Title: MODIFICATION OVER TIME OF PULSE WAVE VELOCITY PARALLEL TO CHANGES IN AORTIC BP AS WELL AS IN 24H-AMBULATORY BRACHIAL BP

Running Title: VARIATION OF AORTIC BP AND ARTERIAL STIFFNESS

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Abstract

Arterial stiffness as assessed by carotid-femoral pulse-wave velocity (cfPWV) is a marker of preclinical organ damage and predictor of cardiovascular outcomes, independently of blood pressure (BP). However, limited evidence exists on the association between long-term variation (\(\Delta\)) on aortic BP (aoBP) and \(\Delta\)cfPWV.

We aimed to evaluate the relationship of \(\Delta\)BP with \(\Delta\)cfPWV over time, as assessed by office and 24h-ambulatory peripheral BP, and aoBP.

AoBP and cfPWV were evaluated in 209 hypertensive patients with either diabetes or metabolic syndrome by applanation tonometry (Sphygmocor®) at baseline (b) and at 12-months follow-up (fu). Peripheral BP was also determined by using validated oscillometric devices (office(o)-BP) and on an outpatient basis by using a validated (Spacelabs®-90207) device (24h-ambulatory BP). \(\Delta\)cfPWV over time was calculated as follows: \(\Delta\)cfPWV=[(cfPWV_{fu}-cfPWV_{b})/cfPWV_{b}]x100. \(\Delta\)BP over time resulted from the same formula applied to BP values obtained with the three different measurement techniques. Correlations (Spearman “Rho”) between \(\Delta\)BP and \(\Delta\)cfPWV were calculated.

Mean age was 62 years; 39% were female; 80% had type 2-diabetes. Baseline oBP (mmHg) was 143±20 / 82±12. Follow-up (12-months later) oBP (mmHg) was 136±20 / 79±12.

\(\Delta\)cfPWV correlated with: \(\Delta\)oSBP (Rho=0.212; p=0.002), \(\Delta\)24h-SBP (Rho=0.254; p<0.001), \(\Delta\)daytime-SBP (Rho=0.232; p=0.001), \(\Delta\)nighttime-SBP (Rho=0.320; p<0.001) and \(\Delta\)aoSBP (Rho=0.320; p<0.001). A multiple lineal regression analysis included the following independent variables: \(\Delta\)oSBP, \(\Delta\)24h-SBP, \(\Delta\)daytime-SBP,
Δnighttime-SBP and ΔaoSBP. ΔcfPWV was independently associated with Δ24h-SBP (β-coefficient=0.195; p=0.012) and ΔaoSBP (β-coefficient= 0.185; p=0.018).

We conclude that changes in both 24h-SBP and aortic SBP more accurately reflect changes in arterial stiffness than do office-BP measurements.
Introduction

Arterial stiffness is increasingly recognized as a marker of preclinical target organ damage\textsuperscript{1,2}. Also it is credited with prognostic value as a predictor of cardiovascular events and of both cardiovascular and all-cause mortality\textsuperscript{3-6} in general population\textsuperscript{7-9} as well as in patients with hypertension\textsuperscript{10}, diabetes\textsuperscript{11} or chronic renal disease\textsuperscript{12}. According to most of these studies, aortic stiffness shows its prognostic value independently of blood pressure (BP) and other cardiovascular risk factors. Several methods to measure aortic stiffness have been developed, but carotid-femoral pulse wave velocity (cfPWV) remains the noninvasive “gold-standard”\textsuperscript{5,13}. High blood pressure and older age are clearly related to high cfPWV, i.e., aortic stiffness, and they are considered to be its main determinants\textsuperscript{14}. Although recent studies suggest that aortic stiffness may be at least in part responsible for the development of hypertension or to contribute to its progression\textsuperscript{15-17}, aortic stiffness has been long thought as the consequence of hypertension. Until now, most studies showing association of BP with cfPWV were carried out by measuring peripheral BP, either office\textsuperscript{18} or ambulatory\textsuperscript{19} and scarce information is reported as for the relationship between cfPWV and central BP\textsuperscript{20}. Since it has been suggested that non-invasive assessment of aortic (central) BP could relate better with preclinical target organ damage\textsuperscript{21-23}, it may indeed be of interest to study the relationship of cfPWV with aortic BP, in addition to the peripheral BP. Moreover, limited evidence exists on the association between long-term variation in aortic BP measurements and changes in preclinical target organ damage. And it is unknown whether these associations are or not stronger than the association between changes in office BP over time and changes in preclinical target organ damage.
We hypothesize that aortic BP estimation may be a better predictor of evolution of preclinical target organ damage, in terms of aortic stiffness, than peripheral BP measurements. Accordingly, we aimed to study the relationship of changes in cfPWV with the variation in BP over time, as assessed by three different methods of measurement, i.e. office and 24h-ambulatory peripheral BP as well as aortic BP.
Patients and Methods

