The out-patient consultative departments, or polyclinics as they may be called, will probably play an increasing part in the development of the Health Service.

The hospitals, particularly those with attached medical schools and research institutions, are the spearheads of research, but a plea is made for associating with certain types of research the local medical officer of health, who is anxious to know the amount and nature of the sickness among the community which he serves. By observing "associations," as did Dr. John Snow, the causes, now unknown to us, of many diseases may be revealed.

Hospitals are the training-grounds for doctors, nurses, and medical auxiliaries. Though the training of doctors is a responsibility of the universities, and not of the National Health Service, the training given in hospitals should emphasize, by the work going on there, the interdependence of the three branches of the Service and how they work together.

I am most grateful to the College for giving me this opportunity of expressing my views on a subject which I regard as of paramount importance to the future of the National Health Service, a service which, if it does what is expected of it, will make a substantial contribution to the happiness and prosperity of the nation. I am grateful, also, to my listeners for giving me so much of their time; and to the many, too numerous to mention individually, who have helped me with advice and suggestions. I must, however, specially thank Mr. C. C. Barnard. Librarian of the London School of Hygiene, for invaluable help in looking out references.

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RELATIONSHIP OF ALIMENTARY LIPAEMIA TO BLOOD COAGULABILITY

BY

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It has been clearly shown that a factor of lipoid nature is essential for the normal process of blood coagulation, although its precise character has not been finally determined (Macfarlane *et al.*, 1941). This finding led us to undertake experiments designed to discover whether the coagulability of blood is altered during the lipaemia which follows the ingestion of fat. The work is conveniently divided into two parts: (1) the influence of alimentary lipaemia on prothrombin times ("accelerated clotting times"); and (2) the effect of such lipaemia on the coagulability of blood estimated in silicone-coated tubes.

1. Influence on Accelerated Clotting Time

The technique adopted was the usual one-stage method of determining the prothrombin time (Fullerton, 1940). For a reason which is referred to later, Russell-viper venom ("stypven") was used as the thromboplastin in the test. The observations may be divided into three groups.

(*a*) In a group of eight patients taking the ordinary ward diet, blood was removed at intervals after meals and the accelerated clotting times were determined. The fat content of these meals varied from 12 to 30 g. The accelerated clotting times showed no significant variation throughout the day.

(*b*) A group of five patients were given a breakfast containing 65 g. of fat (bacon, eggs, bread, and butter). The results are shown in Table I. It is seen that in each case a significantly shorter time was found at the height of the lipaemia, with an average reduction of 4.9 seconds.

TABLE I.—*Accelerated Clotting Times after Fat Intake of 65 g.*

Case	Fasting* (Seconds)	Time† (Hours)	Shortest Reading‡ (Seconds)	Difference§ (Seconds)
1	22·5	3	15·4	7·1
2	23·7	3¼	19·7	4·0
3	23·0	2½	19·8	3·2
4	25·1	3	20·2	4·9
5	25·8	3	20·6	5·2
Means	24·0		19·1	4·9

TABLE II.—*Accelerated Clotting Times After Fat Intake of 85 g.*

Case	Fasting* (Seconds)	Time† (Hours)	Shortest Reading‡ (Seconds)	Difference§ (Seconds)
1	24·5	5	10·9	13·6
2	22·3	4¾	13·4	8·9
3	23·0	3	12·8	10·2
4	24·3	2½	17·5	6·8
5	22·3	3¾	14·8	7·5
6	24·7	3¾	13·1	11·6
Means	23·5		13·7	9·8

* Accelerated clotting times with the patients fasting.
† Time interval at which the shortest accelerated clotting time was reached during the lipaemia.
‡ Actual reading obtained.
§ Differences between the fasting and the shortest times.

