

PAPER

Blood pressure and pulse pressure development in a population sample of women with special reference to basal body mass and distribution of body fat and their changes during 24 years

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OBJECTIVE: To study blood pressure and pulse pressure longitudinally and their association with basal and change of body mass index (BMI) and waist to hip ratio (WHR).

DESIGN: A prospective population study of 1462 women in Gothenburg, Sweden, aged 38–60 y at baseline, with a longitudinal follow-up of 24 y.

OUTCOME MEASURES: Incidence of hypertension, systolic and diastolic blood pressure, and pulse pressure at baseline and after 12 and 24 y of follow-up.

RESULTS: Systolic and diastolic blood pressure as well as pulse pressure increased with age and turned down again at high age. BMI and WHR at baseline were each independently associated with baseline systolic and diastolic blood pressure, but only BMI with pulse pressure. However, baseline BMI and WHR were not associated with change of systolic, diastolic or pulse pressure during 12 or 24 y of follow-up. Increase in BMI during the follow-up period was associated with increase in systolic and diastolic blood pressure but not with increase in pulse pressure. There were no such associations with WHR changes which, were either unrelated or in one analysis inversely related with blood pressure changes. When considering incidence of hypertension during the first 12 y of follow-up, BMI and change in BMI were significant predictors, independent of WHR.

CONCLUSION: Age, BMI and increments in BMI seem to be strong predictors for hypertension and increased systolic and diastolic blood pressure in women. In contrast, WHR plays a lesser and uncertain role in the development of hypertension in middle-aged women. Changes in BMI seem not to be accompanied by changes in pulse pressure during a long time follow-up. *International Journal of Obesity* (2003) 27, 128–133. doi:10.1038/sj.ijo.0802190

Keywords: anthropometry; hypertension; blood pressure; pulse pressure; antihypertensive treatment; population study; women

Introduction

Epidemiological studies show a linear rise in systolic blood pressure with age from 30–80 y and a concurrent early increase in diastolic blood pressure from young age up to the age of 50–60 y^{1,2} where the diastolic pressure levels off or even declines after that. This causes the pulse pressure, which is the gap between systolic and diastolic blood pressure, to increase steeply in people older than 50–75 y which may *per se* accele-

rate the development of arteriosclerosis.¹ Recently it has been pointed out that increased pulse pressure is an independent predictor of cardiovascular events in younger normotensive subjects³ and in subjects with otherwise relatively low cardiovascular risk,⁴ as well as in older hypertensive patients.⁵

Obesity has by tradition been measured as body mass index (BMI) with cut-off point ≥ 25 denoting generalised obesity.⁶ Waist to hip circumference ratio (WHR) has been used as an indicator of centralised fat distribution. These variables are related to increase in blood pressure,^{7,8} metabolic derangement such as hyperinsulinaemia,^{9–11} and are considered as risk factors for diabetes,¹² ischaemic heart disease and premature death.^{13–15} However, information is still lacking on the long term effect of obesity and weight change on blood pressure and pulse pressure development.

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The purpose of this paper is to describe changes of blood pressure in a cohort of women in a follow up period of 12 and 24 y and possible associations between BMI and WHR at baseline and changes of BMI and WHR on one hand, and on the other hand incidence of hypertension and systolic, diastolic and pulse pressure development.

Materials and Methods

Study population

The baseline study of the population sample was carried out in Gothenburg, Sweden in 1968–1969,¹⁶ when altogether 1462 women in five age strata between 38 and 60 y underwent health examinations (Table 1). The subject selection was based on date of birth in order to ensure that the women were representative of the general population of women of the ages studied. The study was carried out during a period of 1 y, starting with the women born at the beginning of the year. This means that the women in each age stratum were examined at the same age and with only a very small range. The participation rate was 90.1%, mainly similar in the different age strata studied. The participants were re-examined after 6 and

