Hypertension in a rural South African white population and the effect of antihypertensive treatment on the risk of coronary heart disease

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Summary

The association between hypertension and coronary risk factors and the effect of antihypertensive treatment on coronary risk were investigated in rural South African whites aged 15 - 64 years. Almost 25% of men (range 1,9 - 46,6%) and almost 27% of women (2,1 - 56,2%) were hypertensive or being treated for hypertension; the prevalence increased with age, particularly among women. Only 25,8% of male and 43,4% of female hypertensives were being treated, and of these only 38% had controlled blood pressure.

Hypertension was associated with a high serum total cholesterol level, a low high-density lipoprotein cholesterol level, a high body mass index, a high uric acid level, a high prevalence of coronary heart disease and, in men, high alcohol consumption. Treated hypertensives had a greater risk of coronary heart disease than untreated hypertensives. Men on β-blockers had significantly lower high-density lipoprotein cholesterol levels than men not on treatment, while uric acid levels in both men and women on diuretics were higher than those of untreated hypertensives.

Hypertension in the study population appears to be inadequately treated, and antihypertensive medication may impact adversely on metabolic risk factors. The goal of antihypertensive therapy should be a net reduction of coronary heart disease risk.

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Hypertension is considered to be one of the three major reversible coronary heart disease (CHD) risk factors, the other two being hypercholesterolaemia and smoking.[1] Hypertension is one of the easier risk factors to control. However, some controlled randomised trials of antihypertensive agents have failed to show that the incidence of CHD is reduced with normalisation of blood pressure,[2,3] while others have shown a reduction in the frequency of coronary events in non-smoking men on β-blocker therapy.[4,5] Experimental studies have reported that commonly used antihypertensive drugs such as thiazide diuretics and both cardioselective and non-cardioselective β-blockers have adverse effects on lipid metabolism that can offset some or even most of the CHD protection achieved by blood pressure reduction.[6,7]

Little information exists on the prevalence of hypertension in white South Africans, a population known to have very high mortality from ischaemic heart disease.[8] In 1980 it was reported that the prevalence of hypertension in urban whites aged 15 - 90 years was 25,6% for men and 20,0% for women,[9] and in a group of white male miners the age-specific prevalence of hypertension varied from 6,1% in young men to 25,9% in those aged over 60 years.[10] A hypertension prevalence study was conducted as part of a community coronary risk factor study in three rural white communities in the south-western Cape.[11] Specific objectives of the hypertension study were: (*i*) to describe the prevalence of hypertension and antihypertensive treatment status in these communities; (*ii*) to compare the coronary risk factors and CHD risk of hypertensives with those of normotensives; (*iii*) to study possible differences in risk factors and CHD between treated and untreated hypertensives; and (*iv*) to examine the blood lipids and uric acid levels of hypertensives taking β-blockers, diuretics, other antihypertensive medication or no medication.

Study population and methods

The general objectives, sampling procedures, response rates, measurements and interpretation of data in the Coronary Risk Factor Study (CORIS) have been reported elsewhere.[11] Briefly, 3 357 men and 3 831 women (representing 82% of the known target population aged 15 - 64 years) from three rural communities in the south-western Cape were studied to establish the prevalence of coronary risk factors in an Afrikaner community.

All participants had to complete a general risk factor questionnaire and the London School of Hygiene questionnaire for chest pain[12] by interview, as well as a self-administered Bortner Short Rating Scale for coronary-prone (type A) behaviour.

After the questionnaires were completed, biological measurements were made. Resting ECGs were taken and classified according to the Minnesota criteria. A person was classified as suffering from CHD if he or she had a history of chest pain and/or an ECG suggestive of CHD (Minnesota codes 1.1 and 1.2). Anthropometric observations in the standing position, included height and weight (light clothing without shoes). The body mass index (BMI) (weight in kg/height in m^2) was calculated from these measurements.

Blood pressure readings were obtained according to the recommendations of the American Heart Association. A standard 12,5 × 23 cm cuff connected to a mercury manometer was used. The respondent was seated with the back supported, the cuff was applied to the right upper arm, and after a 5-minute rest period the lowest blood pressure of at least three intermittent readings was recorded, with the point of muffling of the Korotkoff sounds (phase IV) taken as the diastolic pressure. Three observers were used and their readings were standardised against those of an experienced clinician. Standardisation was checked weekly and inter-observer variation

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did not exceed 5%. Subsequent analysis of the data showed no evidence of end-digit preference.

