the patient's resumption of work then confronts the doctor. In general an early return to work is to be encouraged.

So long as disabling symptoms persist—exertional pain or dyspnoea—a resumption of occupation may be out of the question. For those handicapped by a residual angina of effort, or even minor degrees of congestive failure, retirement from active work may be the only alternative. Recovering satisfactorily from the acute attack and having mild or only trivial symptoms when ambulant, the patient should in general be able to resume some form of active work after a month's convalescence—that is, in approximately eight weeks from the onset. The weeks elapsing after leaving hospital should be devoted to the recovery of physical fitness and improved muscular tone, skeletal and cardiac. Rehabilitation has its psychological as well as its physical aspects. The doctor can contribute much to the recovery of confidence, which should be a constant aim.

There is nothing to be gained and much to be lost by confining a symptomless patient to bed for two to three months and then subscribing to such a graduated convalescence that little or no attempt is made to resume an active working life, even after a further three to four months' rest. The tendency to impose a great restriction of activities is lessening, but patients are still seen who have received much advice of a negative character, such as, "Don't stoop, don't lift, don't strain, don't hurry, don't walk for more than five minutes, don't take more than five steps on the stair," and so on.

The main principle of rehabilitation is to encourage rather than to restrict. The cardiac patient should be advised to do as much, or a little more, than he thinks he can—that is, short of the production of disagreeable symptoms. In brief, the attitude of his adviser should be not "so little" but "so much." Confident reassurance throughout the illness and convalescence can counteract the development of a neurosis, which is all too apt to prove in the long run a greater handicap than the structural damage.

Admittedly much depends on the nature of the employment to which the patient has been accustomed, his mental and physical stamina, and the ease with which symptoms are induced. I have known miners, within a couple of months after an acute myocardial infarction, return to work at the coal-face without harm to themselves. Busy executives may well resume full and increasing responsibility. Capacity for work can be determined for the individual only by the graduated method of trial and error. Fortunately, the great majority recover from the acute attack and are able to lead useful and productive lives, thus dispelling the notion that a damaged heart necessarily leads to permanent disability and incapacity.

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BLOOD COAGULATION AND FIBRINOLYSIS IN RELATION TO CORONARY HEART DISEASE

A COMPARATIVE STUDY OF NORMAL WHITE
MEN, WHITE MEN WITH OVERT CORONARY
HEART DISEASE, AND NORMAL BANTU MEN

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Since the middle of the nineteenth century there have been two main theories regarding the pathogenesis of atherosclerosis. Virchow (1856) postulated that atheromatous lesions are produced by the imbibition of lipids from the circulating blood, while Rokitansky (1841) favoured the view that the primary lesion is a deposition of fibrin on the arterial intima. These two theories continue to dominate modern thought on atherogenesis, and they have led to many studies of the blood lipids and of blood coagulation and fibrinolysis in relation to coronary heart disease.

In Southern Africa, research in this field has been facilitated by comparative interracial studies among the White population, in whom the death rate from coronary heart disease is very high, and the Bantu population, in whom deaths from this disease are extremely rare. Previous reports from this department have dealt with differences in serum-cholesterol levels in white and Bantu men and with the role of dietary fat intake in their production (Bronte-Stewart *et al.*, 1955, 1956; Brock and Bronte-Stewart, 1955). The present report is concerned with a comparison of blood coagulation and fibrinolysis in apparently healthy men of these two races and in white men with clinical evidence of coronary heart disease.

Material of the Investigation

The three groups of subjects who were studied are shown in Table I. The normal white men ("controls") were mainly white-collar workers from a large Cape-town insurance company; a number of professional

TABLE I.—Material of Investigation

	No.	Age (Years)		Mean
		Minimum	Maximum	
Controls	80	20	65	39.8
Coronary	48	38	69	52.3
Bantu	53	20	65	33.8

men and skilled manual workers were also included. The normal Bantu men ("Bantu") were all migrant labourers who had just arrived in Capetown from the Transkei Native Reserves; the majority were examined within 24 hours of their arrival in Capetown; all were examined within five days of their arrival. In neither of these two groups was there any sign of active disease or of previous coronary heart disease as judged by history, clinical examination, erythrocyte sedimentation rate, and electrocardiograph. The white men with coronary heart disease ("coronaries") either had sustained a well-documented attack of cardiac infarction not less than three months before the examination or were suffering from angina pectoris in a stable phase; patients who had been on anticoagulant therapy during the previous three months or who were consuming large quantities of unsaturated fats were excluded from the main study; patients with cardiac failure were also excluded.

It was not possible to age-match the Bantu and the coronary subjects. There were therefore two sets of randomly selected white controls, one age-matched with the Bantu and one with the coronary group (Table II).

TABLE II.—Material of Investigation Divided into Age-matched Groups. Some of the Control Subjects Appear in Both Groups A and B

	Group A		Group B	
	Control	Bantu	Control	Coronary
No.	52	53	52	48
Age range (years)	20-65	20-65	36-65	38-65
Mean age (years)	34.1	34.6	46.0	52.3
Standard deviation	9.8	8.6	6.8	8.0

but some controls were included in both groups. No attempt was made at age-matched pairs of subjects; only the groups were compared. The primitive Bantu seldom knows his age, which has therefore to be assessed by reference to historical events, puberty rites, etc.; at best the figures should be regarded only as approximations.

