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THE EFFECT OF ARTIFICIAL MODIFICATION OF FOOD ON THE SERUM CHOLESTEROL LEVEL*

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THERE ARE SEVERAL good reasons for believing that serum cholesterol is etiologically related to human atherosclerosis. Cholesterol is abundantly present in atheromata;[1] the feeding of cholesterol to a wide variety of experimental animals, including primates, results in the production of atherosclerosis and is associated with striking rises in serum cholesterol.[2] Epidemological studies have shown a good correlation between serum cholesterol levels and coronary heart disease in many countries throughout the world.[3] Many clinical studies have shown a relative increase in serum cholesterol level in persons suffering from the clinical manifestations of atherosclerosis.[4]

There is also much evidence to suggest that individual serum cholesterol levels are significantly influenced by the diet and especially by its fat content. Removal of the cholesterol alone from the diet has relatively little effect on serum cholesterol levels.[5] Removal of fat from the diet results in a sharp drop in serum cholesterol levels,[6] but, as will be shown below, this has a drastic effect on lipid metabolism and transport. Substitution of dietary fats rich in polyunsaturated fatty acids for those rich in saturated fatty acids results in a sharp decline in serum cholesterol levels and appears to be related fundamentally to differences in fatty acid structure (i.e. molecular configuration).[7] The cholesterol-lowering effect is probably not related to trace factors such as vitamins, minerals, sterols or other undefined non-fatty acid substances. There is good evidence to suggest that not only the essential fatty acids, but also a wide array of polyunsaturated fatty acids, possess the ability to lower serum cholesterol levels.[7]

The exact mechanism of action of the unsaturated fatty acids is not agreed upon. The author has presented data which suggest that the hypocholesterolemic effects of corn oil and/or ethyl linoleate concentrate are really due to the removal of the usual dietary fats or of some substance closely associated with them, and that the corn oil or linoleate exercises a bland or neutral effect on the serum cholesterol level, permitting it to attain its natural base-line level.[8] However, Beveridge[9] has demonstrated a cholesterol-depressant action of corn oil with formula-type diets, and Kinsell[10] has shown a similar effect with linoleic acid.

This paper is a report of further observations on the effect of dietary alterations on serum cholesterol levels in man. It is believed that these observations may have important clinical implications in the management of hypercholesterolemic states.

MATERIALS AND METHODS

The subjects were student volunteers in good health who carried on with their regular duties during the experiments. Total caloric intake, as well as the percentage of calories taken as fat, carbohydrate and protein, was calculated from dietary histories taken during the control period. During the study periods, which lasted from one to three weeks, a basal low-fat diet was used to which the various test fats were added. In every instance the protein intake was held constant, and fat was substituted for carbohydrate. The composition of the basal diet is shown in Table I.

TABLE I.—THE AVERAGE DISTRIBUTION OF CALORIES IN, AND CHOLESTEROL CONTENT OF, THE CONTROL AND LOW-FAT DIETS

	Percentage of calories yielded			
Diet	*Protein*	*Fat*	*Carbohydrate*	*Cholesterol (mg.)*
Control.....	14	45*	41	512
Low fat.....	16	4*	80	28

*By analysis; all other figures from tables.

Subjects were weighed twice a week and caloric intake was adjusted to keep the body weight constant during the experiments.

Lipids were estimated twice weekly on serum obtained from patients in the fasting state. Serum cholesterol level was determined by the method of Abell,[11] serum triglycerides by Van Handel and Zilversmit's method,[12] and lipoproteins by Boyd's modification of paper electrophoresis.[13] Beta lipoprotein cholesterol value was determined by Scanu's method.[14]

