherosclerosis. Recently, Tay-

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lor,[17] by feeding monkeys a human-type diet which greatly raised the serum cholesterol for many months, produced tendon xanthomata, widespread atherosclerosis and thrombosis with myocardial infarction in one animal and gangrene of the lower limb in a sibling. This is a dramatic duplication of human familial essential hypercholesterolemia with its clinical sequelae and typical atherosclerotic lesions and complications.

In man there appears to be some relationship between elevated blood lipids and atherosclerotic disease. Conditions such as diabetes, hypothyroidism, nephrosis, hyperlipemia and hypercholesterolemia, in which there are abnormally elevated serum lipid levels, are characterized by an increased incidence of atherosclerotic disease. As a group, humans below age 60 who have clinically evident atherosclerosis have significantly elevated serum lipids.[18] Epidemiological studies of various population groups throughout the world have linked a low incidence of atherosclerotic disease, low-fat diet and low serum lipid level.[19] It must be noted that the accuracy, significance and interpretation of these epidemiological studies are open to criticism.[20]

The link between elevated serum lipid concentration and atherosclerotic lesions in the artery wall is not clear. Direct infiltration of serum lipids into the artery wall with resulting fibrosing effects offers one possible explanation. Stimulation of the blood-clotting mechanism[21, 22] and inhibition of the blood fibrinolysins[23] by the blood lipids (as well as other substances) with the repeated layering of platelets,[24] fibrin and thrombi upon the lining membrane is another possible mechanism, which has been described as the encrustation theory of atherosclerosis. This might also explain in part the large thrombi which frequently occlude the narrowed vessel in the end stage of atherosclerotic disease.[16]

The serum lipids are a mixture of several compounds combined with the serum proteins in various proportions to form a complex series of lipoproteins. The lipid fraction which is most frequently determined quantitatively is the total serum cholesterol. The several phospholipid fractions are usually determined quantitatively as a single group on the basis of the phosphorus content of the serum lipids. The determination of the concentration of the individual phospholipids (lecithin, phosphatidyl ethanolamine, phosphatidyl serine, phosphatidyl inositol and sphingomyelin) is not yet practical. The serum also contains triglycerides (neutral fats) of varying types which vary in concentration much more than the other lipid fractions. After a fat meal, there is an increase in triglycerides which are suspended in the serum as fat droplets or chylomicrons, giving it a cloudy appearance.

An enzyme system, the plasma lipemia clearing factor, hydrolyses the triglycerides, releasing fatty acids, and may be responsible in part for the clearing of triglyceride from the blood within the normal period of eight hours postprandially.[25] A non-esterified fatty acid fraction is also present and is a very important source of energy for body cells.[25] The lipoprotein combinations of all these lipid fractions with proteins are estimated by physical methods such as electrophoresis and ultracentrifugation. Numerous workers have attempted to find which lipid fraction most closely relates to the incidence of atherosclerotic disease. So far, all of the fasting serum lipid fractions, including total, ester and free cholesterol, total phospholipid and the standard S_f 0-400 lipoprotein fractions show approximately equal discriminative values.[18] Although statistically significant in a large group of patients, these correlations are not sufficiently high to permit the use of any serum lipid level as a diagnostic test for clinical atherosclerotic disease in the individual patient.

A major difficulty in human studies arises from the fact that subclinical atherosclerosis is universal in our adult population. The fact that there is even a slight average increase in serum lipids in those with clinical atherosclerosis is all the more significant. The mean total serum cholesterol concentration for "normal" men over 30 years of age, in Toronto, is 210 mg. %,[26] which is considerably greater than the mean level of 150 mg. % reported in areas with a lesser incidence of atherosclerotic disease.[27] The relationship between serum cholesterol and the incidence of atherosclerosis is only an association of findings and not a proved causal relationship. Still, the possibility exists that even the Toronto normal mean serum cholesterol level of 210 mg. % is excessive and conducive to an unnecessarily high incidence of atherosclerotic disease.