The study of plasma fibrinogen was carried out as a separate investigation and included many of the white subjects studied in the main experiment. The Bantu were similar to the ones previously studied. In addition, a group of coronary patients maintained on "long-term" out-patient anticoagulant (phenindione) therapy (three months to five years) was included.

General Procedure

Eight or nine subjects were tested at each session. During one phase of the experiment a coronary group, an age-matched white control group, and a Bantu group were tested together. At other times either a Bantu or a coronary group were tested concurrently with an age-matched white control group. The subjects had been instructed to eat only a very light breakfast containing a minimal amount of fat on the morning of the test. The venepunctures were performed in the early morning and all were carried out by one of the investigators. The order in which the venepunctures were done was determined by random selection, and none of the other investigators or technicians knew from which subject the blood specimens had been obtained. All the blood specimens were collected into silicone-treated syringes, using 18-gauge needles. If difficulty

was encountered with a venepuncture it was abandoned and repeated at another site. The clinical examination, dietary interrogation, and electrocardiography were usually carried out just before or after the venepuncture.

Technical Methods

Serum Chemistry

Serum cholesterol was determined by the method of Abell *et al.* (1952), as modified by Anderson and Keys (1956). Serum albumin and globulin were determined by Weichselbaum's (1946) method as modified by Wolfson *et al.* (1948). Thymol turbidity was estimated by MacLagan's (1944) method as modified by De la Huerga and Popper (1949), zinc sulphate turbidity according to Kunkel (1947), serum urea by the method of Archer and Robb (Harrison, 1947), and serum urate by the spectrophotometric method of Praetorius and Poulsen (1953).

Coagulation Tests

Coagulation times were measured in four glass or four silicone-treated 3 by $\frac{3}{8}$ -in. (7.5 by 1-cm.) tubes as previously described (Merskey and Nossel, 1957).

Prothrombin was measured by the following techniques: (1) One-stage method (Quick, 1957) using acetone-dried human brain as thromboplastin. (2) One-stage method using Russell viper venom ("stypven") 1:20,000 (R.V.V.) as thromboplastin. The test was carried out on both platelet-rich and platelet-poor plasma. (3) The P. and P. method of Owren and Aas (1951). (4) The method of Biggs and Douglas (1953), the so-called "area" method. (5) The method of Hjort *et al.* (1955) using a R.V.V.-cephalin reagent.

Prothrombin Consumption.—The prothrombin present in the plasma was compared with that present in the serum one hour after coagulation had occurred. The prothrombin was measured as previously described (Merskey, 1950) and by the R.V.V.-cephalin method mentioned above.

Factor V was measured by the corrective effect of a 1:10 plasma dilution on normal aged human oxalate plasma.

Factor VII was measured in serum as described by Koller *et al.* (1951). The serum was incubated at 37° C. for eight hours and then stored at -20° C. for 18 hours before being tested. It was then stored at -20° C. until the conclusion of the investigation, when all the specimens could be retested together.

Antihaemophilic globulin was measured as described by Pool and Robinson (1959). This is a modification of the thromboplastin generation test in which a source of lipid (asolectin) is incubated with a dilution of dried human serum, a source of factor V and calcium. After six minutes of incubation, a dilution of alumina-treated plasma is added and tested as in a thromboplastin generation test using citrated human plasma as substrate.

Thromboplastin generation was measured (at one-minute intervals up to 15 minutes) as described by Biggs and Macfarlane (1957). The alumina-treated plasma was diluted 1:10 and the serum 1:20. Three different sources of lipid were used, and to each of these the particular subject's plasma and serum factors were added. The sources of lipid were: the subjects' own platelets freshly prepared; pooled fresh human platelets; and a freshly prepared dilution of asolectin.

Variation in results of tests of blood coagulation between groups was considered valid only when the two groups were tested under similar conditions. For this reason the results in the Bantu and their age-matched white controls were not always comparable with those of the coronary patients with their own controls, and required separate analysis.

Fibrinogen was measured on heparinized plasma diluted 1:10 with saline and clotted with thrombin. The clot was collected, washed in distilled water, dried to constant weight, and weighed.

Fibrinolysis was measured as the clot lysis time by the method described by Fearnley *et al.* (1957) with the following modifications: (a) M/15 phosphate buffer at pH 7.4 was used. (9.47 g. Na_2HPO_4 /litre mixed with 2.27 g. KH_2PO_4 /250 ml.) (b) The blood was diluted first with phosphate buffer 1:10. Two-ml. aliquots of this mixture were then clotted with 0.1 ml. (5 units) of thrombin. The clots were kept in an ice bath for 30–90 minutes before incubation. Lysis of the clot was either observed or recorded photographically as described by Lackner and Goosen (1959).

Diets

The white subjects were asked to describe their food intake during the previous 24 hours and then to indicate how this differed, if at all, from their usual eating habits; from this information an average daily diet was drawn up for each subject. The Bantu subjects were asked to describe their usual food intake in the reserves, and an average daily diet was drawn up for each subject. The approximate calorie and fat content of each of these average daily diets was then estimated from the food tables of Fox and Golberg (1944) and Watt and Merrill (1950); but all these results, and particularly those of the Bantu, should be regarded only as being approximate.

