Reprinted from
Journal of Atherosclerosis Research
Elseviers Publishing Company
Amsterdam
Printed in The Netherlands

CARDIOVASCULAR DISEASE IN THE MASAI

G. V. MANN, R. D. SHAFFER, R. S. ANDERSON AND H. H. SANDSTEAD

with the technical assistance in the field of

H. PRENDERGAST, J. C. MANN, S. ROSE, J. POWELL-JACKSON, S. MOITANIK, J. OL MONAH, S. M. ISAAC, ONESIMO H. MSANGI AND E. FRANK

and in the laboratory of

J. MARTIN, J. LANE, I. RASMUSSEN AND K. DICKS

The Nutrition Division, Vanderbilt University Medical School, Nashville, Tenn. (U.S.A.); the Tanganyikan Ministry of Health, Dar es Salaam (Tanganyika); and the African Medical and Research Foundation, Nairobi (Kenya)

(Received August 9th, 1963)

INTRODUCTION

The hypothetical relationship between dietary behavior and the development of atherosclerosis and consequent cardiovascular complications is not well established. While it is widely said that a large intake of animal fat and particularly of saturated fats produces hypercholesterolemia and by its influence atherosclerosis, there are a number of epidemiologic observations which fail to support this relationship[1–5].

If this hypothesis were true it would seem necessary that groups of people, who for cultural reasons habitually take large amounts of animal fat would have both hypercholesterolemia and extensive cardiovascular disease. With the intent of examining this matter we have carried out a survey of the prevalence of cardiovascular disease and of levels of serum cholesterol among the *Masai* of Tanganyika.

It is of historical interest that one of the early and now classical dietary surveys was done 35 years ago among the *Masai* and their vegetarian neighbors, the *Kikuyu*, by ORR AND GILKS[6]. They observed striking differences of physical form between the two tribes. The advantages of the Masai were attributed to their high protein diet.

While this study was underway a similar kind of study was completed by SHAPER *et al.*[7,8] among the *Samburu* tribe of the Northern Frontier Province of Kenya. Those people are related ethnologically to the *Masai*. They are also pastoral, Nilo-Hamitic people, who were found to have a high intake of animal fat. Among the men this amounted to 300–400 g daily. As among the Masai, the Samburu warrior or murran class at the time of its initiation, about age 14 years, is bound by tribal tradition to a diet of milk and meat for the next two decades. No vegetable products are taken. It is among this warrior class that the animal fat intake is high both in the Samburu and in the Masai.

SHAPER observed a very low prevalence of hypertension and a low prevalence of arteriosclerotic heart disease as evidenced by electrocardiography. That study was based upon a sample of 200 men.

The present study describes the findings in 400 Masai men and some additional women and children examined during a four-week period in 1962 in South Masailand of Tanganyika (Fig. 1). A portion of the sample was re-examined in May 1963 at the peak of the milk flow in the Masai cattle.

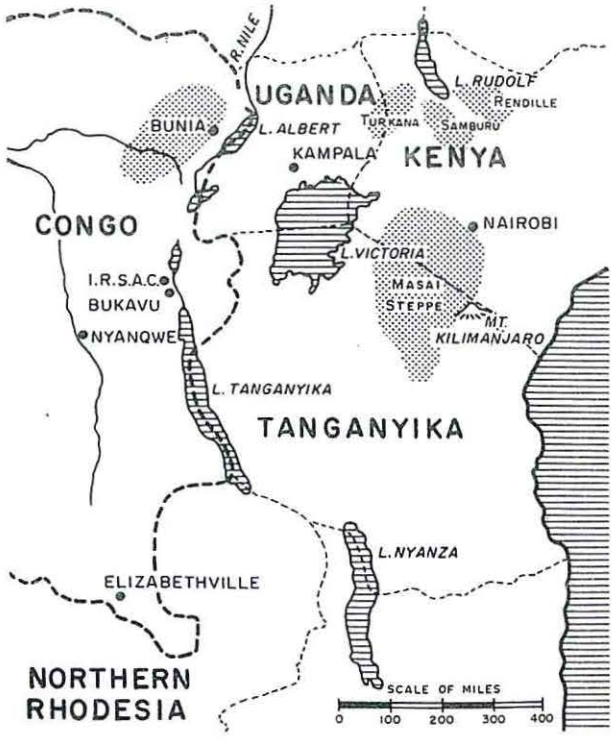

Fig. 1. The studies of the *Masai* were done in Tanganyika at the southern end of the Masai Steppe. SHAPER AND WILLIAMS[8] studied the *Samburu*, the *Rendille* and the *Turkhana* in northern Kenya in the areas indicated. The studies of *Congo Pygmies*[9] were done in 1960 in the Ituri Forest west of Lake Albert.

METHODS

The clinical methods were similar to those which have been described in an earlier publication of work with the *Pygmies* of the *Ituri Forest* region of Congo[9]. A mobile clinic was organized with the facilities of the *African Medical and Research Foundation*. Since the men would not attend the same clinic with the women and children, two units were drawn up near the thornbush enclosed clusters of houses. The cardiovascular survey was carried out along with a venereal disease control program which was the particular interest of the *Ministry of Health* of the Tanganyikan government. It was believed by both the Masai and the officials that increasing in-

fertility among the Masai is caused by extensive venereal disease. For this study blood was obtained for serologic tests and at the time of examination every adult, both men and women, was given an intramuscular dose of benzathine penicillin. The additional inducements for attendance at the clinic were the distribution of plastic containers, jewelry, tobacco and ochre which proved attractive to these people. There is reason to believe that virtually all the men in the three principal areas of the survey were seen. Since there is no census and no ready way of knowing the exact population this can only be surmised.

The men were registered, assigned a serial number, and their age class determined by *Masai Rural Medical Aids*. The height and weight were recorded and skin folds were measured with LANGE calipers at three sites: triceps, scapula and flank. Anthropometric calipers were used to measure the biacromial, the bicristal and the bicondylar widths. The hand strength was measured with a SMEDLEY dynamometer. The subject was then taken into the mobile unit and placed in a supine position on an examining table. He was given a brief physical examination which included a systematic recording of abnormalities of the eyes, skin, teeth, mouth and mucous membranes. Enlarged nodes were sought and the neck was palpated for the thyroid.

The blood pressure was measured in the left arm. The systolic pressure and the pressure at the disappearance of the diastolic sound were recorded. When the pressures were borderline or abnormal ($\geqslant$ 140/90 mm Hg) repeated pressures were measured and the lowest pressure obtained was used in analysis. The point of maximum impulse of the heart and evidences of enlargement were noted. The heart was examined for murmurs by grade, location and quality. The abdomen was examined for liver, spleen and other masses. The extremities were examined for abnormalities of skeletal conformation and edema and the dorsalis pedis pulses were sought. A standard 12-lead electrocardiogram was recorded with a portable, direct writing, battery machine. Two samples of blood were obtained in evacuated tubes. The first was a 15 ml sample anticoagulated with ethylenediaminetetraacetate. Blood was taken from the needle tip for measurement of hemoglobin immediately by the cyanomethemoglobin method[10] and hematocrit using the Yellow Springs electrohematocrit[11]. The serum and plasma were recovered and placed in separate vials for freezing in dry ice. This material was kept frozen and transported to the base laboratory in the United States where the remaining chemical measurements were made.