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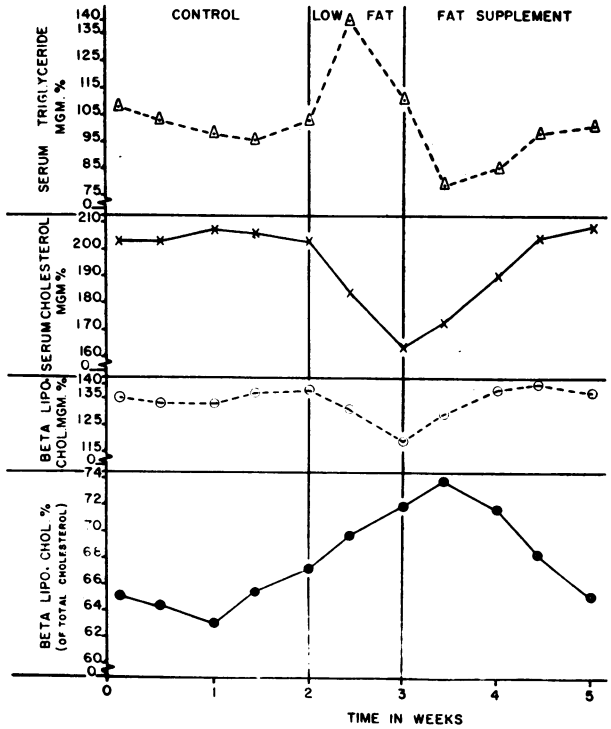

Fig. 1.—Summary of data on eight subjects. During the fat supplement period, four subjects received butter fat and four subjects received partially hydrogenated blended corn oil margarine. As there was no difference in the results between these groups (Fig. 2B), they were pooled to produce this summary figure. Discussion in text.

RESULTS

(A) *The Effects of Rigid Fat Restriction on the Serum Lipids and Lipoproteins*

Fig. 1 shows the effect of a very low fat basal diet (4% of calories from fat) which was also a very high carbohydrate diet (80% of calories from carbohydrate). There was a sharp fall in serum cholesterol level of approximately 20% during the one-week low fat period. The fall in beta lipoprotein cholesterol level was less marked proportionately, and the per cent beta lipoprotein cholesterol therefore rose from 66 to 74%. There was also a sharp rise in the serum triglyceride levels. The addition of a fat supplement (butter in four subjects and corn-oil margarine in four subjects) resulted in prompt return of all indices to the control levels. The rise in serum triglycerides was a constant phenomenon whenever the very low fat diet was used.

(B) *The Effects of Adding Corn Oil to a Low Fat Diet*

This is clearly illustrated in Tables II and III. In Table II we have reported the results of short-term dietary studies in six subjects. After one week on a very low fat diet, there was no further fall in serum cholesterol level when corn oil was added at 40% of calories level, replacing an equicaloric amount of carbohydrate. In Table III, however, we note that corn oil produced a favourable modification of the lipoprotein transport mechanism, increasing the proportion of alpha lipoprotein.

TABLE II.—MEAN VALUES OF SERUM CHOLESTEROL FOR CONSECUTIVE DIETARY PERIODS

	Control period	*Low-fat period*	*Corn-oil period*
Duration of period.....	1 week	1 week	2 weeks
No. of subjects.....	6	6	6
No. of determinations.....	3	2	4
Serum cholesterol mg.%...	204.5*	157.5*	155.7*

*Mean value for each period.

TABLE III.—CHANGES IN BETA LIPOPROTEIN % WITH CONSECUTIVE DIETARY PERIODS

	A		*B*		*C*	
	Control	*Corn oil*	*Control*	*Low fat*	*Low fat*	*Corn oil*
Subjects	8	8	14	14	6	6
Observations	23	25	53	56	19	20
Mean	72.6	66.8	75.45	75.80	71.06	67.25
P	0.1				0.2	

(C) *The Effect of Natural Modification and Processing of Foodstuffs on Serum Cholesterol Levels.*

These data, and that of others, make it clear that the substitution of corn oil for the usual dietary fats almost invariably results in a sustained fall of 20-30% in serum cholesterol levels. From a practical point of view, it would seem reasonable to incorporate the vegetable oil into the diet by modifying or processing natural foodstuffs in such a way as to replace their saturated fatty acids by the unsaturated ones found in corn oil. We have previously reported on the modification of the yolk fats of hens' eggs produced by feeding hens 10% sunflower seed oil.[15] This resulted in a six-fold increase in the unsaturated fatty acids, nearly all of which was due to an increase in linoleic acid. These eggs were then subjected to a very rigorous dietary test using formula-type diets supplying 40% of the calories as egg-yolk fat. Although there seemed to be a lesser cholesterogenic effect from these yolks, this was not impressive.[16] We felt that the very large amounts of cholesterol and phospholipids in the yolk fats counteracted the influence of the increased linoleic acid. Subsequent attempts to reduce the yolk cholesterol by feeding nicotinic acid in very large doses (up to 1.2 g./day) have not been successful.