Statistical Analysis

When the comparison was between more than two groups, analysis of variance was performed; if this was significant—or if there were only two groups to be compared—Student's *t* test was carried out on the two groups. No result was regarded as being statistically significant unless *P* was less than 0.05%. For technical reasons it was not always possible to carry out all laboratory tests on all samples.

Results

Diets

The mean daily calorie intakes and the mean fat:calorie ratios are recorded in Table III. It will be seen that the mean daily calorie intakes were significantly different between both the Bantu and coronary subjects and their controls. The fat:calorie ratio was not significantly different in the control and coronary

TABLE III.—*Estimated Average Daily Calorie Intake and Fat: Calorie Ratio. These Figures Should be Regarded as Only Approximations*

Group	No. of Subjects	Mean Daily Calorie Intake and S.D.	Fat: Calorie Ratio % and S.D.	P Less Than	
				Calories	Fat: Calorie Ratio
Bantu	51	2,734 ± 496	18.4 ± 8.5	0.01	0.001
Control	44	3,045 ± 468	43.2 ± 9.2		
Coronary	43	2,606 ± 685	42.2 ± 16.9	0.05	—
Control	47	2,889 ± 595	44.3 ± 17.3		

groups; in the Bantu group, however, it was very much lower. The two white groups consumed the usual foodstuffs of the Western dietary, but it was noted that since their illness some of the coronary subjects had been attempting to restrict their saturated fat intake. Among the Bantu the dietary fat was derived mainly from maize meal and from soured milk (*amasi*).

TABLE IV.—*Mean Serum-cholesterol Levels in Apparently Healthy Bantu Males and Age-matched White Controls. Means are Significantly Different in All Age-groups Tested. Mean Figures for Whole Group Were: Bantu 147 ± 34 mg./100 ml.; Control, 243 ± 46 mg./100 ml. P Less than 0.001*

	21–30 Years		31–40 Years		41–50 Years	
	Control	Bantu	Control	Bantu	Control	Bantu
No.	16	22	25	19	9	9
Mean age (years)	25	26	36	36	45	45
Serum cholesterol (mg./100 ml.)	231	133	236	147	272	171
Standard deviation	42	32	49	35	43	43
P less than	0.001		0.001		0.001	

TABLE V.—*Mean Serum-cholesterol Levels in Men with Coronary Heart Disease and Age-matched White Controls. Figures for Coronary Group are Higher, but Appear to be Significantly so Only in Age-group 36–45 Years. Mean Figures for Whole Group Were: Coronaries, 285 ± 44 mg./100 ml.; Controls, 262 ± 41 mg./100 ml. P Less than 0.01*

	36–45 Years		46–55 Years		56–65 Years	
	Control	Coronary	Control	Coronary	Control	Coronary
No.	24	9	23	20	5	17
Mean age (years)	40	42	49	49	59	60
Serum cholesterol (mg./100 ml.)	251	303	273	284	268	275
Standard deviation	45	50	37	41	23	25
P less than	0.01		—		—	

Serum Chemistry

Serum Cholesterol.—The mean level in the Bantu (147 ± 34 mg./100 ml.) was very significantly lower than in their age-matched white controls (243 ± 46 mg./100 ml.). As will be seen in Table IV, highly significant differences were present in all three decades from 21 to 50 years. The mean level in the coronary group (285 ± 44 mg./100 ml.) was significantly higher than in their age-matched controls (262 ± 41 mg./100 ml.; *P* less than 0.01). In Table V it will be seen that the mean levels of the control and coronary groups differed significantly only at the age of 36–45 years; over the age of 45 years there was no significant difference. The serum-cholesterol levels of all the subjects over the age of 35 years are shown in Fig. 1; while the individual control values overlap considerably with those of the other two groups, there is no overlap of Bantu and coronary values.

FIG. 1.—Mean serum-cholesterol levels in all subjects over the age of 35 years. There is no overlap between the coronary and Bantu groups.

The serum-cholesterol levels of all the subjects over the age of 35 years are shown in Fig. 1; while the individual control values overlap considerably with those of the other two groups, there is no overlap of Bantu and coronary values.

Serum Albumin (Table VI).—The mean level in the Bantu was significantly lower than in the controls, but neither of these differed significantly from the coronary group.

Serum Globulin (Table VI).—The mean level in the Bantu was significantly higher than in the control and coronary groups.

Thymol Turbidity (Table VI).—The mean value in the Bantu was greater than in the control and coronary groups.

Zinc Sulphate Turbidity (Table VI).—This was greatest in the Bantu and least in the coronary group, while the controls were intermediate; all these values were significantly different.

Serum Urea (Table VI).—The mean value in the Bantu was somewhat less than in the coronary group but did not differ significantly from the controls. There were no significant differences between the two white groups.

Serum Urate (Table VI).—The mean level was lowest in the Bantu and highest in the coronary group; the controls had an intermediate mean level, closer to that of the coronary group than to the Bantu figure. All three values were significantly different.

Blood Coagulation (Table VII)

Coagulation Time and Prothrombin Consumption.—

(a) *Bantu and age-matched white controls*: It will be seen that there was no statistical difference between the coagulation time in glass or in silicone-treated tubes, but the residual prothrombin in silicone-treated tubes was much less in the Bantu. This is true for both methods used for assessing prothrombin. (b) *Coronary patients and age-matched white controls*: No significant differences were found between these two groups.