12 y,^{17,18} and a third follow-up was carried out after 24 y, in 1992–1993.¹⁹ In 1980–1981 two additional groups aged 26 (85 participants) and 38 (122 participants) took part.¹⁸ These age-groups were again invited to participate in the study in 1992–1993. Furthermore, in 1980–1981 those women born in 1930 (50 y of age in 1980–1981) who had moved to the study area since the initial examination and who fulfilled the inclusion criteria with respect to date of birth were invited to participate (59 participants in this group in 1980–1981) in order to obtain a representative cross-sectional sample, and for the same reason, women born in 1922, who had moved to the study area since the initial examination in 1968–1969 and fulfilled the inclusion criteria with respect to date of birth, were included in 1992–1993, and 32 women in this group participated. The procedure was mainly the same in all the four studies. Only data from the basal study and the 12 y and 24 y follow-ups will be included in the present analysis. The 24 y follow-up study included 836 out of the 1462 women studied at baseline.¹⁹ Of the participants in the baseline study, 280 (19.2%) had died before the 24 y follow-up study. Another 89 women had moved from the city and did not participate 255 women refused to participate and two were not accessible at the time of the study. The participation rate was 70.7% among those who had participated in the baseline study and who were alive 24 y later. Data on mortality were obtained for all who participated in the baseline examination. Further details about the studies have been given in previous reports.^{16–19}

Table 1 Blood pressure and pulse pressure as measured in the basal study in 1968–1969

Age (y)	n	Systolic blood pressure		Diastolic blood pressure		Pulse pressure	
		Mean	s.d.	Mean	s.d.	Mean	s.d.
38	372	123	15	79	9	44	10
46	431	131	19	82	10	48	13
50	398	138	22	86	11	52	15
54	180	143	24	87	12	56	16
60	81	154	27	89	12	65	19

Methods

At the baseline visit, the women were invited to participate in a comprehensive medical examination.¹⁶ They were interviewed about history of arterial hypertension, and about use of antihypertensive drugs. The women were examined in the morning in the fasting state. Blood pressure was measured

Table 2 Changes in BMI, WHR, blood pressure and pulse pressure (increase, measured as mmHg) during the 24 y period between 1968–1969 and 1992–1993

	Age at the years 1968–1969					P-values*
	38	46	50	54	60	
<i>Changes from 1968–1969 to 1980–1981</i>						
Number	308	332	325	140	49	
BMI	1.6	1.2	0.7	0.8	0.3	< 0.0001
WHR	0.08	0.08	0.07	0.07	0.05	0.0004
Systolic blood pressure	12.0	13.9	10.6	14.3	11.0	0.88
Diastolic blood pressure	5.6	4.2	1.5	2.9	0.6	< 0.0001
Pulse pressure	6.4	9.6	9.1	11.4	10.4	0.0044
<i>Changes from 1968–1969 to 1992–1993</i>						
Number	252	263	218	79	18	
BMI	3.5	2.8	1.7	1.5	–0.3	< 0.0001
WHR	0.11	0.10	0.10	0.08	0.07	< 0.0001
Systolic blood pressure	25.1	27.8	23.4	27.0	9.7	0.18
Diastolic blood pressure	2.8	–0.5	–5.0	–3.6	–10.4	< 0.0001
Pulse pressure	22.6	28.3	28.2	30.9	18.4	0.0065

*P-values for test of correlation between age and changes.

Table 3 Partial correlation analysis ($n = 1462$) relating BMI and WHR at baseline to baseline values of systolic and diastolic blood pressures and pulse pressure. Age and WHR are taken into consideration as background variables when correlating BMI to blood pressure values, and age and BMI when correlating WHR with blood pressure values

Variables studied	BMI		WHR	
	Partial correlation coefficient	P-value	Partial correlation coefficient	P-value
Systolic blood pressure	0.21	0.0001	0.07	0.011
Diastolic blood pressure	0.24	0.0001	0.09	0.0004
Pulse pressure	0.12	0.0001	0.03	0.284

in the seated, supine and standing positions after about 5 minutes rest to the nearest 2 mmHg. Only blood pressure measured in the seated position is included in this report. All blood pressure measurements were made by the same physician (CB). Hypertension was defined as ongoing anti-hypertensive treatment and/or a systolic blood pressure ≥ 160 mmHg and/or a diastolic blood pressure > 95 mmHg. At the 12 y and 24 y follow-ups, the blood pressure measurements were made with the subjects in the seated position by different examiners. Pulse pressure was calculated as the difference between the systolic and the diastolic blood pressure. Body weight was measured to the nearest 0.1 kg by a balance scale. The women wore only briefs when being weighed. Body height without shoes was measured to the nearest 0.5 cm. BMI was calculated as weight/height² (kg/m²). Waist circumference was measured to the nearest 1 mm at the level midway between the lower rib margin and the iliac crest using a steel tape measure. Hip circumference was measured with the same steel tape measure to the