Because of the ability of the bovine rumen to hydrogenate unsaturated fatty acids, it did not appear to be profitable to attempt modification of dairy fats by altering the diet of dairy cows. However, with the collaboration of the Dairy Science Department, we were able to prepare liquid milk and ice cream products in which the fat content was replaced by corn oil. Basically, these products consisted of milk solids (non-fat) plus corn oil. The milk contained 4% of corn oil and the ice cream 13.25%. Both products contained 10% milk solids. They were very well received by the test subjects and well tolerated during the trial period.

TABLE IV.

Product	Iodine value	% of total fatty acids as: Linoleic (L)*	Saturates (S)†	Oleic‡	L/S ratio
"Special" corn oil margarine	95.0	27.9	21.0	51.1	1.3
"Plain" margarine	88.4	9.3	16.1	74.6	0.6

*Based on the spectrophotometric method following alkali isomerization of the linoleic acid present.
†Based on the results of two unrelated direct gravimetric tests giving values in good agreement.
‡Obtained by difference: 100—linoleic—saturates = oleic.

In addition to testing the corn-filled milk and ice cream products, we also carried out tests on a newly produced corn oil margarine.* The experimentally produced corn oil margarine was prepared by blending liquid non-hydrogenated corn oil into a mixture of partially hydrogenated soy and cottonseed oils. This product had an iodine number of 95.0 as compared with a standard commercial margarine with an iodine number of 88.4 (Table IV). (It should be noted that corn oil has an iodine number of 122-125 and butter of 30-40.) The results from four separate dietary studies are shown in Fig. 2, A, B, C and D, and Fig. 3.

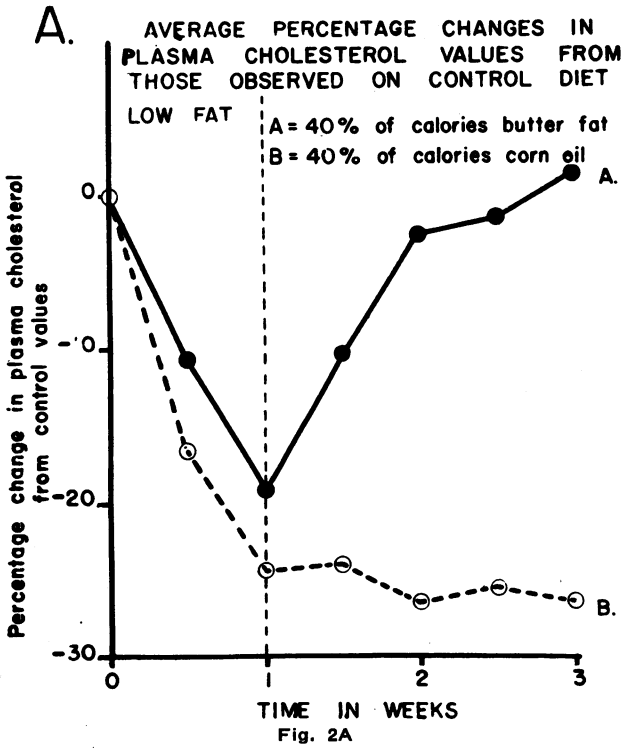

Fig. 2A

Fig. 2.—In each study shown above, there were four subjects in group A and four in group B. Discussion in text.

