Journal of the American College of Cardiology
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Published by Elsevier Science Inc.

Vol. 37, No. 5, 2001
ISSN 0735-1097/01/$20.00
PII S0735-1097(01)01166-4

Peripheral Vascular Disease

Comparative Effects of Aging in Men and Women on the Properties of the Arterial Tree

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OBJECTIVES We measured the properties of the arterial tree, seeking differences between men and women as they aged.

BACKGROUND There are many differences between men and women, besides menopause, which might account for such disparities. These include body height, heart rate, stroke volume and smaller arterial diameters. Any gender differences in arterial stiffness could influence pulse pressure (PP), now recognized as a cardiovascular risk factor.

METHODS A total of 530 patients (347 men and 183 women) were classified by age into quartiles: ≤40, 41–47, 48–54 and ≥55 years. The middle groups represented the menopausal years. Studies included brachial artery blood pressure (BP), aortic pulse wave velocity (PWV), B-mode ultrasonography and wave form analysis of the common carotid artery (CCA), with its conversion to the aortic wave form. Standard echocardiography provided left ventricular dimensions and flows. Calculated values included CCA compliance and distensibility, systemic compliance, stroke volume and peripheral resistance.

RESULTS At all ages, women had higher heart rates but lower BP than men. Pulse pressure, however, was lower in young women and higher in older women. Measurements influenced by body size, such as CCA diameter, compliance and systemic compliance, were lower in women. Those related to arterial wall properties, such as CCA and aortic distensibility, were the same. Although aortic PWV rose similarly with aging, PWV had more of an influence on PP in women than did mean BP. The reverse was true in men.

CONCLUSIONS Despite lower mean BP and similar arterial distensibility, women develop a higher degree of pulsatility with aging, as compared with men. This is mainly due to their smaller physical characteristics, independent of the role of menopause and its related hormonal changes. (J Am Coll Cardiol 2001;37:1374–80) © 2001 by the American College of Cardiology

It is well recognized that aging leads to progressive stiffening of the arterial tree in both men and women. However, multiple factors affecting the arterial tree differ between the two genders. Obviously, estrogenic hormone levels, believed to affect arterial distensibility (1), fluctuate widely in women as they relate to pregnancy and menopause (2,3), with no counterpart in men. However, there are also nonhormonal differences that affect the behavior of the arterial tree. These include discrepancies in body height, heart rate (HR), stroke volume, arterial compliance and distensibility, all of which influence systolic blood pressure (SBP), diastolic blood pressure (DBP) and pulse pressure (PP). This last one is important, because PP has recently been shown to be a predictor of cardiovascular risk in unselected populations (4–6), hypertensive patients (7) and those with myocardial infarctions or congestive heart failure, or both (8,9).

This report compares the influence of a number of nonhormonal factors that affect the arterial tree in men and women, by seeking gender and age differences in a large cohort with a wide age span. The results are statistically analyzed to provide insight into the simultaneous effects of gender and aging on the development of arterial stiffness in men and women.

METHODS

Study cohort. From January 1995 to June 1997, ~1,500 patients entered the Department of Internal Medicine of Broussais Hospital for a routine cardiovascular evaluation ordered by their general practitioner or cardiologist. These evaluations were requested on the basis of one or more risk factors, including high blood pressure, smoking, dyslipidemia, diabetes mellitus and/or a family history of premature cardiovascular disease, with or without previously identified atherosclerotic disease. From these 1,500 patients, 190 normotensive subjects and 340 subjects with essential hypertension agreed to be studied (n = 530). Hypertension was defined as SBP >140 mm Hg and/or DBP >90 mm Hg, measured on three occasions in the supine position by sphygmomanometry during the preceding month. Patients with all forms of secondary hypertension were excluded on the basis of standard laboratory and

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Manuscript received June 7, 2000; revised manuscript received November 15, 2000, accepted January 4, 2000.

