In the post-natal group the daily haemoglobin rise was fairly equal with all three methods, and was more than one would expect with the dosage given, a matter at present under investigation. It will be seen that treatment with oral iron is as satisfactory as by the other two methods, and this method can be used in all cases of iron-deficiency anaemia in the puerperium in the expectation of a good and rapid response.

Intramuscular Iron Therapy

Altogether 208 patients were treated with intramuscular iron. In the first 135 cases serum iron levels were estimated immediately before and 18 and 48 hours after the first injection of 100 g. The results are shown in Table II.

TABLE II.—Serum Iron Levels ($\mu\text{g./100 ml.}$)

	No. of Cases	Time After First Injection of Imferon		
		0 Hours	18 Hours	48 Hours
Anaemia of pregnancy ..	14	20-90 (Mean 56)	100-455	220-960
Iron-deficiency anaemia ..	40	20-135 (Mean 66)	100-550	190-900
Post-partum anaemia, third day	81	20-190	90-700	190-1,110

Several workers have reported serum iron levels in patients receiving imferon. A comparison of results is difficult. Different dosage, times of collecting specimen, and methods of estimation of serum iron have been used. Cappell *et al.* (1954) report serum iron levels in the range 1,000-2,000 $\mu\text{g.}$ per 100 ml. 24-48 hours after injection of 200-250 mg. of iron. With a dose of only 100 mg. our patients had levels approaching 1,000 $\mu\text{g.}$ per 100 ml. In the series of 50 patients with anaemia of pregnancy reported by Scott and Govan (1954), serum iron levels of 335-820 $\mu\text{g.}$ were found 24 hours after injection of 100 mg. of iron as imferon.

In our patients with anaemia of pregnancy serum iron levels were in the range 100-455 $\mu\text{g.}$ 18 hours after and 220-960 $\mu\text{g.}$ 48 hours after the first injection. A number of patients in each group did not absorb iron as well as in other reported series. An attempt was made to correlate absorption with haemoglobin levels. Of 91 patients, 15 had serum iron levels below 33 $\mu\text{g.}$ per 100 ml. after 48 hours, by our method. These patients showed a mean haemoglobin rise of 1.25% a day. Thirty patients who showed serum iron levels of more than 500 $\mu\text{g.}$ per 100 ml. after 48 hours showed a mean haemoglobin rise of 1.16% a day. Thus there was no significant difference between these two groups.

Although there does not seem to be a direct relationship between the haemoglobin rise and the immediate rise in the serum iron, nevertheless a considerable rise in the serum iron level is noted in practically all cases treated with intramuscular iron.

In our series intramuscular iron is equally as effective as intravenous iron in the treatment of all types of iron-deficiency anaemia, though the response is slightly slower in some. It has proved effective in those cases which were refractory to oral iron, and in the doses we have used has given rise to no reactions.

Summary

A therapeutic comparison is made of the values of oral ferrous succinate with a similar amount of iron given intravenously and intramuscularly in the treatment of hypochromic anaemia in pregnancy, the puerperium, and in medical patients free from complication which would interfere with haemopoiesis.

Ferrous succinate taken orally is shown to be as effective as the other two methods in all but a few refractory cases, but the response was less rapid in the antenatal and medical cases, though equally as rapid in the post-partum cases.

In 208 cases treated with intramuscular iron there were no untoward side-effects.

All the cases refractory to oral iron responded satisfactorily to intramuscular iron.

Intramuscular iron was as effective as intravenous iron, although it was not so rapid in the medical series.

In 82 cases treated with intravenous iron good responses were obtained in all except two which were abandoned owing to fairly severe reactions.

We are grateful to the members of the nursing staff of the maternity department and medical wards of the Dudley Road Hospital for their co-operation in this trial, also to Calmic Limited for the generous supply of ferromyn tablets.