Triglyceride concentration in the serum of each individual is quite variable and rather difficult to estimate. Unfortunately, because of this, studies of its relationship to atherosclerosis have been neglected. A recent report indicates that serum triglyceride concentration was more closely correlated with atherosclerotic disease than total serum cholesterol.[28] Inability to determine the separate phospholipid fractions accurately has prevented their study in atherosclerosis. This is particularly unfortunate since the phospholipid complex appears to contain both a stimulator and an inhibitor of coagulation. Although their identity is controversial, phosphatidyl ethanolamine may be the stimulating factor and phosphatidyl serine may be the inhibitor.[29]

Of special interest to practising physicians and to dietitians are the nutritional factors which affect the concentration of serum lipids. Unfortunately, most of the investigations in this field have utilized only total serum cholesterol as an index of the serum lipid concentrations. It is necessary to know the effect of various foods on all the serum lipid and lipoprotein fractions and not just cholesterol, since unusual elevations of triglycerides or other

fractions may occur as a result of dietary changes despite normal serum cholesterol levels.[30, 31]

The influence of dietary cholesterol on human serum lipid levels is still controversial. Endogenous production of cholesterol by the liver is greater than the amount contained in natural diets richest in this substance. Therefore, cholesterol *per se* has been considered of little importance in human diet. However, recent experiments by Beveridge[32] have shown that over the lower ranges of cholesterol ingestion, beginning at zero level, there is a positive relation between intake and serum cholesterol concentration. This is modified by the type of triglyceride also present in the diet. It remains to be seen if this dietary effect would persist longer than the eight-day period of these experiments or if, in the natural human diet, these amounts of cholesterol are important.

A period of weight loss and negative caloric balance usually results in a decreased serum cholesterol concentration, whereas weight gain and positive caloric balance have the opposite effect. This is important in experimental design and means that caloric balance must be maintained to avoid misinterpreting the effects of various foods on serum lipids. There is as yet no therapeutic justification for prolonged nutritional deprivation except in the presence of obesity.

A low fat diet, containing 10-20% of its calories as fat, will lower serum cholesterol in many cases. In our country, such a diet is unpopular and difficult to maintain because we are accustomed to diets with approximately 40% of calories as fat. The "low fat diet" has become obsolete and unnecessary with the discovery that certain types of fat tend to lower serum lipids while other types of fat tend to produce serum lipid elevation. Among the fats with the greatest hyperlipidemic effect are egg yolk, milk fat and coconut oil.[30] (Coconut oil is a constituent of some butter substitutes.) Many fats such as those from meats, peanuts and olives have a lesser hyperlipidemic effect. Corn oil, cotton seed oil and marine oils tend to lower serum lipids under certain conditions. Substitution of the fats with hypolipidemic effect for those with hyperlipidemic effect, in a diet containing 40% of calories as fat, will effectively lower serum lipids.[30, 31, 33] This may be most aptly termed an "altered fat diet".

The isolation of the factor or factors in foods responsible for raising and lowering serum lipids is the subject of much current research. It would appear that several different factors may be responsible. Saturated fatty acids and short-chain fatty acids, as in butter and coconut oil, tend to raise serum lipids, whereas the essential fatty acids such as linoleic and arachidonic as well as other polyunsaturated fatty acids tend to lower serum lipids.[30] Hydrogenation of oils, as in margarines, alters the structure of the fatty acids, and the saturated acids which are produced are hypercholesterolemic. The sterols may also be important. Cholesterol, the animal sterol, raises serum lipids,[32] while the plant sterols lower serum lipids.[34] Extreme variations of dietary protein affect serum lipids,[35] but this probably does not apply to the more than adequate protein content of the Canadian diet. Unusually high carbohydrate diets have resulted in marked elevations of the serum triglyceride fraction without elevation of cholesterol.[30] By practical necessity a low fat diet is a high carbohydrate diet and although this may lower serum cholesterol concentrations, serum triglycerides may become elevated. Since there is no evidence that elevated serum triglyceride levels are harmless, there is no justification for blindly prescribing a low fat diet. Preliminary animal experiments have shown that different dietary carbohydrates have a varied effect on the rate of lipid absorption, serum lipid concentrations and atherogenesis.[36-38] Similar studies on humans have not as yet been reported.

The effect on serum lipids of feeding various phospholipids has been investigated to some extent. As mentioned above, eggs and dairy fats tend to raise serum lipid levels, and in experimental animals they have produced atherosclerotic lesions. Eggs and dairy fat contain phospholipids in addition to cholesterol and other fats. Mustard[39] has fed a lipid extract rich in phosphatidyl ethanolamine to rabbits and produced elevated serum lipids and atherosclerosis, whereas material rich in phospatidyl serine had no such effect. Both of these extracts contained small amounts of cholesterol and other lipids. He points out that eggs and dairy fats contain phosphatidyl ethanolamine in greater amounts than phosphatidyl serine, and it is possible that the phospholipids, in addition to cholesterol, may account for the hyperlipidemic effect of these foods. Amatuzio and Hay[40] found that egg yolk phospholipids were hyperlipidemic for patients with primary hyperlipemia.