Abbreviations and Acronyms

CCA	= common carotid artery
DBP	= diastolic blood pressure
HR	= heart rate
IMT	= intima-media thickness
LV	= left ventricle
MBP	= mean blood pressure
PP	= pulse pressure
PWV	= pulse wave velocity
SBP	= systolic blood pressure

radiology tests. Subjects with cancer, insulin-dependent diabetes or severe renal insufficiency (creatinine >120 μmol/liter) were also excluded. The study group was then composed of 236 hypertensive men, 104 hypertensive women, 111 normotensive men and 79 normotensive women. In all hypertensive subjects, pharmacologic therapy had been discontinued at least one month before the study. Subjects had no signs, symptoms or history of stroke or major cardiac or renal disease other than hypertension. No patient received antidiabetic or hypocholesterolemic drugs, hormone replacement therapy or any other cardiovascular drug during the study. Written, informed consent was obtained from each subject after a detailed description of the procedure.

Classification of subjects. In a previous study, 30,966 French women completed a health questionnaire about age, body weight and height and the presence or absence of menopause. Data thus derived showed that menopause occurred 100% of the time between the ages of 40 and 55 years. Men and women in the present study were therefore classified into four age groups: ≤40 years, 41–47 years, 48–54 years and ≥55 years. This age classification not only divided the cohort into nearly equal quarters, but allowed comparison of premenopausal and postmenopausal data (groups 1 and 4), as well as observations on the middle groups, whose number of menopausal women increased with advancing age.

Blood pressure and pulse wave velocity (PWV) measurements. Each subject was studied at 10:00 AM in a controlled environment at a room temperature of 22 ± 2°C. After 20 min of rest in a supine position, brachial SBP, DBP and mean blood pressure (MBP), PP and HR were determined every 3 min with an oscillometric recorder (model 845, Dinamap, Critikon, Tampa, Florida) placed on the left arm. Pulse wave velocity was then measured.

In all 530 subjects, aortic PWV was determined using an automatic device, the Complior (Colson, Garges les Gonesses, France), which allowed on-line pulse wave recording and automatic calculation of PWV. Common carotid artery (CCA) and femoral artery pressure wave forms were recorded noninvasively using a TY-306 Fukuda pressure-sensitive transducer (Fukuda, Tokyo, Japan). Measurement was repeated over 10 different cardiac cycles, and the mean value was used for the final analysis. The distance traveled by the pulse wave was measured over the body surface as the distance between the two recording sites (D), while pulse transit time (t) was automatically determined by the Complior. Pulse wave velocity was automatically calculated as PWV = D/t. Details, as well as validation of this automatic method and its reproducibility, have been reported previously, with intraobserver and interobserver repeatability coefficients >0.90 (10).

In 460 patients, ultrasonography of the heart and CCA was performed. Echocardiographic studies were performed using a Hewlett-Packard Sonos 1000 device equipped with a 2.25-MHz probe, allowing M-mode, two-dimensional and pulsed wave Doppler measurements. Left ventricular (LV) outflow and aortic velocities were recorded from the apical position. Measurements were performed by two physicians according to the methods described by the American Society of Echocardiography (11). Techniques, as well as interobserver and intraobserver reproducibilities, have been reported elsewhere (12). The mass of the LV was calculated according to the Penn convention (13). Stroke volume was calculated from the LV outflow velocity integral (cm/s) and aortic outflow area, and cardiac output was calculated as the product of stroke volume and HR.

The pressure wave of the CCA was recorded noninvasively with a pencil-type probe, using a high fidelity Millar strain gain transducer (SPT-301, Millar Instruments, Houston, Texas). The pressure wave form and amplitude of the CCA were recorded by applanation tonometry and converted to a calculated aortic wave form and amplitude, using the Sphygmograph (PWV, Sydney, Australia) and the generalized transfer function (14). The carotid pulses were calibrated assuming that the MBP and DBP, as measured by oscillometry of the brachial artery (vide supra), were the same. The time constant of the last two-thirds of diastolic decay was calculated from the derived aortic pressure curve, as previously reported (15). The diastolic diameter of the CCA was measured by a high resolution B-mode (7.5-MHz transducer) echo-tracking system (Wall-Track System). A complete detailed description of this system, as well as the reproducibility of the measurements, has been published previously (16). The accuracy of the system is