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ALIMENTARY LIPAEMIA IN MEN WITH CORONARY ARTERY DISEASE AND IN CONTROLS

BY

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For more than a century it has been known that the intimal plaques of atheroma contain cholesterol (Vogel, 1847). Much evidence has now accumulated of an association between atherosclerosis and abnormalities of the blood lipids. Most of the work on this subject has been concerned with cholesterol metabolism. This preoccupation with cholesterol values is understandable. Intimal deposits contain a high proportion of cholesterol and its esters, in certain animals arterial lesions are produced by cholesterol feeding, and in some xanthomatoses premature arterial disease is frequent. Furthermore, the blood cholesterol is fairly easy to measure in the laboratory. Barr, Russ, and Eder (1951) and Oliver and Boyd (1953) found that patients with cardiac infarction had higher blood cholesterol values than controls. Many patients in the coronary artery disease group, however, had blood cholesterol values within the normal range and there was no clear dividing line between the two groups.

Attention has not been confined to serum cholesterol concentrations. Gofman *et al.* (1950) used the ultra-centrifuge to investigate the serum lipids in atherosclerosis. They described a class of lipoprotein molecules, an increase of which was closely related to clinical evidence of atheroma. This result was not affected by the taking of a meal.

Barr *et al.* (1951) also investigated the linkage of lipids with the serum proteins. They found that in patients with atheroma more lipid was bound to the beta-globulin and less to the alpha-globulin than in

controls. This finding has been supported by the results of electrophoretic studies of the serum lipids. Nikkilä (1953) estimated cholesterol in the electrophoretic fractions and found a greater proportion travelling with the beta-globulin in cases of myocardial infarction than in controls. Using the technique of Sudan black colouring of all the serum lipids after filter-paper electrophoresis, Rosenberg, Young, and Proger (1954) reached a similar conclusion. Our own work by this method suggested that increased beta-lipoprotein in cases of coronary thrombosis in the fasting state was accompanied by an absolute increase in the total serum lipids. Observations on the serum lipids before and after a fatty meal seemed desirable.

Previous workers had measured the intensity and duration of the lipaemia following a fatty meal in cases of coronary thrombosis. Schwartz, Woldow, and Dunsmore (1952) measured the optical density of post-prandial serum and found a significant increase in the visible lipaemia three and five hours after a standard fatty meal in cases of cardiac infarction. Becker, Meyer, and Necheles (1949) found increased chylomicron counts up to 24 hours after a fatty meal in 30 patients (average age 76 years) compared with 30 young subjects (average age 18 years). They suggested that there was an association between prolonged chylomicronaemia and atheroma. Pomeranze, Beinfeld, and Chessin (1954) estimated the lipids by chemical means. Serum neutral fat rose higher and took longer to return to the fasting level in elderly patients with atheroma compared with normal young subjects.

In the present investigation the lipids have been measured both by optical and by chemical methods.

Material

Three groups of patients have been investigated. The first series consisted of 35 men who had a myocardial infarction with electrocardiographic confirmation. Patients with congestive heart failure were excluded, as were those who died within six weeks of the onset of their illness, those over 70 years of age, and one man with essential hyperlipaemia whose fasting serum was milky. The majority had no evidence of arterial hypertension. Their ages ranged from 32 to 66, with a mean of 51.9 years.

The second series was of male controls. Thirty-three men who were admitted to hospital for minor surgical procedures such as ligation of varicose veins or herniorrhaphy were studied. None had hypertension, all had normally palpable peripheral arterial pulses, and none admitted having chest pain or leg pain on exertion. Their ages ranged from 30 to 68, with a mean of 50.4 years.

The third series was of pre-menopausal women controls. They were selected on the same basis as the men controls. Their ages ranged from 27 to 41 years.

Methods

The control patients were usually studied a few days after operation, when the majority were again ambulant and were taking a normal ward diet.

Patients in the coronary series were usually studied four to eight weeks after the onset of their illness, when they had been taking a normal ward diet for at least three weeks. In three cases the infarct had occurred more than three months previously. In some patients the test was performed during the period of bed rest. In others the test was delayed until the patient was ambulant again.

