THE AMERICAN JOURNAL OF CLINICAL NUTRITION
Vol. 23, No. 10, October, 1970, pp. 1353-1363
Printed in U.S.A.

International Nutrition

Editor: Robert B. Bradfield, Ph.D.

Dietary Factors Causing Hypertension in India

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THERE ARE MANY STUDIES that show that important and interesting differences exist in the patterns of blood pressure and the incidence of hypertension, not only between countries but from group to group within the same country (1–8). Such big differences among groups suggest that the causes may be environmental.

According to previous reports, cardiovascular disease is more common in South India as compared with North India (9–11). The present investigation, therefore, was planned to determine whether there are any significant differences in the prevalence of hypertension in these two population groups, and if so, what are their causes.

METHOD OF INVESTIGATION

Five railway centers, namely Delhi, Ajmer, and Ratlam in the North; and Madras and Waltair (Fig. 1) in the South, were selected for the enquiry.

For the sake of comparability, we included in our main survey, which concerned Delhi and Madras, sedentary male railway employees, between the ages of 20 and 58 years in the Accounting Section of the Finance Branches of these two railway centers, namely, Delhi in the North and Madras in the South. The socioeconomic status, the nature of work, and the wage bracket of the clerks in both the geographical areas are identical. We had 1,046 North Indians and 1,106 South Indians in this trade; all have been included in the survey by retrieving, by a subsequent examination (Table 1), those absent at the time of the survey. In the case of the other centers, the subjects included were as follows: Ajmer (North), 296 accounting clerks; Ratlam (North), 1,000 railway employees in different trades; for example, drivers, firemen, fitters, clerks, carpenters, and sweepers; and in the case of Waltair (South), we had 114 sweepers only. Thus, the method of selection of the subjects was varied, but in each place we included all the employees on the rolls of the particular trade. This method had the advantage of comparing arterial pressures, not only in geographically disparate groups but even in groups working in different trades and work levels according to whether they were sedentary or physically active.

In the principal survey (Delhi versus Madras), blood pressure was recorded by six physicians, all men, using two mercurial manometers. One reading of blood pressure was made in the seated subject after 10 min rest in accordance with the technique for routine blood pressure (12). The study reported here was designed to throw light on any etiological factor in hypertension, the defining of geographical differences being merely one facet of the investigation. Therefore, records were made for all subjects for height, weight, smoking, diet and eating patterns, and consumption of alcohol. Urine was examined for sugar, albumin, pus cells, and casts in 208 out of 278 persons with hyper-

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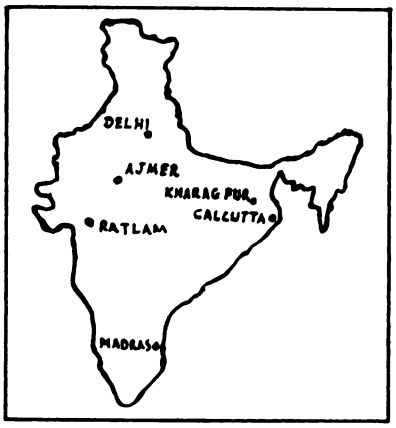

FIG. 1. Map of India showing the various railway centers included in the survey.

tension and in 208 age-matched controls from the original populations in Delhi and Madras.

Deming and his colleagues (13) have observed that hypotensive drugs lower blood pressure and serum cholesterol simultaneously, suggesting a common etiological factor. We have shown elsewhere that serum cholesterol levels are closely related to fecal urobilinogen levels which, in turn, are dependent upon the pattern of diet and eating (14–17). It seemed of interest to us to obtain data in persons with hypertension and in age-matched controls. We, therefore, carried out these determinations on hypertensive individuals from our accounting clerks attending the Cardiovascular Clinic of our Hospital at Calcutta and age-matched controls from the same department. Both the hypertensives and the controls were Bengalese. Since the object was to study the differences in blood cholesterol and fecal urobilinogen in a group of persons with elevated blood pressure, comparable in other respects, there is no reason why this mode of investigation should invalidate our conclusions. Serum cholesterol was determined by Sackett's (18) method and fecal urobilinogen (milligrams per 100 g stool) by the method described by Maclagan (19).