Table 1. Population Description

	Men (n = 347)	Women (n = 183)
Age (yrs) (p = NS)	48.2 ± 10.6	49.9 ± 11.5
Height (cm) (p < 0.0001)	174 ± 7	162 ± 6
Weight (kg) (p < 0.0001)	78.9 ± 11.6	63.5 ± 11.6
SBP (mm Hg) (p = 0.02)	146.41 ± 18.62	142.27 ± 22.16
DBP (mm Hg) (p < 0.0001)	90.21 ± 12.73	85.36 ± 13.38
MBP (mm Hg) (p = 0.002)	109.7 ± 14.17	104.93 ± 15.49
Heart rate (beats/min) (p = 0.02)	68.64 ± 11.62	71.23 ± 12.27
Pulse pressure (mm Hg) (p = NS)	56.20 ± 11.12	56.91 ± 15.01
PWV (m/s) (p = NS)	11.56 ± 2.97	11.21 ± 3.23

Data are presented as the mean value ± SD.
DBP = diastolic blood pressure; MBP = mean blood pressure; PWV = pulse wave velocity; SBP = systolic blood pressure.

JACC Vol. 37, No. 5, 2001
April 2001:1374–80

Table 2. Gender and Age Comparisons

A.	Women		Men	
Age Group	Mean (± SD) Age	HTN	Mean (± SD) Age	HTN
≤40 years	32 ± 6	37%	35 ± 6	58%
41–47 years	44 ± 2	64%	46 ± 2	70%
48–54 years	50 ± 2	61%	51 ± 2	67%
≥55 years	61 ± 5	65%	61 ± 5	76%

B.	Height (cm)			
p < 0.0001 (gender)	Women		Men	
p = 0.001 (age)	n	Mean ± SD	n	Mean ± SD
≤40 years	45	163 ± 6	74	177 ± 6
41–47 years	47	162 ± 7	87	174 ± 8
48–54 years	44	162 ± 7	90	174 ± 7
≥55 years	45	160 ± 6	88	172 ± 7
	Weight (kg)			
p < 0.0001 (gender)	Women		Men	
p = 0.09 (age)	n	Mean ± SD	n	Mean ± SD
≤40 years	45	61.7 ± 12.4	74	77.4 ± 0.38
41–47 years	47	64.6 ± 12.4	87	79.9 ± 0.35
48–54 years	44	66.0 ± 11.3	90	79.5 ± 0.34
≥55 years	45	61.6 ± 9.7	88	78.5 ± 0.35

C.	MBP dinamap (mm Hg)			
p = 0.00007 (gender)	Women		Men	
p = 0.0002 (age)	n	Mean ± SD	n	Mean ± SD
≤40 years	46	97.82 ± 2.21	75	103.91 ± 1.60
41–47 years	45	107.11 ± 2.24	85	110.89 ± 1.51
48–54 years	41	107.22 ± 2.35	91	108.64 ± 1.46
≥55 years	43	108.08 ± 2.29	89	112.12 ± 1.47
	Heart Rate (beats/min)			
p = 0.02 (gender)	Women		Men	
p = NS (age)	n	Mean ± SD	n	Mean ± SD
≤40 years	46	72.7 ± 11.2	74	68.4 ± 11.6
41–47 years	45	71.4 ± 13.6	87	67.3 ± 12.0
48 ± 54 years	41	70.1 ± 10.1	90	68.0 ± 11.8
≥55 years	43	70.6 ± 13.9	88	70.6 ± 11.0

Interactions between gender and age were not significant for the data presented throughout this Table.
HTN = hypertension; MBP = mean blood pressure.

±30 μm for the carotid diameter. The repeatability coefficient of the measurements is ±0.273 mm. Measurements were carried out in the right CCA, 2 cm beneath the bifurcation. The intima-media thickness (IMT) of the CCA was measured on the far wall at the same level as the diameter measurements, with computer-assisted acquisition, processing and storage. The repeatability coefficient of the IMT measurement was ±60 μm. Compliance and distensibility of the CCA were determined from changes in the inner CCA diameter during systole (Ds − Dd; where Ds is the CCA diameter at end systole and Dd is the diameter at end diastole) and the simultaneously measured PP (ΔP) of the CCA, using the following formulas:

$$\text{CCA compliance} = (\pi \text{Dd}\,[\text{Ds}-\text{Dd}]/2)/\Delta P\ (\text{m}^2 \cdot \text{kPa}^{-1} \cdot 10^{-7})$$

$$\text{CCA distensibility} = 2([\text{Ds} - \text{Dd}/\text{Dd}])/\Delta P\ (\text{kPa}^{-1} \cdot 10^{-3})$$

The repeatability coefficients of the measurement were $\pm 1\ \text{kPa}^{-1} \cdot 10^{-3}$ for CCA distensibility and $0.52\ \text{m}^2 \cdot \text{kPa}^{-1} \cdot 10^{-7}$ for CCA compliance.