(*c*) In a group of six patients similar observations were made following a meal containing 85 g. of fat (two eggs, 2½ oz. (70 g.) of bacon, 1 to 1½ oz. (28 to 42 g.) of butter, bread, tea with milk and sugar). The results are shown in Table II. Again in each case a marked reduction in the accelerated clotting time is evident during the lipaemia, and its degree is much greater than with a fat intake of 65 g. The effect is probably due to an increase in the essential lipoid factor during lipaemia. The other forms of thromboplastin ordinarily used in the accelerated clotting test—for example, acetone-dried extracts of rabbit brain (Quick, 1938) and viper venom plus ovolecithin (Witts and Hobson, 1940)—do not reveal this reduction during alimentary lipaemia. Presumably this is because these thromboplastins contain large amounts of lipoid, so that alterations in the plasma content of lipoid are obscured (Fullerton and Anastasopoulos, 1949).

It was realized that the findings set out in Tables I and II could not be interpreted as indicating that a real increase in coagulability occurs during lipaemia. The inference that naturally occurring thromboplastin is influenced by lipaemia in a manner similar to viper venom is not justified. Accordingly, similar experiments were performed using the clotting time in silicone-treated tubes as a measure of the coagulability of blood.

2. Influence on Clotting Time Measured in Silicon-coated Tubes

Method

"Teddol," a silicone-like material, was used. It reacts vigorously with water, yielding hydrochloric acid with heat evolution and a solid silicone formed by the intercondensation of the resulting hydrolysis product. Tubes measuring 3 by ⅜ in. (7.5 by 1 cm.) were filled with "teddol," and after ten minutes the solution was poured off and the tubes were inverted and allowed to drain for two minutes. They were then washed thoroughly in five changes of distilled water and were dried at room temperature. In estimating the clotting time with these tubes a needle, without a syringe, was inserted into a vein at the elbow and the first few millilitres of blood was run into a tube containing oxalate solution. This sample was later centrifuged, and the degree of lipaemia was estimated macroscopically. The first sample presumably contained any thromboplastin resulting from the trauma of the venepuncture. Then approximately 2 ml. was run into each of two teddol-coated tubes, and immediately these tubes were placed in a water-bath at 37° C.

Each tube was examined by tilting every five minutes, and the end-point taken was the time at which the tube could be inverted without flow of the blood. The object of using two tubes was to assess possible variations due to different degrees of silicone coating. Occasionally, if clotting appeared to be almost but not quite complete at any reading, another examination was made before the next interval of five minutes had elapsed. It may be pointed out here that the difference in the readings given by the two tubes did not exceed five minutes in any case.

Results

(*a*) With this technique the changes in the clotting time of whole blood were observed during the phase of lipaemia following the ingestion of a meal rich in fat (80–85 g.) composed of bacon, eggs, bread, and butter. Table III shows the preliminary results in four normal subjects. It is seen that in each case the clotting time three hours after the meal was considerably shorter than the fasting time. Macroscopic lipaemia was present at three hours in each case.

TABLE III

Case	Fasting	3 Hours	Difference
	mins.	mins.	mins.
1	25, 25	15, 15	10
2	30, 30	15, 20	13
3	30, 30	10, 15	18
4	25, 25	15, 20	8

(*b*) The experiment was then expanded to include readings at 3, 3½, and 4 hours after the meal in 11 subjects. The results are presented in Table IV. It is seen that in Cases 1–9 much shorter clotting times occurred following the high-fat meal. The maximal reduction varies from five to fifteen minutes and averages ten minutes. In Cases 1–9 macroscopic lipaemia was present following the meal. In Cases 10 and 11 a significant reduction in clotting time did not occur; in neither of these cases was macroscopic lipaemia observed at 3, 3½, and 4 hours after the meal.

TABLE IV

Case	Fasting	3 Hours	3½ Hours	4 Hours	Difference
	mins.	mins.	mins.	mins.	mins.
1	25, 27	20, 20	20, 23	15, 20	9
2	25, 25	15, 15	15, 15	10, 10	15
3	20, 20	10, 15	10, 10	14, 10	10
4	20, 25	20, 20	20, 15	15, 20	5
5	20, 20	10, 13	10, 10	10, 15	10
6	20, 20	15, 18	15, 12	15, 12	7
7	20, 25	10, 10	15, 15	—	12
8	25, 20	15, 20	20, 20	5, 10	15
9	15, 20	15, 10	10, 15	15, 20	5
10	20, 20	20, 25	20, 20	25, 20	0
11	15, 15	15, 15	12, 10	10, 14	4