RESULTS

The mean systolic and diastolic pressures were found to be higher by 5 mm or more in the South Indians as compared with the North Indians in all the age groups (Table I). But in the individual population groups, there was no difference between the mean arterial pressures in different age groups. Hypertension (systolic above 160 mm and diastolic above 95 mm) was present in 15.2% of the South Indians but in only 6.2% of the North Indians ($P < 0.001$) (Table II). The ratio of

TABLE I

Mean arterial pressures of men in South and North India included in the principal survey (Delhi versus Madras)

Age group, years	Arterial pressure	South		North		Difference between means	t	P
		Number of men	mm Hg, m[a]	Number of men	mm Hg, m[a]			
30	Systolic	65	125.8 ± 2.04	28	120.4 ± 1.72	5.46	1.62	< 0.1
	Diastolic		82.8 ± 1.38		76.4 ± 1.42	6.49	2.78	< 0.01
30–34	Systolic	142	126.4 ± 1.14	206	124.1 ± 1.03	2.31	1.48	< 0.1
	Diastolic		83.0 ± 0.67		76.7 ± 0.69	6.23	6.20	< 0.001
35–39	Systolic	149	127.8 ± 1.29	209	125.4 ± 1.20	2.36	1.40	< 0.1
	Diastolic		82.6 ± 0.72		77.1 ± 0.75	5.51	5.13	< 0.001
40–44	Systolic	233	130.4 ± 1.46	292	126.0 ± 0.87	4.21	2.58	< 0.01
	Diastolic		83.4 ± 0.65		79.4 ± 0.48	4.05	5.13	< 0.001
45–49	Systolic	224	135.0 ± 1.47	223	127.9 ± 1.16	7.11	3.79	< 0.001
	Diastolic		85.9 ± 0.72		80.4 ± 0.69	5.47	5.47	< 0.001
50 and above	Systolic	109	140.6 ± 2.18	131	132.9 ± 1.67	7.73	2.86	< 0.001
	Diastolic		87.7 ± 1.05		81.2 ± 0.90	6.45	4.69	< 0.001

[a] Values are means ± SE.

cigarette smoking was three times more among the North Indians as compared with the South Indians; among the smokers, the average daily per capita consumption of cigarettes was 8 in the South Indians and 19 in North Indians. Although we could find no differences in the number of persons taking alcohol between our two groups, we cannot rely on this information because drinking is considered a social evil and information on this is neither readily volunteered nor correctly given.

As regards obesity, we used the simple ponderal index (weight in pounds per height in inches). Tables III,A and III,B show a definite correlation between obesity and arterial pressure in both the groups.

Diet

Wide variations exist in the pattern of diet and eating between the two populations that have been extensively studied (10, 14, 20, 21). These differences have been summarized in Table IV. The salient points are *a*) the South Indian staple diet is non-masticatory ragi or rice, whereas the North Indian diet is mainly masticatory wheat; *b*) the fat content of the South Indian diet is nine times lower than the North Indian diet; and although the fats of South Indian diets are from unsaturated seed

TABLE II

Showing the incidence rates of hypertension (BP above 160/95 mm Hg) at six railway centers included in the survey

Place	Population	Work grade	Number of men	Number with elevated pressure, diastolic, 2	% Of hypertensives
I					
Delhi	North Indians	Sedentary clerks	1,046	67	6.2
Madras	South Indians	Sedentary clerks	1,106	168	15.2
II					
Ajmer	North Indians	Sedentary clerks	296	19	6.0
Waltair	South Indians	Heavy physical activity	114	18	15.4
III					
Ratlam	North Indians	Mixed	1,000	63	6.3
Khargpur	South Indians	Mixed	374	42	11.2
IV					
Khargpur	North Indians	Mixed	296	14	6.0

TABLE III, A

Effect of obesity on mean arterial pressure (Southern Railway—Madras)

Ponderal index	Number of cases	Systolic[a]	Diastolic[a]	*t* Test between different ponderal indices	Systolic		Diastolic	
					t	*P*	*t*	*P*
1.70	150	124.15 ± 17.34	84.11 ± 9.46	1.70 and 1.71–1.89	1.79	NS	1.672	NS
1.71–1.89	109	128.86 ± 23.22	86.10 ± 9.60	1.70 and 1.90	5.621	0.001	3.708	<0.001
1.90	241	135.92 ± 24.01	90.36 ± 10.70	1.71–1.89 and 1.90	2.61	0.001	6.104	<0.001

NS = not significant. [a] Values are means ± SD.