At the completion of the physical examination the patient was treated for complaints and amenable signs. These were principally malaria, anemia, trachoma and skin infections. Each man was given 2.4 mega units of benzathine penicillin intramuscularly and a bright copper disc on which was inscribed his examination serial number. This was firmly fastened to his jewelry.

The frozen sera and plasma returned to the laboratory were used for examination of serum total cholesterol level by the method of MANN[12], for total plasma protein with the biuret method using an Autoanalyzer[13] and for plasma protein electrophoresis on cellulose acetate paper by the method of BRECKENRIDGE[14]. Samples of serum were

forwarded to the *Venereal Disease Research Laboratory of the Communicable Disease Center, U.S. Public Health Service, Atlanta, Georgia*. Under the direction of Dr. BRITTAIN MOORE AND Mrs. VIRGINIA FALCONE, serological tests for syphilis were carried out. Those sera which showed positive reactions to either the VDRL[15] or the Rapid Plasma Reagin Card Test[16] were then subjected to the *Treponema pallidum* immobilization test (TPI) for confirmation[17].

ECG

The electrocardiograms were read by the criteria which are used in the *Framingham Study*[18]. After one reader had reviewed the tracings all abnormal or doubtful tracings were reviewed by Dr. T. R. DAWBER, Director of the *Framingham Study*. The frequency of the so-called "juvenile pattern"[19] lead us to categorize these separately. Following the experience of GRUSIN[20] and of GOTTSCHALK[21] four types of juvenile pattern were classified as follows.

(*1*) Elevated ST segment in leads V_1–V_3 of 2 mm or more.

(*2*) High-peaked T-waves in the chest leads amounting in V_4 to 40% or more of the sum of the R- and S-waves in V_4.

(*3*) Inversion of T-waves through V_3.

(*4*) Isolated T-wave inversion in V_4.

TABLE I

MASAI-SAMPLE EXAMINED

Class	*Age (years)*	*Number examined*	
Children	1–13		45*
Junior	14–19	36	
Middle	20–24	95	
Senior	25–29	27	
Murran			158
Nyangusi	30–43		80
Derito	44–55	150	
Dareto	>55	3	
Elders			153
		Total	436**

* The 45 male children were bled but not examined in May 1963.

** 9 of the 400 adult men were not Masai, mostly Wadorobo. 400 + 45—9 = 436.

Age

The matter of assigning the age of these people requires comment. Since there is no written record of birth the only practical procedure is to deduce age from the known "age-set" or cohort which each man joins when he is initiated at puberty[22]. The nature of the age structure of the South Masailand people and a description of the sample examined is shown in Table I. While it is not supposed that these ages

TABLE II

MASAI MEN ANTHROPOMETRY

Anthropometric values	*Age groups (years) and number examined*					
	14–19 (36)	*20–24 (95)*	*25–29 (27)*	*30–43 (80)*	*44–55 (150)*	*55+ (3)*
Height (cm)	165.6 ± 1.42	171.6 ± 0.74	173.3 ± 1.27	170.9 ± 0.71	172.0 ± 0.56	160.5 ± 3.89
Weight (kg)	46.2 ± 1.2	56.1 ± 0.72	60.8 ± 1.2	57.3 ± 0.69	58.8 ± 0.63	48.4 ± 1.5
Biacromial (cm)	32.3 ± 0.44	34.9 ± 0.21	35.6 ± 0.46	35.0 ± 0.20	35.3 ± 0.16	30.2 ± 1.53
Bicristal (cm)	23.8 ± 0.31	25.4 ± 0.22	24.6 ± 0.25	26.0 ± 0.16	26.6 ± 0.13	27.8 ± 3.7
Bicondylar (cm)	6.69 ± 0.13	7.59 ± 0.17	7.77 ± 0.17	7.80 ± 0.09	7.43 ± 0.07	7.67 ± 0.88
Skin folds						
triceps (mm)	4.58 ± 0.18	5.36 ± 0.17	6.28 ± 0.34	5.98 ± 0.22	6.40 ± 0.27	4.00 ± 0.58
flank (mm)	6.65 ± 0.25	7.72 ± 0.22	10.2 ± 0.46	8.13 ± 0.28	8.74 ± 0.49	5.17 ± 0.09

TABLE III

CHANGE OF SKIN FOLDS IN MASAI MEN 1962–63

	Age (years)	*Number*	*Year*	*Triceps*			*Flank*		
				$\bar{x}$ *mm*	*Change*	*Significance P*	$\bar{x}$ *mm*	*Change*	*Significance P*
				A. Comparison of same men					
Murran	14–29	8	1962	5.56	+ 1.07	> 0.3	8.31	— 1.94	> 0.20
			1963	6.63			6.37		
Nyangusi	30–43	16	1962	6.34	+ 1.44	> 0.2	8.50	+ 0.25	> 0.80
			1963	7.78			8.75		
Derito	44–55	40	1962	7.78	+ 1.62	> 0.2	10.77	— 0.47	> 0.50
			1963	9.40			9.80		
				B. Comparison of all men					
Murran	14–29	20	1962	5.34	+ 1.81	< 0.01	7.91	— 0.71	> 0.10
			1963	7.15			7.20		
Nyangusi	30–43	34	1962	5.98	+ 1.69	< 0.01	8.13	+ 0.56	> 0.10
			1963	7.67			8.69		
Derito	44–55	40	1962	6.40	+ 2.49	< 0.01	8.74	+ 0.61	> 0.50
			1963	8.89			9.35		

are accurate within more than 3–5 years, they do, within those limits, categorize the subjects. There were nine of the 400 men examined who were not Masai, usually instead Wadorobo and they have not been included in the classifications. It is noteworthy that there were few aged men.

RESULTS

Anthropometric values

The anthropometry in Table II shows that these men are not unusually tall but approximate the average height in Caucasian people[23]. The widespread belief that they are exceptionally tall persons is fostered by their unique dress and body type which is of linear proportions. It is also notable that they are lean people, few of them exceeding 135 pounds. This leanness is confirmed by the skin fold measurement. An unfatted, double thickness of skin has a 3 mm thickness. Most American men of this age range would show skin fold thickness at the triceps area of 11–14 mm and obese men would measure over 20 mm[24].

An extensive drouth followed by disastrous floods occurred in parts of East Africa in 1961. While this affected the Masai in Kenya it did not cause important inanition in South Masailand. Evidence for this is shown in the comparative skin fold measurements in Table III. In May 1963 additional skin fold measurements were made on 54 of the men examined in August 1962 and 40 additional men. These were made at the end of the rainy season when the grass was lush and the milk flow copious. If famine residue had persisted in August 1962 we would expect to see consistent and larger differences in the skin folds at the two seasons.

The bicristal widths which are shown (Table II) as a measurement of laterality

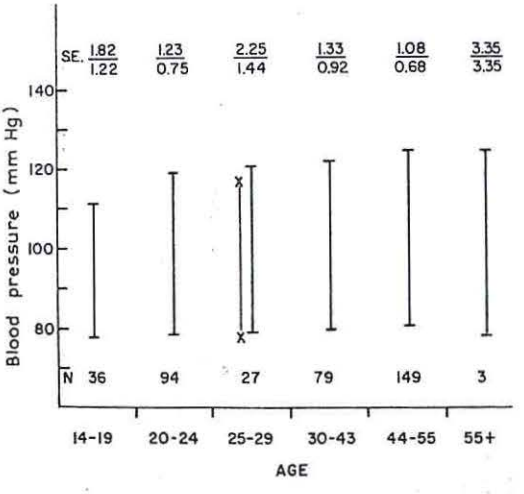

Fig. 2. The mean blood pressures by age for Masai men are shown with the standard errors of these means above. The blood pressures recorded by ORR AND GILKS[6] in 1928 for young Masai men are shown as the x values.

indicate first that the young murran have not reached their adult dimensions and that the adults after age 20 are several centimeters narrower than would be found in American males of these ages[25].