Hypertension was diagnosed in subjects with systolic and/or diastolic blood pressure equalling or exceeding 160/95 mmHg, or those who had normal blood pressure but were on antihypertensive medication. Controlled hypertension was defined as a blood pressure of less than 160/95 mmHg while on antihypertensive medication. To examine the possible differences in blood lipids and uric acid levels related to treatment of hypertension, the hypertensives were subdivided into five different treatment categories as follows: (*i*) those on diuretcis; (*ii*) those on β-blocking agents for hypertension and angina; (*iii*) those on diuretics and β-blockers ('combination' treatment); (*iv*) those on other antihypertensive drugs; and (*v*) those not on any antihypertensive medication. 'Other' drugs for hypertension included sympatholytic agents, peripheral vasodilators, α-adrenergic blocking agents and enzyme-inhibiting drugs.

Non-fasting blood samples were taken and analysed for total cholesterol (TC) and high-density lipoprotein cholesterol (HDL-C) after precipitation of the apoprotein B-containing lipoprotein with dextran sulphate-magnesium chloride,[13] using the Boehringer CHOD-PAP enzymatic method. The remaining serum was analysed for uric acid (Boehringer Uricaquant method).

Means of variables across categories were compared by adding the probabilities obtained from age-specific *t*-tests for the relevant categories, using Fisher's method.[14] The strength of the association of hypertension and antihypertensive treatment status with CHD was obtained by calculating odds ratios and their 95% confidence intervals from stepwise multivariate logistic regression analysis. Firstly it was adjusted for age, and subsequently also for TC, HDL/TC ratio, BMI, smoking, alcohol intake and uric acid. Metabolic effects of the different antihypertensive therapies were estimated using analysis of covariance, adjusting for age and BMI.

Results

In both sexes there was a gradual increase in the mean systolic and diastolic blood pressure with increasing age. This was accompanied by a marked increase in the prevalence of hypertension (Table I). Men aged 25 - 44 years had a higher prevalence of hypertension than women of the same age, whereas in older age groups this situation was reversed. Only 25,8% of all male and 43,4% of all female hypertensives reported using any antihypertensive medication, and only 38% of these (i.e. 10% of all male and 16,9% of all female hypertensives) had controlled blood pressure.

The age-adjusted coronary risk factor profiles of hypertensives and normotensives are shown in Table II. Hypertensives had significantly higher TC and uric acid levels and lower HDL/TC ratios than normotensives. BMIs were significantly higher in hypertensives of both sexes than in normotensives, while Bortner scores did not differ between hypertensives and normotensives. More male hypertensives than normotensives were drinkers, and they also consumed significantly more alcohol than the normotensives. Male hypertensives had a 1,86 times and female hypertensives a 1,93 times greater odds ratio for CHD than normotensives, with 95% confidence intervals of 1,41 - 2,46 and 1,36 - 2,74 respectively.

Among men, treated hypertensives were older than untreated hypertensives and had significantly lower systolic and diastolic blood pressures, lower HDL-C levels, HDL/TC ratios and alcohol consumption, and higher BMIs (Table III). Treated hypertensive women were older and had lower systolic and diastolic blood pressures, lower TC levels, and higher BMIs

TABLE I. MEAN SYSTOLIC AND DIASTOLIC BLOOD PRESSURE, PREVALENCE OF HYPERTENSION* AND TREATMENT STATUS OF HYPERTENSIVES IN THE STUDY POPULATION

	Men						Women					
Age (yrs)	No.†	SBP (mmHg) (mean ± SD)	DBP (mmHg) (mean ± SD)	Prevalence (%)	Treated‡ (%)	Controlled‡ (%)	No.†	SBP (mmHg) (mean ± SD)	DBP (mmHg) (mean ± SD)	Prevalence (%)	Treated‡ (%)	Controlled‡ (%)
15 - 24	635	125 ± 12	75 ± 9	1,9	0	0	658	120 ± 11	76 ± 9	2,1	7,1	7,1
25 - 34	634	130 ± 12	84 ± 10	12,3	10,3	5,1	713	124 ± 13	80 ± 9	7,6	25,9	22,2
35 - 44	641	134 ± 16	88 ± 11	23,7	13,2	4,6	813	131 ± 18	85 ± 10	20,3	33,9	13,3
45 - 54	705	140 ± 20	90 ± 12	34,1	27,9	12,1	845	144 ± 22	91 ± 12	41,0	41,3	17,3
55 - 64	742	149 ± 24	91 ± 12	46,6	34,3	12,4	802	155 ± 25	92 ± 12	56,2	51,7	17,5
15 - 64	3 357	136 ± 19	86 ± 12	24,7	25,8	10,0	3 831	136 ± 23	85 ± 12	26,9	43,4	16,9