TABLE III, B

Effect of obesity on mean arterial pressure (Northern Railway—Delhi)

Ponderal Index	Number of cases	Systolic[a]	Diastolic[a]	*t* Test between different ponderal indices	Systolic		Diastolic	
					t	*P*	*t*	*P*
1.70	488	126.79 ± 14.1	83.03 ± 18.93	1.70 and 1.71–1.89	0.2697	NS	0.4936	NS
1.71–1.89	116	126.26 ± 13.15	83.66 ± 7.91	1.70 and 1.90	0.039	NS	0.047	NS
1.90	2	144.00 ± 30.00	89.00 ± 15.00	171–1.89 and 1.90	0.038	NS	0.053	NS

NS = not significant. [a] Values are means ± SD.

TABLE IV

Pattern of food consumption of North Indians versus South Indians, grams per consumption unit

Foods	Madras, g	Udaipur, g
Rice	425	0
Wheat	0	440
Other cereals	140 (Ragi)	40 (Barley or maize)[a]
Pulses	15	62
Leafy vegetables	0	50
Nonleafy vegetables	122 (Onions or brinjal)	88
Seed oils	7 (Ground-nut)	20 (Mustard or sesame)
Ghee	0	55
Milk, buttermilk, or curd	12	150
Meat and fish	30 (Beef)	5
Condiments	15	5
Sugar and jaggery	12	50
Total calories	2,334	2,786
Total fats, g	7	75
Calories from fats, %	3.5	23
Composition of fats	Chiefly long-chain fatty acids of seed oils	Chiefly short-chain fatty acids of ghee and milk fats
Saturated fatty acids, %	2	44

Fats and oils are the amounts of these foodstuffs available for consumption as such, not the total amount of fat derived from all dietary constituents. [a] Optional.

oils with a preponderance of long-chain fatty acids, the fats of North Indian diets are of animal origin, such as milk, fermented milk, especially yogurt in large quantities, and ghee, which are saturated but have a preponderance of short-chain fatty acids (C_4, C_6); *c*) the South Indian diets are poor in their roughage and vegetable fiber content as compared with the wheat, whole beans, dalls, and vegetable diets of North India, which are rich in cellulose, vegetable fibers, and roughage.

The mean levels of blood cholesterol and fecal urobilinogen were significantly higher in the hypertensives as compared with age-matched controls, the respective values being 212.5 mg/100 ml and 185.3 mg/100 ml for blood cholesterol, and 58.8 mg/100 g and 35.0 mg/100 g for fecal urobilinogen ($P < 0.001$) (Tables v and vi).

Other Etiological Factors

Age. Studies, such as those of Miall (22) and the United States Public Health Service (23) have shown that mean blood pressure levels and prevalence rates for elevated blood pressure tend to increase with age, at least over the decades from young adulthood through middle age. But our data for the two groups do not show any gross association between age and arterial pressures within the group, which might indicate an etiological relationship. From this aspect, therefore, our data are in consonance with the conclusions of Shaper (5) and Lovell (7), who found no etiological effect of age on mean blood pressure levels.

Body weight. Prevalence rates for hypertension are significantly related to body weight (adjusted for height), i.e., to overweight or obesity (24). On the other hand, Shaper (5) in his East African studies found that although the Samburu and Rendille tribes showed no difference in systolic or diastolic pressures in any of the ponderal index groups, the Turkana showed a significant rise in both systolic and diastolic pressures from the two lower ponderal index groups to the heavier group, and in this last group the systolic and diastolic pressures were higher than the corresponding Samburu and Rendille groups.

Although our results of the effect of obesity on arterial pressure (Tables III,A and B) support the association of obesity with arterial pressure among the South Indians, there being a significant increase in both systolic as well as diastolic pressures with increasing ponderal index, the data from North India have shown only a weak association between obesity and arterial pressure.

In view of the conflicting results of our data as well as the data of others, it is difficult to draw any definite conclusion on the role of obesity in hypertension.

Diabetes mellitus. Frank clinical diabetes mellitus is associated with higher prevalence rates of hypertensive disease than encountered in the general population (25, 26). Our results of the effect of clinical diabetes (using urine glycosuria as the test) in 208 hypertensive individuals and 208 age-matched controls are in consonance with these and we found that among the hypertensives there were 5.1% glycosuriacs, whereas among the normal controls there were only 2.2% glycosuriacs. It may be argued that since there were more South Indians among the hypertensives, the correlation may be due to the possibility that diabetes may be more common in South Indians. To test this, we carried out urine studies in our 96 hypertensives and 98 controls, all Bengalese, from Calcutta, and we found a similar association; hypertensives showed glycosuria in 9.4% and the controls only in 3.3% ($P < 0.001$).