Blood pressure

The blood pressures in Table IV indicate that definite hypertension was unusual among these men. There were 6 men with systolic blood pressures equal or over 160 mm Hg and only 5 in the entire group with diastolic pressures of 100 mm Hg or more.

TABLE IV

DISTRIBUTION OF BLOOD PRESSURES BY AGE
(MASAI MEN)

Age	*Total number*	*Number with systolic blood pressure (mm Hg)*							
		90–100	*100–110*	*110–120*	*120–130*	*130–140*	*140–150*	*150–160*	*160 +*
14–19	36		6	10	9	11			
20–24	94	3	8	31	28	20	3		1
25–29	27		4	4	12	4	3		
30–43	79		9	21	21	22	5		1
44–55	149		10	36	42	43	8	6	4
55 +	3				2	1			

		Number with diastolic blood pressure (mm Hg)						
		60–70	*70–80*	*80–90*	*90–100*	*100–110*	*110–120*	*120 +*
14–19	36	4	13	18	1			
20–24	93	7	36	45	5			
25–29	27	2	12	11	2			
30–43	79	4	20	46	7	1	1	
44–55	149	6	58	64	17	4		
55 +	3		1	2				

TABLE V

CLINICAL IMPRESSION OF HEART DISEASE

Age	*Total number*	*Rheumatic*	*Congenital*	*Hypertensive*	*Luetic*	*All other*	*Total number with heart disease*
14–19	35	1					1
20–24	95	2					2
25–29	26	2	1				3
30–43	79	1				3	4
44–55	148	1		1	1		3
55 +	3						
Total	386	7	1	1	1	3	13
Percentage		1.81	0.25	0.25	0.25	0.77	3.36

Furthermore, there was very little, if any, evidence of the age trend of blood pressure which is generally found in American or Western European males (Fig. 2). It is probable that these blood pressures overestimate the extent of real high blood pressure because of the apprehension caused by the examination.

Heart disease

There was, in general, remarkably little heart disease (Table V). Most of those diagnoses were based upon murmurs louder than grade 2 which the examiners attributed to "rheumatic heart disease". Since there is no way of differentiating that cause of murmur from endomyocardial fibrosis[26] it may be that this has been confused. However, there are no recorded autopsy observations on the prevalence of endomyocardial fibrosis among the Masai. There was one instance of an inter-atrial septal defect and there was one man who met the criteria for hypertensive heart disease. There was one instance in a man 30–43 years (Nyangusi) of "pulseless" or TAKAYUSHU's disease[27].

The electrocardiographic evidence is summarized in Table VI. The most prevalent abnormality was non-specific T-wave change. Aside from these there were 13 instances of conduction defects which included one atrial arrhythmia, one WENCKEBACH's

TABLE VI

MASAI ELECTROCARDIOGRAPHY
CLASSIFICATION OF FINDINGS

	Age groups (years) and numbers examined						*Total (391)*	*Rate/ 1000*
	14–19 (36)	*20–24 (95)*	*25–29 (27)*	*30–43 (80)*	*44–55 (150)*	*55 + (3)*		
Normal c̄ PVB*			1	3	6		10	26
Juvenile pattern	17	26	18	21	34		116	247
ST elevated	7	15	16	10	15		63	161
peaked T	2	4	1	8	16		13	79
inverted T_3	6	5	1	2	3		17	43
isolated T inversion	2	2		1			5	13
*Total normal***	36	92	26	68	134	3	358	916
Doubtful abnormal		2		8			10	26
Total abnormal		1	1	4	16		23	59
NST†		1		3	4		8	20
1°HB††			1		4		5	13
RBBB					4		4	10
Atrial arrythmia				1	1		2	5
Left ventricular hypertrophy					2		2	5
Inc. RBBB					1		2	2.5

* Premature ventricular beats.
** Total examined less "doubtful" and "abnormal".
† Non-specific T-wave changes.
†† Heart block.

phenomenon and one instance of premature ventricular beats with persistent bigemini. There were 2 instances of left ventricular hypertrophy by ECG. There were no instances of atrial fibrillation.

The problem of classifying non-specific T-wave changes occurring over the right and central chest is a complicated one in this Negro population. It has been known for 20 years that Negroes tend to have a higher prevalence of persistence of the "juvenile pattern", in the precordial chest leads[19–21]. Whether this represents a racial characteristic and not an abnormality or whether it is somehow associated with the development of heart disease is not established. GRUSIN has shown in South Africa that this disorder, which he classifies in three types, is present in as many as 60 % of an apparently well hospital population[20]. His three classes were: (*1*) depression of the ST-segments and inverted T-waves to V_4 comprising a third of all the abnormalities; (*2*) elevation of the ST segments with peaked T-waves comprising 25 % of all the abnormalities; and (*3*) a rounded or flat T-wave resembling that called "non-specific T-wave changes" in Caucasians.

TABLE VII

PERSISTENCE OF ECG ABNORMALITIES OVER A 9-MONTH PERIOD

Type of ECG change	*Number*	*Unchanged*		*Changed*		*Comment*
		Number	%	*Number*	%	
NST*	5	4	80	1	20	Now normal
Juvenile pattern						
ST elevated	4	4	100			
Peaked T	6	4	67	2	33	(1) Now LVH** (2) Now normal
Inverted T_3	3	3	100	0		
Small Q^1, AVL	2	2	100	0		

* Non-specific T-wave changes.
** LVH, left ventricular hypertrophy.

TABLE VIII

COMPARISON OF MASAI-ECG'S WITH U. S. AVIATORS
(ABNORMALS/1000)

	Population	*Total*	*NST**	*1°HB**	*RBBB*	*LVH***	*?MI†*	*Other*
Masai	391	59	20	13	10	5		7.5
U.S. White Aviators	67,375	37.6	8.6	5.2	1.6	<1.0	1.0	20.2
U.S. Negro Aviators	410	90	51	17	2.4			20

* See explanation under Table VI.
** Left ventricular hypertrophy.
† Doubtful myocardial infarction.