*Blood pressure ⩾ 160/95 mmHg and/or on antihypertensive treatment.
†No. of participants.
‡% of all hypertensives.
SBP = systolic blood pressure; DBP = diastolic blood pressure.

TABLE II. MEAN AGE, AGE-ADJUSTED† MEANS (± SE) OF SELECTED CORONARY RISK FACTORS AND CHD RISK IN HYPERTENSIVES AND NORMOTENSIVES (AGED 15 - 64 YEARS)

	Men		Women	
	Hypertensive ($N = 829$)	Normotensive ($N = 2\,529$)	Hypertensive ($N = 1\,030$)	Normotensive ($N = 2\,805$)
Age (yrs)	49,9 ± 0,37**	37,0 ± 0,29	51,2 ± 0,30**	36,6 ± 0,26
SBP (mmHg)	154 ± 0,92*	129 ± 0,23	154 ± 0,89*	127 ± 0,22
DBP (mmHg)	100 ± 0,73*	82 ± 0,14	99 ± 0,45*	81 ± 0,14
TC (mmol/l)	6,67 ± 0,07*	6,10 ± 0,02	6,69 ± 0,08*	6,30 ± 0,02
HDL-C (mmol/l)	1,25 ± 0,02	1,24 ± 0,01	1,46 ± 0,02*	1,55 ± 0,01
HDL/TC ratio (%)	19,4 ± 0,43*	21,3 ± 0,13	23,1 ± 0,52*	25,6 ± 0,13
BMI	27,6 ± 0,24*	25,4 ± 0,07	28,0 ± 0,38*	24,9 ± 0,09
Type A behaviour (Bortner score)	53,0 ± 0,46	52,6 ± 0,19	50,5 ± 0,65	51,8 ± 0,19
Uric acid (mmol/l)	0,39 ± 0,006*	0,36 ± 0,002	0,30 ± 0,004*	0,27 ± 0,001
Smokers				
%	48,2	49,0	18,1	18,6
Cigarettes/d	22 ± 0,74	20 ± 0,35	15 ± 0,59	15 ± 0,47
Drinkers				
%	85,3	76,4	48,3	56,6
Alcohol/d (g)	20,9 ± 1,14*	15,5 ± 0,49	6,1 ± 0,75	5,7 ± 0,23
CHD				
Prevalence (%)	12,2	6,4	7,5	3,2
Odds ratio‡	1,86	1,00	1,93	1,00
95% CI	1,41 - 2,46		1,36 - 2,74	

†Age-adjusted against 1980 census; SA white population of study areas.
‡Adjusted for age, TC, HDL/TC ratio, BMI, smoking, alcohol intake and uric acid.
*$P < 0,01$ compared with normotensives; Fisher's method.
**$P < 0,001$ compared with normotensives; *t*-test.
SBP = systolic blood pressure; DBP = diastolic blood pressure; CI = confidence intervals.

TABLE III. MEAN AGE, AGE-ADJUSTED† MEANS OF SELECTED CORONARY RISK FACTORS AND CHD RISK IN TREATED AND UNTREATED HYPERTENSIVES (AGED 25 - 64 YEARS)