After a normal evening meal the patient ate nothing until the first sample of venous blood (24 ml.) had been with-

drawn at 9.15 a.m. the following day. This sample was divided into two parts for duplicate estimations. A standard meal was then given. It consisted of 4 oz. (114 ml.) of "double cream" with 4 oz. (114 ml.) of milk, taken with cornflakes and a cup of tea. The fat content was estimated as 60 g. No further food was allowed until the last blood sample had been collected. Smoking was allowed. The delay between withdrawing the first sample of blood and the completion of the meal varied, but was approximately 45 minutes. Further samples of venous blood (12 ml.) were taken at 1, 3, and 5 p.m., and were called the three, five, and seven-hour samples. The blood was allowed to clot in a plain tube, and the serum was separated and allowed to stand overnight for the disturbed red cells to settle. Fig. 1 shows examples of the sera from two patients.

The optical densities of the sera were read in a spectrophotometer at 680 millimicrons. The mean value of the two fasting samples was taken.

The total serum lipids of each sample were then estimated gravimetrically by Bloor's method. 5 ml. of serum was introduced into a 3 to 1 ethanol-ether mixture in a 100-ml. volumetric flask. The mixture was heated in a waterbath until boiling occurred, and then cooled. After cooling, the mixture was made up to the mark with ethanol-ether, shaken, filtered, and 75 ml. evaporated in a weighed beaker. After drying, the weight of alcohol-ether extractable matter was calculated. A second extraction was then performed with petroleum ether. As most of the fasting samples were duplicated it was possible to determine the error of the method in our hands. Thirty-seven pairs of results were analysed. In only two pairs did the difference exceed 120 mg. per 100 ml. of serum. The difference did not increase with the height of the reading. The error of a single reading has been regarded, therefore, as ± 60 mg. per 100 ml. serum.

FIG. 1.—Sera from two patients at the time of taking 60 g. of fat and at three, five, and seven hours after. Upper test, from a healthy man of 50. Lower test, with a fifth sample at nine hours after the meal, from a man of 51 years five weeks after cardiac infarction.

FIG. 2.—Mean values of optical density readings after 60 g. of fat in the three series.

Results

The complete results of the optical density readings are given in the Table. The optical density of the serum in

the coronary series was greater than in the controls before and after the fatty meal. At each time the range of readings overlapped.

Before the meal the optical density varied a good deal in all three series. The mean value in the coronary cases was 0.074 and in the male controls 0.060 (Fig. 2). The difference was statistically significant ($0.001 < P < 0.005$).

After the meal the highest values were usually found at three hours, but in four men controls and 12 coronary cases the five-hour reading was equal to or greater than the three-hour reading.

The greater optical density of the coronary series at three hours was not statistically significant when compared with the male controls ($0.25 > P > 0.1$). At five and seven hours, however, the differences were highly significant ($0.001 < P < 0.005$).

Comparison of the male controls and female controls shows that the serum of the men has a higher optical density than that of the women at all times before and after the meal. None of these differences reaches statistical significance in these numbers ($0.05 > P$).

In none of the women was there a very dense alimentary lipaemia. At three hours the optical density exceeded 0.550 in only one woman, compared with nine men controls and 18 coronary cases. No woman control had a reading as high as 0.450 at five hours, but eight men controls and 17 coronary cases exceeded this figure.

Body Weight and Lipaemia.—No clear relationship was found between body weight and the duration of the lipaemia. An analysis of 19 cases in the coronary series relating weight to the five-hour density reading gave a correlation coefficient of -0.01 , and of 22 cases in the male control series -0.16 . This suggests only that the lipaemia tends to be a little less intense in heavier people, and as the mean body weight

was greater in the coronary series this can have played no part in giving rise to the difference between the two series.

Age and Lipaemia.—Similarly there was no significant correlation in any group between age and lipaemia. Any tendency relating to age seemed to show an increased lipaemia in younger people. The male control series was analysed in this respect, the correlation coefficient between increasing age and the five-hour density reading being -0.13 , which is not significant.

Repeatability.—The test was performed three times in one of the controls and twice in four coronary patients. The results are shown in Fig. 3. The intensity of the lipaemia is seen to vary to some extent in the same person on different occasions.

Confinement to Bed.—Most of the controls were ambulant in the ward when the test was taken. In the coronary cases

FIG. 3.—Results of three tests in one control subject and of two tests in four coronary patients.