Plasma and Serum Factors.—(a) *Bantu and age-matched white controls*: There was no difference between the two groups in the one-stage prothrombin test using human brain thromboplastin; when R.V.V. was used on platelet-rich or on platelet-poor plasma the results were also similar. Three of the methods used for assessing prothrombin (or prothrombin plus factor VII) showed more prothrombin in the white controls than in the Bantu, though they achieved statistical significance only in the area test. There was no difference in factor V between the two groups, but the Bantu had less factor-VII-like activity. Antihaemophilic globulin activity was greater in the Bantu than in their white controls. (b) *Coronary patients and age-matched white controls*: No significant differences between these two groups could be demonstrated by any of these tests.

Thromboplastin Generation.—(a) *Bantu and age-matched white controls*: With the exception of the first three minutes (when the results were virtually identical) the amount of thromboplastin produced by the Bantu was greater than that produced by the white controls (Fig. 2); this was true whatever the source of lipid. Study of the standard error of the difference of the two means (Table VIII) shows that these apparent differences were significant at the 5% level on only two occasions. By combining figures for each series over approximately five-minute periods, significant differences between the two groups could be shown (Table IX). (b) *Coronary patients and age-matched white controls*: The coronary group form more thromboplastin than the controls (Fig. 3), but the differences reached a 5% level of significance on only one occasion (Table VIII). There were no differences of significance when the figures were combined over approximately five-minute periods (Table IX). (c) *Coronary patients, age-matched white controls, and Bantu patients*: At one stage of the

TABLE VI.—Serum Chemistry in Control, Coronary, and Bantu Groups. $\pm$ Refers to S.D.

	Controls		Coronary		Bantu		P Less Than		
	No.	Mean	No.	Mean	No.	Mean	Control Coronary	Control Bantu	Coronary Bantu
Serum albumin (g./100 ml.)	77	4.9 $\pm$ 0.5	46	4.8 $\pm$ 0.5	48	4.7 $\pm$ 0.3	—	0.01	—
" globulin	77	2.4 $\pm$ 0.5	46	2.4 $\pm$ 0.6	48	2.9 $\pm$ 0.6	—	0.001	0.001
Thymol turbidity (units)	80	1.2 $\pm$ 0.5	48	1.2 $\pm$ 0.4	52	1.5 $\pm$ 0.7	—	0.05	0.01
Zinc sulphate turbidity (units)	80	6.8 $\pm$ 0.3	48	5.3 $\pm$ 2.6	52	11.5 $\pm$ 3.8	0.01	0.001	0.001
Serum urea (mg./100 ml.)	54	25.0 $\pm$ 8.8	28	28.0 $\pm$ 10.2	30	21.6 $\pm$ 6.5	—	—	0.05
Serum urate (mg./100 ml.)	75	5.28 $\pm$ 0.86	45	5.69 $\pm$ 1.10	49	4.12 $\pm$ 0.99	0.02	0.001	0.001

TABLE VII.—Blood-coagulation Tests in the Three Groups. The Bantu and Coronary Groups have been Separately Compared with Different Age-matched White Control Groups

	Bantu			White Control (a)			P Less Than	Coronary			White Control (b)			P Less Than
	No.	Mean	S.D. $\pm$	No.	Mean	S.D. $\pm$		No.	Mean	S.D. $\pm$	No.	Mean	S.D. $\pm$	
Coagulation time (minutes):														
Glass tubes	28	7.53	1.5	27	8.05	1.9	—	20	8.67	1.6	27	8.05	1.9	—
Silicone	53	30.45	6.0	52	32.23	5.8	—	28	34.95	4.9	27	33.96	6.8	—
Prothrombin consumption (%):														
Glass tubes	28	15.58	9.0	27	16.57	9.5	—	20	16.84	12.0	27	16.57	9.5	—
Silicone { (a)	53	58.47	28.3	52	81.73	26.4	0.001	28	101.32	33.3	27	97.59	25.8	—
(b)	25	67.91	28.7	25	90.97	26.3	0.01							
Prothrombin complex:														
Quick's 1 stage (seconds)	44	15.13	2.3	44	14.41	1.7	—	28	14.50	1.4	27	14.18	1.7	—
Stypven (seconds)														
Platelet rich	50	17.21	2.4	50	16.51	3.2	—	28	16.33	2.4	27	16.29	2.5	—
" poor	50	21.48	4.3	50	21.80	4.5	—	28	21.43	4.4	27	21.51	4.1	—
P. and P. (%) (Owren)	53	91.82	28.1	52	103.77	36.1	0.06	28	63.09	15.1	27	68.83	23.7	—
Area (squares)	53	715.55	359.7	52	883.37	398.5	0.03	28	1269.39	507.7	27	1303.04	471.8	—
Stypven cephalin %:														
Plasma	52	95.89	32.2	52	103.81	34.6	—	28	100.00	21.2	27	103.01	17.2	—
Serum	25	35.83	15.3	25	47.43	20.5	0.03	28	74.62	26.6	27	71.62	28.9	—
Factor 5 (%)	28	98.36	43.1	27	101.30	42.4	—	20	100.00	37.2	27	101.30	42.4	—
VII (%) (serum)	53	74.94	25.4	52	91.94	23.3	0.001	27	104.86	43.5	24	86.98	66.5	0.08
Antihaemophilic globulin (%)	50	162.83	78.7	49	119.67	73.3	0.005	28	116.24	33.2	27	104.63	36.4	—