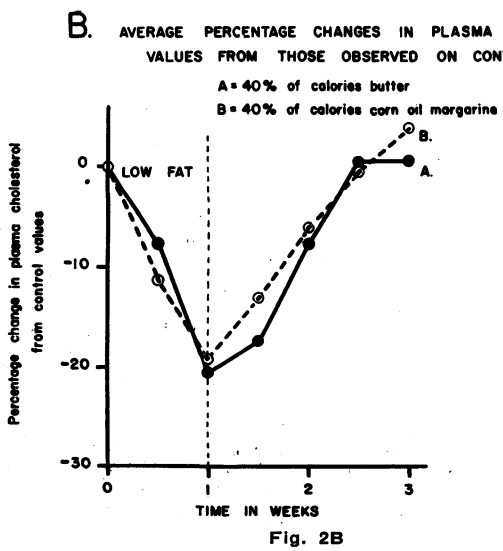

Fig. 2B

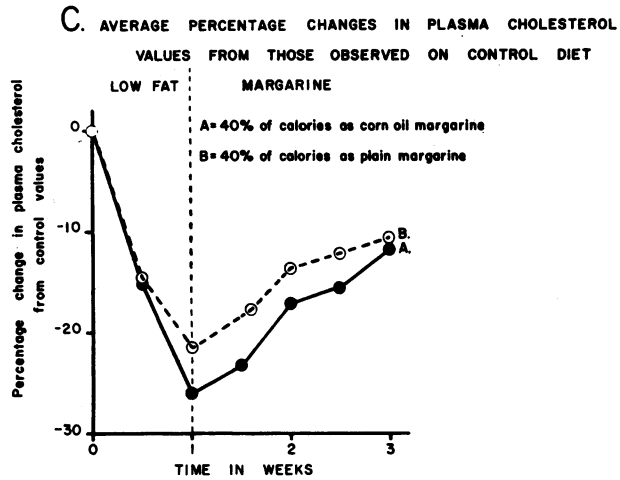

Fig. 2C

Series A. Comparison of corn-oil-filled milk and ice cream and the natural products (Fig. 2A).—With the corn-oil-filled products there was no rise from the low serum cholesterol levels reached on the low fat diet. Use of the natural products, on the other hand, resulted in a sharp return of serum

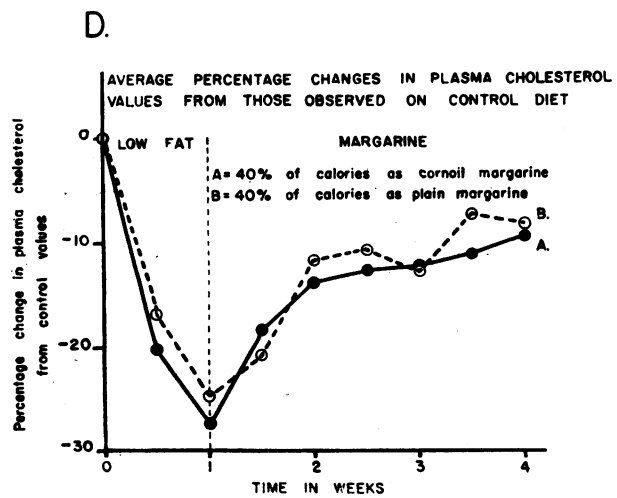

Fig. 2D

*An experimental product supplied by Corn Products Corp.

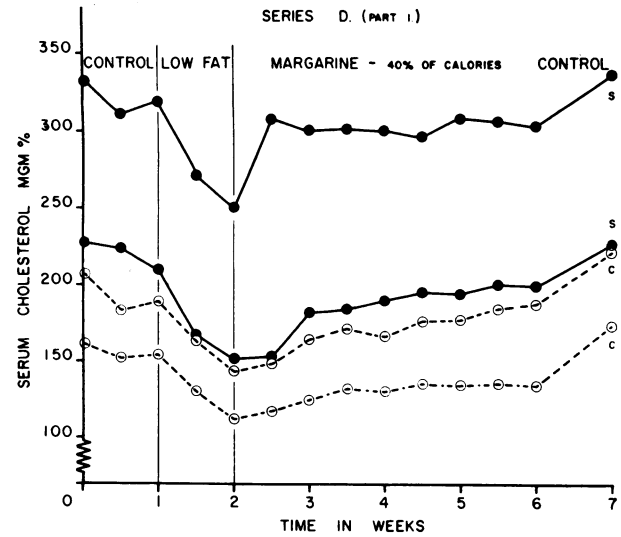

Fig. 3A.—Data for four subjects who received margarine for four weeks (weeks 2-6) and were returned to their self-selected control diet for the final week of the study. Subjects "s" received the special corn oil margarine, while subjects "c" received the plain commercial margarine.

cholesterol to original control levels over a two-week period.