Another approach to the study of diet and serum lipids has been based on the correlation of results of analyses of the diets of groups of individuals with their serum lipid values. From the results of such studies, there appears to be a relationship between the average amount of fat eaten and the average serum lipid concentrations in people from different world areas.[19] This relationship is an association which is not necessarily causal. Many variables other than diet also might account for these differences in serum lipids, such as race, exercise, climate and other factors. Because of this, it is interesting to note that in two groups of men, one with coronary atherosclerotic heart disease and the other a matched control group, both living in Toronto and both similar in nearly every respect except that the coronary group is slightly less active physically, the serum lipids are significantly higher in those with coronary atherosclerosis. Yet Table I shows that the control group tends to consume more calories while both groups eat the same percentage of calories as fat. These data are based on week-long dietary records of everything each

TABLE I.—DIETARY ANALYSIS AND TOTAL SERUM CHOLESTEROL IN HEALTHY CONTROLS AND MALES WITH CORONARY HEART DISEASE

Decade	*4th*				*5th*			
	No.	*Total calories*	*Fat as % of calories*	*Total serum chol.*	*No.*	*Total calories*	*Fat as % of calories*	*Total serum chol.*
Control								
Mean	16	2565	36.9	206.0	14	2260	39.8	212.0
± S.E.		72	1.3	8.0		185	1.1	10.7
Coronary								
Mean	17	2250	35.8	273.0	19	2154	37.3	259.0
± S.E.		132	2.2	10.4		136	1.3	6.4
p		<.05	<1.0	<.001		<1.0	<.5	<.001
Decade	*6th*				*7th*			
	No.	*Total calories*	*Fat as % of calories*	*Total serum chol.*	*No.*	*Total calories*	*Fat as % of calories*	*Total serum chol.*
Control								
Mean	22	2109	37.2	203.0	18	2148	38.1	214.0
± S.E.		85	1.1	6.5		145	1.3	9.6
Coronary								
Mean	23	1977	36.5	244.0	19	1612	36.5	228.0
± S.E.		111	.94	10.0		94	1.3	8.3
p		<.5	<1.0	<.001		<.01	<1.0	<0.1

individual eats. The only difference in the average consumption of eggs, milk, cream, ice cream, butter or cheese is that the control group tends to eat more foods containing milk fat than the coronary group, as shown in Table II. Under the conditions of this experiment, it is not possible to say that the kind and amount of fat in the diet of both groups is identical in every respect but it appears unlikely that there are any important average differences. The most probable explanation for the higher serum lipids in those with coronary disease is that they are more susceptible to the effects of dietary fats with an unusual hypercholesterolemia.[32] Our own unpublished studies, in agreement with others,[31] indicate that substitution of corn oil for eggs and butter in the diet of a diabetic lowers serum cholesterol significantly. From this and the fact that diabetics are unusually susceptible to atherosclerosis, it would seem reasonable to question the traditional use of eggs and dairy products in their diets. A long-term trial of the effect of other dietary fats on the serum lipids and the incidence of atherosclerotic disease in diabetics would constitute a potentially valuable study.

TABLE II.—WEEKLY AMOUNTS OF EGGS AND MILK PRODUCTS CONSUMED BY MEN IN FOURTH AND FIFTH DECADES AND THE TOTAL SERUM CHOLESTEROL (MEAN ± S.E.)

Group No.	*Eggs*	*Cheese oz.*	*Butter g.*	*Ice cream g.*	*Whole milk oz.**	*Total serum chol. mg.%*
Control 30	6.13 ±.87	4.13 ±.72	162.0 ±18.5	34.0 ±12.0	73.2 ±11.8	209.2 ±6.5
Coronary 36	6.30 ±.66	2.29 ±.34	140.0 ±25.0	41.0 ±10.0	40.0 ±5.8	265.4 ±6.0

*On the basis of its fat content, all milk and cream was converted to oz. of whole (4%) milk, i.e. 2 oz. of 2% milk equals 1 oz. of 4% milk.

hyperlipidemic dietary fats because of some inherent difference in metabolism.

There are three recognized inherited metabolic conditions in which fasting serum lipids are abnormal and in which there are abnormal responses to fat foods. The fasting serum lipid values for each one of these diseases are shown in Table III.