TABLE VIII.—Thromboplastin Generation Test. Thromboplastin Generation and Standard Errors of the Mean at Each Minute After Incubation. The First Pair of Results Refer to Fig. 2, the Next Pair to Fig. 3, and the Final Group to Fig. 4. When only Two Groups are being Compared the Standard Error of the Difference Between the Means is Shown. The Points at which the Differences are Significant at the 5% Level are Shown,† while the 10% Level is Shown*. When the Comparison is Between Three Groups the Standard Error of the Mean of Each Set is Shown Separately. Analysis of Variance Showed no Significant Differences in these Three Groups

	Time after Incubation (Minutes)														
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
Control (%)	6.7	22.1	48.9	66.2	73.2	77.9	77.7	75.5	76.2	74.1	71.2	68.9	67.1	66.4	65.3
Bantu (%)	6.5	19.1	47.2	68.9	82.4	83.1	85.9	82.0	81.0	78.3	77.6	74.8	72.7	70.2	67.8
S.E. (diff.)	0.76	4.04	5.67	5.48	4.84*	4.37	4.01†	3.74*	3.53	3.34	3.13†	3.25*	3.04*	3.15	2.98
Control (%)	5.4	23.7	58.3	73.4	81.9	85.2	86.3	82.6	82.6	83.3	81.5	78.2	75.6	73.6	73.9
Coronary (%)	7.9	27.8	64.1	86.7	88.3	88.6	86.6	84.7	84.3	85.1	82.7	81.3	79.1	73.9	72.6
S.E. (diff.)	1.46*	6.51	8.00	6.58†	4.83	4.33	3.74	3.89	3.88	3.64	3.61	3.65	3.66	4.22	3.82
Control (%)	7.2	20.8	44.4	62.9	78.9	86.9	86.4	83.3	84.6	80.1	76.0	73.7	71.4	72.0	70.0
S.E. (diff.)	0.83	4.55	5.91	6.01	5.25	4.39	3.79	3.50	3.05	3.37	3.17	3.28	3.09	3.29	3.00
Bantu (%)	7.3	20.6	48.5	74.4	90.3	91.7	95.5	91.1	86.6	85.6	84.7	81.1	78.8	76.1	72.5
S.E. (diff.)	0.94	3.96	5.64	5.54	4.80	4.31	4.07	3.68	3.50	2.90	2.76	2.96	2.84	2.88	2.78
Coronary (%)	7.8	21.4	50.4	81.4	90.6	95.2	93.7	93.1	91.3	87.7	86.9	82.6	78.8	75.3	73.4
S.E. (diff.)	1.06	5.08	6.53	7.00	6.29	5.60	4.83	4.31	4.01	3.99	4.38	3.72	4.23	4.48	4.44

investigation age-matched Bantu males were being tested at the same time as white controls. Simultaneously, some older coronary patients were also being tested, and these results are shown in Fig. 4. There was little difference between the coronary and Bantu patients; both these groups showed greater thromboplastin generation than the controls. Analysis of variance did not show any significant differences

FIG. 2.—Thromboplastin generation test; Bantu and control. The Bantu produces more blood thromboplastin than the controls after the first three minutes.

FIG. 3.—Thromboplastin generation test; coronary and control. The coronaries produce more blood thromboplastin than the controls, but the differences are less striking than in Fig. 2.

between the three groups at any one minute. Fig. 2 includes all this information together with additional data so far as Bantu and their age-matched controls are concerned. In Fig. 3 many of these coronary patients have been retested together with age-matched controls. For these reasons the information has not been further analysed. The main purpose of this part of the investigation was to provide a direct comparison between Bantu and coronary patients even though it was recognized that they would not be age-matched.

Plasma Fibrinogen

*Bantu and Age-matched White Controls (Table X).—*There were no significant differences between these two groups.

TABLE IX.—Thromboplastin Generation Test. Data From Which Figs. 2 and 3 were Derived Have Been Combined Over Approximately Five-minute Periods

	Time after Incubation (Minutes) Inclusive			
	1-5	5-10	11-15	1-15
Bantu:white:				
Mean difference (%)	1.35	6.36	4.87	3.99
P less than ..	—	0.01	0.01	0.01
Coronary/control:				
Mean difference (%)	6.34	2.63	1.40	3.18
P less than ..	—	—	—	—

FIG. 4.—Thromboplastin generation test; Bantu, coronary and control. More blood thromboplastin is produced by the Bantu and the coronary groups than by the controls, but there are no significant differences between the Bantu and coronary groups.

Coronary Patients and Age-matched White Controls (Table XI).—The mean plasma fibrinogen was significantly higher for patients over the age of 50 in both coronary groups when compared with controls, these differences not being present under this age. There were no significant differences between these coronary patients and patients on long-term phenindione therapy as out-patients (Table XI).