TABLE V

Comparison of blood cholesterol levels in hypertensives and normal cases, mg/100 ml[a]

	Hypertensives (96)	Controls (98)
Mean ± SE	212.5 ± 5.30	185.3 ± 4.30
P	< 0.001	

Numbers in parentheses refer to number of cases.
[a] These patients and controls are from a different population, consisting of Bengalese accounting clerks located in Calcutta.

TABLE VI

Comparison of fecal urobilinogen concentration in hypertensives with normal cases, mg/100 g feces[a]

	Hypertensives (96)	Controls (98)
Mean ± SE	58.8 ± 6.0	35.0 ± 3.54
P	< 0.001	

Numbers in parentheses refer to number of cases.
[a] These patients and controls are from the same group as in Table V.

Smoking. As shown in several studies, smoking habits and blood pressure are not correlated (for references, see Stamler (24)). Karvonen (3) found that pressures were significantly lower in smokers than nonsmokers. Our findings are in agreement with those of Karvonen but their significance vis-à-vis the mode of action of smoking is less clear.

Chronic pyelonephritis. Higher prevalence rates of bacteriuria have been found in hypertensive persons (27). Almost certainly, chronic pyelonephritis may lead to hypertension, i.e., a true secondary hypertension, although this is by no means an invariable development.

In our 208 cases of hypertension (71.2% of total hypertensives), evidence of pyelonephritis was present in 6.4%. When we examined an equal number of nonhypertensive age-matched individuals, we could find evidence of kidney infection only in 3.2%. Although these results are in consonance with the hypothesis of the role of pyelonephritis in the causation of hypertension, the proportion of cases showing evidence of pyelonephritis is too small to be regarded as having any causative role. Moreover, because of the exceptional susceptibility of the hypertensives to pyelonephritis, it becomes a very difficult matter to decide which is the cause and which is the effect. The results of the bacteriuria in our Calcutta subjects confuse the issue further, because, although bacteriuria was present in 4 of 98 controls, none of the hypertensives showed this.

Sociologic, cultural, behavioral, psychological factors and the prevalence of hypertension. An important hypothesis of the causation of hypertension suggests that the disease fundamentally originates in the central nervous system. Basically, this hypothesis holds that negative life experiences in susceptible individuals generate psychoemotional disturbances leading to hypertension. It is very difficult to measure psychological stress because every individual reacts differently to the same situation. If the stress of work alone is considered as our parameter, our two groups are subject to identical work stress and, therefore, work stress is unlikely to have produced the differences in the prevalence rates of hypertension seen in our study. It must be recognized that this may be an oversimplification of the problem and this whole area of psychocultural factors and hypertension is, at this juncture, relatively unexplored. Stamler (24) has found the tendency for prevalence rates among several strata, stratified by sociopsychological variables, to be similar, at least when groupings are along simple demographic lines. One interesting observation negates the hypothesis of psychological stress as having an etiological influence. We investigated eight cases of endogenous depression in which the patients were under extreme agitated depression with anxiety, and without exception all these patients were suffering from hypotension instead of hypertension. In one case, where the agitation-depression complex had been present for over 5 yr, the blood pressure levels were as low as 90/60. Quite possibly this may be the effect of drugs, but not wholly, because with temporary withdrawal of drugs, the pressure rose but not sufficiently to cause hypertension.

Dietary salt intake. Some epidemiological studies have shown a relationship between level of habitual salt intake and prevalence rates of hypertensive disease (24, 28–31). However, no such association has been demonstrated in other studies (32, 33). Our data show a reverse association as the consumption of salt in North Indians is higher than in South Indians. The consumption in South Indians is 8 g/day per person as compared with 12–15 g/day per person in North Indians. Thus, the facts in this area appear to be inconclusive and contradictory at the present time, and, therefore, clearly no relationship of cause and effect can be established between

dietary salt intake and prevalence of hypertensive disease.

DISCUSSION AND CONCLUSIONS

In this study, an attempt has been made to determine the incidence rates of essential hypertension in a 100% survey of a comparable socioeconomic, as well as occupational group, of males between the ages of 20–58 years in two different geographical areas of India that have shown big differences in the prevalence of cardiovascular and certain other chronic diseases, such as peptic ulcer (21), gastric and hepatic cancers (10), and cholelithiasis (34). Our results show that wide variations exist in the incidence rates of essential hypertension as well as in the mean levels of blood pressure between the South Indians as compared with North Indians (Tables I and II) and that these differences are dependent on dietary factors (Fig. 2). Factors such as age, body weight, smoking, physical activity, consumption of alcohol, and work stress do not seem to have any etiological association. Diabetes showed a definite correlation mirroring the observations of several other workers.