Series B. Comparison of corn-oil margarine and butter (Fig. 2B).—Both products produced a rapid return of serum cholesterol levels to the control values by the end of the two-week test period.

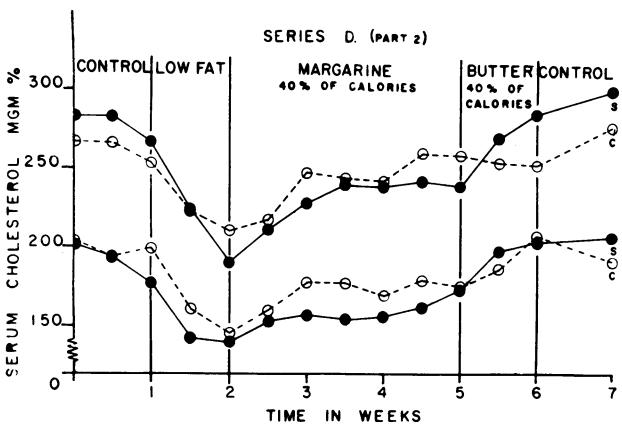

Fig. 3B.—Legend same as for Fig. 3A, except for the fact that subjects received margarine for three weeks only (weeks 2-5). They received butter fat for one week and were then returned to their control diet for the final week. Again, subjects "s" received the special corn oil margarine, while subjects "c" received the plain commercial margarine.

Series C. Comparison of corn-oil margarine and commercial margarine (Fig. 2C).—With both products there was a slow rise in serum cholesterol levels during the two-week test period, the final cholesterol levels being still below the original control values. Since there was an obvious discrepancy between the behaviour of the corn-oil margarine in Series B and C, it was decided to repeat this study.

Series D (Fig. 2D and Fig. 3) was identical in design with Series C, except that the margarine test period was prolonged to three weeks. Two of the four subjects in each group were continued on the same regimen for another week, and two received butter as a replacement for the fat previously used. From Fig. 3A, it may be seen that the results were identical with those in Series C up to the end of the second test week. During the third and fourth weeks on margarine, the two groups behaved similarly, with one member in each group showing a continuing rise in serum cholesterol level and one member remaining stabilized. Resumption of a normal diet for the final week of the experiment resulted in each instance in a rise in serum cholesterol to control levels. Fig. 3B reveals similar behaviour up to the end of the third week on margarine. When butter was given for one week instead of margarine, the serum cholesterol levels rose sharply in three subjects. Further slight changes occurred on transition to a control diet.

The results of test series C and D suggested that there was no difference between the two margarines used in these trials. Both resulted in a gradual rise of serum cholesterol level during the test period. In half of the subjects studied, the serum cholesterol stabilized at levels still below control. In contrast are the results in Series A, in which pure corn oil was used, with no rise at all from the cholesterol levels achieved on the low fat diet.

Discussion and Conclusions

Very low fat diets—which are in essence high carbohydrate diets—have a striking effect on lipid transport mechanisms. There is a sharp rise in serum triglyceride levels and a relative increase in the proportion of beta lipoprotein cholesterol, although the total cholesterol declines.

Hatch *et al.*,[17] in their studies of the rice diet (which is very low in fat and cholesterol), found that approximately one-fifth of their subjects showed an appreciable rise in serum neutral fat. This was associated with a substantial rise in the S_f 20-100 fraction of the lipoproteins. Nichols *et al.*[18] studied normal persons, using natural base diets very low in fat, and observed an increase in the S_f 20-400 fraction. They attributed this to the increased carbohydrate intake, as it was not seen when both fat and carbohydrate were restricted on weight-reducing diets. Nichols *et al.* remark, "It is of special interest that although S_f 20-100 and 100-400 are high in triglyceride content (approximately 50% of the lipoprotein being triglyceride), the serum levels of these classes were elevated on a diet low in triglyceride." The triglyceride is presumably synthesized in the liver from the excess carbohydrate intake. Stetten and Boxer[19] and Masoro *et al.*[20] have shown this to be true in rats at both normal and high levels of carbohydrate intake regardless of the absence of fat or protein from the diet.