Statistical analysis. Statistical analysis was carried out using the NCSS program, release 6.0.21 (©1996 by Jerry Hintze). Covariance analyses used SPSS program, release 4.0.3 (1990) (Chicago, Illinois). As described earlier, we classified all of the study subjects into four age groups, as defined by approximate quartiles of the age distribution. We compared mean values for men and women by using the *t*

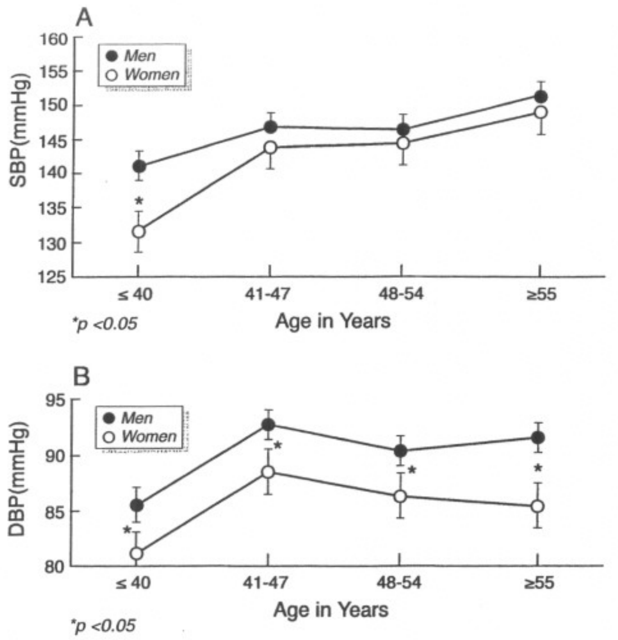

Figure 1. **(A)** Systolic blood pressure (SBP) by gender ($p = 0.03$) and evolution by age ($p < 0.001$). **(B)** Diastolic blood pressure (DBP) by gender ($p < 0.01$) and evolution by age ($p < 0.001$). Pairwise comparisons were made by using a Bonferroni test.

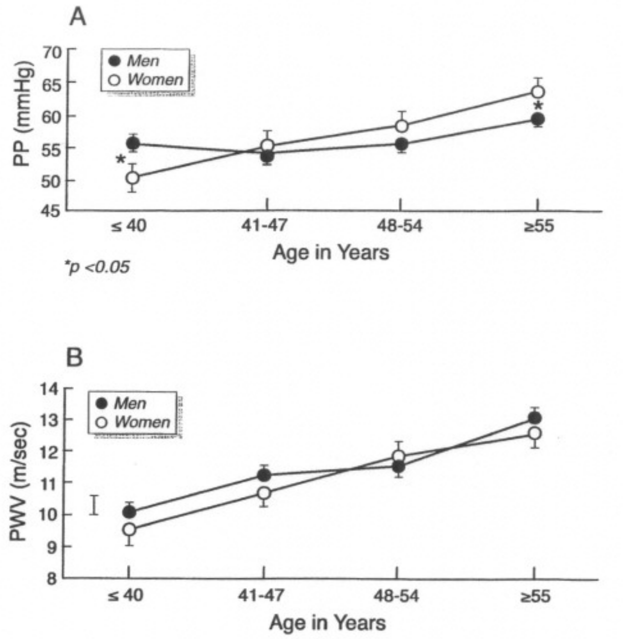

Figure 2. **(A)** Pulse pressure (PP) by gender (p = NS), evolution by age ($p < 0.001$) and age-gender interaction ($p = 0.02$). **(B)** Pulse wave velocity (PWV) by gender (p = NS) and evolution by age ($p < 0.001$). Pairwise comparisons were made by using a Bonferroni test.

test at a nominal two-tailed level of significance ($p = 0.05$), whereas differences between mean values for the factors of age and gender were evaluated by analysis of variance *F* tests, also at $p = 0.05$. In conjunction with testing whether the regression slopes of PP on PWV differed between men and women in an age class, we used age values within the class as a covariate. We explored relationships between explicative variables and PP by using stepwise multiple regression, with the two-tailed partial p value <0.05 as the criterion for variable selection.