Complete Results of Optical Density Readings and Total Fasting Lipids in the Three Series

Coronaries							Men Controls							Women Controls						
Age	Wt. Kg.	Optical Densities				Fasting Lipids mg./ 100 ml.	Age	Wt. Kg.	Optical Densities				Fasting Lipids mg./ 100 ml.	Age	Wt. Kg.	Optical Densities				Fasting Lipids mg./ 100 ml.
		0 Hrs	3 Hrs	5 Hrs	7 Hrs				0 Hrs	3 Hrs	5 Hrs	7 Hrs				0 Hrs	3 Hrs	5 Hrs	7 Hrs	
32		0.078	0.517	0.722	0.420	1,025	30		0.043	0.315	0.092	0.053	975	27		0.047	0.550	0.365	0.110	763
41*	80	0.058	0.292	0.258	0.086	995	33	64	0.051	0.404	0.127	0.081	786	28	62	0.077	0.453	0.349	0.192	1,030
41*		0.062	0.662	0.635	0.251	725	33	94	0.063	1.180	0.630	0.125	820	29	59	0.038	0.210	0.072	0.040	651
43	65	0.052	0.451	0.277	0.116	908	37		0.094	1.080	0.840	0.200	1,065	30		0.058	0.540	0.054	0.074	944
45*	76	0.085	0.575	0.300	0.325	1,033	40		0.055	0.600	0.455	0.051	828	31	54	0.049	0.252	0.169	0.073	1,045
45*	64	0.076	0.690	0.690	0.242	1,208	41		0.085	0.310	0.280	0.088	985	33	80	0.058	0.295	0.335	0.116	855
45*		0.080	0.850	0.460	0.180	1,070	43	64	0.046	0.555	0.260	0.155	768	33	46	0.046	0.078	0.205	0.067	868
46*	75	0.077	0.660	0.310	0.097	1,100	45	69	0.079	0.295	0.542	0.330	1,343	34		0.028	0.310	0.150	0.108	692
47		0.072	0.491	0.594	0.381	1,050	46	70	0.075	0.307	0.102	0.087	1,108	35	57	0.085	0.406	0.243	0.071	957
47		0.057	0.475	0.251	0.071	883	47	70	0.035	0.340	0.065	0.037	648	37		0.044	0.720	0.167	0.065	
47	70	0.062	0.372	0.135	0.066	903	47	62	0.058	0.980	0.245	0.142	1,198	40		0.068	0.294	0.205	0.075	608
47*		0.055	0.475	0.855	0.125		48	64	0.072	0.425	0.385	0.095	898	40	46	0.067	0.475	0.422	0.160	
47*		0.070	0.225	0.235	0.105	750	49	67	0.047	0.689	0.605	0.142		40		0.056	0.415	0.208	0.067	772
49	86	0.099	0.555	1.030	0.533	1,160	50	76	0.058	0.280	0.120	0.058	937	41	70	0.070	0.300	0.360	0.090	820
51	67	0.064	1.080	0.305	0.117	889	50	67	0.044	0.396	0.202	0.102	822							
51	74	0.072	0.760	0.342	0.102	1,100	51	60	0.058	0.192	0.096	0.103	682							
52	76	0.067	0.780	0.575	0.167	1,175	51	76	0.055	0.240	0.127	0.068	892							
52	67	0.082	0.695	0.420	0.126	1,008	52	65	0.064	0.545	0.540	0.382	1,050							
53*	57	0.068	1.150	0.960	0.208	1,440	52	65	0.073	0.340	0.180	0.056	1,109							
53*		0.050	0.595	0.510	0.215		53	64	0.073	0.590	0.220	0.126	1,058							
54*	67	0.056	0.483	0.279	0.184	1,140	53	70	0.050	0.240	0.250	0.050	748							
54*	102	0.118	0.385	0.310	0.135		53		0.052	0.168	0.135	0.126	1,100							
55	58	0.069	0.390	0.479	0.366	1,045	54	92	0.067	0.480	0.089	0.072	839							
55*		0.075	1.060	0.656	0.255	1,150	55	67	0.063	0.352	0.111	0.060	853							
55*		0.137	0.560	0.780	0.262	1,430	57		0.043	0.251	0.097	0.075								
58*		0.076	0.325	0.560	0.100	1,045	57		0.060	0.300	0.655	0.160	760							
58*		0.064	0.800	0.230	0.132	1,140	60		0.045	0.590	0.210	0.085								
59		0.078	0.349	0.312	0.168	1,223	61		0.073	0.292	0.240	0.098	830							
59*	89	0.130	0.700	0.660	0.275	1,462	62	73	0.047	0.755	0.308	0.068	961							
61	54	0.069	0.258	0.503	0.303	1,181	63	51	0.069	0.352	0.200	0.080	1,086							
62	64	0.076	0.598	0.332	0.065	685	65	94	0.055	0.380	0.068	0.058	691							
63		0.042	0.192	0.072	0.094	753	67		0.042	0.248	0.210	0.067	995							
63*		0.090	0.135	0.280	0.220	1,090	68	54	0.074	0.295	0.720	0.480	729							
64		0.067	0.740	1.120	0.515	1,027														
66	63	0.063	0.438	0.407	0.147	854														
Mean		0.0742	0.5647	0.4808	0.2044	1,051.5			0.0596	0.4475	0.2850	0.1200	918.8			0.0565	0.3784	0.2360	0.0934	833.8
Standard error		0.0035	0.0415	0.0432	0.0209	34.1			0.0024	0.0435	0.0374	0.0171	30.8			0.0042	0.0438	0.0304	0.0110	41.3
Standard deviation		0.0205	0.2454	0.2557	0.1234	193.1			0.0140	0.2497	0.2149	0.0984	168.5			0.0158	0.1638	0.1139	0.0410	143.2