GRUSIN'S work, extended by THOMAS at *Meharry Medical College*[28] in Nashville and by GOTTSCHALK AND CRAIGE[21] at the *University of North Carolina* supplies no evidence that this kind of persistence of the "juvenile pattern" should be grounds

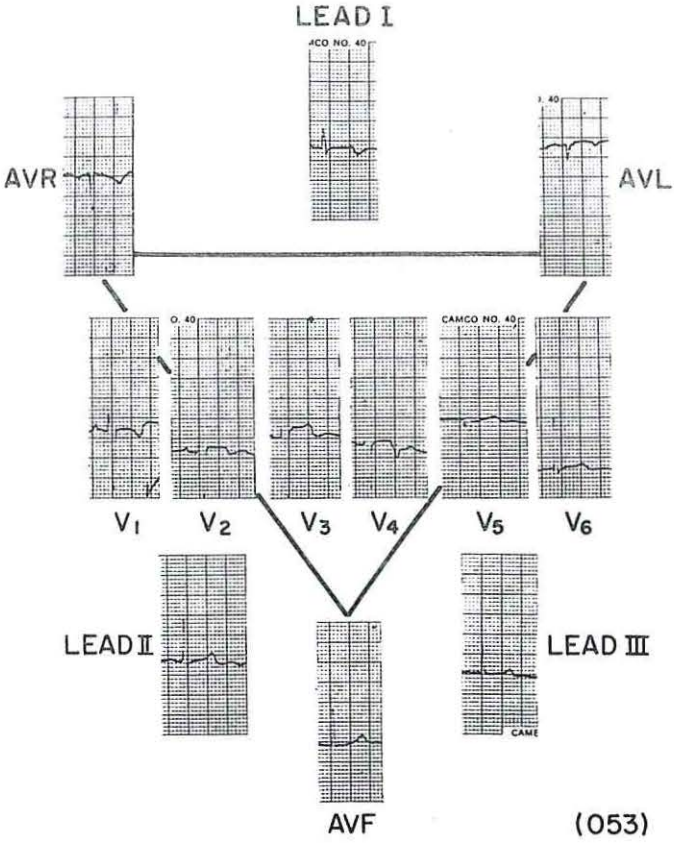

Fig. 3. A juvenile pattern with elevation of ST segment in V_2 and V_3 and inversion of T in V_4. This was recorded in a warrior age 22, well developed but thin, with BP 130/78 and serum cholesterol of 78 mg/100 ml.

for diagnosing heart disease. The age distribution of the classification of this family of patterns is also shown in Table VI. It was by no means restricted to young men. The observations were repeated 10 months later in 20 of these men showing one or another of the original ECG changes. The results are shown in Table VII. The instance of a non-specific T-wave abnormality which changed to "normal tracing" may reflect the difficulty of applying criteria to this abnormality. The category of juvenile patterns with peaked T-waves is obviously subject to change. One of these was on the second observation interpreted as left ventricular hypertrophy in a man of age 40 with a blood pressure of 156/90 mm Hg.

In Table VIII the electrocardiographic abnormalities noted in the Masai population are compared with two other reference groups. The first is the group of 67,375 U.S. Naval aviators who were examined with electrocardiograms by AVERILL AND LAMB at Pensacola[29]. These were predominantly white men but among them were 410

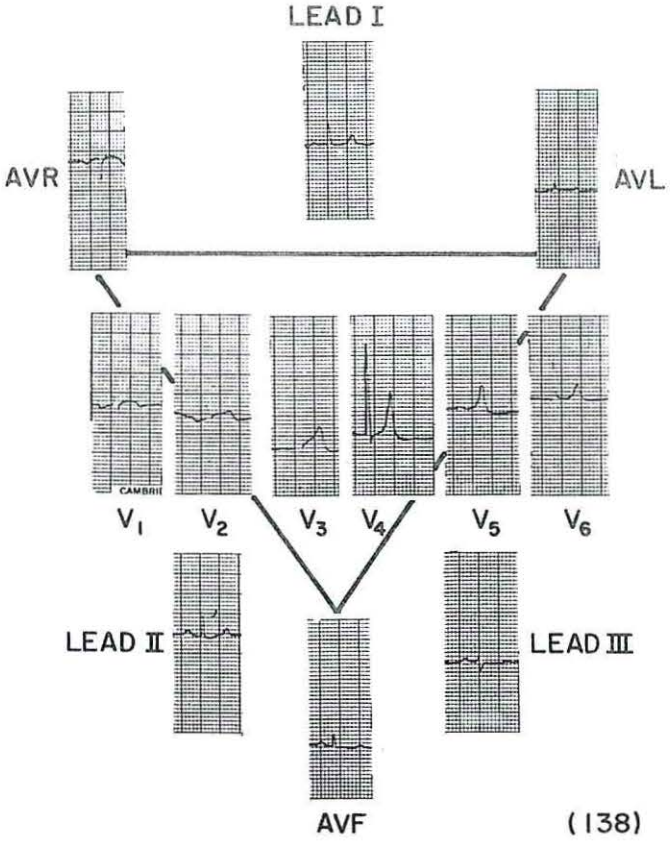

Fig. 4. A juvenile pattern with peaked T-waves in leads V_3–V_5. This was in a man age 55, PB 110/60, serum cholesterol 58 mg/100 ml.

Negro Americans. The relevant comparison in Table VIII is perhaps between the Masai and the sub-sample of U.S. Negroes although there must have been much more selection by health criteria for the aviators than for the Masai. When this is done it is apparent that the proportion of abnormalities in the Masai, expressed here as rate per thousand, is about 2/3 that for the Americans. The U.S. Negroes had much larger proportions of non-specific T-wave changes. This lower rate in the Masai may result because the juvenile patterns are extracted from the group of non-specific T-wave abnormality, as seems to us reasonable. The difficulty with this comparison is the uncertainty of comparison of criteria for non-specific T abnormalities.

Representative examples of the ECG abnormalities encountered and of the sub-types of "juvenile pattern" are shown in Figs. 3–6.

The heart murmurs encountered among the Masai men are summarized in Table IX. Despite their lean chest walls and hyperactive hearts, murmurs of clinical significance were not often heard. The systolic murmurs were attributed to rheumatic heart

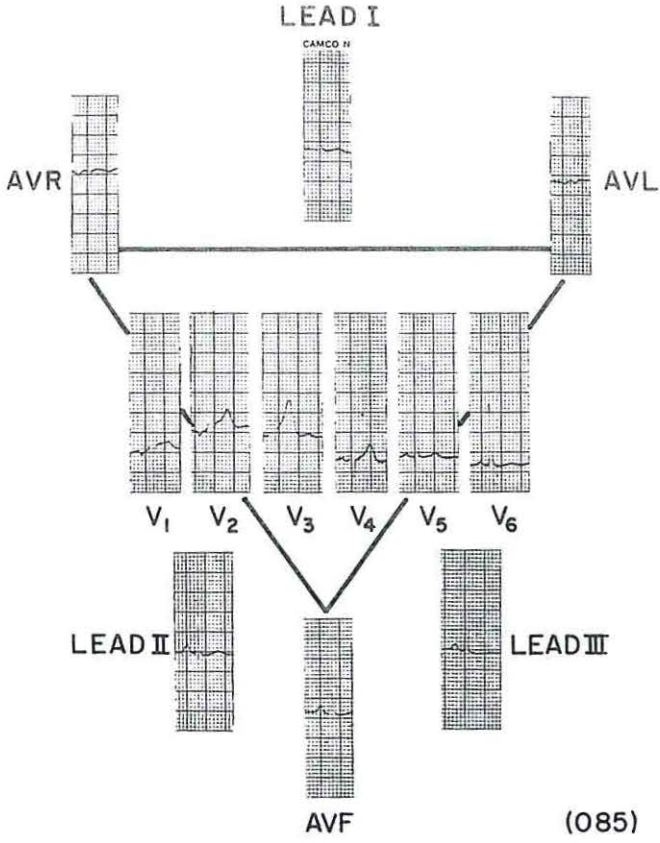

Fig. 5. Non-specific T-wave abnormalities in a young elder, age 43, thin, but well developed, BP 140/90, serum cholesterol 152 mg/100 ml. The T-waves are low to flat in I, III, V_5 and V_6.

disease in 5 instances and an atrial septal defect in the sixth. There were only two definite diastolic murmurs, one thought to represent mitral stenosis and the other luetic aortitis in a man of 50–60 years[5]. The third murmur was of dubious authenticity and unclassified.