RESULTS

The data in Table 1 compare the measured variables for men and women in the overall population. Although the mean ages were the same, HR was higher in women ($p = 0.02$), but body weight, height, SBP, DBP and MBP were significantly lower. These results were consistent in the subgroups in which carotid and cardiac studies were performed. Arterial distensibility, as measured by PWV, was the same for both genders.

In Table 2 (A), the mean ages and proportion of hypertensive patients for each group are listed. Table 2 (B and C) and Figures 1 and 2 compare the measured variables from Table 1 for the different age groups in men and women. The following values increased with age in both men and women: SBP, DBP, MBP, PP and PWV. In women, body weight increased initially, then decreased over age 55 years (group 4). Body height decreased with age for both genders. There were gender differences at all ages for body weight and height, DBP and MBP, all of which were lower in women. In contrast, HR was higher in women, except for those >55 years old. Figure 1 shows that, whereas DBP was lower in women than in men in all age groups, SBP was lower in women than in men <40 years of age ($p < 0.05$), but reached the same level above this age. As a consequence, as seen in Figure 2 (upper panel), PP was lower ($p < 0.05$) in women than in men <40 years old and higher in women than in men >55 years old ($p < 0.05$).

Figure 2 (lower panel) indicates that PWV increased with age similarly in men and women. Figure 3 shows that the regression slopes of the relationship between PP (*y* axis) and PWV (*x* axis) are higher in women than in men <47 years old ($p < 0.05$; $p < 0.01$) and then became independent of gender beyond this age.

The statistically significant independent variables in multiple regression analysis, with PP as the dependent variable, are shown in Table 3. Statistically insignificant variables, not shown in Table 3, include height, weight, gender and age. In the overall population (Table 3, A), PP was most

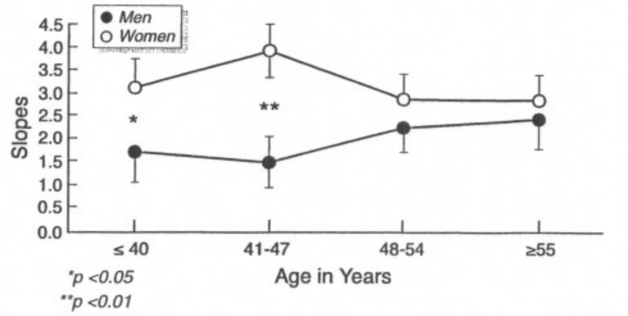

Figure 3. Slopes of regression between PP (*y* axis) and PWV (*x* axis) by gender and evolution by age. Pairwise comparisons were made by using a Bonferroni test.

Table 3. Linear Multiple Regression Analysis of Pulse Pressure as Dependent Variable

	Regression Coefficient ± SE	p Value	Correlation Coefficient	r^2 (variance part)
A. Global				
PWV (m/s)	1.57 ± 0.17	<0.001	0.38	0.113
MBP (mm Hg)	0.28 ± 0.04	<0.001	0.33	0.083
Weight (kg)	−0.10 ± 0.03	0.004	−0.11	0.011
Heart rate (beats/min)	−0.11 ± 0.04	0.005	−0.10	0.010
B. Men				
MBP (mm Hg)	0.29 ± 0.04	<0.001	0.37	0.133
PWV (m/s)	1.18 ± 0.21	<0.001	0.30	0.091
Heart rate (beats/min)	−0.12 ± 0.05	0.007	−0.13	0.021
C. Women				
PWV (m/s)	1.98 ± 0.34	<0.001	0.43	0.170
MBP (mm Hg)	0.26 ± 0.07	0.0002	0.27	0.081

MBP = mean blood pressure; PWV = pulse wave velocity.

influenced by MBP and PWV, but there was a small, negative role for body weight and HR. In men (Table 3, B), the contribution of both MBP and PWV to the global variance was 22%, similar to the 25% rate in women (Table 3, C). However, the contribution of PWV alone was 17% in women and 9% in men, whereas the contribution of MBP was 8% in women and 13% in men. This illustrates the reduced influence of MBP on compliance and pulsatility in women as compared with men. Finally, the contribution of HR to the variance of PP was observed in men, but not in women.