* Ambulant at time of test.

the test was as often performed before the patient was allowed up. Comparison of the results from those coronary patients who were ambulant with those confined to bed shows no obvious differences. Limited physical activity seems, therefore, to be a relatively unimportant factor.

Results of the Chemical Estimation of Total Lipids

Fasting Values.—In men who had cardiac infarction the fasting serum contained more lipid than in controls (see Table). Again there was a wide range of results in all three series, with a considerable overlap. The mean value in the coronary series was 1,052 and in male controls 919 mg. per 100 ml. of serum. This difference was statistically significant ($0.005 < P < 0.01$).

Lipaemic Values.—Measurement of the intensity and duration of the lipaemia by total lipid estimations was unsatisfactory because of the size of the error of the method. Thus a rise in total lipid of 120 mg. per 100 ml. could be discounted by the error. In spite of this failure to measure the rise and fall of total lipids with accuracy it is clear that in general the rise and fall closely parallels increasing or diminishing serum turbidity after a fatty meal. In most cases lipaemic turbidity of the serum matched the chemical lipaemia closely. In a few cases the difference between the expected chemical figure and the result obtained exceeded the error of the method. The mean values for the rise in total lipids after the meal in the three series are shown in Fig. 4.

FIG. 4.—Mean values of the rise in total lipids after 60 g. of fat in the three series.

Discussion

After a meal of fat the serum becomes milky or turbid in appearance. Optical measurement of this lipaemic turbidity has shown that in men with coronary artery disease lipaemic turbidity is more intense and prolonged than in apparently healthy men of a similar age. Because of the effect of prolongation of the lipaemia the greatest difference between the two series of men occurs after the main peak of the lipaemia has passed. In these tests the greatest difference has therefore been found at five hours.

If an attempt were made to use this test selectively to distinguish those with established arterial disease from controls, the best single figure would be the seven-hour reading. At this time the overlap between the two series is less. Of the 33 male controls only four have seven-hour lipaemic turbidity greater than 0.160, compared with 19 of 35 coronary cases. Twenty male controls have a seven-hour reading less than 0.100, but only six coronary cases fall to so low a figure. If the seven-hour figure is combined with the sum of the three- and five-hour figures a clearer distinction is possible. In only one of the controls does the seven-hour reading exceed 0.160 and the sum of the three- and five-hour figures exceed 1.100. These combined values are exceeded in 12 of the 35 men with coronary artery disease. It appears probable that such values are always abnormal.