	Men		Women	
	Treated ($N = 214$)	Untreated ($N = 603$)	Treated ($N = 446$)	Untreated ($N = 570$)
Age (yrs)	53,8 ± 0,55***	49,0 ± 0,42	53,4 ± 0,39***	50,3 ± 0,40
SBP (mmHg)	155 ± 3,55**	161 ± 1,32	157 ± 2,15**	166 ± 1,42
DBP (mmHg)	98 ± 1,86**	102 ± 0,66	96 ± 1,16**	101 ± 0,63
TC (mmol/l)	7,08 ± 0,25	6,93 ± 0,10	7,26 ± 0,14*	7,42 ± 0,13
HDL-C (mmol/l)	1,16 ± 0,04**	1,28 ± 0,03	1,47 ± 0,04	1,53 ± 0,03
HDL/TC (%)	17,1 ± 0,78**	19,2 ± 0,70	21,1 ± 0,75	21,4 ± 0,57
BMI	28,5 ± 0,61**	28,0 ± 0,33	29,8 ± 0,62**	28,2 ± 0,43
Type A behaviour (Bortner score)	52,1 ± 1,30	51,5 ± 0,76	49,9 ± 1,04	50,4 ± 0,83
Uric acid (mmol/l)	0,41 ± 0,017	0,38 ± 0,007	0,33 ± 0,010**	0,30 ± 0,007
Smokers				
%	42,9	49,0	16,0	15,9
Cigarettes/d	22 ± 2,62	25 ± 1,30	14 ± 1,89	14 ± 1,70
Drinkers				
%	75,7	81,0	40,2	50,6
Alcohol/d (g)	22,4 ± 4,14*	24,2 ± 2,54	6,5 ± 1,34	6,5 ± 1,05
CHD				
Prevalence (%)	32,8	10,3	17,4	6,2
Odds ratio[1]	4,30	1,00	3,22	1,00
95% CI	2,89 - 6,40		2,10 - 4,93	
Odds ratio[2]	1,22	1,00	1,10	1,00
95% CI	1,11 - 1,35		1,01 - 1,20	

†Age-adjusted against all hypertensives of the study population.
*$P < 0,05$ compared with untreated; Fisher's method.
**$P < 0,01$ compared with untreated; Fisher's method.
***$P < 0,001$ compared with untreated; *t*-test.
SBP = systolic blood pressure; DBP = diastolic blood pressure; odds ratio[1] = adjusted for age; odds ratio[2] = adjusted for age, TC, HDL/TC ratio, BMI, smoking, alcohol intake and uric acid; CI = confidence intervals.

and uric acid levels than untreated hypertensive women. In addition to age-adjustment, stepwise multiple logistic regression adjustment for other coronary risk factors (TC, HDL/TC ratio, BMI, smoking, alcohol intake and uric acid) resulted in a decrease in the odds ratio for CHD of treated versus untreated hypertensives from 4,3 to 1,22 for men, and from 3,22 to 1,12 for women. From the 95% confidence interval shown in Table III it is evident that all these odds ratios maintained a significance level of 5% after adjustment for the covariate risk factors.

Analysis of covariance, comparing the blood lipids and uric acid of the various antihypertensive treatment groups (Table IV), revealed that hypertensive men on β-blockers and the combination treatment had significantly lower HDL levels and HDL/TC ratios than untreated hypertensive men. Men on the combination treatment also had significantly higher TC levels than those on no treatment, and men on diuretics and on the combination treatment had significantly higher uric acid levels than the untreated group. Among women there were no significant differences in TC or HDL-C levels between the treatment groups, but the mean uric acid level of women on diuretics was significantly higher than the level for women on no treatment. Other antihypertensive drugs, whether they were taken in combination with diuretics and/or β-blockers or not, had no significant effect on either lipids or uric acid levels; therefore only the means of this 'other' medication group are shown in Table IV.

Discussion

The mean prevalence of hypertension of 24,7% in men and 26,9% in women observed in this community study may be misleading in certain aspects. Firstly, it masks the low prevalence (approximately 2%) of hypertension in young people (15 - 24 years), raising the question of adequacy of the criteria used for defining hypertension in young people. Considering the high prevalence of hypertension in older age groups in the same population, it may be advisable to use lower blood pressure levels in the young age groups to identify individuals at risk of developing hypertension later in life. From the Pooling Project[1] it is known that the risk of CHD is higher than average when blood pressure levels exceed 140/90 mmHg in middle-aged men. In younger men and women, a blood pressure of 140/90 mmHg may carry a much higher relative risk than the same pressure in older persons.

Secondly, the mean prevalence of hypertension does not focus attention on the alarmingly high rate of hypertension (46,6% for men and 56,2% for women) among older people (55 - 64 years), particularly women. Even if cross-sectional studies overestimate the prevalence of hypertension in a community study, this crude rate of approximately half of the older age group being hypertensive remains unacceptably high.