Table 4 summarizes the findings of carotid, aortic and cardiac variables. Men and women had similar values of the following CCA variables: IMT, wall to lumen ratio, PP and distensibility. In contrast, CCA diameter, distention and compliance were lower in women. With regard to the calculated aortic variables, the slope of the calculated aortic diastolic pressure decay in women was higher and the systemic compliance lower than that in men. This confirmed the compliance data in the CCA.

DISCUSSION

In this study, we sought to define the role of factors, not directly hormone-related, that could explain the differences in circulatory function between men and women. Our interest focused on factors that influence pulsatility, because PP has recently been recognized as a cardiovascular risk factor in diverse clinical populations. To do so, we examined a number of variables that describe the function of the central circulation and the peripheral arterial tree in a cohort of men and women of widely diverse ages. These data were analyzed to detect similarities and differences between the genders in how the arterial tree ages and to determine how these differences influence PP.

Comparisons between men and women of all ages. With regard to the entire patient group, women had a smaller stature and a faster HR as compared with men. This is true at all ages. A smaller body height has been previously described as a cardiovascular risk, because of the early return

Table 4. Arterial and Cardiac Variables

	Men (n = 265)	Women (n = 195)	p Value*
CCA variables			
Pulse pressure (mm Hg)	48 ± 15	53 ± 19	NS
Diastolic diameter (mm)	7.3 ± 1.0	6.8 ± 0.7	<0.01
Distension (%)	7.2 ± 2.5	6.0 ± 1.4	<0.05
Intima-media thickness (μm)	712 ± 103	678 ± 103	NS
Wall/lumen (ratio)	0.25 ± 0.03	0.25 ± 0.04	NS
Compliance ($m^2 \cdot kPa^{-1} \cdot 10^{-7}$)	7.4 ± 2.9	5.7 ± 2.3	<0.001
Distensibility ($kPa^{-1} \cdot 10^{-3}$)	17.8 ± 8.7	15.6 ± 7.1	NS
Calculated aortic variables			
Diastolic slope (s^{-1})	0.537 ± 0.011	0.596 ± 0.01	<0.01
Systemic compliance (ml/mm Hg)	2.00 ± 0.79	1.66 ± 0.70	<0.01
Cardiac variables			
End-diastolic LV diameter (mm)	5.2 ± 0.4	4.8 ± 0.4	<0.001
Ejection fraction (%)	65.3 ± 6.1	72.1 ± 6.1	<0.001
Cardiac output (ml/min)	5,586 ± 1,231	5,056 ± 1,314	NS
Aortic VTI (cm/beat)	20.6 ± 4.2	22.8 ± 3.8	<0.05
Aortic valve opening diameter (mm)	1.98 ± 0.18	1.74 ± 0.19	<0.001
LV mass index (g/m^2)	116 ± 31	95 ± 15	<0.001
Stroke volume (ml)	63.2 ± 15.8	54.2 ± 12.7	<0.05
Total peripheral resistance ($dynes \cdot s \cdot cm^{-5}$)	3,026 ± 850	2,858 ± 703	NS

*By the Student *t* test. Data are presented as the mean value ± SD.
CCA = common carotid artery; LV = left ventricular; VTI = velocity-time integral.

of reflected waves to the central aorta in systole rather than diastole (17), and could therefore help to explain the increased PP in older women as compared with older men. A shorter stature also implies reduced length of the arterial tree, a factor believed to be responsible for a faster HR (18), a shorter diastolic period, a shorter diastolic time constant and, at the same peripheral resistance, lower arterial compliance.

As expected, because of their smaller size, women's CCA dimensions, CCA compliance and calculated systemic compliance are all significantly lower than those in men. However, CCA distensibility, which better reflects the properties of the arterial wall as a material, is no different between men and women. This is supported by the similarity in CCA wall thickness and PWV, confirming a recent report (19). Furthermore, although the CCA diameters were smaller in women, the CCA wall/lumen ratios were identical in men and women, indicating similar values for Young's modulus. The importance of reduced body height in women was stressed by London et al. (20), who demonstrated an increased CCA augmentation index in women, as well as the differential effects on peripheral and central blood pressures with aging in men and women. Hayward and Kelly (21) also found similar differences in the CCA wave form between men and women, and ascribed them to the differences in body height. Other values reflecting a smaller body size in women than in men include smaller LV diameter, stroke volume, cardiac output, aortic outflow diameter and LV mass. Together, these findings imply that the process of aging on the arterial wall is similar in men and women, and that the differences observed in the arterial tree have more to do with diameter and length than with hormonal changes.