We found a close association between arterial pressure and blood cholesterol on the one hand and fecal urobilinogen on the other. This association between arterial pressure and blood cholesterol agrees with the observations of Deming et al. (13), but more evidence is necessary before a statement can be made that these are a factor in the production of hypertension. One way of testing the truth of this conclusion is to compare the levels of blood cholesterol and fecal urobilinogen among South Indians versus North Indians in the general population. Our previous studies on healthy adult railway sweepers show that although there are no differences in the levels of blood cholesterol (14), the fecal urobilinogen concentration (milligrams per 100 grams stool) levels were significantly higher in South Indians as

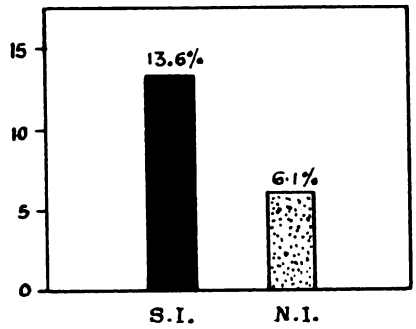

FIG. 2. A total of 4,232 persons were surveyed. Of 1,594 South Indians from three railway centers, namely, Madras, Waltair, and Khargpur, 328 were hypertensive. Of 2,638 North Indians from four railway centers, namely, Delhi, Ajmer, Ratlam, and Khargpur, 163 were hypertensive.

compared with North Indians (14, 34), the respective figures being 64.4 mg/100 g for South Indians and 19.8 mg/100 g for North Indians ($P < 0.001$), and that these differences were due to the differences in the diet and eating patterns. As a second, and perhaps a more convincing, test of an etiological role of blood cholesterol and fecal urobilinogen, we examined the effect of two hypotensive agents in combination on blood pressure, blood cholesterol, and fecal urobilinogen in 31 patients suffering from hypertension from our Calcutta sample. Unfortunately, the drug effect could not be studied in the hypertensives in our original populations due to the difficulties of distances, nor in all the 96 patients attending our hypertension clinic at our hospital in Calcutta. We can, however, see no reason why this should make any difference to the validity of the results since the main purpose is to study the effect of hypotensive agents on blood cholesterol and fecal urobilinogen in persons with elevated blood pressures. These determinations were done while the patients, all of them rice- and fish-eating Bengalese, were on their habitual diet and at work. Although the blood cholesterol levels fell slightly in 20 of 31 patients by the administration of these drugs, fecal urobi-

TABLE VII

Levels of serum cholesterol and fecal urobilinogen excretion in hypertensive patients

Patient	Serum cholesterol, mg/100 ml		Fecal urobilinogen excretion, mg/24 hr	
	Pretreatment	Posttreatment	Pretreatment	Posttreatment
1	200	160	110.2	72.9
2	175	195	148	32.1
3	300	270	126	48.6
4	232	260	108	106
5	240	232	158	65.8
6	286	216	113	20.8
7	336	240	172	38.8
8	240	280	160.6	92
9	230	216	182	75
10	184	170	130	50.2
11	320	290	160	50.6
12	216	200	140	60.4
13	192	182	41.38	37
14	204	184	31	34
15	160	148	70.6	52.42
16	258	210	178.6	41
17	280	232	128.7	111.7
18	300	270	Not done	65
19	144	208	42.6	40.8
20	184	220	56.8	63.2
21	264	280	60.8	86.7
22	200	192	123.1	79.2
23	219	224	178.6	41
24	248	264	174.2	134.2
25	330	320	174.2	134.2
26	256	240	53.9	75
27	392	328	255.2	255
28	256	248	100.0	180
29	225	200	192.7	65
30	280	264	120	70.8
31	364	260	194	105

Patients were all Bengalese from Calcutta. They were treated with Serpasil plus dichlotride combination.

linogen excretion (milligrams per 24 hr) fell markedly in 24 of 29 patients (86%) treated with the combination of *Rauwolfia serpentina* and chlorothiazide; in one patient, the fecal urobilinogen excretion studies could not be carried out (Table VII). As a further proof of this association between arterial pressure and fecal urobilinogen, we noticed that when the drugs were stopped, there was a rise in mean daily fecal urobilinogen excretion (milligrams per 24 hr) as well as the arterial pressures to the pretreatment levels nearly 4 weeks after discontinuing the drugs.