Study Population

Hypertensive subjects with impaired glucose metabolism were consecutively recruited by investigators attending specialized consults on Hypertension in Spain. Hypertension was defined by at least 3 BP measurements taken in separate occasions with systolic (SBP) and/or diastolic (DBP) blood pressure higher or equal to 140 mm Hg and/or 90 mm Hg, respectively, or if the patient was under antihypertensive treatment for at least 6 months. Patients were treated according to clinical practice during the monitoring period. Baseline clinical and anthropometric features as well as laboratory analyses were recorded, and all patients underwent BP measurements both at baseline and at 12 months follow-up as detailed below. Diabetes mellitus was diagnosed if patients had a minimum of two fasting plasma glucose determinations more than 126 mg/dl or when they were under antidiabetic treatment. Metabolic syndrome was diagnosed, in accordance with the consensus document then in force\textsuperscript{24}, when the patient had two or more of the following in addition to elevated BP: abdominal obesity, as defined by a waist circumference more than 102 cm in men or more than 88 cm in women, fasting serum glucose ≥100 mg/dl, high-density lipoprotein plasma cholesterol 40 mg/dl or less in men or 50 mg/dl or less in women, plasma triglycerides >150 mg/dl, or if the patient was under current treatment for any of them. Urinary albumin excretion (UAE) (measured by turbidimetry in local laboratories according to current recommended standards) was determined as the average of urinary albumin/creatinine ratio from three first-morning void urine samples obtained in separate days. Exclusion criteria were serious life-threatening
co-morbidity for the next 12 months, patients already enrolled in another clinical
trial or follow-up clinical records not available. The study protocol conforms to the
principles outlined in the Declaration of Helsinki and was approved by all
correspondent research ethic committees. All participants gave written informed
consent before their inclusion in the study.

**BP Measurement**

*Office brachial BP (oBP)* was obtained by trained personnel as the average of
triplicate measurements taken at intervals of 1 min after an initial 5 min of seated
rest, using validated oscillometric devices, with regular or large adult cuffs used as
needed. Furthermore, after 10 min of rest in the supine position at a comfortable
room temperature, *aortic BP (aoBP)* was measured with the SphygmoCor® device
(AtCor Medical, Sydney, Australia). Mean arterial pressure was determined by
mathematical integration of the radial pressure waveform obtained by applanation
tonometry and calibrated by using the oscillometric value of brachial SBP and DBP,
as has been previously validated\(^25\). AoBP measurements were carried out at each
investigation centre by either nurses or doctors who were specifically trained to
perform this technique according to current standardized recommendations\(^5\). They
were closely monitored throughout the study to ensure accuracy in recording aoBP
(details are given elsewhere)\(^26\). Moreover, the averaged values of two consecutive
valid (operator index higher than 80%) measurements at each visit were required.
Intraobserver aoBP measurement coefficients of variation were 7.8 and 8.9% for
SBP and DBP, respectively. The interobserver aoSBP and aoDBP coefficients of
variation were 11.6 and 15.1%, respectively. Finally, *24h-ambulatory brachial BP*
recordings were taken with a validated device (Spacelabs®-90207) at 20-minute intervals throughout both the self-reported awake and asleep periods.

