Prognostic Significance of Left Atrial Enlargement in a General Population
Results of the PAMELA Study

Michele Bombelli, Rita Facchetti, Cesare Cuspidi, Paolo Villa, Dario Dozio, Gianmaria Brambilla, Guido Grassi, Giuseppe Mancia

Abstract—We estimated the risk of cardiovascular events, cardiovascular mortality, and all-cause mortality associated with left atrium (LA) enlargement alone or combined with echocardiographic left ventricular hypertrophy (LVH) in 1785 representatives of the general population of Monza recruited for the Pressioni Arteriose Monitorate E Loro Associazioni (PAMELA) study. LA enlargement was assessed by measuring LA diameter via echocardiography. LA enlargement was defined as a LA diameter ≥2.3 cm/m², whereas LVH was defined as a left ventricular mass index ≥114 g/m² and 99 g/m² in men and women, respectively. Death certificates and hospital diagnoses were collected over an average 148 months follow-up. During follow-up, there were 175 deaths (of which 59 for cardiovascular causes) and 139 cardiovascular fatal and nonfatal events. Compared with subjects with neither LA enlargement nor LVH, subjects with isolated LA enlargement exhibited a significant increase in the adjusted risk of combined fatal and nonfatal cardiovascular events (hazard ratio, 2.0; confidence interval, 1–4.1; P=0.04), although not of cardiovascular death or all-cause death. The adjusted (for baseline covariates, including ambulatory blood pressure) risk of fatal and nonfatal cardiovascular events, cardiovascular death, and all-cause death was significantly increased also in subjects with isolated LVH (hazard ratio, 2.2, 3.4, 2.1, respectively; P=0.001 for all), whereas no further increase was seen in subjects with both LA and left ventricular abnormalities. Thus, like LVH, LA enlargement is an independent long-term predictor of cardiovascular events. The cardiovascular risk, however, is not further increased when LA enlargement is superimposed on an increase of LV mass. (Hypertension. 2014;64:1205-1211.) • Online Data Supplement

Key Word: heart atria

Several studies have shown that alterations in left ventricular (LV) structure and function, such as an increased LV mass (LVM), a reduction of systolic contractility, and an impairment of LV diastolic function, have an adverse prognostic value (ie, that they are associated with an increased risk of cardiovascular morbid and fatal events, independently on other cardiovascular risk factors).1,2 More recently, evidence has been obtained that this is the case also for anatomic alterations of the left atrium (LA), the enlargement of which has been found to predict independently the development of heart failure,3 and other cardiovascular events.4,5 LA enlargement, however, frequently occurs with an increased LVM or a frank LV hypertrophy (LVH),6,7 both changes being commonly generated by an increase in blood pressure (BP),8 and limited information exists on whether LA enlargement increases cardiovascular risk also when associated with or superimposed on LV structural changes.

This has been the aim of the present study, which has examined the long-term risk of cardiovascular morbid and fatal events (as well as of all-cause mortality) in a population in which LA enlargement and LV hypertrophy were present in isolation or combination, their combined absence representing the control group. A strength of the study is that BP was measured in and outside the physician’s office, which allowed to correct the data for the effect of BP elevation on cardiovascular risk more accurately than by using office BP.9

Methods

Subjects and Measurements

The details of the Pressioni Arteriose Monitorate E Loro Associazioni (PAMELA) study have been reported elsewhere.10 Briefly, the study was performed in 1990 to 1991 on 3200 individuals aged between 25 and 74 years, who were selected to be representative of the Monza population, based on the criteria of the World Health Organization Monitoring Diseases (WHO-MONICA) project performed in the same geographic area.11 The participation rate was 64%, and the demographic characteristics and medical history of nonparticipants were similar to those of participants. All subjects underwent a
medical visit at the outpatient service of the local University Hospital (San Gerardo) where the following data were obtained: (1) 3 systolic BP (SBP) and diastolic BP values by a mercury sphygmomanometer, with the subjects in the sitting position since ≥25 minutes; (2) a heart rate value (palpatory method) after each BP measurement; (3) subject’s height and weight, which allowed to calculate body mass index by the ratio of weight and height squared to body surface area; (4) 24-hour ambulatory BP by a device (SpaceLabs 90207) set to read BP and heart rate values every 20 minutes; (5) 2 home BP and heart rate values by a validated semiautomatic device (Philips Model HP 5331), approximately at 7.00 AM and at 7.00 PM. The ambulatory BP measuring device was applied in the morning, and the subjects were instructed to attend at their usual activities (only avoiding strenuous exercise) to keep the arm extended at the time of the BP measurements and to return to hospital for the device removal the following day; and (6) serum glucose, total cholesterol, high-density lipoprotein cholesterol, and triglycerides by a blood sample withdrawn from the antecubital vein.