Are we justified in concluding that an etiological association exists between arterial pressure and blood cholesterol on the one hand and fecal urobilinogen on the other? If this is so, what is the mechanism involved in this association? Because data collected in a number of ways have led to the same conclusion that, when the fecal urobilinogen rises, the arterial pressure rises too, we conclude that a rise in fecal urobilinogen is a factor, and an important one, in the production of hypertension. The evidence of its mode of action is less clear. Since the amount of fecal urobilinogen depends upon the amount of bile entering the intestinal lumen (35), it seems likely that the amount of bile entering the intestinal lumen is in some way related to arterial pressure. Dietary factors seem to be concerned in this relationship because it has been shown both in epidemiological studies as well as in feeding experiments that the patterns of diet and eating regulate the amount of fecal urobilinogen (16, 17).

The amount of bile salts in the intestinal lumen in turn determines not only the rate (36), but also the route (37), of absorption of the long-chain fats. Thus ordinarily the long-chain fats have an absolute dependence on the presence of

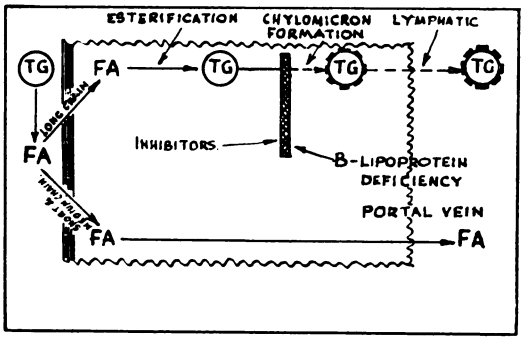

FIG. 3. Pathways of lipid transport in the mucosa.

bile salts for their digestion and are hydrolyzed by bile salts into fatty acids, which are then resynthesized in the wall of the intestine into fatty acid triglycerides, in the form of complex chylomicrons—partly characteristic of the species; short-chain fatty acid triglycerides are absorbed directly into the blood stream without chylomicron formation (Fig. 3) even in the complete absence of bile salts (38). The diet of North Indians has a preponderance of short-chain fatty acids as compared with the diet of South Indians (10, 14, 20, and Table IV). The relative proportion of the short-chain versus long-chain fatty acids in the plasma depends upon their relative proportion in the dietary fats (39). Also larger amounts of bile enter the intestinal lumen of the hypertensive than the normals, as well as South Indians versus North Indians. Because of these factors, differences in the size, nature, and type of chylomicrons in the blood of hypertensives as compared with normals, and South Indians as compared with North Indians, are not only probable but highly likely. Therefore, it may be reasonable to suggest that the number and the complex structure of chylomicrons, depending upon whether they represent complex long-chain triglyceride acids or simple short-chain fatty acids in the plasma, depending upon the amount of bile salts in the intestinal lumen on the one hand and the chain-length of the fatty acids of dietary fats on the other, may in some manner be related to arterial pressure and may explain the significantly higher incidence rates of hypertension among South Indians as compared with North Indians. There is evidence that the increase in peripheral resistance seen in patients with essential hypertension is attributable to hemodynamic changes (40). Further studies are needed to evaluate the role of the preponderance of complex chylomicrons, derived from the long-chain complex triglyceride acids, on hemodynamics due to alterations brought about in the viscosity of the blood and peripheral resistance and to arrive at a meaningful interpretation of the same, vis-à-vis the patterns of diet and eating.

SUMMARY

The prevalence of hypertension was studied in 2,638 North Indian and 1,860 South Indian railway workers, all men, between 20 and 58 years of age, by determining casual arterial pressure. The main survey included 1,046 North Indian and 1,106 South Indian clerks from the Accounting Section of the Finance Branch, comparable in their socioeconomic status, nature of work, and wage bracket. The South Indian population showed a much higher prevalence of hypertension as well as higher mean arterial pressures, probably due to environmental rather than ethnic reasons.

In neither of these populations was the prevalence of hypertension related to age, occupation, body weight, smoking, chronic pyelonephritis, dietary salt intake, or psychological factors.

The differences appeared to be due to hemodynamic changes probably brought about by dietary factors (especially the long-chain versus short-chain fatty acids in fats consumed) by virtue of their effect on the size and nature of chylomicrons, blood viscosity, and peripheral resistance.

I am deeply indebted to Dr. A. K. Jha; Dr. R. K. Baijal and his colleagues for taking part in the field survey; Dr. D. C. Chakravorty of my laboratory for the biochemical investigations; and Mr. Jacob Thomas, Statistical Consultant, Johns Hopkins University, for facilities to check the statistical results. I am also very grateful to Dr. J. R. Gadeock, Chief Medical Officer, Northern Railway; Dr. A. Ramdas, Chief Medical Officer, Southern Railway; and Dr. R. C. Vevaina, Chief Medical Officer, Western Railway for allowing us to carry out the survey work in centers situated in their respective railway zones.