Table 5 Odds ratio (OR) and 95% confidence interval (CI) for risk of developing hypertension during 12 and 24 y per unit increase in baseline level and change in body mass index (BMI) and waist to hip ratio (WHR) for women non-hypertensive at baseline

Variable studied	OR	95% CI of OR	P-value
<i>During 12 y</i>			
BMI ^a	1.08	1.02–1.13	0 < 0.001
WHR $\times 100^a$	1.03	1.00–1.06	0.080
Change of BMI ^b	1.08	1.00–1.17	0.042
Change of WHR ^b	1.01	0.97–1.03	0.76
<i>During 24 y</i>			
BMI ^a	1.05	1.00–1.11	0.056
WHR $\times 100^a$	1.03	1.00–1.07	0.067
Change of BMI ^b	1.05	0.995–1.12	0.08
Change of WHR ^b	1.01	0.98–1.04	0.50

^aFrom logistic regression model with age, BMI and WHR as independent variables; ^bFrom logistic regression model with age and change in BMI and change in WHR as independent variables.

nearest 1 mm at the widest point between the iliac crest and buttock. The circumferences were measured in a standing position. WHR was calculated as waist circumference divided by hip circumference.

Statistical methods

Standard statistical methods have been used for all analyses. Simple tests of correlation were used to study the effect of age on the change of the studied variables (Table 2). Linear regression models are used to study the partial correlation between BMI/WHR and change in BMI/WHR to blood pressure (Tables 3 and 4). Binary logistic regression models were used to study predictors for incidence of hypertension during 12 and 24 y (Table 5).

Table 4 Partial correlation coefficients (r) between basal BMI and basal WHR on one hand and change of blood pressure on the other, and between changes of BMI and changes of WHR on one hand and change of blood pressure on the other, as observed between the 12 y period between 1968–1969 and 1980–1981 ($n = 916$ – 923) and during the 24 y period between 1968–1969 and 1992–1993 ($n = 659$ – 701), confined to women defined as non-hypertensive at the baseline study in 1968–1969

	Systolic blood pressure change		Diastolic blood pressure change		Pulse pressure change	
	r	P	r	P	r	P
<i>Changes within 12 y</i>						
BMI at baseline ^a	0.001	0.97	–0.016	0.63	0.011	0.72
WHR at baseline ^b	0.044	0.18	0.044	0.19	0.026	0.43
BMI change ^c	0.176	< 0.0001	0.27	< 0.0001	0.036	0.28
WHR Change ^d	–0.022	0.50	–0.069	0.036	0.017	0.61
<i>Changes within 24 y</i>						
BMI at baseline ^a	0.007	0.86	0.017	0.66	–0.014	0.72
WHR at baseline ^b	0.050	0.18	0.006	0.87	0.052	0.17
BMI change ^c	0.124	0.0013	0.243	< 0.0001	–0.014	0.72
WHR Change ^d	0.024	0.53	0.025	0.53	0.012	0.76

^aWHR additional background variable; ^bBMI additional background variable; ^cBMI at baseline and WHR change as additional background variables; ^dWHR at baseline and BMI change as additional background variables.

Results

Blood pressure, pulse pressure and antihypertensive treatment during 24 y

Blood pressure and pulse pressure at baseline, irrespective of being on antihypertensive treatment or not, is shown in Table 1. Table 6 shows prevalence of pharmaceutical antihypertensive treatment in 1968–1969 (at baseline), in 1980–1981 and in 1992–1993, respectively. As shown in Table 6, the prevalence of antihypertensive treatment increased with age, as observed on all three occasions, while there was no obvious secular trend, the percentages being mainly similar at corresponding ages on the three occasions.