TABLE V

Case	Fasting	3 Hours	3½ Hours	4 Hours	Difference
	mins.	mins.	mins.	mins.	mins.
1	15, 15	15, 20	15, 15	15, 15	0
2	15, 15	15, 20	15, 15	15, 15	0
3	20, 20	23, 25	20, 20	25, 25	0
4	30, 33	25, 27	25, 27	25, 25	6
5	20, 20	20, 18	20, 20	20, 22	1
6	25, 25	22, 25	25, 20	25, 25	3
7	25, 25	25, 25	25, 25	25, 25	0
8	30, 27	25, 25	25, 25	25, 25	3
9	20, 20	10, 15	10, 10	10, 15	10

(*c*) As a control, observations were made in nine patients before and after the ordinary ward breakfast (fat content 12–30 g.). The results (Table V) show that, with the possible exception of Case 4, a significant reduction occurred in only one case (No. 9). In Cases 1–8 no macroscopic lipaemia was observed at 3, 3½, and 4 hours after the meal; in Case 9 macroscopic lipaemia was present at these times and was absent in the fasting sample.

Comment.—The results in Tables III, IV, and V show that the coagulability of blood as measured by this technique increases markedly after a high-fat intake if this produces macroscopic lipaemia. Conversely, meals low in fat have no influence on the clotting time unless macroscopic lipaemia develops. These results would seem to have greater significance than those given in section 1, since they demonstrate that clotting induced by naturally occurring thromboplastin is accelerated by lipaemia. Whether this effect is due to an increase of the essential lipoid factor or to an alteration in the physical state of the plasma dependent upon its high content of chylomicrons during lipaemia has not been determined, and in any event the distinction does not influence the significance of the findings. We have discovered only one reference to observations similar to those described above. Duncan and Waldron (1949), using collodion-lined tubes, demonstrated a shortening of the clotting time in dogs and in humans, after the ingestion of cream. The implications of the results were not discussed.

Discussion

Lipaemia and Thrombosis

The results of these experiments seem striking enough to justify speculation regarding the pathogenesis of thrombosis, both arterial and venous.

Lipaemia is common in diabetes mellitus (Bloor, 1921; Joslin, 1927), pregnancy (Boyd, 1934), leukaemia (Pernokis

and Freeland, 1941), and after ether anaesthesia (Boyd, 1936) and acute blood loss (Boyd, 1934; Horiuchi, 1920). In all these circumstances thrombosis is frequent, although, of course, additional factors must be borne in mind; for example, the arterial degeneration in diabetes, venous stasis produced by the gravid uterus in pregnancy, the increased viscosity of blood in leukaemia, and immobilization after anaesthesia and blood loss. Nevertheless it seems possible, in view of the results described above, that the increased coagulability produced by lipaemia may be a factor responsible for the frequency of thrombosis in the conditions mentioned. It is known that coronary thrombosis not infrequently follows ingestion of a large meal (Master *et al.*, 1940; Smith *et al.*, 1942; Boas, 1942). It is impossible, however, on the evidence available, to determine whether this association is due to the decrease in coronary blood flow accompanying gastric distension (Gilbert *et al.*, 1940) or to the increased coagulability of blood which we have shown follows the ingestion of meals rich in fat.

Perhaps stronger evidence in favour of our thesis is given by the numerous observations which relate thrombosis to the dietary intake of fat. For example, Dock (1946) has noted that diets rich in eggs, cream, and butter are particularly common in men who are neither diabetic nor hypertensive, but who develop myocardial infarction. Malmros (1949) also considers that excessive consumption of eggs, butter, and other foods rich in fat may be the cause of the large numbers of cases of myocardial infarction now being observed. He points out that in Denmark the consumption of fat has been high for a long time and did not decrease in the second world war, whereas in Norway the consumption of fat and eggs was very low during the war. He thinks that this may explain why there was a fall in the incidence of myocardial infarction in Norway but not in Denmark during that period. Closs and Dedichen (1949) showed a similar fluctuation in the incidence of thrombo-embolic disease in general during the war in Norway.