ADDENDUM

Since this paper was written and during the course of our study on cerebrovascular disease

among North Indians versus South Indians we came across 241 South Indian general clerks (not belonging to the Accounting Section) at Khargpur who had settled down there for at least two generations and had changed their dietary habits to conform with the North Indian dietary pattern. It is interesting to note that the incidence rate of hypertension in these South Indian clerks was 4.9%, a figure very similar to the incidence rates in North Indians. This observation is consistent with the dietary hypothesis in the etiology of hypertension presented in this paper.

REFERENCES

1. Bays, R. P., and N. S. Scrimshaw. Facts and fallacies regarding the blood pressure of different regional and racial groups. *Circulation* 8: 655, 1953.
2. Rantaharju, P. M., M. J. Karvonen and A. Keys. The frequency of arteriosclerotic and hypertensive heart disease among an ostensibly healthy working population in Finland. *J. Clin. Diseases* 13: 426, 1961.
3. Karvonen, M. J. Arterial pressure in the East and West of Finland. In: *Epidemiology Reports on Research and Teaching,* edited by J. Pemberton. London: Oxford Univ. Press, 1963, p. 124–130.
4. Epstein, F. H. Epidemiological studies on the nature of high blood pressure. *Proc. 15th Ann. Conf. Kidney.* Swampscott, Mass., October, 1963.
5. Shaper, A. G. Blood pressure studies in East Africa. In: *Epidemiology of Hypertension,* edited by J. Stamler, R. Stamler and T. N. Pullman. New York: Grune & Stratton, 1967, p. 139–145.
6. McDonnough, J. F., G. E. Garrison and C. Hanes. Blood pressure and hypertensive disease among negroes and whites in Evans County, Georgia. In: *Epidemiology of Hypertension,* edited by J. Stamler, R. Stamler and T. N. Pullman. New York: Grune & Stratton, 1967, p. 167–187.
7. Lovell, R. R. H. Race and blood pressure, with special reference to Oceania. In: *Epidemiology of Hypertension,* edited by J. Stamler, R. Stamler and T. N. Pullman. New York: Grune & Stratton, 1967, p. 122–139.
8. Shaper, A. G., and A. W. Williams. Cardiovascular disorders at an African hospital in Uganda. *Trans. Roy. Soc. Trop. Med. Hyg.* 54: 62, 1960.
9. Padmavati, S. Epidemiology of cardiovascular disease in India. *Circulation* 25: 711, 1962.
10. Malhotra, S. L. Geographical aspects of acute myocardial infarction in India with special reference to the pattern of diet and eating. *Brit. Heart J.* 29: 777, 1967.
11. Malhotra, S. L. Epidemiology of ischaemic heart disease in India with special reference to causation. *Brit. Heart J.* 28: 895, 1967.
12. Burgess, A. M., Jr., Z. Fejfar and A. Kagan. *Arterial Hypertension and Ischaemic Heart Disease.* World Health Organ., Geneva, 1963.
13. Deming, Q. B., M. E. Hodes, A. Baltazar, J. G. Edreira and S. Torosdag. The changes in concentration of cholesterol in the serum of hypertensive patients during anti-hypertensive therapy. *Am. J. Med.* 24: 882, 1958.
14. Malhotra, S. L. Serum lipids, dietary factors and ischemic heart disease. *Am. J. Clin. Nutr.* 20: 462, 1967.
15. Malhotra, S. L. Geographical distribution of gastrointestinal cancers in India with special reference to causation. *Gut* 8: 361, 1967.
16. Malhotra, S. L. Effect of diet on faecal urobilinogen excretion and its possible relationship to the pathogenesis of peptic ulceration. *Gut* 9: 183, 1968.
17. Malhotra, S. L. Effect of pattern of eating and antacids on faecal urobilinogen excretion. *Gut* 9: 38, 1968.
18. Sackett, G. C. Modification of Bloor's method for the determination of cholesterol in whole blood or blood serum. *J. Biol. Chem.* 64: 203, 1925.
19. Maclagan, N. F. Faecal urobilinogen — clinical evaluation of a simplified method of estimation. *Brit. J. Exptl. Pathol.* 27: 190, 1946.
20. Indian Council Medical Research. *Diet Atlas of India.* New Delhi: Govt. Printing Press, 1964.
21. Malhotra, S. L. Peptic ulcer in India and its aetiology. *Gut* 5: 412, 1964.
22. Miall, W. E. Some personal factors influencing arterial blood pressure. In: *Epidemiology of Hypertension,* edited by J. Stamler, R. Stamler and T. N. Pullman. New York: Grune & Stratton, 1967, p. 60–67.
23. U.S. Dept. of Public Health, Education and Welfare, Public Health Service. *Health Diseases in Adults, United States, 1960–62.* Data from National Health Survey, Ser. 11, no. 5, 10, Washington, D.C., 1964.
24. Stamler, J. *Lectures on Preventive Cardiology.* New York: Grune & Stratton, 1967, p. 246.
25. Ostrander, L. D., Jr., T. Francis, N. S. Hayner, M. O. Khelsberg and F. H. Epstein. The relationship of cardiovascular disease to hyperglycemia. *Ann. Internal Med.* 62: 1188, 1965.
26. Epstein, F. H., L. D. Ostrander, Jr., B. C. Johnson, M. W. Payne, N. S. Hayner, J. B. Keller and T. Francis, Jr. Epidemiological studies of cardiovascular disease in a total community — Tecumseh, Michigan. *Ann. Internal Med.* 62: 1170, 1965.