Our own data, reported above, confirm the sharp increase in triglycerides in persons on a low fat diet. The relative increase in beta lipoprotein cholesterol fits well with the observed increases in low-density lipoproteins. We have also observed that the addition of fat to the diet results in a return of triglyceride levels and of the proportion

of beta lipoprotein cholesterol to normal. This phenomenon was observed with all the fats tested (corn oil, butter fat, corn-oil margarine and commercial margarine). Only corn oil, however, both corrected the lipoprotein abnormalities and kept the cholesterol levels low. Presumably the restoration of a proper balance between carbohydrate-fat-protein makes unnecessary the large-scale conversion of carbohydrate to fat and therefore the rise in circulating triglyceride.

That the type of lipid disturbance produced by a low fat diet may be atherogenic in rabbits has been demonstrated by Malmros.[21] The data cited above suggest that very low fat diets are probably harmful and may be atherogenic in man. Supplementation with corn oil prevents the deleterious effects of the diet and has the added advantage of maintaining a low cholesterol level.

In our experiments the addition of corn oil to a low fat diet, after a minimum period of one week, resulted in little additional fall in the serum cholesterol levels. In previous work,[8] we had shown that similar results were also obtained when a concentrate rich in linoleic acid was used rather than corn oil *per se*. These results led us to postulate that corn oil and similarly constituted vegetable oils did not actively depress the cholesterol levels, but had a neutral effect. The observed fall in serum cholesterol levels was therefore thought to be due to elimination from the diet of cholesterogenic factors associated with the dairy, meat and egg fats.

Of interest in this regard is the recent work of Beveridge *et al.*[22] Using formula-type diets, they first subjected their volunteers to an eight-day fat-free period during which the cholesterol values fell approximately 20%. Groups of ten subjects were then fed for another eight days a formula diet containing various test fats at a 40% of calories level. Only corn oil produced a further decline in cholesterol levels of 16.4%, while hydrogenated corn oil (I.V.69.6), safflower oil, safflower oil and sitosterol, cottonseed oil and soyabean oil failed to effect any significant change in cholesterol levels from those achieved on the fat-free diet. The authors state, "It is suggested that these results may provide an explanation why so many investigators have reported a hypocholesterolemic effect for a variety of oils when these were added to diets containing hypercholesterolemic fat. It is further suggested that a valid measure of the hypo- or hypercholesterolemic property of any fat is best obtained by determining the change in plasma level that occurs following transfer from a fat free diet to one in which the fat is substituted equicalorically for carbohydrate." The different results noted by Beveridge and ourselves with respect to corn oil may perhaps be related to experimental design, since the use of natural base diets as opposed to formula diets may introduce complicating factors. Nevertheless, the basic conclusions are much the same, namely, that with the possible exception of corn oil, the vegetable oils do not possess an active cholesterol-lowering effect, and the postulated active factor in corn oil is probably therefore unrelated to the unsaturated fatty acids.

The substitution of corn oil for butter fat in milk and ice cream is not an original idea with us In 1957, Malmros and Wigand[23] reported the use of such substituted materials in long-term feeding trials with sustained reduction in cholesterol levels for periods of one year or more. Our own studies indicated that such products were not difficult to prepare, were stable and esthetically acceptable, and even when consumed in very large amounts produced a sustained fall in serum cholesterol levels. The substitution of corn oil or other vegetable oils for the butter fat in milk, cream, ice cream and cheese should make it possible to diversify greatly the present low-fat corn oil diet which is being increasingly widely used for the treatment of hypercholesterolemia.

Little useful data are available on the effects of hydrogenation of vegetable oils on their special properties with respect to serum lipid levels. Bronte-Stewart *et al.*[24] observed that feeding hydrogenated ground nut fat (I.V.55) to a Bantu resulted in a slight rise in serum cholesterol on two separate occasions, whereas the unhydrogenated product (I.V.89) resulted in a fall of cholesterol. Ahrens *et al.*[25] have reported data on three subjects in which formula diets were used and partially hydrogenated oils fed at 40% of calories level. The most clearcut response was shown by patient No. 20 in whom the feeding of natural corn oil (I.V.126) had resulted in a fall of serum cholesterol from an original level of 347 to a base line cholesterol value of 200 mg. %. The substitution of corn oil hydrogenated to an I.V.80 resulted in a rise of the cholesterol to 273 mg. % and corn oil hydrogenated to I.V.58 resulted in a further rise to 298 mg. %.