Echocardiographic Variables

Echocardiographic variables were obtained, as previously reported. In brief, M-mode and 2-dimensional echocardiographic images were obtained by a commercially available instrument (Acuson 128 CF, computer sonography). End-diastolic and end-systolic LV internal diameters, interventricular septum thickness, and posterior wall thickness were measured from 2-dimensionally guided M-mode tracings recorded at 50 to 100 cm/s speed during ≥3 consecutive cycles according to the requirements of the American Society of Echocardiography. Relative wall thickness was defined by the ratio of posterior wall plus interventricular septum thickness to LV internal diastolic diameter. LVM was estimated by the corrected American Society of Echocardiography method and indexed for the body surface area. LA diameter was assessed by the parasternal long axis view, using a leading edge to leading edge measurement of the maximal distance between the posterior aortic root and the posterior LA wall at end systole. Two skilled operators were involved in the collection of echocardiographic tracings, which were read off-line by a third person. LVH was defined as a Left Ventricular Mass Index ≥144 g/m² in men and 99 g/m² in women. These cutoff values were derived from sex-specific upper limits of normality (mean+1.96 SD) for LVM index in 675 healthy individuals with documented in and out of office heart rates). Most correlations retained statistical significance when adjusted for age and sex (Table 2, right part), whereas fewer exhibited an independent relationship with

Results

Table 1 shows the demographic, clinical, and echocardiographic variables in 1785 subjects in whom the full set of data was available. Mean age was 50.6 years, male prevalence 50.9%, and average LA diameter 3.5±0.5 cm, with a higher value in males than in females (P<0.0001) and a near normal distribution in both sexes and the whole population (Figure 1). As shown in Table 2, left part, in the univariate analysis, LA diameter exhibited a significant direct relationship with several variables (age, in and out of office BP, LVM, body mass index and serum total cholesterol, triglycerides, and glucose) while showing a significant inverse relationship with few others (serum high-density lipoprotein cholesterol and in and out of office heart rates). Most correlations retained statistical significance when adjusted for age and sex (Table 2, right part), whereas fewer exhibited an independent relationship with
LA diameter in the multivariable analysis, the most important being body mass index, followed in descending order of importance by LVM, age, male sex, and 24-hour heart rate (negative relationship; Table 3).

The relationship between LA diameter and morbid or fatal events is shown in Figures 2 to 4, as well as in Table S1 in the online-only Data Supplement. Figure 2 shows that quartiles of increasing LA diameter were accompanied by a progressive increase in the incidence of cardiovascular death (n=59), all-cause death (n=175), and fatal or nonfatal cardiovascular events (n=139), as well as of cause-specific events such as stroke (n=47), coronary events (n=57), heart failure (n=23), and cardiovascular events of other nature (n=12; Table S1). The incidence of all 3 types of events increased progressively from subjects with neither LA enlargement nor LVH, to the ones with only LA enlargement, those with only LVH, and those with both LA enlargement and LVH (Figure 3). As shown in Figure 4, compared with individuals with neither LA enlargement nor LVH, the fully adjusted (see Methods) risk of events exhibited an increase in subjects with only LA enlargement, although the difference was statistically significant only for the fatal and nonfatal cardiovascular events together. The risk was more markedly and consistently increased in subjects with only LVH (in whom the difference was statistically significant for all events), whereas no further increase occurred when the 2 echocardiographic alterations were present in combination.