Only 25,8% of hypertensive men and 43,4% of hypertensive women were on treatment; 74,2% of male hypertensives and 56,6% of female hypertensives were therefore undiagnosed or untreated, or had stopped taking their previous antihypertensive medication. Markedly more hypertensive men than women were untreated. This difference was also observed in the Hypertension Detection and Follow-up Programme (HDFP),[15] where 41% of hypertensive men and 65% of hypertensive women were on treatment, but total treatment figures were higher than those found in this study. Also, in the HDFP 28% of hypertensive men and 52% of hypertensive women had controlled blood pressures, in comparison with the low 10,0% of men and 16,9% of women in this study. These comparisons should be interpreted with caution because the differences could have resulted from differences in age or socio-economic status between the populations studied, but their magnitude does suggest that rates of treatment of hypertension and percentages of hypertensives with controlled blood pressure in this study were markedly lower than those in the HDFP.

The interrelation of TC, HDL/TC, BMI, uric acid and blood pressure (see Table II) concurs with findings of other observational studies.[16,17] The higher BMI of the hypertensives could have contributed, at least in part, to their higher TC and uric acid levels and lower HDL/TC ratios. Hypertensives should therefore attempt to keep their body weight within the normal range to prevent unnecessary increases of their blood lipid levels. In fact, more than 65% of hypertensives (men and

TABLE IV. MEANS (± SE) AND *P*-VALUES* FOR DIFFERENT ANTIHYPERTENSIVE TREATMENT GROUPS

	Type of treatment				
	No treatment	Diuretics	β-blockers	Combination†	Other‡
Men	***N* = 603**	***N* = 34**	***N* = 70**	***N* = 19**	***N* = 22**
TC (mmol/l)	6,90 ± 0,06	7,11 ± 0,24	7,03 ± 0,17	7,88 ± 0,32	6,74 ± 0,30
		(*P* = 0,4156)	(*P* = 0,4364)	(*P* = 0,0029)	(*P* = 0,6189)
HDL-C (mmol/l)	1,28 ± 0,01	1,26 ± 0,06	1,11 ± 0,04	1,09 ± 0,08	1,25 ± 0,07
		(*P* = 0,7048)	(*P* = 0,0001)	(*P* = 0,0139)	(*P* = 0,6498)
HDL/TC (%)	19,2 ± 0,24	18,4 ± 1,01	16,4 ± 0,69	15,2 ± 1,33	18,9 ± 1,24
		(*P* = 0,4584)	(*P* = 0,0002)	(*P* = 0,0032)	(*P* = 0,8517)
Uric acid (mmol/l)	0,38 ± 0,004	0,44 ± 0,017	0,37 ± 0,011	0,47 ± 0,022	0,40 ± 0,020
		(*P* = 0,0003)	(*P* = 0,3783)	(*P* = 0,0001)	(*P* = 0,3061)
Women	***N* = 570**	***N* = 79**	***N* = 73**	***N* = 38**	***N* = 68**
TC (mmol/l)	7,44 ± 0,06	7,18 ± 0,17	7,31 ± 0,18	7,29 ± 0,24	7,24 ± 0,18
		(*P* = 0,1348)	(*P* = 0,4662)	(*P* = 0,5059)	(*P* = 0,2514)
HDL-C (mmol/l)	1,52 ± 0,02	1,52 ± 0,05	1,50 ± 0,05	1,51 ± 0,07	1,48 ± 0,05
		(*P* = 0,9489)	(*P* = 0,7186)	(*P* = 0,8943)	(*P* = 0,4175)
HDL/TC (%)	21,2 ± 0,29	22,0 ± 0,78	21,6 ± 0,81	21,7 ± 1,12	21,3 ± 0,84
		(*P* = 0,3102)	(*P* = 0,6467)	(*P* = 0,6669)	(*P* = 0,9224)
Uric acid (mmol/l)	0,30 ± 0,004	0,33 ± 0,010	0,31 ± 0,011	0,33 ± 0,014	0,31 ± 0,011
		(*P* = 0,0112)	(*P* = 0,3734)	(*P* = 0,0828)	(*P* = 0,4653)