TABLE X.—Mean Plasma Fibrinogen in Bantu and Age-matched White Controls. There are no Significant Differences

Age	Bantu		White Controls	
	No.	Fibrinogen (mg./100 ml.)	No.	Fibrinogen (mg./100 ml.)
17-30	23	350	32	332
31-40	19	376	25	358
41-50	7	356	21	356
Total	49	361	78	347

TABLE XI.—Mean Plasma Fibrinogen in Control Group Compared with Two Groups of Patients with Coronary Heart Disease, One of the Latter Being on "Long-term" Out-patient Phenindione Anticoagulant Therapy. There are Significant Differences Over the Age of 50 Years

Age:		38-50 Years		Over 50		Total	
		No.	Mean	No.	Mean	No.	Mean
A	White control	28	366	11	351	39	362
B	Coronary	14	375	15	441	29	409
C	Long-term anti-coagulant	13	413	25	409	38	410
D	All coronaries	27	393	40	421	67	410
A B	P less than ..	—		0.01		0.02	
A C	P	—		0.05		0.01	
B C	P	—		—		—	
A D	P	—		0.02		0.01	

TABLE XII.—Mean Plasma Fibrinogen in Patients with Coronary Heart Disease. Patients Have Been Subdivided into Three Groups, Depending on Time that Has Elapsed Since the Last Clinical Episode Suggesting Cardiac Infarction. There are No Significant Differences

	Time Elapsed Since Last History of Infarct							
	Less than 6 Months			Over 6 Months			Angina Only	
	No. of Patients	Mean Plasma Fibrinogen (mg./100 ml.)	Standard Error	No. of Patients	Mean Plasma Fibrinogen (mg./100 ml.)	Standard Error	No. of Patients	Mean Plasma Fibrinogen (mg./100 ml.)
No anticoagulants ..	—	—	—	26	409.3	16.2	3	406.0
Anticoagulant-treated groups	9	403.3	23.4	24	417.2	15.9	5	390.0
Total ..				50	413.1	11.2	8	396
								32.0

Since it has been shown by Meyers (1948) and Gilchrist and Tulloch (1952) that plasma fibrinogen is elevated immediately after a cardiac infarction (and that this elevation can persist for at least three months) the fibrinogen data have been further subdivided into three groups (Table XII): (a) No history of infarction; angina only. (b) History of cardiac infarction within six months of plasma fibrinogen estimation. (c) History of cardiac infarction more than six months prior to test. There were no significant differences between these three groups.

Fibrinolysis

Bantu and Age-matched White Controls (Fig. 5).—The mean lysis time for the Bantu was 5.6 hours, which

was almost half that of their own controls (10.4 hours), this difference being statistically significant ($P < 0.01$). With one exception all the Bantu specimens lysed in under 16 hours.

Coronary Patients and Age-matched White Controls (Fig. 6).—There were no statistically significant differences between clot lysis time in the coronary group (12.3 hours) and their age-matched white controls (11.7 hours). A lysis time of over 16 hours occurred in 16.8% (21 out of 125) of the control and coronary groups, the longest recorded time being 124 hours in a coronary patient. For purpose of analysis the upper limit was regarded as being 60 hours.

Discussion

Control and Coronary Groups

The coronary group and their age-matched controls were roughly comparable with respect to socio-economic status and geographical environment; dietary calories and total fat consumption were similar and there were no material differences in their serum proteins, liver-function tests, and serum urea. The higher mean serum-cholesterol level in the coronary group is in accordance with the findings in several other surveys (Steiner *et al.*, 1952; Oliver and Boyd, 1953; Gertler and White, 1954; Lawry *et al.*, 1957). The observa-

FIG. 5.—Clot lysis times in the Bantu and control groups. The Bantu has more rapid clot lysis.

FIG. 6.—Clot lysis times in the coronary and control groups. There are no significant differences.

tion of Gertler and White (1954) that the mean serum-urate level is higher in persons with coronary heart disease than in normal controls is also supported by the present study. The implications of this abnormality and of the low mean serum-urate level in the Bantu group will be considered elsewhere (Sougin-Mibashan, 1959). Thus from both the sociological and the biochemical aspects the subjects of the control and coronary groups may be regarded as being fairly representative of their respective populations, and a comparison of their coagulation functions and fibrinolytic activity seems to be justifiable.

We have used a large number of tests in an attempt to assess efficiency of blood coagulation. Few of these

tests actually measure specific factors, even though the titles used for these tests appear to imply such specificity. We would prefer the term "specific factor activity," but this might add even more confusion. Among the many tests of the clotting mechanism which were carried out, only the measure of the plasma-fibrinogen level gave any indication of an increased coagulability of the blood in the coronary group, and this only in patients over the age of 50 years. In their recent comparison of the blood of coronary patients and healthy subjects in London, McDonald and Edgill (1957) also found a higher plasma-fibrinogen level in the coronary subjects. In addition, however, they found increased plasma thromboplastin generation and an increased prothrombin time with Russell viper venom (findings we could not confirm) and increased platelet stickiness (a test we did not do) in their patients with coronary heart disease.

The significance of the increased plasma-fibrinogen levels in the coronary cases is not clear; even if it is connected with the development of coronary heart disease it could be the result rather than the cause of the disease. It is noteworthy that the difference in plasma-fibrinogen levels between the coronary and control groups is statistically significant only in those over the age of 50. There was no evidence that long-term therapy with anticoagulant drugs like phenindione decreased this elevated level; nor could it be related to the temporary elevation of plasma fibrinogen which occurs in the first month after an acute episode of cardiac infarction. There were no significant differences in the level of plasma fibrinogen in patients who had had their last symptoms of sustained myocardial ischaemia within the preceding six months when these were compared with those in whom this period was exceeded or when compared with those patients whose presenting symptoms had only been argina of effort.