Discussion

Our study provides several findings of interest. (1) LA diameter predicted the long-term incidence and risk of fatal and nonfatal cardiovascular events, independently on the role played by several other well-known cardiovascular risk contributors. (2) The increase of risk associated with LA enlargement was somewhat less important than that associated with LVH, which was demonstrable not only for combined fatal and nonfatal cardiovascular events but also for fatal cardiovascular events or all-cause deaths alone. (3) The 2 cardiac structural alterations did not have an additive effect on cardiovascular risk because in subjects in whom they were both present, risk was increased to a degree that was not significantly greater than that seen when only LA enlargement or LVH was present. This confirms previous findings on the adverse prognostic importance of LA structural alterations with an extension to a follow-up period (12 years) longer than that available in previous studies. It also shows that the adverse prognostic role played by LA enlargement has upper limit (ie, it does not further contribute to cardiovascular risk when superimposed to structural alterations of the LV ventricle, such as LVH).
Our study provides detailed information on the behavior of LA structure in a general population. (1) In the overall population, as well as in males and females, LA diameter exhibited a normal distribution, which means that, as for many other biological variables, use of cutoff values to distinguish abnormality from normality is arbitrary. (2) As reported in previous studies,22,23 LA diameter showed a direct or inverse relationship with several factors (age, sex, in and out of office BP, and heart rate) and metabolic alterations, which means that its detection should alert physicians on the likelihood of a multifactorial cardiovascular risk profile. (3) The negative relationship between LA diameter and heart rate (presumably because of the dependence of atrial dimension on the duration of the diastolic phase and the atrial filling) has a specific methodological relevance because it implies that comparisons of atrial diameter between individuals or at different times within individuals should be preferably done at similar heart rate values. (4) Alterations of LA or LV structure were detected in ≈19% (n=323) of the subjects, with LVH accounting for ≈3 quarter (75.2%) of the cases. An isolated LA enlargement, however, was by no means rare because it affected about a quarter (24.7%) of the individuals with any cardiac structure alteration. Given its adverse prognostic significance, this means that echocardiographic examinations must include an assessment of LA and that if this is not done, a high cardiovascular risk condition will be missed in several subjects (based on our study ≈1 of 21). (5) It is widely believed that whenever LVH is present, alterations of diastolic function and intracardiac hemodynamics make frank LA structural abnormalities almost unavoidable.24 However, this is not in line with our present findings that in the 243 subjects who exhibited LVH, only 41 (16.9%) had a concomitant LA enlargement. This may depend on the fact that (1) in presence of LVH, the increase of LA often remains below the cutoff value used in our study

**Table 2. Correlation Coefficients Between Left Atrium and Variables**

<table>
<thead>
<tr>
<th>Variables</th>
<th>r</th>
<th>P Value</th>
<th>Adjusted r</th>
<th>P Value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>0.321</td>
<td>&lt;0.0001</td>
<td>...</td>
<td></td>
</tr>
<tr>
<td>Body mass index, kg/m²²</td>
<td>0.478</td>
<td>&lt;0.0001</td>
<td>0.416</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Relative wall thickness, cm</td>
<td>0.11</td>
<td>&lt;0.0001</td>
<td>−0.013</td>
<td>0.584</td>
</tr>
<tr>
<td>Left ventricular mass, g</td>
<td>0.542</td>
<td>&lt;0.0001</td>
<td>0.406</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Serum glucose, mg/dL</td>
<td>0.189</td>
<td>&lt;0.0001</td>
<td>0.093</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Total cholesterol, mg/dL</td>
<td>0.103</td>
<td>&lt;0.0001</td>
<td>0.027</td>
<td>0.249</td>
</tr>
<tr>
<td>Triglycerides, mg/dL</td>
<td>0.213</td>
<td>&lt;0.0001</td>
<td>0.109</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>HDL cholesterol, mg/dL</td>
<td>−0.275</td>
<td>&lt;0.0001</td>
<td>−0.141</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Office systolic blood pressure, mmHg</td>
<td>0.348</td>
<td>&lt;0.0001</td>
<td>0.182</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Office diastolic blood pressure, mmHg</td>
<td>0.317</td>
<td>&lt;0.0001</td>
<td>0.181</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Office heart rate, beats/min</td>
<td>−0.095</td>
<td>&lt;0.0001</td>
<td>−0.033</td>
<td>0.159</td>
</tr>
<tr>
<td>Home systolic blood pressure, mmHg</td>
<td>0.365</td>
<td>&lt;0.0001</td>
<td>0.17</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Home diastolic blood pressure, mmHg</td>
<td>0.295</td>
<td>&lt;0.0001</td>
<td>0.161</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Home heart rate, beats/min</td>
<td>−0.135</td>
<td>&lt;0.0001</td>
<td>−0.035</td>
<td>0.161</td>
</tr>
<tr>
<td>24-h systolic blood pressure, mmHg</td>
<td>0.309</td>
<td>&lt;0.0001</td>
<td>0.158</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>24-h diastolic blood pressure, mmHg</td>
<td>0.233</td>
<td>&lt;0.0001</td>
<td>0.01</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>24-h heart rate, beats/min</td>
<td>−0.176</td>
<td>&lt;0.0001</td>
<td>−0.055</td>
<td>0.020</td>
</tr>
</tbody>
</table>