27. SWITZER, S. Bacteriuria in a healthy population and its relation to hypertension and pyelonephritis. *New Engl. J. Med.* 264: 7, 1961.
28. CORT, J. H., V. FENCL, Z. HEJL AND J. JIRKA (editors). *The Pathogenesis of Essential Hypertension. Proc. Prague Symposium.* Prague: State Medical Publishing House, 1960.
29. FREGLY, M. J. In: Seminar on the role of salt in cardiovascular hypertention. *Am. J. Cardiol.* 8: 526, 684, 863, 1961.
30. DAHL, L. K. Effects of chronic excess salt ingestion—experimental hypertension in the rat: correlation with human hypertension. In: *Epidemiology of Hypertension,* edited by J. Stamler, R. Stamler and T. N. Pullman. New York: Grune & Stratton, 1967, p. 261.
31. KENETA, S., K. ISHIGURO, S. KOYAISHI AND E. TAKAHASHI. An epidemiological study on nutrition and cerebro-vascular lesions in Tohoku area of Japan. *Tohoku J. Exptl. Med.* 83: 398, 1964. Quoted in *Lectures on Preventive Cardiology* by J. Stamler. New York: Grune & Stratton, 1967, p. 1261.
32. DAWBER, T. R., W. B. KANNEL, A. KAGAN, R. K. DONABEDIAN, P. M. MCNAMARA AND G. PEARSON. Environmental factors in hypertension. In: *Epidemiology of Hypertension,* edited by J. Stamler, R. Stamler and T. N. Pullman, New York: Grune & Stratton, 1967, p. 269.
33. PAUL, O., M. H. ZEPPER, W. H. PHELAN, G. W. DUPERTINS, A. MACMILLAN, H. MCKEON AND H. PARK. A longitudinal study of coronary heart disease. *Circulation* 28: 20, 1963.
34. MALHOTRA, S. L. An epidemiological study in cholelithiasis among railroad workers in India with special reference to causation. *Gut* 9: 290, 1968.
35. VARLEY, H. *Practical Clinical Biochemistry* (3rd ed.). London: Heinemann, 1963, p. 296.
36. KERN, J. F., AND B. BÖRGSTROM. The effect of a conjugated bile salt in oleic acid absorption in the rat. *Gastroenterology* 49: 623, 1965.
37. DAWSON, A. M., AND D. R. SAUNDERS. The effect of bile salts on the pathway of fat absorption in the rat. In: *Symp. NATO Advanced Study Inst. Biliary System.* Oxford: Blackwell, 1965.
38. HUGHES, R. H., AND E. J. WIMMER. The absorption of soluble, volatile fatty acids. *J. Biol. Chem.* 108: 141, 1935.
39. AHRENS, E. H., JR., J. HIRSH, W. INSULL, JR. AND M. L. PETERSON. *Dietary Fats and Human Serum Blood Levels. Chemistry of Lipids as Related to Atherosclerosis.* Springfield, Ill.: Thomas, 1958, p. 222.
40. FROHLICH, E. D., R. C. TARAZI AND H. P. DUSTAN. Reexamination of the hemodynamics of hypertension. *Am. J. Med. Sci.* 257: 9, 1969.