All BP measurements were recorded for each included patient at baseline \((b)\) and after 12 months of follow-up \((fu)\).

*Carotid-femoral pulse wave velocity (cfPWV) measurement*

cfPWV was evaluated by noninvasive applanation tonometry on carotid and femoral arteries (Sphygmocor® device, AtCor Medical, Sydney, Australia)\(^{27}\) at baseline \((b)\) and after 12 months of follow-up \((fu)\). The values of two valid consecutive measurements were averaged at each visit. cfPWV was computed as the distance travelled by the pulse wave divided by pulse wave transit time. Travel distance was measured to the nearest centimetre with an external tape measure over the body surface. The transit time was determined as the time difference between the feet of carotid and femoral arterial waveforms gated to ECG.

cfPWV variation \((\Delta)\) over time was calculated as follows: \(\Delta \text{cfPWV} = \left[\frac{\text{cfPWV}_{fu} - \text{cfPWV}_{b}}{\text{cfPWV}_{b}}\right] \times 100\). BP variation over time was calculated with the same formula applied to BP values obtained with the different measurement techniques.
Statistical analysis

A minimum number of 194 subjects were required for a bilateral contrast with an alpha risk of 0.05, a statistical power of 80% and an estimated correlation coefficient between changes in aortic SBP and cfPWV of 0.2.

Continuous variables are summarized by mean ± SD if normally distributed or by median [interquartile range] otherwise. Categorical variables are described as frequencies and percentages. Comparisons of clinical characteristics between baseline and follow-up were performed by paired t tests in continuous data, by the non-parametric Wilcoxon rank sum test in asymmetrically distributed data, and by McNemar’s test in categorical data. The association between Δ BP and Δ cfPWV was assessed with the Spearman’s correlation coefficient (Rho). A multiple lineal regression analysis was performed to ascertain the BP parameter(s) that were independently associated to variation of cfPWV over time. The SPSS for Windows version 19.0 software (IBM Corp. Armonk, NY, USA) was used for statistical analyses.
Results

A total of 209 hypertensive patients entered the study. They were 127 (61%) men and 82 (39%) women, with a mean age of 61.8 ± 11.2 years. The prevalence of type 2 diabetes mellitus was 80%, and the remaining 20% had metabolic syndrome. Dyslipidemia accounted for 69% of the patients and 13% were current smokers. At baseline, arterial stiffness (cfPWV >10 m/s) was diagnosed in 40.7% (85 out of 209) patients. Other clinical characteristics at baseline and at 12-months follow-up are shown in Table 1 and none of these characteristics showed a statistically significant change at 12-months follow-up.

Regarding BP measurements, results on office, 24h-ABPM and aoBP at baseline and at 12-months follow-up are shown in Table 2. Overall, both office-BP (p<0.001 for all estimators) and aortic -SBP and -DBP (p=0.002 and p=0.001, respectively) were significantly lower at follow-up. As regards ambulatory-BP estimators, only day- and night- DBP, but not SBP, showed a statistically significant decrease at 12-months follow up (p<0.01 for both).

Table 3 shows the correlations between △ cfPWV and △ BP as assessed by brachial office BP, brachial ambulatory BP and aortic BP. Changes in cfPWV at follow-up significantly correlated with variation of both SBP and DBP as measured by either office brachial BP and aortic BP, as well as by brachial 24h-, daytime- and nighttime- ambulatory BP. The variation of pulse pressure (PP) as regards to aortic PP and brachial ambulatory PP, but not in terms of office brachial PP, also showed statistically significant correlations with changes in cfPWV.