In a second patient (No. 18) with a high control cholesterol level (331-358 mg. %) serum lipids were equally depressed on unhydrogenated corn or cottonseed oils, but the subsequent feeding of cottonseed oil hydrogenated to an I.V.68 produced a small but significant rise in cholesterol and phospholipids.

In a third subject (No. 30) who was initially normocholesterolemic, the ingestion of corn oil hydrogenated to an I.V.80 produced no increase in cholesterol levels over those observed with the unhydrogenated oil. It may be that hypercholesterolemic individuals behave differently in this regard from those with normal cholesterol levels.

Malmros and Wigand[23] found that coconut fat (hydrogenated or non-hydrogenated) had no effect on serum cholesterol levels in volunteers eating a natural base diet low in fat. Transfer to a diet in which whale oil supplied 40% of the calories resulted in a fall of approximately 40 mg. % cholesterol, whereas transfer from a diet containing coconut oil to one containing hydrogenated whale

oil resulted in no significant change in cholesterol levels.

We have already referred to Beveridge's[22] results in which hydrogenated corn oil (I.V.69.6) fed at 40% of calories level produced no significant increase in the serum cholesterol levels over those observed on a fat-free regimen. In contrast we have reported above that ingestion of an experimental margarine comprised of a blend of unhydrogenated corn oil and partially hydrogenated soy and cottonseed oils results in a gradual rise in serum cholesterol levels. These results were obtained in three separate experimental trials. In two trials (C and D) we compared a commercial margarine (I.V.88.4) and a corn oil margarine (I.V.95). These products differed not only in iodine number but also in the constituent oils from which they were fabricated. Partial hydrogenation of the soy and cottonseed oils would have been expected to produce a wide range of isomeric products with variable effects on lipid metabolism. In view of this, it is interesting to note that both margarines behaved in a very similar manner with respect to the cholesterol levels. We can only deduce from our experiments that hydrogenation, as carried out commercially, results in the production of substances, probably isomers, which tend to raise the blood cholesterol levels. In a previous study[26] in which we fed ethyl stearate to volunteers, there was no increase, but rather a moderate fall in the cholesterol levels. It would seem therefore that it is not the degree of saturation of double bonds which is decisive, but rather the biochemical or steric configuration of the products produced during hydrogenation. Brown[27] has enumerated some of the changes known to occur during the hydrogenation process:

1. Addition of hydrogen to double bonds forming more saturated compounds of higher melting point.

2. Shift of double bonds within the molecule. Thus oleic acid with one double bond between the 9-10 C atoms may shift either toward the carboxyl to the 8 position, or away from the carboxyl to the 10 position producing iso-oleic acids.

3. Formation of transacids and conjugated CIS acids which are found only infrequently in nature. For example, cottonseed oil has an iodine value of 110 with 50% linoleic acid. Hydrogenation to an I.V.75-80 results in a product with only 10% linoleic acid. In addition, the product now contains from 25-40% of trans acids and many other unnatural isomers.

It would seem imperative that a closer analysis be made of the products of commercial hydrogenation and their effects on lipid metabolism should be carefully studied.

Finally, we should note that in only one of the three trials in which margarine was used (B) did the serum cholesterol levels return to control levels. In the other two (C, D) it seemed to stabilize at a level roughly 10% below control. In two trials with butter (A, B), the cholesterol levels returned to control values during the two-week trial period. This suggests that butter fat is a more potent stimulant to cholesterol levels than are the hypothetical isomeric substances in margarine. In experiment D (section 2), the substitution of butter for margarine in the last week of the experiment resulted in a sharp rise in serum cholesterol levels in three out of four subjects. Beveridge[28] has suggested that the potent cholesterol elevating factors in butter may be the short-chain fatty acids and the cholesterol content. In margarine, one would expect the potent factors to be isomers of partly or fully saturated long-chain fatty acids. Again it becomes clear that we need to know much more about the relationship of steric structure and properties of the molecule, such as chain length, before we can make any confident assumptions about the relationship between any individual dietary fat and the effect on serum cholesterol levels.