The commonest one is diabetes mellitus. Diabetics as a group have increased fasting serum lipids, the greatest proportion being in the triglyceride fraction.[41] They may respond to certain

Familial essential hypercholesterolemia is a condition characterized by clear fasting serum, elevated serum cholesterol, relatively low or normal serum triglyceride, active plasma lipemia clearing factor and a marked tendency for atherosclerotic disease at an early age. Serum cholesterol can be decreased by avoiding eggs and dairy fats and by substitution of corn oil and other unsaturated fats as in the case illustrated in Fig. 1. It is usually not possible to achieve the very low serum lipid levels obtainable by diet in normal people.

TABLE III.—SERUM LIPID FRACTIONATIONS IN HEALTHY MALES AND PATIENTS WITH METABOLIC DISEASE

Patients	*121 men 30 - 80 years*	♀ *34 years Familial ess. hyper-cholesterol-emia*	♀ *25 years Familial primary hyper-lipemia*	♂ *40 years*
Diagnosis	*Healthy*			*Untreated diabetic*
	Mean and 95% Limits			
Total cholesterol mg.%	211 143-279	343	225	331
% free cholesterol	28.4 25.6-31.2	26	44	30
Phospholipid mg.%	251 187-315	288	250	350
Std. Sf liproproteins				
0-12 mg.%	290 186-394	580	161	285
12-20 mg.%	53 29-77	32	40	48
20-100 mg.%.....	70* 23-209	6	182	296
100-400 mg.%	20* 3-126	0	141	409
Triglyceride fatty acid, mEq./l. (45)	6.4** 3.9-9.2	6	41	49
Plasma clearing factor, units (46)	.15*** .026-.274	.25	.00	.10
Fasting serum	clear	clear	creamy	creamy

*Geometric mean, log normal distribution.
**On 26 normal men, aged 30-55, non-parametric mean.
***Non-parametric mean.

Primary hyperlipemia is another inherited metabolic defect in which there is decreased or absent plasma lipemia clearing factor, cloudy or milky serum due to very high serum triglyceride levels, high or low serum cholesterol levels and the likelihood of early onset of atherosclerotic disease. In these patients, after a fat meal, there is very slow clearing of triglyceride from the blood. Therefore, "spaced fat feedings" have been recommended[42] whereby most of the day's quota of fat is included in one of the three meals, thus allowing 24 hours for clearing the blood. Some of these persons appear to be unusually sensitive to the ingestion of eggs, dairy fats, coconut oil and alcohol, which is followed by rapid, large increases in serum triglyceride.[40] Therefore, an altered fat diet is an important measure in the care of this group of patients as well.

Many patients with coronary atherosclerotic heart disease before the age of 50 have one of these three metabolic disorders. Actually, 95% of patients

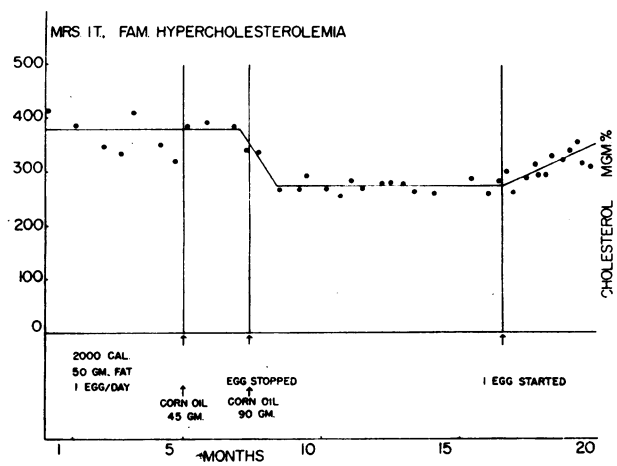

Fig. 1.—The effect of diet on the serum cholesterol level of a 34-year-old female with familial essential hypercholesterolemia (serum lipid fractionation shown in Table III). The dietary changes were isocaloric with no resulting weight changes.

dying with coronary heart disease are over 50 years of age,[43] and most of their fasting serum cholesterol levels and other fasting serum lipid fractions are similar to or only slightly greater than the average in our so-called normal population.[18] An altered fat diet nevertheless produces some decrease in serum lipids in these older patients. Whether or not this will delay the progress of atherosclerotic disease and prevent recurrences of coronary thrombosis has not been proved. In any case, the physician who prescribes such a diet should follow the patient's serum lipid levels to determine whether the diet is effective and observe the patient's clinical state for signs of continuing atherogenesis or dietary insufficiency. The dietitian should be certain that the diet has a full complement of all essential food factors.