*Compared with no treatment; analysis of covariance, adjusted for age and body mass index.
†Diuretics and β-blockers.
‡Sympatholytic agents, peripheral vasodilators, α-adrenergic blocking agents and enzyme-inhibiting drugs.

women) were also hypercholesterolaemic (TC $\geqslant$ 6,5 mmol/l), a tendency also observed in other studies.[6]

Both systolic and diastolic blood pressures were positively associated with increasing alcohol consumption among users of alcohol, more so in men than in women. Male users of alcohol as a group had significantly higher systolic and diastolic blood pressures than non-drinkers ($P < 0{,}005$; Fisher's method), while female users had significantly higher diastolic pressures ($P < 0{,}01$), but significantly lower systolic pressures ($P < 0{,}005$) than non-users. These results provide evidence, along with the results of other population studies,[18] of a strong link between alcohol and blood pressure.

As expected, treated hypertensives had lower systolic and diastolic blood pressures than untreated hypertensives; they were also significantly older, weighed more, and had a higher prevalence of CHD. Comparison of plasma lipid and lipoprotein levels in subjects being treated for hypertension with those in untreated persons indicated that HDL levels and HDL/TC ratios were lower in treated men, but that the lower TC in women had little effect on their HDL/TC ratio. Since this was a cross-sectional study, these differences in risk factor status between treated and untreated hypertensives probably represent a combination of the pre-existing risk factor profiles of hypertensives and the effects of the treatment on some of the risk factors. Although treated hypertensives had lower mean systolic and diastolic blood pressures, most of the other coronary risk factors appeared more unfavourable, and the prevalence of CHD considerably higher, than in untreated hypertensives. Also, the risk of CHD remained significantly higher for treated than for untreated hypertensives, even after controlling for all the various coronary risk factors. Stepwise analyses revealed that of all these risk factors, age and HDL/TC ratio for men and BMI for women were the only variables other than treatment that had a significant effect on the odds ratio for CHD. Removing the effect of these variables still resulted in a significantly higher odds ratio for the treated hypertensives (Table III). A shortcoming of this study design is the non-availability of lipid levels before antihypertensive treatment commenced. It is, however, unlikely that selection of the type of treatment was influenced in any way by the pre-existing lipid levels. Even though patients with severe hypertension were more likely to have been placed on treatment, the increased CHD rates for treated hypertensives in this study underline the observations of other researchers that changes in plasma lipid and lipoprotein levels associated with antihypertensive treatment may offset in part the decrease in risk of CHD that can be expected to result from blood pressure reduction.[6,7] These findings once again emphasise the importance of attention to other coronary risk factors before and during antihypertensive therapy.

The results of this study indicate that the effect of β-blockers was more pronounced in men, particularly on HDL-C, since HDL and HDL/TC ratios were significantly lower in those receiving β-blockers or the combination treatment than in the untreated group. These effects of antihypertensive treatment in men are consistent with several reports on the effects of β-blockers on plasma lipoproteins and lipids.[6,7] Because antihypertensive therapy with β-blockers was associated with differences in lipoprotein levels in this study, routine investigation of plasma lipid and lipoprotein levels in hypertensive patients, especially men, before and during antihypertensive treatment is important. As reported in other studies,[19,20] diuretics (taken on their own or in combination with β-blockers) were associated with elevated uric acid levels in both men and women, even after controlling for age and BMI. It is therefore recommended that serum uric acid levels should be studied after antihypertensive treatment with diuretics is started, especially in persons with a history of gout.

Treatment of hypertension should therefore not only aim at lowering blood pressure, because drugs that do this effectively may actually increase the risk of CHD through adverse effects on serum lipids and uric acid. Therapeutic agents without known adverse effects on plasma lipids or lipoproteins should be considered for patients with pre-existing hyperuricaemia and plasma lipid or lipoprotein abnormalities or for those whose plasma lipid levels react adversely to β-blocking and thiazide diuretic therapy.

Our results emphasise the need for the detection and successful treatment of hypertension in these and other communities with similar hypertension rates. Hypertension screening programmes, using existing infrastructures, e.g. general practitioners, schools and workplaces, can be used effectively to identify younger hypertensives, most of whom are undiagnosed and untreated at present. Individuals with a clustering of risk factors should be identified to reduce the overall risk of CHD. Finally, treatment of high blood pressure should avoid adverse effects on plasma lipid, lipoprotein and uric acid levels.

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