SUMMARY

The ingestion of a natural base low-fat diet (less than 4% of calories from fat) for periods of one week or more resulted in striking changes in the pattern of the circulating lipids. There was a fall in total cholesterol of approximately 20% but a disproportionate increase in the lipoprotein cholesterol. Serum triglycerides showed a substantial increase.

The incorporation of corn oil, butter, partially hydrogenated blended corn oil margarine or commercial margarine at a level of 40% of calories resulted in every instance in a return of triglyceride levels to control values and a correction of the disturbed lipoprotein ratios. Only corn oil maintained the cholesterol levels at the low point achieved on the low-fat diet.

It proved feasible to produce processed milk and ice cream in which the butter fat was replaced by corn oil. These products, which were esthetically acceptable, could be ingested in large amounts without causing any rise in serum cholesterol from the low values obtained with the low-fat diet.

Both a commercial margarine and a specially prepared "corn-oil margarine" resulted in a rise in serum cholesterol from low-fat basal levels.

The data presented reinforce our view that vegetable oils exercise a neutral effect on serum cholesterol levels and that the active cholesterol elevating factors may consist of such substances as the short-chain fatty acids in milk fats and the isomers formed by long-chain fatty acids during the hydrogenation procedure.

The principles arising from this research permit the design of attractive and palatable low-fat diets supplemented with corn-oil-filled products for the treatment of hypercholesterolemic states.

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ABDOMINAL DECOMPRESSION DURING THE FIRST STAGE OF LABOUR
A PRELIMINARY REPORT

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THIS IS a preliminary report of abdominal decompression as a method of reducing the pain of parturition and of accelerating the first stage of labour. Also reported is a description of a new type of decompression apparatus which we believe offers several important improvements over previous equipment.

HISTORY

This work was undertaken as a result of the original publication in April 1959 by Professor O. S. Heyns[1] of Witwatersrand University, Johannesburg, South Africa. Observation, during experiments using muscle relaxants, suggested that a marked acceleration of the first stage of labour would occur with abdominal wall relaxation. A search was undertaken in an effort to find a safe method capable of relaxing the abdominal musculature. From this has evolved the technique of abdominal decompression.

THEORY OF ACTION

In the resting state the uterus is approximately ellipsoid in shape, with the upper segment at an angle backward from the lower segment. During a contraction the uterus tends to become spherical and rises forward, eliminating the angle between upper and lower segments. A tense abdominal wall will resist these changes, and the contracting uterus will expend a portion of its energy in overcoming this resistance.

A number of methods now in use attempt to solve this difficulty by encouraging the mother to relax. These include relaxation exercises, the natural childbirth of Grantly Dick-Read,[2] hypnosis, and sedative drugs. Decompression produces relaxation of the abdominal wall by mechanical means which, unlike the others, is largely independent of the mother's emotional status. Its use requires only a patient who is co-operative and intelligent enough to operate the apparatus. A language barrier is not a drawback to its use.

Our results below will show that this method considerably relieves the pain of labour, but as yet we have no definite explanation for this.

ORIGINAL DECOMPRESSION SUIT

The mother is zippered inside a plastic suit which extends from the axilla downwards to enclose the feet. The suit is separated from the abdomen by a rigid spacer, and air is pumped out, creating a partial vacuum over the body. The mother works the pump herself when the contraction begins. This results in a sucking force on the abdomen which causes it to bulge outwards where the wall is muscular and able to stretch. Experiments began at St. Mary's Hospital in the summer of 1959. The South African equipment was not available to us at this time, so we constructed our own following the description of the original. A further modification of the original model was tested. This suit extended from the axilla to the level of the upper thigh but was rejected as unsatisfactory.

DISADVANTAGES OF THE SUIT

The suit was used in the first 40 cases. The initial 20 are not included in our statistical analysis, because of incomplete documentation during the initial part of this investigation. The disadvantages evidenced by our experience with these 40 cases were:

1. A feeling of pressure on the chest at the upper end of the suit. This made breathing difficult and proved unbearable in some cases, requiring removal of these patients from the suit.

2. Inability to examine the patient rectally or vaginally or to auscultate the fetal heart without partially dismantling the apparatus.

3. Accumulation of liquor amnii within the plastic suit.

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