A multiple lineal regression analysis was performed to explore the association between the variation of the different BP parameter(s) and the variation of cfPWV.
over time. Variables entered into the model were Δ in office SBP, Δ in 24-hour SBP, Δ in daytime SBP, Δ in nighttime SBP and Δ in aortic SBP. We applied the stepwise backward method to build the multiple regression model, in order to minimize the impact of multicollinearity. Table 4 shows the results of this analysis. As can be observed, the independent variables that showed a statistically significant association with variation of cfPWV at 12 months follow-up were the variation over time of 24-hour SBP (β-coefficient, 0.195; p=0.012) and the variation over time of aortic SBP (β-coefficient, 0.185; p=0.018).
Discussion

The main finding of our study is that the BP measurements that better predict the variation of $\text{cfPWV}$ over time are changes in aortic SBP and 24-hour SBP, but not office brachial BP changes. Accordingly, it should be reasonable to speculate that targeting a different BP estimation instead of the currently accepted office brachial BP values could be better to prevent the progression of arterial stiffness. Although it is well known that sustained high BP favors the development and/or progression of preclinical target organ damage as measured by several markers, arterial stiffness amongst them, there is some evidence that treating hypertension according to office peripheral BP values does not clearly prevent from the progression of arterial stiffness. Actually, in a recent longitudinal study therapies aimed to reduce BP in a large cohort of old men did not show to be effective in $\text{cfPWV}$ reduction$^{28}$. As regards peripheral ambulatory BP monitoring (ABPM), Karpettas et al.$^{19}$ showed in a small study that changes in 24h-ambulatory BP were a better predictor of changes in $\text{cfPWV}$ than variation of office BP. This is indeed not really surprising, since ABPM has largely demonstrated a stronger association with target organ damage than office BP values$^{29,30}$. However, the relationship between 24h-ABPM variation over time and changes in $\text{cfPWV}$ or other markers of preclinical organ damage has generally been poorly documented$^{19,31}$. On the other hand, as regards aortic BP, there is a gap in knowledge concerning the relationship between variation of aortic BP and changes in preclinical target organ damage over time. To our knowledge this is the first time that the association between aortic BP changes over time and variation of $\text{cfPWV}$ is reported. As shown, our results demonstrate that the best parameters to predict
cfPWV changes at one year are variation of peripheral 24h-SBP and variation of aortic-SBP. This finding may have two important consequences. First of all, there is a BP value determined at office, i.e. aortic BP as measured by applanation radial artery tonometry, which is a reliable predictor of changes in arterial stiffness, at least as good as is 24h-ABPM. The main advantage is that this BP measurement is more comfortable for the patient than the 24h-ABPM and the result is available immediately, allowing physicians to make the most appropriate therapeutic decisions right then. Secondly, as different antihypertensive classes have different effect on aortic BP, it may be hypothesized that drugs specifically targeting aortic BP could be more effective to reduce arterial stiffness, beyond the effect of these antihypertensive drugs on office BP.

The most important limitation of this study is that antihypertensive treatment was not homogeneous and could change during the observation period. However, all the patients were treated according to clinical practice along follow up, and the number of antihypertensive drugs did not change in the observation period, which makes unlikely that the treatment is a factor that has significantly influenced these results. Another point to consider is the fact that the majority of patients evaluated in this study were diabetic. Patients with type 2 diabetes mellitus or abnormal glucose metabolism have increased arterial stiffness, even before the onset of diabetes and independently of both conventional cardiovascular risk factors and hyperglycemia or hyperinsulinemia32, so we believe that this is a target population in whom assessing the influence of changes of BP over time on cfPWV would be relevant. Certainly, our results should not be extrapolated to other populations until further confirmation on them. We must add some more possible limitation to the study. Although an accurate training and monitoring of aortic BP
measurement was implemented, the interobserver aoSBP and aoDBP coefficients of variation were slightly higher than expected. Finally, it is possible that a longer observational period of time could be better to reaffirm these results. Strengths of the study include a direct comparison of the three BP measurement methods (office, 24h-ABPM and aortic BP) being performed in the same subjects. Also it is remarkable that there was no change in body mass index over time (30.9 kg/m², both baseline and at 12-months follow up) because there is recently published evidence that weight loss may reduce cfPWV, and it could have been a confounding factor in our study.