HDL indicates high-density lipoprotein. *Adjustment for age and sex high-density lipoprotein.

**Table 3. Multivariable Analysis**

<table>
<thead>
<tr>
<th>Variables</th>
<th>β</th>
<th>SE</th>
<th>P Value</th>
<th>R²adj</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>2.16173</td>
<td>0.1026</td>
<td>&lt;0.0001</td>
<td></td>
</tr>
<tr>
<td>Body mass index, kg/m²²</td>
<td>0.03201</td>
<td>0.00241</td>
<td>&lt;0.0001</td>
<td>0.0634</td>
</tr>
<tr>
<td>Left ventricular mass, g</td>
<td>0.00304</td>
<td>0.000258</td>
<td>&lt;0.0001</td>
<td>0.0499</td>
</tr>
<tr>
<td>Age, y</td>
<td>0.00462</td>
<td>0.000688</td>
<td>&lt;0.0001</td>
<td>0.0163</td>
</tr>
<tr>
<td>Sex (0=F;1=M)</td>
<td>0.12495</td>
<td>0.02055</td>
<td>&lt;0.0001</td>
<td>0.0133</td>
</tr>
<tr>
<td>Heart rate 24 h, beats/min</td>
<td>−0.00314</td>
<td>0.00107</td>
<td>0.0034</td>
<td>0.0031</td>
</tr>
</tbody>
</table>

R²adj = 0.3975

Sex, age, body mass index, left ventricular mass, blood glucose, serum triglycerides and HDL cholesterol, office systolic blood pressure, and 24-hour heart rate were the independent variables, whereas left atrium was the dependent variable. F indicates female; M, male; R²adj, adjusted R squared is a corrected goodness-of-fit (model accuracy) measure for linear models. It identifies the percentage of variance in the target field that is explained by the input or inputs; and R²adj, squared semipartial correlation coefficients computed using type II sums of squares.
define LA enlargement and that (2) only marked increases of LV mass are more consistently associated with LA dilatation. In this context, it should be mentioned, however, that this may have a limited practical importance because the superimposition of LA enlargement to LVH does not seem to worsen patient’s risk compared with the LVH alteration alone.

Our study has strengths and limitations. Strengths are unquestionably that (1) the study had long follow-up, (2) there were almost no subjects with atrial fibrillation (ie, a factor that modifies atrial volume and directly contributes to the risk of stroke, thereby acting as confounder), and (3) data collection included both home and ambulatory BP. Because these pressures are more closely associated with cardiac structural alterations and cardiovascular events than office BP,20,25 adjustment for their values allowed to determine the role of LA diameter as an independent risk factor more stringently than in previous studies. The limitations consist in (1) the limited number of cardiovascular morbid and fatal events exhibited by our population and (2) the fact that LA size was defined by a single measure and it was based on diameter rather than on tridimensional imaging, with thus potential errors in volume determination. However, the adverse prognostic role of LA enlargement was seen also when the analyses focused on the considerably greater number of all-cause deaths. Furthermore, regardless the potential inaccuracies of our LA volume determination, our data show that information of prognostic relevance can be obtained by a simple echocardiographic assessment of LA dimension.