Table 2 shows changes in BMI, WHR, blood pressure and pulse pressure during the 24 y study period. In Table 2, consideration is not taken as to whether individuals are on antihypertensive treatment or not. The systolic blood pressure increased by about 12 mmHg during the first 12 y period and by about another 12 mmHg during the following follow-up period, and was similar in the different age strata studied. The only age group in whom the blood pressure development was different was the oldest one, in which there was a decrease in systolic blood pressure between the examinations in 1980–1981 and 1992–1993 (between the age of 74 and 84 y). The pulse pressure increased with age, again with an exception for the eldest age group during the latest 12 y period.

Both BMI and WHR increased in all age groups studied, but the increase was significantly less in the older age groups.

Antihypertensive treatment and blood and pulse pressures at baseline in relation to BMI and WHR at baseline

Women who were overweight, with BMI ≥ 25 , were more commonly treated with antihypertensive drugs compared to women with BMI < 25 (odds ratio 2.56, 95% CI 1.55–4.22, $P < 0.001$). Women with WHR ≥ 0.80 compared to those with WHR < 0.80 were also more often treated for hypertension (odds ratio 2.94, 95% CI 1.72–5.03, $P < 0.001$). Table 3 shows associations between basal values of BMI and WHR on one hand, and basal blood pressure and pulse pressure on the

other. In addition to age, WHR was taken into consideration as a background variable when associations were made with BMI and vice versa. There were rather high and statistically significant partial correlations between BMI and blood pressure and pulse pressure, while the partial correlations with WHR were lower and not statistically significant for pulse pressure.

Incidence of hypertension in relation to basal BMI and WHR and change of BMI and WHR during the follow-up period

Logistic regression was used to predict 12 and 24 y incidences of hypertension in women, controlling for age and WHR when studying the influence of BMI, and controlling for age and BMI when studying the influence of WHR. During the first 12 y period only, there was an increased risk associated with both higher initial BMI and change in BMI, which was independent of initial WHR and change in WHR. The converse was not true for WHR at baseline and change in WHR, neither of which was a BMI-independent predictor of incident hypertension (Table 5).

Blood pressure and pulse pressure changes in relation to BMI and WHR at baseline and changes of BMI and WHR

As shown in Table 4 there were no partial correlations of statistical significance between on one hand basal BMI and WHR and on the other hand change of blood pressure and pulse pressure during the periods studied. The analyses were made on participants who were defined as non-hypertensive at the time of the basal study, but consideration was not taken as to whether they were on antihypertensive treatment or not later on, and WHR was in addition to age a background variable when analysing the associations with BMI and vice versa.

When associating changes in BMI and WHR with changes in blood pressure and pulse pressure, partial correlations of statistical significance were observed for changes in BMI, both with systolic and diastolic blood pressure, but not with pulse pressure change. However, a change of WHR was observed to have a negative partial correlation of statistical significance to change of diastolic blood pressure during the first 12 y period.

Discussion

In agreement with previous results from other studies, blood pressure increased with age, the increase being about 1 mmHg per year for the systolic blood pressure. At higher ages (about 80 y), the systolic blood pressure seems to decrease again. The turning down trend starts earlier for the diastolic blood pressure. This means also that the pulse pressure increases with age, which is in accordance with results from other studies.^{1,2,20} However, again, at upper

Table 6 Prevalence of pharmaceutical antihypertensive treatment in 1968–1969, 1980–1981 and 1992–1993, respectively

1968–1969		1980–1981		1992–1993	
Age	Prevalence	Age	Prevalence	Age	Prevalence
—	—	26	0 (0.0%)	38	1 (1.9%)
—	—	38	1 (0.8%)	50	8 (9.4%)
38	3 (0.8%)	50	26 (7.3%)	62	44 (16.4%)
46	14 (3.3%)	58	45 (13.7%)	70	69 (23.6%)
50	18 (4.5%)	62	64 (19.8%)	74	57 (27.8%)
54	19 (10.6%)	66	37 (27.0%)	78	25 (31.6%)
60	13 (16.0%)	72	21 (42.9%)	84	7 (41.2%)

ages the decrease in systolic blood pressure seems to be more pronounced than that of diastolic blood pressure, leading to a decrease also in pulse pressure. Despite age-related increments in pulse pressure and baseline associations between pulse pressure and BMI, changes in BMI over 12 and 24 y were not accompanied by changes in pulse pressure.