TABLE IV.—ALTERED FAT DIET

Breakfast
1/2 oz. corn oil
1 fruit exchange
3 slices back bacon
2 bread exchanges
1 tbsp. jam
1/2 cup skim milk
coffee

10:00 a.m.
tea or coffee

Lunch:
1/2 oz. corn oil
3 meat exchanges—chicken or fish
1 vegetable exchange—List 2, List B
2 bread exchanges
1 fruit exchange
tea or coffee

3:00 p.m.
1 oz. walnuts
tea or coffee

Dinner:
1/2 oz. corn oil
3 meat exchanges
1 vegetable exchange—List 2, List B
2 bread exchanges
1 fruit exchange
tea or coffee

Bedtime:
2 bread exchanges
1/2 cup skim milk

P = 81 g.; C = 196 g.; F = 94 g.
Calories—1954 with 43% as fat and 63% of this as corn oil.
Exchange lists from the Canadian Diabetic Association Meal Planning Booklet.

Altered fat diets should be constructed with the same rigidity and limitations as a diabetic diet, with the amount of calories, carbohydrate, protein, fat and kind of fat food definitely specified. A sample diet of this type is shown in Table IV. As in the case of diabetes, it is necessary to teach the patient about dietary principles and to recall him periodically for estimations of his serum lipid concentrations. Inability to obtain a decrease in serum lipids, or the occurrence of an initial decrease followed by an increase, signifies some fault in the diet which requires detection. This may be caused by the use of baked goods rich in egg yolk, or of a margarine instead of an oil. The use of unsaturated oils in the diet requires ingenuity. Oil may be used in baking or frying, in gravies and in French

dressing. It may be brushed on vegetables and bread with a sprinkling of salt in the same way that one uses melted butter. It can be stirred into hot cereals. Certain nuts, such as walnuts, are rich in unsaturated fats.[44] By persistent dietary trial and error, it should be possible to lower most patients' serum lipid level unless some unrecognized metabolic disease such as diabetes, myxedema or nephrosis is present.

SUMMARY

Because of the high incidence of degenerative disease in our ageing population, the role of nutrition in the etiology and prevention of these conditions warrants investigation. Some of the available information on this subject has been reviewed and some details of the author's studies and dietary methods have been presented. It has been established that alteration of the type of fat in human diets, with substitution of certain fats for eggs and dairy fats, will lower serum lipids, but it is not yet proved that this will lessen morbidity or mortality from atherosclerotic disease and its thrombotic complications. The causal relationship between diet and atherosclerosis is plausible, but the proof is incomplete. In patients with this disease, alteration of the diet may be undertaken under the clinical supervision of the doctor and dietitian in an attempt to delay progress of the condition and to improve the prognosis. Prophylactic changes in dietary fat are not yet recommended for the entire population. Popular articles have represented the dietary lowering of serum lipids as a simple, established procedure. Actually, it is complicated, difficult and incompletely understood.

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WHAT MAKES YOUR DAY?

Our children's physician arrived unusually late for lunch, and remarkably contented with life. The morning had produced a clinical pearl, some rare syndrome with real honest-to-goodness physical signs that she could demonstrate to the students. It had made her day, shedding a glamour over an outpatient clinic normally recruited from the young owners of running noses and capricious appetites. We congratulated her and then asked each other to describe what transformed a routine session into a memorable one. What makes your day?

The radiologist admitted to an inordinate, if private, satisfaction when an x-ray revealed a lesion which had been unsuspected or overlooked by the clinicians. The pathologist enjoyed drawing blood (and marrow) from metaphorical stones. Our neurologist's cup of happiness overflows whenever he is able to establish an exact diagnosis: apparently this happens excessively rarely. The surgeon was spoiled for choice. All his performances were spectacular—we gathered that was the reason operations were done in theatres. But there were subtle joys—deftly setting a transfusion going into some secret, invisible, impalpable, fully-patented-in-all-major countries venule at the base of the little toe, after the anesthetist had ploughed up all the official veins. The anesthetist said, somewhat bitterly, that the sight of a surgeon including his own little finger in a ligature, or his screams as he snipped off the tips of his glove in the course of some fancy piece of scissor-work, rendered an operating list more than tolerable.

And we know, too, what makes a child psychiatrist's day. He told us, when he had worked his way through the 2s. 8d. luncheon, as he left for an unexpected afternoon's golf. It is a last-minute cancellation of outpatient appointments en masse.—(Peripatetic correspondent) *Lancet*, 2: 977, 1960.