Serum cholesterol values

The values found for serum cholesterol are shown in Table X for the Masai men. These are remarkably low being exceeded in this only by values obtained in our previous study of the *Pygmies* of the Ituri Forest which are shown for comparative

purposes in the 5th column of the same table. The levels are considerably lower than those that SHAPER *et al.* found among the *Samburu*[7], a suprising finding since it is believed that the nature of the dietary regimen was similar for these two populations. There can be no doubt that this is a real population difference not assignable to

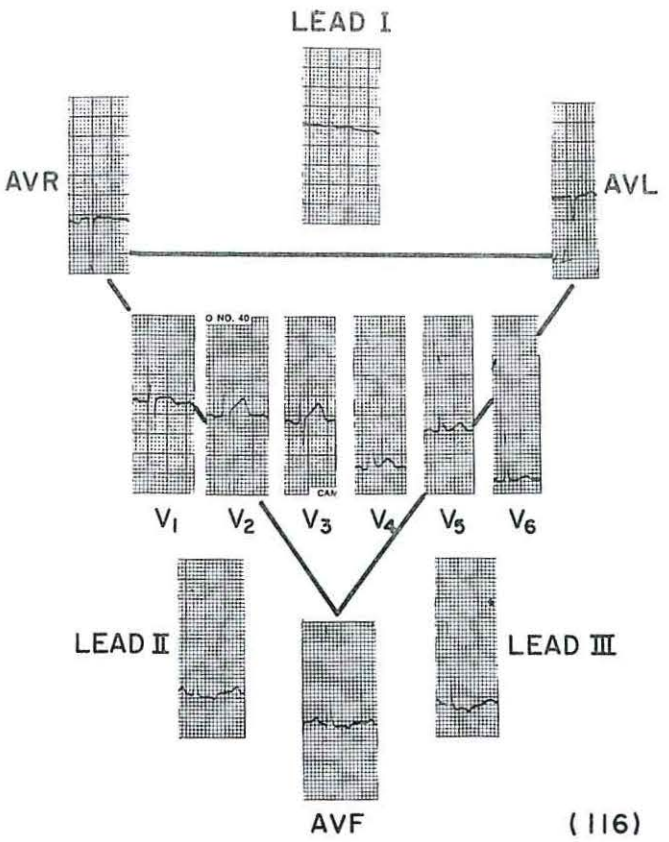

Fig. 6. Indications of left ventricular hypertrophy in a vertical heart observed in a 55 year Masai elder with a BP of 120/80 and serum cholesterol of 178 mg/100 ml.

differences of methodology. The last column in Table X shows values that were found in American males and children of these ages. It is noteworthy that there were only eight among the 388 Masai men measured who showed values of over 200 mg/100 ml (Table XI).

The levels of cholesterol found in 45 children are higher than those of adult men. An age curve of cholesterol level for the Masai is a reciprocal of that for Americans being highest in childhood and minimal in youths and young adults. It is of interest also that many ($\frac{1}{3}$) of the sera obtained from children were lipemic. The relation of the last feeding to the time of bleeding is unknown in the Masai material.

TABLE IX

HEART MURMURS (NUMBER OF MASAI MEN)

Age	*Number examined*	*Systolic (grade)*			*Diastolic (grade)*	
		I and II	*III*	*IV*	*I*	
14–19	35	13	1			
20–24	95	30		1		
25–29	26	11	2		1	
30–43	79	31				
44–55	148	24	2		2	
55 +	3	0	0			
Total	386	109	5	1	3	
Location						
apical		90	4	1	1	
pulmonic		17	1		1	
aortic		2			1	basal
Impression		all functional	5 RHD	RHD	1	mitral stenosis (?RHD)
					1	luetic aortitis
					1	septal defect

TABLE X

MASAI-SERUM CHOLESTEROL (MG/100 ML) WITH COMPARISONS

(MEAN ± STANDARD ERROR)

Age	*Number*	*Masai*	*Samburu*[7]	*Pygmy*[9]	*U.S.A.*[30,38]
1–4	15	133.4 ± 8.33			202
5–9	14	131.4 ± 8.53		99	210
10–12	16	143.9 ± 9.08		110	208
14–19	36	114.7 ± 5.62	164	104	179
20–24	94	114.8 ± 2.60	168	89	198
25–29	27	130.2 ± 5.23	170	104	218
30–43	79	129.9 ± 3.57	168	99	238
44–55	149	129.6 ± 2.53	153	111	244
55 +	3	113.7 ± 16.36	144		234

Serum protein values

In the studies of the *Pygmies* previously reported[9] it was observed that many showed hyperproteinemia. These Pygmy data are summarized in Table XII along with the protein values for the Masai examined in the present study and some comparative data found in the literature for Americans of different cultural groups. The 363 Masai men measured showed an average protein value of 9.41 g/100 ml. When this protein was electrophoresed, 47.7% of the total protein was albumin and 22.4% was γ-globulin. The Pygmies had somewhat less total protein but a larger

proportion as γ-globulin. There is a steady diminution in γ-globulin concentration as one moves from the data of the American Indian (the data of KLEIN *et al.*[32]) through the American Negro to the American white. It is of interest in the last

TABLE XI

DISTRIBUTION OF CHOLESTEROL LEVELS

Level (mg/100 ml)	*Number of men*
≤ 60	3
61–70	5
71–80	19
81–100	55
101–120	86
121–140	115
141–160	60
161–170	15
171–190	19
191–210	5
211–220	4
≥ 221	2

column to note the cholesterol values that are observed as one progresses through this range of protein values. It is not proposed that there is some meaningful relationship between these quantities but it is an interesting fact that they change in this inverse manner.

The other blood chemistries for the Masai men are summarized in Table XIII.

TABLE XII

MASAI SERUM PROTEINS WITH COMPARISONS

	Number	*Total protein (g/100 ml)*	*Albumin (%)*	*γ-Globulin (%)*	*Total cholesterol (mg/100 ml)*
Pygmy	67	8.50	40.1	40.0	101
Masai	363	9.41 ± 0.04	47.7 ± 0.40	22.4 ± 0.38	124.6 ± 1.55
U.S. Indian[32]	45		62.7	16.9	
U.S. Negro	45		61.3	16.4	
U.S. White	45	6.50	68.8	11.5	242

Diseases

The Masai, like the Pygmies, while nearly free of cardiovascular disease carry a heavy load of other diseases which may have some bearing upon their immunity to cardiovascular disease. Splenomegaly, occurring in a third of all patients observed, is believed to be largely due to malaria. There is said to be brucellosis in this area and this may account for part of it. Of 383 men examined only 9 had enlarged livers

TABLE XIII

MASAI BLOOD CHEMISTRIES

(MEAN ± STANDARD ERROR)