At the baseline examination there was a strong correlation between BMI and WHR and between hypertension and arterial blood pressure, after taking age into consideration.

The prospective part of our analysis showed that high BMI at baseline predicted risk of hypertension whereas WHR did not, during long time follow-up. On the other hand, the women increased their systolic, diastolic and pulse pressure at the same rate irrespective of their BMI and WHR values at baseline. This means that lean and obese women had similar blood pressure rise during the study period. The obese women were, however, more often diagnosed as hypertensive, probably because they had higher blood pressure levels at the start of the study, as mentioned above. However, increase in BMI correlated strongly with elevation in blood pressure and incidence of hypertension, even after adjusting for baseline BMI and WHR change.

WHR at baseline examination did not predict hypertension, but we found a statistically significant inverse association between change in WHR and change in diastolic blood pressure. This was unexpected, as previous reports from this study have shown a stronger association between WHR and cardiovascular morbidity and death, than between BMI and these end-points.¹⁴ This inversed association indicating a fall in diastolic blood pressure is most likely explained by increased arteriosclerosis in this group of women causing large artery stiffness. Haemodynamic studies have shown that the fall in diastolic blood pressure is most likely caused by such arterial stiffness.^{1,21}

Obesity is an increasing problem world-wide²² and it is therefore important to clarify its impact on various risk factors and morbidity. In this sample of middle aged women we found a strong influence of generalised obesity and weight gain on blood pressure. A previous report from this study showed a correlation between obesity parameters BMI and WHR, and raised fasting s-insulin.¹⁰ This is in line with other studies suggesting that total body fat is a major contributor to the metabolic sequelae of obesity, but deep subcutaneous and visceral adipose tissue also make a significant, but smaller contribution.¹¹ Several studies have demonstrated that weight changes in women correlate much less or not at all to their changes in WHR compared to men.^{23,24} Furthermore change in WHR did not relate to changes in metabolic factors.²⁵

Most of the large epidemiological studies with long time follow-up have been performed on males. The main strength of this study is its focus on women's health and the high participation rate at baseline and during all phases of the study^{16–19} guaranteed the participants were representative of women in Gothenburg in the age strata studied. Furthermore,

the organisation of the study and most of the blood pressure measurements were done by the same person (CB) initially and during the 24 y of follow-up.²⁶ Another strength is the long and thorough follow-up.¹⁹

A main weakness is the limited number of participants in the upper age groups. The fact that it is difficult to take the effect of blood pressure treatment into consideration when studying blood pressure development is also to some extent a problem. In order to minimise this problem, prevalence and incidence of hypertension, including pharmaceutical antihypertensive treatment as a criterion, have been included in the statistical analyses together with blood pressure and pulse pressure changes.

The main reason for non-participation at 12 and 24 y follow-up was death, and there was an increased death rate in those with basal hypertension.^{18,19} Further drop-out analysis comparing subjects who participated in the first two examinations with those who dropped out after the first showed a significant difference in basal systolic blood pressure in those who dropped out (higher values) compared to those who participated. We cannot exclude the fact that some selection bias has occurred since the first examination and that we may have lost proportionately more women with hypertension.

As a considerable number of statistical tests have been carried out in this study, the matter of 'multiple significance tests' must be taken into consideration. A reasonable approach is to consider other supporting or contradictory results in the final judgement.

We conclude that BMI and increase in BMI are strong predictors for hypertension and increased blood pressure in women. Pulse pressure increases with age, but change in the variables measuring obesity do not seem to influence pulse pressure. WHR as an independent risk factor plays an uncertain role in the development of hypertension in middle-aged women and seems to be of less importance than BMI with respect to blood pressure development.

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