In conclusion, we have shown that changes in both 24h-SBP and aortic SBP more accurately reflect changes in arterial stiffness than do office BP measurements, in hypertensive patients with impaired glucose metabolism. It remains to be explored if different antihypertensive drugs targeting these BP estimators would differently modify cfPWV over time. And, we should further evaluate whether changes in cfPWV in accordance to targeted 24h-SBP or aortic SBP results in an improvement of cardiovascular risk.

Disclosures/Conflicts of interest:

None to declare.

Acknowledgements:

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Summary Table

What is known about the topic

- Arterial stiffness, as measured by carotid-femoral pulse wave velocity (cfPWV), is a marker of preclinical target organ damage and a predictor of cardiovascular outcomes and mortality, independently of blood pressure.
- Peripheral blood pressure is reported to be associated with cfPWV.
- Limited evidence exists on the association between long-term variation in aortic (central) BP measurements and changes in preclinical target organ damage, and specifically cfPWV.

What this study adds

- Evaluation of a cohort of patients with altered glucose metabolism shows that the BP measurements that better predict the variation of cfPWV over time are changes in both aortic and 24-hour systolic BP, but not office brachial BP changes.
- This finding allows the hypothesis that different antihypertensive drugs targeting these BP estimators could differently modify cfPWV over time.
References


   Central or peripheral systolic or pulse pressure: which best relates to target organs and future mortality? *J Hypertens* 2009; **27**: 461-467.

23. Roman MJ, Okin PM, Kizer JR, Lee ET, Howard BV, Devereux RB. Relations of central and brachial blood pressure to left ventricular hypertrophy and geometry: the Strong Heart Study. *J Hypertens* 2010; **28**: 384-388.


Table 1. Demographic and clinical characteristics of patients at baseline and at 12 months follow-up.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Baseline n=209</th>
<th>Follow-up (12 months)* n=209</th>
</tr>
</thead>
<tbody>
<tr>
<td>Current smokers, %</td>
<td>13.4</td>
<td>14.8</td>
</tr>
<tr>
<td>BMI (Kg/m$^2$)</td>
<td>30.9 ± 4.4</td>
<td>30.9 ± 5.0</td>
</tr>
<tr>
<td>Obesity, BMI ≥30 Kg/m$^2$, %</td>
<td>54.5</td>
<td>55.0</td>
</tr>
<tr>
<td>Abdominal obesity*, %</td>
<td>74.2</td>
<td>72.7</td>
</tr>
<tr>
<td>UAE (mg/g)</td>
<td>14.3 [5.6 - 59.2]</td>
<td>9.7 [4.8 - 35.2]</td>
</tr>
<tr>
<td>UAE &gt;30 mg/g, %</td>
<td>34.4</td>
<td>27.6</td>
</tr>
<tr>
<td>eGFR, ml/min/1.73m$^2$</td>
<td>79.9 ± 30.3</td>
<td>81.3 ± 31.7</td>
</tr>
<tr>
<td>eGFR &lt;60 ml/min/1.73m$^2$, %</td>
<td>28.2</td>
<td>27.4</td>
</tr>
<tr>
<td>cfPWV (m/s)</td>
<td>10.01 ± 3.50</td>
<td>10.19 ± 3.21</td>
</tr>
<tr>
<td>cfPWV &gt;10 m/s, %</td>
<td>40.7</td>
<td>41.1</td>
</tr>
<tr>
<td>Number of antihypertensive drugs</td>
<td>3 [1 - 4]</td>
<td>3 [1 - 4]</td>
</tr>
</tbody>
</table>

Data are given as mean ± SD, median [IQR].

Abbreviations: BMI, body mass index; cfPWV, carotid-femoral pulse wave velocity; eGFR, estimated glomerular filtration rate (according to the Modification of Diet in Renal Disease equation); UAE, urinary albumin excretion.

*aAbdominal obesity defined as waist circumference >102 cm (men) or >88 cm (women).