	Age groups (years)						*All ages*
	14–19	*20–24*	*25–29*	*30–43*	*44–55*	*55 +*	
Hemoglobin (g/100 ml)	10.92 ± 0.20	12.00 ± 0.14	12.34 ± 0.26	12.02 ± 0.12	11.61 ± 0.10	11.53 ± 1.77	11.77 ± 0.06
Hematocrit (%)	38.43 ± 0.60	42.07 ± 0.50	43.63 ± 0.80	42.30 ± 0.42	41.71 ± 0.36	41.47 ± 4.42	41.67 ± 0.22
Cholesterol (mg/100 ml)	114.7 ± 5.62	114.8 ± 2.60	130.2 ± 5.23	129.9 ± 3.57	129.6 ± 2.53	113.7 ± 16.36	124.6 ± 1.55
Serum protein (g/100 ml)	9.54 ± 0.11	9.33 ± 0.08	9.24 ± 0.13	9.48 ± 0.08	9.43 ± 0.08	8.93 ± 0.34	9.41 ± 0.04
Albumin (% total)	45.05 ± 1.12	46.68 ± 0.64	50.86 ± 1.87	47.78 ± 0.87	47.12 ± 0.68	51.80 ± 0.50	47.74 ± 0.40
γ-Globulin (% total)	23.36 ± 1.13	21.96 ± 0.76	19.63 ± 1.31	22.34 ± 0.89	23.01 ± 0.61	17.45 ± 1.00	22.38 ± 0.38

and only one of these, in a junior murran, was over 2 fingerbreadths below the costal margin. Only two instances of jaundice were seen. There were 15 unexplained abdominal masses, 12 occurring in men beyond age 35. The masses were non-pulsating and believed not to be aneurysms. There were four ventral hernias and 12 men, of which 10 were *Derito*, showed a large obese pannus. Obesity was uncommon and when an obese belly was seen it was usually on the head man of the clan. Skin infections and skin rashes were common. Ectoparasites consisting of either ticks or lice were also seen. Trachoma was a common ailment and perhaps the most debilitating disease seen since it frequently leads to blindness (Table XIV). This was seen in all stages but frequently in stages III and IV[33] with some limitation of vision.

TABLE XIV

EYE DISEASES

Group	*Age*	*Total number*	*Corneal scars*	*Trachoma*	*Blind eye*	*Blind eyes*
Murran	14–19	36	2	7	0	1
	20–24	95	1	22	2	2
	25–29	27	–	10	3	1
Nyangusi	30–43	80	3	32	3	2
Derito	44–55	150	6	73	5	6
Dareto	> 55	3	1	2	0	0
Total		391	13	146	13	12
Percentage		100	3.3	37	3.3	3.1

Lesions of the mouth, tongue and teeth were most common among the elders after age 35, perhaps reflecting their adoption of a vegetable diet. No smooth tongues were seen but atrophy of the margins was not uncommon. Enlargement of superficial lymph nodes was not common. Malformation of the thoracic cage and emphysematous changes were common, increasing with age. Coughing and spitting was frequently observed. The smoke filled interiors of the windowless houses must cause both eye and bronchial irritation. No goitres were seen and only two instances of discrete, thyroid nodules were found.

The Masai do not use salt. Their language contains no word for salt. Whereas their vegetarian neighbors and their cattle crave salt, these carnivores seem to obtain sufficient salt from the flesh and milk which they eat.

Disorders of the extremities were largely the result of trauma. Six instances of DUPUYTREN'S contracture were seen.

There was one instance of hemiplegia with shortening suggesting an injury in early life. There was one tumor of the shin suggesting a gumma. There was one instance of extensive varicosity of the veins of the lower legs. There was a single instance of arthritis of the knee with swelling and heat suggesting gonococcal infection.

There was no evidence of rheumatoid or hypertrophic arthritis of the hands or feet and no signs of gout or tophi. There was one instance of minimal pedal edema with a normal heart. In the *Derito* group, age 45–55 and up, there were two men with diminished dorsalis pedis pulses in one foot and one man with these pulses diminished in both feet. There was another man with absent pulses in both feet with a normal heart (BP 134/68 mm Hg, Hct 49, TC 105 mg/100 ml) and no signs of atrophy of cutaneous structures. There was one instance of pulseless disease[27].

TABLE XV

PARASITES IN MASAI WOMEN AND CHILDREN

	Number		*Totals*	*Percentage*
Malaria				
Total examined			36	
Women	23			100
Children	13			100
Positive smears				
Women	1			4
Children	3			23
Stool specimens				
Total examined			89	100
Women (w)	66			
Children (c)	23			
Positive				
Taenia sp.	w	17	19	21
	c	2		
Necator sp.	w	8	9	10
	c	1		
Strongyloides	w	1	2	2
	c	1		
Trichiuris	w	0	1	1
	c	1		
E. histolytica	w	5	6	7
	c	1		
G. lamblia	w	1	1	1
	c	0		

The men were not examined for the prevalence of intestinal parasites but a systematic sub-sample (1 : 20) of women and children were examined. Of 36 women and children whose thick smears of peripheral blood were examined for malaria there were three children and one woman with positive smears. There were 89 stool samples obtained from 66 women and 23 children. Of these the distribution of positive findings and the nature of the parasite are shown in Table XV. *Taenia, Strongyloides, Trichiuris* and *Entameba* were all common parasites.

The serological findings are shown in Table XVI. The examiners were not impressed with evidences of either early or late syphilis. It appears from the serological findings

that syphilis is not as prevalent as has been supposed. In summary there were 9.3% of the 431 persons examined who had serological evidence of syphilis. While 17.2% have positive VDRL reactions half of these were shown to be falsely positive when re-examined by the TPI. The prevalence of both syphilis and false positive serologies might have been expected in an area of endemic malaria and with hypergammaglobulinemia prevalent.

TABLE XVI

MASAI SEROLOGY

Test	*Total examined*	*Men*	*Women*
VDRL	431	377	54
Positive	74 (17.2 %)	63 (16.7 %)	11 (20.4 %)
TPI	71	61	10
Positive	40 (9.3 %)	35 (9.3 %)	5 (9.3 %)

Flocculation screening tests (34) were all negative in 43 children ages 1–14 who were examined in 1963.

Dietary intake

The accurate measurement of dietary intake of these people proved extraordinarily difficult. We were able to make only limited measurements. This difficulty is because of the erratic intake of food, there being no fixed meal patterns in the families, because there are no uniform units of measurement or utensils and because of the disruption of usual behavior in the presence of an observer.

Milk is collected and consumed from a gourd and no two gourds are of a similar size. Meat is generally cut and eaten directly from a quarter or large piece of the beef. To circumvent these difficulties in measuring diet, several of the men were issued plastic drainage bags with screwcaps which had been treated with a small amount of thymol and glacial acetic acid. The men were asked to carry these on their sword belts and to collect a full day's urine in this container and return it. Ten of the men did this and when the urine was examined for total nitrogen and creatinine, the results in Table XVII were obtained. Complete collections as judged by creatinine excretion were obtained from eight of the ten men. It appears that many of the men have very large nitrogen intakes consonant with a large intake of milk. If one assumes nitrogen balance and that the food intake on this day among the murran was all milk, a nitrogen excretion of 20 grams or more would have to represent something over 3 liters of milk. Analysis of samples of milk which were obtained from Masai cows in various stages of their production cycle indicated the protein content varied from 3.8 to 4.4%. The fat content of this milk was found, as has been previously reported, to be slightly less than 6%. Thus the Masai excreting 20 g nitrogen must have taken 3125 ml milk which would have supplied him with 187.5 g dairy fat. The murran take either milk or meat in a given day, never mixing. Milk days are more frequent than meat days in the ratio 3 : 1.