**Perspectives**

Our prospective population-based study shows that, in absence of LVH, an increase of LA diameter is a predictor of combined fatal and nonfatal cardiovascular events, independently of other
Do not further increase cardiovascular risk when LVH is abnormal. It also shows, however, that LA enlargement, isolated left ventricular hypertrophy, and with both left atrial enlargement and left ventricular hypertrophy. The reference group is represented by subjects without left atrial enlargement and left ventricular hypertrophy. Data are shown unadjusted (left), and sex and adjusted (middle), and adjusted for history of CV events, body mass index, serum total cholesterol, blood glucose, smoking, 24-hour average systolic blood pressure (SBP), and antihypertensive treatment. BMI indicates body mass index; CI, confidence interval; LAE, left atrial enlargement; and LVH, left ventricular hypertrophy.

Disclosures

G.M. has received honoraria for participation as speaker/chairman in national and international meetings from Bayer AG, Böhringer Ingelheim, CVRx, Daiichi Sankyo, Medtronic, Novartis, Recordati, and Servier.

References


16. Lang RM, Bierig M, Devereux RB, Flachskampf FA, Foster E, Pellikka PA, Picard MH, Roman MJ, Seward J, Shanewise JS, Solomon SD, Spencer KT, Sutton MS, Stewart WJ. Chamber Quantification Writing Group; American Society of Echocardiography’s Guidelines and Standards Committee; European Association of Echocardiography. Recommendations for chamber quantification: a report from the American Society of Echocardiography’s Guidelines and Standards Committee and the Chamber Quantification Writing Group, developed in conjunction with...


---

**Novelty and Significance**

**What Is New?**

- An enlargement of left atrium diameter, in absence of left ventricular hypertrophy, is an independent predictor of the long-term (>12 years) risk of cardiovascular morbidity and fatal events in the general population.
- The magnitude of the increased risk accompanying isolated left atrial enlargement is substantial (2-fold).
- No further substantial increase in risk occurs when left atrium enlargement is superimposed on left ventricular hypertrophy.

**What Is Relevant?**

- The present observations call for an accurate assessment of left atrium by echocardiographic examinations. This allows to identify individuals with normal left ventricle who are at increased risk because of alterations of left atrium.

**Summary**

The results of the Pressioni Arteriose Monitorate E Loro Associazioni (PAMELA) study provide evidence on the importance of left atrium enlargement as an independent cardiovascular risk factor in a general population. They also describe the clinical, anthropometric, and metabolic variables most frequently correlated independent fashion with this echocardiographic abnormality.
Prognostic Significance of Left Atrial Enlargement in a General Population: Results of the PAMELA Study
Michele Bombelli, Rita Facchetti, Cesare Cuspidi, Paolo Villa, Dario Dozio, Gianmaria Brambilla, Guido Grassi and Giuseppe Mancia

Hypertension. 2014;64:1205-1211; originally published online September 8, 2014;
doi: 10.1161/HYPERTENSIONAHA.114.03975
Hypertension is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2014 American Heart Association, Inc. All rights reserved.
Print ISSN: 0194-911X. Online ISSN: 1524-4563

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://hyper.ahajournals.org/content/64/6/1205

Data Supplement (unedited) at:
http://hyper.ahajournals.org/content/suppl/2014/09/08/HYPERTENSIONAHA.114.03975.DC1
http://hyper.ahajournals.org/content/suppl/2016/04/10/HYPERTENSIONAHA.114.03975.DC2

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Hypertension can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Hypertension is online at:
http://hyper.ahajournals.org//subscriptions/
PROGNOSTIC SIGNIFICANCE OF LEFT ATRIAL ENLARGEMENT IN A GENERAL POPULATION.