Fibrinolysis times in the two groups did not differ significantly, and from this and from the results of the coagulation tests it must be concluded that with the exception of the raised fibrinogen level this phase of the investigation failed to show any material difference in the blood of the coronary and control groups.

These results do not, however, exclude the possibility that increased coagulability of the blood plays some part in the development of coronary heart disease; it may merely indicate that the two groups of subjects were inadequately contrasted for the available tests to reveal a difference in their blood coagulability. Thus, though the control group were free from *clinical* evidence of coronary heart disease, many probably had quite substantial coronary atherosclerosis and could be regarded as potential cases of overt coronary heart disease. In fact, two subjects who were originally selected as controls were found to have electrocardiographic evidence of cardiac ischaemia, while another subject who was apparently quite well at the time of the investigation developed angina pectoris with electrocardiographic changes about one week later. These three subjects were excluded from the control group, but they illustrate the difficulty of ensuring that any member of a "privileged" Western community is free from severe coronary atherosclerosis.

Another factor which may have tended to obscure the differences between these two groups is the diet. In Capetown many practitioners are advising their

coronary patients to restrict their dietary fat intake or to replace saturated fats by unsaturated fats; in addition, reports in the lay press about the possible "dangers of saturated fats" have sometimes been misleading and often misinterpreted. As a result some coronary patients in this area are attempting to modify their diets and are either reducing their fat intake or replacing saturated fat by unsaturated fat. In this investigation, in our initial selection of material, we excluded those who were consuming large dietary supplements of unsaturated fats; but many of our coronary subjects had been practising some form of dietary restriction since the onset of their disease, and this may have played some part in accounting for the absence of a significant difference between the fat:calorie ratio of this group and their controls.

A number of workers have claimed that a fat-containing meal shortens the coagulation time—for example, Fullerton *et al.* (1953) and Keys *et al.* (1957)—and though work in this department and elsewhere has failed to confirm this (Merskey and Nossel, 1957; Tulloch *et al.*, 1953; Manning and Walford, 1954; Sohar *et al.*, 1957), lipids do play a part in the blood-coagulation process *in vitro* (Poole, 1955, 1958). Furthermore, Greig (1956) has shown that the consumption of saturated fats decreases fibrinolytic activity. It is therefore just possible (but very unlikely) that, as a result of their dietary manipulations, the coronary subjects may have modified a previous state of hypercoagulability or diminished fibrinolytic activity of their blood.

A third factor which should not be overlooked is that the existing methods for testing hypercoagulability are inadequate. Moreover, *in vitro* tests of coagulation should not be regarded as being synonymous with tests of the formation of intravascular thrombi.

Control and Bantu Groups

It seemed to us that a more satisfactory contrast would be obtained by comparing the blood coagulation and fibrinolysis of white controls with Bantu subjects; the latter consume a relatively constant diet of low-fat content (Walker and Arvidsson, 1954; Bronte-Stewart *et al.*, 1955); they very rarely present with clinical evidence of coronary heart disease, and it is uncommon to find severe coronary atherosclerosis in them at necropsy (Higginson and Pepler, 1954; Brock and Gordon, 1959). In order to avoid as far as possible the effects of a "white" environment, subjects were selected from those who had been living in the native territories and who had only just arrived in Capetown. As these men were all relatively young, physically fit unskilled labourers, it was not possible to age-match them with coronary subjects; the age matching with white controls, however, was achieved adequately.

From the available data it appears that the subjects studied were a representative sample of rural Bantu. Their dietary fat:calorie ratio of 18% was much the same as that found in previous surveys (Walker and Arvidsson, 1954; Bronte-Stewart *et al.*, 1955). Their serum-cholesterol levels were generally much lower than in the white controls. In the Bantu subjects the serum-albumin level was lower and the serum-globulin level was higher than in the white controls, while both the zinc-sulphate-turbidity and the thymol-turbidity tests were higher than in the controls—that

is, changes which frequently are observed in apparently healthy Bantu subjects (Bersohn *et al.*, 1954).

The results of the coagulation tests are difficult to interpret. Coagulation times did not differ significantly in the two races, nor did the plasma levels of factor V and fibrinogen. As judged by the lower plasma levels of prothrombin (by the area method and possibly the P. and P. method) and the lower serum factor VII levels, the blood of the Bantu could be regarded as less coagulable than that of the white controls; on the other hand, the better prothrombin consumption and the increased levels of plasma antihaemophilic globulin might suggest that the blood of the Bantu is potentially more coagulable than that of white controls. Studies of thromboplastin generation showed that the Bantu certainly did not produce less blood thromboplastin than the white controls; in fact, what differences there were suggested greater production. Because of these conflicting results, and until more is known of the relative importance of the above-mentioned factors as criteria of coagulability, we can only conclude that no clear evidence has been found to show that the comparative immunity of the Bantu to severe coronary atherosclerosis and to clinical coronary heart disease is due to a lesser coagulability of his blood.