The Masai alternate this intake of milk and meat as the supply varies. The meat feasts are sometimes gargantuan. We have no measurements of this intake but on market days when cattle are killed or on celebration days these men may take 2–5 kg or more of fatty beef and have been observed to do so[35]. In order to augment their capacity for meat, they drink tea (*kiloriti*) made of the bark of a local tree (*Acacia abyssinica* or *Acacia albica*) with the meat. It would be of some interest to determine

TABLE XVII

URINARY EXCRETION IN 24 HOURS IN MASAI MEN

Age	*Subject No.*	*Diet*	*Volume*	*Creatinine (mg)*	*Nitrogen (g)*	*Sulfur (mg)*	*N/S ratio*
	1	peanuts + meal	1400	1702	24.0	1355	17.7
Elders	128	?	600	1213	13.6	663	20.5
	268	meal	800	2046	15.6	854	18.3
	367	?	incomplete	—*	—	—	21.8
	068	?	1124	1800	16.1	762	21.1
	273	meat	800	778*	6.98	396	17.6
	277	meat	900	2080	20.2	968	20.8
Murran	278	meat	1250	1398	15.7	938	16.7
	530	milk 4 qts.	1325	1681	18.1	1108	16.3
	531	milk	2600	2007	21.7	2056	10.6

* Known or assumed to be incomplete.

whether this infusion contains some proteolytic enzyme which may confirm the native belief in its efficacy.

The legendary role of blood in the Masai diet may have been overemphasized. Venesection of the cattle is practiced occasionally but usually as an emergency procedure when the milk supply is depleted and the alternative would be to kill a valuable animal or at the start of the high milk flow season when the blood makes a large milk intake more agreeable.

DISCUSSION

These studies, like those of SHAPER among the *Samburu*[8] and of GSELL AND MAYER among the *mountain Swiss*[5] show no support for the contention that a large intake of dairy fat and meat necessarily causes either hypercholesterolemia or coronary heart disease. Indeed, if such dietary habits are causes of hypercholesterolemia, atherosclerosis and clinical cardiovascular disease, one must invoke overriding protective mechanisms among the Masai. It cannot be a failure to reach the susceptible age. We should have found coronary heart disease among the 233 Masai men examined who were 30 years or over if the prevalence rate is near that of American men[18].

Its prevalence by objective signs, *i.e.*, not including angina pectoris, in the *Framingham* experience for men aged 30–44 was 2.8‰ and for men aged 45–62 it was 26.6‰. Thus we would expect to find 4–5 cases of coronary heart disease (excepting angina pectoris) in the Masai men we examined if the incidence and survival experience was similar to that in the U.S.A. While the Masai men do not have clinical cardiovascular disease of the atherosclerotic type there is no autopsy data to confirm their anatomical situation. One might consider two possibilities. Either such a diet does not in fact contribute to those consequences or the Masai have some other protecting mechanism which allows them to eat these foods with immunity. One possibility for the latter might be a large amount of exercise. We have shown that strenuous exercise prevents the hypercholesterolemic effects of over-eating[36] and GOLDING has shown the influence of exercise in lowering serum cholesterol[37]. While the Masai murran walk long distances the elders seem sedentary. To evaluate muscular development the SMEDLEY hand dynamometer was used in a sample of 99 Masai men. Table XVIII shows the measurements by age compared with performances of Negro Americans[25]. The Masai were distinctly less powerful.

TABLE XVIII

MUSCLE STRENGTH IN THE HANDS OF MASAI MEN
(SMEDLEY DYNAMOMETER)

Age group	*Years*	*Number*	*Kg*	
			Right	*Left*
Murran				
Junior	14–19	4	17.5 ± 2.06	16.0 ± 2.16
Middle	20–24	5	37.4 ± 3.57	34.4 ± 2.71
Senior	25–29	11	39.5 ± 1.54	36.0 ± 1.31
Total		20	34.6 ± 2.46	31.6 ± 2.01
Nyangusi	30–43	34	33.5 ± 1.04	32.6 ± 1.14
Derito	44–55	39	33.3 ± 1.01	29.2 ± 0.97
Dareto	> 55	6	25.8 ± 1.36	25.3 ± 2.22
American Negroes (25)		100	50.2 ± 0.54	46.92 ± 0.61

One might argue that the Masai are relatively freer of emotional and competitive stresses which, some believe, contribute to hypercholesterolemia and cardiovascular disease. More than most primitive people the Masai find subsistence easy, labor light. Competition is negligible and, some might think, frustrations limited. They have few responsibilities and a quite different attitude toward the world and people about them than do most of us. It remains to be demonstrated that this attitude toward life and its complexities is the nature of their immunity. It would be especially valuable to follow the occasional young Masai who now "goes civilized" in the sense of going to school and adapting the ways of urban "Europeans".

These *Masai* men showed lower cholesterol levels than do the *Samburu*, their *Nilo-Hamitic* neighbors to the north. There is no available explanation for this difference. SHAPER *et al.*[7] assigned the relatively low cholesterol levels and freedom from cardiovascular disease among the *Samburu* to an irregular or fluctuating food intake. This explanation has little basis for support. There is evidence in experimental animals that alternate feasting and famishing lead to *higher* serum lipid levels than are produced in the same animal when he is fed small meals regularly[39]. One might expect from this that the *Samburu* would have higher lipid levels if they had more irregular feeding but there is no evidence to suggest that the *Masai* in Tanganyika eat more regularly than do the *Samburu*. We are presently unable to explain these observed differences. It might be possible to identify the causes by comparative studies of these two pastoral and related tribes, existing on a similar diet. The other important need is for the collection of anatomical material which will indicate whether even with this low level of serum cholesterol, atherosclerotic plaques develop in *Masai* men. In the meantime, we add additional evidence to suggest that the hypothesis relating saturated animal fat to the causation of hypercholesterolemia and cardiovascular disease remains dubious.

ACKNOWLEDGEMENTS

This study was supported by research grants from the U.S. Public Health Service, National Heart Institute, U.S.A., the American Heart Association, New York, The National Live Stock and Meat Board, Chicago and with the supporting personnel of the Ministry of Health and Labor of the Tanganyikan Government. The equipment, special services and personnel of the African Medical and Research Foundation Nairobi, were generously loaned and indispensable. The assistance and encouragement of Mr. MICHAEL WOOD, Director of A. M. R. F. were especially important.

The Charles F. Pfizer Company furnished several kinds of drugs for clinic treatments. The John Wyeth and Sons Ltd. furnished benzathine penicillin at a special rate.

The field work was made possible by PARKEPO, chief of the Masai in South Masailand. We admire and are grateful for his astute assistance and foresight in persuading the murran to be examined and bled. This is an unprecedented accomplishment among Masai.

Dr. G. V. MANN was an Established Investigator of the American Heart Association. He is now a Career Investigator sponsored by the National Heart Institute at Vanderbilt University Medical School.

Dr. R. D. SHAFFER was the regional medical officer of the Tanganyikan Ministry of Health assigned to Masailand. He is now Medical Director of Mobile Medical Facilities of the African Medical and Research Foundation, Nairobi.

Dr. R. S. ANDERSON is Professor of Medicine, Meharry Medical College, Nashville, Tennessee.