Lipaemia and Atherosclerosis

Although the pathogenesis of atherosclerosis remains obscure despite intensive investigations for many years, some recent work has tended to discount the view of Virchow that the essential lesion is a primary degeneration in the arterial wall and has lent support to the earlier work of Rokitansky. This point of view has been emphasized in recent years by Duguid (1949, 1952), who holds that the lesion in atherosclerosis does not arise initially within the arterial wall but follows the incorporation into the vessel wall of fibrin deposits which are covered by vascular endothelium and later show hyaline and fatty change. If this conception be correct then it follows that any factor which leads to intravascular fibrin formation may be of importance in the pathogenesis of atherosclerosis. The results of our experimental work have led us to consider whether alimentary lipaemia may be a factor of importance in this process.

A large volume of work indicates that the level of the serum cholesterol probably bears some significant relationship to the development of atherosclerosis (Boas *et al.*, 1948; Gubner and Ungerleider, 1949; Duff and McMillan, 1951; Gould, 1951; Keys, 1951a, 1951b; and Katz, 1952). Numerous investigations on these lines have been carried out since Anitschkow (1933) showed that arterial lesions similar to atheroma followed cholesterol feeding in rabbits. As Keys *et al.* (1952) have pointed out, this finding cannot be applied directly to the problem of atherosclerosis in humans, since the amount of cholesterol needed to produce the lesions in rabbits is fantastically high in comparison with the cholesterol content of the diet in man. He concludes that "from the animal experiments alone the most reasonable conclusion would be that the cholesterol content of human diets is unimportant in human atherosclerosis." In any event, it is now clear that in man there is no relationship between the serum cholesterol level and the habitual dietary intake of cholesterol (Keys, 1949; Keys *et al.*, 1950; and Gertler *et al.*, 1950) but marked changes in the cholesterol level occur when the intake of fat is altered (Keys *et al.*, 1950; Hildreth *et al.*, 1951; and Keys, 1952). It is thus apparent that the large amount of evidence which links high serum cholesterol levels with the development of atherosclerosis may simply mean that a high-fat intake is related to atherosclerosis, and it is our suggestion that the high-fat intake acts by inducing increased coagulability of blood during phases of lipaemia.

Any theory of the pathogenesis of thrombosis and atherosclerosis must account for the notable variation in the age of onset and also for the tendency for the lesions to increase with age. Therefore it would be necessary, if our theory is to withstand criticism, to adduce evidence that the degree of lipaemia following ingestion of fat shows considerable individual variation and that in general it tends to be greater in the elderly than in the young. It is not difficult to find experimental work which supports both these suggestions (Necheles *et al.*, 1949; Morrison *et al.*, 1949; and Moreton, 1950). In our own studies we have noted great variation from case to case in the macroscopic degree of lipaemia following the standard high-fat breakfast, and Morrison and his co-workers (1949) devised a fat-tolerance test which showed that the rise in the serum lipid constituents following a high-fat meal varied within wide limits and was particularly high in 25 patients with recent coronary thrombosis. Necheles *et al.* (1949), by a study of the chylomicron counts after the ingestion of fat, found that this measure of lipaemia was altered much more in elderly individuals than in the young.

We suggest, therefore, that the pathogenesis of atherosclerosis may not be so obscure as it has appeared in the past. The conception that lipaemia, by leading to increased coagulability, produces fibrin deposits which are incorporated into the arterial wall accords well with our experimental findings and with a large amount of circumstantial evidence relating atherosclerosis to fat intake and serum cholesterol levels. It does not of course explain the apparent increase of atherosclerosis in the coronary arterial tree, but it is important not to let this particular aspect overshadow the general problem. We know of no convincing evidence that the overall incidence of atherosclerosis is increasing to a greater extent than can be accounted for by the general increase in longevity; the cause of its frequent and early localization in the coronary arteries may well be a problem separate from that of atherosclerosis in general.

It should be pointed out that our findings suggest that it is the occasional high intake of fat that is likely to be important rather than the average daily intake, since we found that the normal ward breakfast was followed by an increase in coagulability in only one case. If further work supports the theory set out above, studies of fat tolerance will become of increasing importance. In this way it might be possible to use the information thus obtained to determine which individuals are likely to develop atherosclerosis if a high dietary intake of fat is continued.