An increased number of fat particles seem to persist in the blood of the coronary patients throughout the 24 hours, for there is an increased optical density of the fasting serum. The relationship of the fasting optical density and the total lipids estimated chemically is not a very close one. Statistical testing shows the correlation coefficient to be $P=0.62$, the probability of this happening by chance being less than 0.01. No closer correlation could be expected, as so little of the total lipid is in a visible form in the fasting state. The range of fasting optical densities varies between 0.035 and 0.137 in the two series, and it is unlikely that any figure below 0.078 can be held to imply a high fasting total lipid value. There were 11 cases in this series with a fasting figure above 0.078, and their total fasting lipids vary from 957 to 1,462 mg. per 100 ml. of serum. Stricter supervision of the diet on the day before the test would perhaps reveal a closer relationship.

A relationship is also found between the prolonged alimentary lipaemia of the coronary series and the fasting total lipids. Eleven of the coronary cases with highest optical densities throughout the seven hours have total fasting lipids of 725 to 1,462 mg. per 100 ml. of serum, with a mean value of 1,170 mg., compared with 21 other cases with a mean value of 989 mg. per 100 ml. This difference proved to be significant at just less than the 1% level of confidence. Thus the total fasting serum lipids are related to prolonged and intense visible lipaemia after a fatty meal.

Nature of the Abnormality.—From this study no direct evidence emerges concerning the nature of the abnormality responsible for the prolongation of lipaemia in coronary artery disease. It would seem certain that failure on the part of the controls to absorb the fatty meal plays no part in giving rise to this difference. Delayed absorption from the gut in the coronary series could play a part in the difference. Certainly there is a stronger tendency for the peak of the lipaemia to occur later than three hours in the coronary series than in the controls, but this may not be due to delayed absorption. The fact that the intensity of the lipaemia is greater in the earlier as well as in the later hours of the test suggests that delayed absorption is not an important factor. Further evidence on this point was sought by taking some additional readings at one and a half hours. At this time the mean value for optical density in six coronary cases was 0.197 and in seven men controls 0.200. There seems, therefore, to be no significant delay in the onset of the lipaemia in the coronary series. The most likely explanation is that there is a relative failure to clear the blood of fat particles in the coronary series. This would presumably lead to a rapid rise in the concentration of fat particles as absorption took place in the gut and a slow resolution of the turbidity after the peak of the lipaemia was passed. These are the differences between the two series.

Clearing Action of Heparin.—Previous evidence was available which suggested that the clearing of alimentary lipaemia in coronary artery disease was abnormal. Block, Barker, and Mann (1951) gave a small intravenous injection of heparin at the height of a lipaemia in cases of arterial disease and in controls and measured the degree of clearing of the plasma which took place in the following fifteen minutes. Significantly less clearing occurred in the atherosclerotic group. Oliver and Boyd (1953) repeated this experiment and found that the amount of clearing after heparin in the coronary group was half that which occurred in the controls. In the present study the degree of clearing between three and five hours in the male control series is 36.4% and in the coronary group 14.9%. We have therefore shown a similar delay in clearing over a longer interval of time without the intervention of intravenous heparin.

Relationship to Coronary Artery Disease

The mechanism by which this abnormality of fat metabolism is related to coronary artery disease remains obscure. Moreton (1947) advanced the view that an excess of fat particles in the blood of atherosclerotics over the years would lead to infiltration of fat through the arterial

intima and perhaps to the inclusion of the cholesterol moiety of the chylomicrons in the intimal tissues.

An altogether different possibility is suggested by the studies of Fullerton *et al.* (1953), Waldron and Duncan (1954), Poole (1955), and O'Brien (1955) concerning the relationship between lipaemic plasma and accelerated clotting-times. The first two groups of workers have claimed to find significant acceleration of the clotting-time after a fatty meal, especially in the presence of pronounced visible lipaemia. Poole has shown that rapid centrifugation, with the removal of fat particles from the plasma, increases recalcified plasma clotting-times by the order of two and a half times in the absence of platelets. Subsequent addition of the fat particles restored the clotting-time to its former value. If this work is confirmed then the present finding of increased visible lipaemia in men with atherosclerosis will appear to reveal an obvious clue to the possible aetiology of arterial thrombosis, at least in a proportion of cases.

Oliver and Boyd (1953) preferred to explain the relative resistance in atherosclerotics to the clearing action of heparin by diminished production of a heparin-like substance from the mast cells of a diseased arterial system.