Results of the Pamela study

Michele Bombelli¹,², Rita Facchetti², Cesare Cuspidi²,³, Paolo Villa¹, Dario Dozio¹, Gianmaria Brambilla¹, Guido Grassi¹,²,⁴, Giuseppe Mancia³,⁵.

¹Clinica Medica, Università Milano Bicocca, Monza (Monza e Brianza), ²Dipartimento di Scienze della Salute, Università Milano-Bicocca, Monza (Monza e Brianza); ³Istituto Auxologico Italiano IRCCS; ⁴IRCCS Multimedica, Sesto San Giovanni (Milano); ⁵Università Milano-Bicocca, Milano, Italy

Running title: Left atrial enlargement in a general population

Corresponding Author:

Giuseppe Mancia
IRCCS Istituto Auxologico Italiano
Via L. Ariosto, 13
20145 MILANO

tel.: +39 02 619111

e-mail: giuseppe.mancia@unimib.it
S1- Progressive increase in the incidence of stroke, coronary events, and heart failure from quartile with the lowest (1) to quartile with the highest (4) left atrium diameter.

<table>
<thead>
<tr>
<th>Quartiles of LA diameter</th>
<th>N.</th>
<th>Stroke</th>
<th>N. events</th>
<th>%</th>
<th>Coronary events</th>
<th>N. events</th>
<th>%</th>
<th>Heart Failure</th>
<th>N. events</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1</td>
<td>402</td>
<td>7</td>
<td>1.74%</td>
<td>7</td>
<td>1.74%</td>
<td>2</td>
<td>0.50%</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>424</td>
<td>8</td>
<td>1.89%</td>
<td>9</td>
<td>2.12%</td>
<td>3</td>
<td>0.71%</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>442</td>
<td>10</td>
<td>2.26%</td>
<td>12</td>
<td>2.71%</td>
<td>7</td>
<td>1.58%</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>437</td>
<td>22</td>
<td>5.03%</td>
<td>29</td>
<td>6.64%</td>
<td>11</td>
<td>2.52%</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*p-value*$_{trend}$ 0.0037  <0.0001  0.0055

LA: left atrium; CV: cardiovascular
流行病学人群（摘要）

中心动脉硬化、血压与年龄的纵向前瞻观察研究

Longitudinal Perspective on the Conundrum of Central Arterial Stiffness, Blood Pressure, and Aging

Angelo Scuteri*, Christopher H. Morrell*, Marco Orrù, James B. Strait, Kirill V. Tarasov, Liana Anna Pina Ferrelli, Francesco Loi, Maria Grazia Pilia, Alessandro Delitala, Harold Spurgeon, Samer S. Najjar, Majd AlGhatri, Edward G. Lakatta*

贾一农 译

与年龄相关的动脉僵硬度的增加长期以来被认为伴有或导致年龄相关的血压升高。然而，不同性别和年龄的个体中动脉硬化的指标脉搏传导速度（pulse wave velocity, PWV）和血压轨迹随时间变化而未被分析对比。这项研究通过对>4000例住在撒丁岛年龄在20-100岁之间的男性和女性9.4年的随访，确定了血压和动脉PWV的演变轨迹。线性混合效应模型分析显示在观察期内PWV随时间增长，整个年龄段的男性和女性的长度的速率大致相同。在男性中，血压随时间变化的增长度率与PWV的增长速率并不平行；年龄>40岁收缩压（systolic BP, SBP）和脉压（pulse pressure, PP）先以稳定速率增长，而SBP随年龄增长的速率下降。在女性中，收缩压、舒张压和平均血压随全年龄段以稳定速率增长，导致PP的增长速率明显增加。因此，动脉僵硬度增加与年龄相关的SBP和PP的增长有关。这些发现表明PWV不能代替血压，而且除了发生动脉硬化以外动脉的性质也会随着年龄和性别发生改变，还能随年龄的增长调节BP的轨迹变化，但在男性中PWV、PP、SBP的变化轨迹会有所分离。

(Hypertension. 2014;64:1219-1227.)