The physiologic effects of a smaller body size in women is a hemodynamic liability that is life long. This phenomenon of decreased compliance in women with similar distensibility, as compared with men, becomes more important as PWV increases secondary to arterial stiffening from advancing age. Reduced arterial compliance explains the lower DBP in women in all age groups. The lower SBP in young women may relate to their lower stroke volume, but could also be due to greater amplification of SBP in the brachial artery of young men, due to their greater body height (17,20). However, with progressive aging and associated arterial stiffening, women's SBP rises to that of men.

Comparisons between men and women according to age groups. The different changes in SBP and DBP with aging of men and women produce a lower PP in young women, but a higher PP in older women. In contrast, PP in men rose less with aging, probably due to greater amplification of the pulse in men <40 years old (vide supra). In a recent repeat analysis of the Framingham data, Franklin et al. (22) pointed out that the gender differences of PP as a coronary heart disease risk factor are most striking in those <40 years old. In young males only, there was an inverse relationship between PP and risk, but the risk of increased PP rose in both men and women as they aged. Therefore, it appears from our data that DBP in women is a major determinant of PP throughout life, whereas SBP only becomes important in this regard after menopause and with aging. Together, this information and our data on PP, when used to evaluate cardiovascular risk in men and women, explain the different mechanisms for lower risk at a young age in women and men, and the reversal of that trend with aging.

Those measured values that differ between men and women and change the most during the middle years may be suspected of having a menopausal influence. Figure 3 shows that aortic PWV, a surrogate for distensibility, is more important in predicting PP in young women than in young men. The difference persists between ages 41 and 47 years, but is lost at higher ages. This finding is supported by the multiple regression data, which demonstrate that the influence of MBP and PWV on PP are different between men and women. In men, a higher MBP is more predictive than PWV of the variations in PP, whereas the reverse is true in women. If these speculations are correct, there is no need to invoke the effect of hormonal changes in women with aging to explain the findings.

Effects of menopause. The published evidence supports the unfavorable effects of a lack of estrogen due to menopause, as well as the favorable circulatory effects of administered estrogen (23–26). The presence of circulating estrogen in young women could account, in part, for their lower SBP and PP. However, the circulatory effects of estrogen are complex, not only involving the endothelium but also raising angiotensinogen levels while lowering renin levels, with possible opposing effects on atrial natriuretic peptides (27,28). The question remains whether the gradual loss of estrogenic function due to menopause translates into brachial arterial blood pressure changes, which can be used to predict cardiovascular risk. This is difficult to demonstrate, because the menopausal changes must somehow be separated from the effects of aging. Faced with these and other problems, Casiglia et al. (29) were unable to demonstrate an effect of menopause, apart from aging, on blood pressure. In contrast, Staessen et al. (30–32) demonstrated an increased rate of rise in SBP versus age in women versus men >50 years old. This was found in both cross-sectional and longitudinal analyses. However, even this group of investigators was cautious in ascribing the findings to the hormonal effects of menopause alone.

Study limitations. The present study suffers from some of the same limitations as many other previous studies. Our study group includes more hypertensive than normotensive subjects and assumes that the process of aging is similar for both. Therefore, these data may not be applicable to the general population, less of whom are hypertensive. The present study has a cross-sectional design, but uses men as comparisons rather than control subjects. It does provide information on many previously uncorrelated factors (i.e., CCA compliance and distensibility, systemic arterial compliance, cardiac output, stroke volume, ejection fraction, peripheral vascular resistance and aortic PWV), many of which are related to aging.

Conclusions. It is clear from the published data that estrogen levels affect some areas of the arterial tree, such as the aorta, endothelium and microvasculature. However, it is likely that the menopausal effects on the arterial tree of women who are aging, though present, are sufficiently small and gradual so as to be nearly impossible to distinguish from the concomitant effects of aging alone. At a given LV ejection fraction, the major determinants of PP are MBP and arterial distensibility. Despite the same distensibility and lower MBP, women, as compared with men, develop a greater degree of pulsatility with aging. Therefore, their increased pulsatility is largely influenced by their smaller body size and arterial dimensions, independent of menopause and its related hormonal changes.

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