Summary

Following the administration of a meal of 60 g. of fat the extent and duration of the lipaemia has been measured by optical and chemical methods in 35 men with cardiac infarction, 33 healthy men matched for age, and 14 healthy pre-menopausal women.

The coronary and male control series have been compared. Results of the optical density readings showed significantly greater degrees of visible lipaemia at five to seven hours after the meal in the coronary series. A wide range was encountered in both series, leading to a considerable overlap.

The fasting serum also showed greater optical density in the coronary series.

Results of the chemical estimations showed total lipids in the fasting state to be significantly higher in the coronary series. The difference in the mean values was 133 mg. per 100 ml. of serum. Total lipids after the meal tended to rise and fall with changes in the visible lipaemia.

In the female controls the lipaemia was generally less than in the men. These differences did not reach statistical significance.

It is concluded that in some men with coronary artery disease the clearing of visible lipaemia after a fatty meal is delayed and that the relative failure of the clearing mechanism may continue to exert its influence after many hours.

Possible ways in which this phenomenon may be related aetiologicaly to atherosclerosis are briefly discussed.

I am indebted to Professor C. Bruce Perry for his interest in this investigation. The physicians and surgeons of the United Bristol Hospitals have allowed me to investigate their patients. Dr. Fraser Roberts, of the London School of Hygiene, generously gave statistical advice, and Miss Duncan, of the Department of Public Health in Bristol, carried out all the statistical calculations. Miss Margaret Haynes performed the chemical estimations.

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Medical Memoranda

Hypertension Due to Hydronephrosis: Relief after Nephrectomy

The following case is reported because of the problem which it poses in the aetiology of hypertension due to renal disease.

CASE REPORT

In September, 1953, I was asked to see a European man, then aged 31, because of the discovery of a non-functioning left kidney on excretion pyelography. This had been found by his own doctor during an investigation for hypertension, which had been discovered when the patient had attended about a month previously complaining of waking at night "with his heart thumping" and shortness of breath. At such times he suffered nausea but did not vomit. These symptoms had arisen two weeks before he had consulted his physician, and had never occurred previously. When aged 13 he had been confined to bed for nine months with rheumatic fever. He had later been treated with Lugol's iodine for "thyroid disease," but had since played games without ill effect. While on active service in 1943 he had had an attack of left renal colic lasting a few hours, but no investigations were undertaken because of the exigencies of the situation.

On examination the relevant findings were an apparently healthy mesomorph showing no evidence of disease except a blood pressure of 170/110. He smoked 40 cigarettes a day, and weighed 187 lb. (84.8 kg.). An E.C.G. tracing revealed moderate left ventricular stress, and the optic fundi showed grade 1 hypertensive retinopathy. Urinalysis disclosed nothing abnormal.

In view of the rapid onset and nature of the symptoms, I thought it advisable to exclude the possibility of phaeochromocytoma: an amylobarbitone sedation test favoured primary hypertension, a benzodioxane test was negative, and presacral perirenal pneumography did not show any abnormality of the adrenal glands. Ascending pyelography showed a large pelvic hydronephrosis of the left kidney, and the appearances suggested an "aberrant" artery as the likely cause.

Operation.—On October 22 I removed the left kidney through an incision in the bed of the twelfth rib; freeing the upper pole did not affect the blood pressure. The kidney was largely a hydronephrotic sac, with just a thin rim of renal tissue. The primary cause appeared to be a large aberrant artery arising from the distal aorta and passing to the lower pole of the kidney; the true renal vessels were a little smaller than normal.

Histology.—"The kidney is the seat of a large internal hydronephrosis, with marked thinning of the surrounding parenchymal kidney tissue. Sections through the thinned kidney tissue show a marked degree of chronic interstitial nephritis, fibrosis and hyaline degeneration of the majority of the glomeruli, narrowing and obliteration of many of the tubules and dilatation of others, which contain pink-staining hyaline material. The arterioles show the changes associated with hypertension—namely, thickening of the walls due to elastosis, and intimal thickening and consequent narrowing of the lumina. The characteristic arteriolonecrotic lesions of malignant hypertension are not found here."