The mean fibrinolysis time in the Bantu was significantly shorter than in white controls. Fig. 6, however, shows that there was considerable overlap between the two groups, and it appears that the longer mean lysis time of the controls was partly due to the presence of a number of samples taking a long time to lyse. Thus, among the Bantu only one sample took longer than 16 hours to lyse, whereas 16.8% of samples from the coronary and control subjects took over 16 hours to lyse and 12.8% took over 20 hours.

We can do no more than speculate on the significance of our findings in relation to atherogenesis. Rokitsky's (1841) conception of a deposition of fibrin on the arterial intima as being the initial lesion of atherosclerosis has been canvassed with enthusiasm by several modern workers, notably Duguid (1955) and Morgan (1956). In the healthy organism a dynamic equilibrium exists between the fibrin-deposition mechanism and the fibrinolytic process (Astrup, 1956). It is tempting to suggest that during phases of prolonged lysis time—that is, when fibrinolysis is inhibited—unrestricted deposition of fibrin occurs and atherogenesis is promoted. Thus in the white race, who appear to be prone to phases of prolonged lysis, severe atherosclerosis is more common than in the Bantu.

It remains to consider the reason for the difference in fibrinolytic activity in the two races. On the basis of Greig's (1956) observations, it is possible that the relatively high fat consumption of the whites inhibits their fibrinolysis. On the other hand, Kwaan *et al.* (1956) and Gillman *et al.* (1957) have reported that fibrinolytic activity is increased in liver disease, and it may be that rapid lysis, together with low plasma-prothrombin and serum-factor-VII levels are, like the high serum-globulin level, manifestations of hepatic dysfunction in the Bantu. The assessment of the role of genetic factors, dietary factors, chronic infections, liver dysfunction, etc., in relation to blood chemistry and the pathogenesis of various diseases presents a most complicated problem whose further discussion is beyond the scope of this paper.

Finally, it will be recalled that acute anxiety and injections of adrenaline increase fibrinolytic activity (Macfarlane and Biggs, 1948). It would be reasonable to expect that the apprehension and excitement produced by a large-scale medical examination, including venepuncture, would be greater in rural Bantus who had just come to a big city than in locally resident white men who are more accustomed to such adventures; this may partly account for the more rapid lysis times in the Bantu subjects.

Summary

A comparative study has been made of a number of blood-coagulation factors and of blood fibrinolysis in age-matched groups of healthy white men and in white men with overt coronary heart disease. The coronary cases differed from the controls in having higher plasma-fibrinogen levels in patients over the age of 50. No other significant differences could be recorded.

The same factors were compared in healthy rural Bantu men and in age-matched healthy urban white men. The Bantu differed from the controls in having lower plasma-prothrombin and serum-factor-VII levels and in having better prothrombin consumption and higher plasma levels of antihaemophilic globulin. They generated more plasma thromboplastin than did the controls. Fibrinolytic activity was generally faster in Bantu subjects.

No differences in thromboplastin generation could be shown between Bantu subjects and patients with coronary heart disease.

The significance of these observations in relation to coronary heart disease is discussed.

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First-aid boxes, cases, and cupboards for all workers in factories, at docks, or on building-sites throughout the country have had important changes made in their contents from January 1, when new regulations made by the Minister of Labour under the Factories Acts came into operation. These changes have arisen from the advances made in medical and first-aid treatment of injuries during the last quarter of a century. For example, antiseptics, special burn dressings, and cocaine eye-drops will no longer be required. A leaflet giving advice on treatment, which has been revised in the light of these advances, must be included in each first-aid box. All materials have to be of a grade and quality not lower than the standard specified by the *British Pharmaceutical Codex*, and specifications for adhesive dressings for wounds and for eye ointment, for which the regulations provide, are given in certificates of approval of the Chief Inspector of Factories (Ministry of Labour).

BLEEDING OESOPHAGEAL VARICES TREATED BY OESOPHAGEAL COMPRESSION TUBE

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Oesophageal tamponade for the arrest of haemorrhage from oesophageal varices was introduced by Westphal (1930), who applied local pressure by means of an oesophageal sound. Later an inflatable rubber balloon on a modified Miller-Abbott tube was used to compress the coronary veins in the fundus of the stomach which drain into the oesophageal varices (Rowntree *et al.*, 1947; Bixby, 1948; Tocantins, 1948; Barnett and Cohen, 1949). The introduction by Sengstaken and Blakemore (1950) of a tube with two balloons enabled local pressure on the varices by the oesophageal balloon to be added to the effect on the fundal veins of the gastric balloon (Fig. 1).

FIG. 1.—Sengstaken oesophageal compression tube.

The purpose of this paper is to describe and discuss the indications for and the results obtained with this apparatus in relation to a group of 101 patients with alimentary bleeding associated with portal hypertension seen at Hammersmith Hospital during the years 1954 to 1958.

Apparatus and Indications for Use.—The Sengstaken tube was a standard three-lumen instrument made of stiff rubber with latex rubber balloons (manufactured by J. G. Franklin and Sons Ltd., London). If possible a new apparatus was used for every second patient, and when not in use it was stored in the refrigerator to maintain its stiffness. The indication for use was haemorrhage from oesophageal varices uncontrolled by liberal blood transfusion and bed rest.

Introduction of the Tube

The apparatus was thoroughly tested for leaks. The alert and co-operative patient was told the purpose of the manoeuvre and his co-operation obtained. The pharynx was sprayed with 4% lignocaine, the patient being propped up in bed. A bandage was placed over