Summary

Alimentary lipaemia increases the coagulability of blood as measured by the "accelerated clotting time" and the clotting time in silicone-coated tubes.

The possible relationship of this finding to the pathogenesis of thrombosis and atherosclerosis is discussed.

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ASSOCIATION BETWEEN PERIOD OF GESTATION AND LENGTH OF MENSTRUAL CYCLE

BY

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It has often been stated that the period of gestation may be a multiple of the length of the menstrual cycle. This possibility is suggested by evidence, in experimental animals and in man, of cyclic activity in the ovary during pregnancy, and by a supposed association between the length of the cycle and the duration of pregnancy. The association can by no means be said to be established in man, the lack of good data being due largely to the difficulty of establishing the length of the menstrual cycle. In the course of an inquiry in which pregnant women were investigated from the first antenatal attendance until two years after the birth of the child, we have recorded both the period of amenorrhoea and the length of the cycle for 1,227 mothers of single births, and the data are here used in an examination of the association between the two variables.

Period of Amenorrhoea Related to Length of Cycle

We have attempted to collect fairly extensive data in respect of pregnancies of all mothers domiciled in the County Borough of Smethwick whose children were born between April 1, 1949, and March 31, 1950. We are here concerned only with the period of gestation and the length of the menstrual cycle.

(*a*) *Period of Gestation.*—Our only indication of the period of gestation is the duration of amenorrhoea. The date of onset of the last menstrual period was recorded at the first antenatal attendance,* and the date of birth was extracted from the records of notification of birth submitted to the public health department.

(*b*) *Length of Menstrual Cycle.*—Either on leaving hospital or at the first home visit of a health visitor, mothers were given a calendar on which they were asked to cross off each day of bleeding during the year after the birth of the child. This calendar was printed on a child's record card, on which were entered other items of more interest to the mother (such as the weight of the child). Mothers were subsequently interviewed at a welfare centre or at home within two weeks of each quarter during the year following birth, at which time the record card was inspected and the menstrual history copied on to another document. These documents provided the data used in the analysis which follows.

As a means of excluding some pregnancies of abnormal length, the examination is restricted to the 1,227 mothers of single births whose children survived to the end of the first year. (For example, this device excludes several pregnancies whose duration is influenced by the presence of a malformed foetus.) For 362 of the 1,227 mothers the data were considered incomplete, either because the period of amenorrhoea was not reliable or because fewer than three menstrual cycles were recorded in the year after birth.

Of the 865 cases in which the data were thought to be complete enough, 149 were excluded because the menstrual history was irregular. It is not possible to give any precise indication of what constituted an irregular history, except to say that two observers inspected the data independently, and with few exceptions decided upon exclusion of the same cases.

Table I shows the association between the length of the cycle and the period of amenorrhoea for the 716 women who had at least three regular cycles, and for whom the period of amenorrhoea was recorded unequivocally. The shortest mean length of cycle was 23 days (six women) and the longest 45 days (one woman).

The data suggest that the period of amenorrhoea increases slightly with the length of the cycle. The association is, however, somewhat irregular. This irregularity is attributable largely to the fact that the mean length of cycle for each woman has been calculated from all cycles during the year. Inspection of the records indicates that many women whose menstrual cycles are in general quite regular have one or two cycles which are grossly inconsistent with the others, and whose inclusion must make the mean less accurate. We have therefore recalculated the mean length of cycle for each of the 716 women, after excluding cycles whose lengths were judged to be inconsistent with the other observations. Table II gives the number of recorded cycles upon which the adjusted means are based: thus for 37 women the mean is based on three cycles, and for 34 on four.

TABLE I.—*Period of Amenorrhoea Related to Length of Cycle. Length of Cycle is Given as a Crude Mean*

Mean Length of Cycle (Days):	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	Over 38	Total
Mean period of amenorrhoea (days)	271·5	271·0	280·0	277·5	278·9	278·1	280·8	281·7	285·4	280·7	279·3	285·2	286·0	283·0	281·8	290·1	—	280·9
No. of women	6	6	24	51	76	113	126	111	65	51	34	18	12	6	8	7	2	716

*70% of the mothers were supervised during the antenatal period by one of us (T. D.).