Dr. H. H. SANDSTEAD, Assistant Surgeon, U.S. Public Health Service, was assigned to assist with the field work from his duty station at NAMRU III – Cairo by his parent

organization, the Interdepartmental Committee on Nutrition for National Defense. We appreciate this cooperation and his assistance.

SUMMARY

A field survey of 400 Masai men and additional women and children in Tanganyika indicates little or no clinical or chemical evidence for atherosclerosis. Despite a long continued diet of exclusively meat and milk the men have low levels of serum cholesterol and no evidence for arteriosclerotic heart disease. The reasons for this disagreement with the popular hypothesis relating animal fat intake to coronary disease are examined. The authors concede that some overriding protective mechanism such as freedom from emotional stress or abundance of physical exercise may be present. They favor the conclusion that diet fat is not responsible for coronary disease.

RÉSUMÉ

Un groupe de 400 hommes Masaï, plus quelques femmes et enfants du Tanganyika, montre peu, ou pas de signe clinique ou chimique de l'athérosclérose. En dépit d'un régime prolongé contenant exclusivement de la viande et du lait, les hommes présentent des taux bas de cholestérol sérique et ne montrent aucun signe de cardiopathie artérioscléreuse. Les auteurs examinent les raisons de ce désaccord avec l'hypothèse populaire selon laquelle la consommation de graisses animales conduit à la maladie coronaire. Ils admettent la présence de mécanismes protecteurs, tels que l'absence de stress ou l'abondance d'exercices physiques. Ils sont amenés à conclure que les graisses de régime ne sont pas responsables de la maladie coronaire.

ZUSAMMENFASSUNG

Eine Untersuchung im Felde von 400 Masai-Männern und einer Anzahl Frauen und Kinder in Tanganyika zeigte wenig oder keine klinische oder chemische Zeichen von Atherosklerose. Ungeachtet einer langen kontinuierlichen Diät von ausschliesslich Fleisch und Milch haben die Männer niedrige Serumcholesterinspiegel und keine Anzeichen arteriosklerotischer Herzkrankheit. Die Gründe für diese Differenz mit der geläufigen Auffassung, dass tierische Fette zu Koronarerkrankung führen, wurden untersucht. Die Verfasser geben zu, dass irgendein unabhängiger schützender Mechanismus, wie das Fehlen emotionaler Beanspruchung oder eine ausgiebige Körperbewegung, eine Rolle spielen kann. Sie bevorzugen die Schlussfolgerung, dass die Diätfette nicht verantwortlich sind für das Entstehen von Koronarerkrankungen.

REFERENCES

1 W. J. Zukel, R. H. Leurs, R. E. Enterline, R. C. Painter, L. S. Ralston, R. M. Fawcett, A. P. Meredith and B. Peterson, *Am. J. Public Health*, 49 (1959) 1630.
2 M. Friedman and R. H. Rosenman, *Circulation*, 16 (1957) 339.

3 T. R. DAWBER, G. V. MANN AND G. PEARSON, in preparation.
4 A. D. CHARTERS AND B. P. ARYA, *Lancet*, i (1960) 288.
5 D. GSELL AND J. MAYER, *Am. J. Clin. Nutr.*, 10 (1962) 47.
6 J. B. ORR AND J. L. GILKS, *Rept. Med. Res. Council*, 155 (1931).
7 A. G. SHAPER, M. JONES AND J. KYOBE, *Lancet*, ii (1961) 1324.
8 A. G. SHAPER, *Am. Heart J.*, 63 (1962) 437.
9 G. V. MANN, O. A. ROELS, D. L. PRICE AND J. M. MERRILL, *J. Chronic Diseases*, 15 (1961) 341.
10 R. K. CANNAN, *Clin. Chem.*, 1 (1955) 151.
11 J. A. KERNEN, H. WURZEL AND R. OKADA, *J. Lab. Clin. Med.*, 57 (1961) 635.
12 G. V. MANN, *Clin. Chem.*, 7 (1961) 275.
13 T. E. WEICHSELBAUM, *Am. J. Clin. Pathol.*, 7 (1946) 40; also see J. F. FAILING, M. W. BUCKLEY AND B. ZAK, *Am. J. Clin. Pathol.*, 33 (1960) 83.
14 C. J. BRECKENRIDGE, *Anal. Chem.*, 32 (1960) 1353.
15 A. HARRIS, A. A. ROSENBERG AND E. R. DEL VECCHIO, *J. Vener. Dis. Inform.*, 29 (1948) 72.
16 J. PORTNOY, J. H. BREWER AND A. HARRIS, *U. S. Public Health Rept.*, 77 (1962) 645.
17 A. HARRIS, V. H. FALCONE, L. S. TRICE AND W. J. BROWN, *U.S. Public Health Rept.*, 73 (1958) 210.
18 T. R. DAWBER, F. E. MOORE AND G. V. MANN, *Am. J. Public Health*, 57 (1957) 4.
19 D. LITTMAN, *Am. Heart J.*, 32 (1946) 370.
20 H. GRUSIN, *Circulation*, 9 (1954) 860.
21 C. W. GOTTSCHALK AND E. CRAIGE, *Southern Med. J.*, 49 (1956) 453.
22 H. A. FOSBROOKE, *African Studies*, 15 (1956) 188.
23 L. B. PETT AND G. F. OGILVIE, *Can. Bull. Nutr.*, 5 (1957).
24 J. BROZEK AND A. KEYS, *Brit. J. Nutr.*, 5 (1951) 194.
25 G. WOLFF AND M. STEGGERDA, *Human Biol.*, 15 (1943) 127.
26 A. W. WILLIAMS, J. D. BALL AND J. N. P. DAVIES, *Proc. Roy. Soc. Trop. Med. Hyg.*, 48 (1954) 290.
27 R. S. ROSS AND V. A. MCKUSICK, *A.M.A. Arch. Internal Med.*, 92 (1953) 701.
28 J. THOMAS, E. HARRIS AND G. LASSITER, *Am. J. Cardiol.*, 5 (1960) 468.
29 K. E. AVERILL AND L. E. LAMB, *Am. J. Cardiol.*, 6 (1960) 76.
30 E. R. LAWRY, G. V. MANN, A. PETERSON, A. P. WYSOCKI, R. R. O'CONNELL AND F. J. STARE, *Am. J. Med.*, 22 (1957) 605.
31 R. C. HODGES, W. M. SPERRY AND D. H. ANDERSON, *Am. J. Diseases Children*, 65 (1943) 858.
32 G. C. KLEIN, M. M. CUMMINGS AND J. F. HAMMARSTEN, *Proc. Soc. Exptl. Biol. Med.*, 111 (1962) 298.
33 EXPERT COMMITTEE ON TRACHOMA, *WHO Techn. Rep. Ser.*, 106 (1956).
34 J. PORTNOY, J. H. BREWER AND A. HARRIS, *U.S. Public Health Rept.*, 77 (1962) 645.
35 A. JACOBS, personal communication.
36 G. V. MANN, K. TEEL, O. HAYES, A. MCNALLY AND D. BRUNO, *New Engl. J. Med.*, 253 (1955) 349.
37 L. A. GOLDING, *Federation Proc.*, 21 (1962) 96.
38 C. G. HAMES AND B. G. GREENBURG, *Am. J. Public Health*, 51 (1961) 374.
39 C. COHN AND D. JOSEPH, *Metabolism*, 9 (1960) 492.