(*) p-value NS for all comparisons.
Table 2. Blood pressure measurements at baseline and at 12 months follow-up.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Baseline n=209</th>
<th>Follow-up (12 months) n=209</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Office (o) BP</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>o-SBP (mmHg)</td>
<td>143.1 ± 19.8</td>
<td>136.0 ± 19.9</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>o-DBP (mmHg)</td>
<td>81.8 ± 12.2</td>
<td>79.1 ± 11.8</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>o-PP (mmHg)</td>
<td>61.3 ± 16.6</td>
<td>56.9 ± 16.6</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td><strong>24h-Ambulatory BP</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>24h-SBP (mmHg)</td>
<td>129.1 ± 13.3</td>
<td>128.0 ± 14.5</td>
<td>0.243</td>
</tr>
<tr>
<td>24h-DBP (mmHg)</td>
<td>73.7 ± 9.5</td>
<td>72.3 ± 9.5</td>
<td>0.007</td>
</tr>
<tr>
<td>24h-PP (mmHg)</td>
<td>55.3 ± 12.4</td>
<td>55.7 ± 13.1</td>
<td>0.446</td>
</tr>
<tr>
<td>day-SBP (mmHg)</td>
<td>132.0 ± 14.0</td>
<td>130.7 ± 14.4</td>
<td>0.171</td>
</tr>
<tr>
<td>day-DBP (mmHg)</td>
<td>76.4 ± 10.1</td>
<td>74.7 ± 9.8</td>
<td>0.003</td>
</tr>
<tr>
<td>day-PP (mmHg)</td>
<td>55.6 ± 12.9</td>
<td>56.0 ± 13.1</td>
<td>0.510</td>
</tr>
<tr>
<td>night-SBP (mmHg)</td>
<td>121.5 ± 15.3</td>
<td>121.3 ± 17.3</td>
<td>0.836</td>
</tr>
<tr>
<td>night-DBP (mmHg)</td>
<td>66.8 ± 9.4</td>
<td>66.4 ± 10.2</td>
<td>0.426</td>
</tr>
<tr>
<td>night-PP (mmHg)</td>
<td>54.7 ± 12.5</td>
<td>54.9 ± 14.0</td>
<td>0.681</td>
</tr>
<tr>
<td><strong>Central (aortic) BP</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>aortic-SBP (mmHg)</td>
<td>128.8 ± 17.5</td>
<td>124.2 ± 18.6</td>
<td>0.002</td>
</tr>
<tr>
<td>aortic-DBP (mmHg)</td>
<td>81.3 ± 11.8</td>
<td>78.9 ± 11.5</td>
<td>0.001</td>
</tr>
<tr>
<td>aortic-PP (mmHg)</td>
<td>47.4 ± 14.6</td>
<td>45.5 ± 16.4</td>
<td>0.064</td>
</tr>
</tbody>
</table>

BP = blood pressure; DBP = diastolic blood pressure; PP = pulse pressure; SBP = systolic blood pressure.
Table 3. Correlations of variation of cfPWV with variation of BP (\(\Delta BP\)) according to different BP measurements.

<table>
<thead>
<tr>
<th>Measurement method</th>
<th>(\Delta) Systolic BP (\Delta) cfPWV</th>
<th>(\Delta) Diastolic BP (\Delta) cfPWV</th>
<th>(\Delta) Pulse pressure (\Delta) cfPWV</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>rho</td>
<td>p</td>
<td>rho</td>
</tr>
<tr>
<td>office peripheral BP</td>
<td>0.212</td>
<td>0.002</td>
<td>0.286</td>
</tr>
<tr>
<td>24h-ABPM</td>
<td>0.254</td>
<td>&lt;0.001</td>
<td>0.273</td>
</tr>
<tr>
<td>daytime-BP</td>
<td>0.232</td>
<td>0.001</td>
<td>0.265</td>
</tr>
<tr>
<td>nighttime-BP</td>
<td>0.320</td>
<td>&lt;0.001</td>
<td>0.248</td>
</tr>
<tr>
<td>aortic-BP</td>
<td>0.320</td>
<td>&lt;0.001</td>
<td>0.183</td>
</tr>
</tbody>
</table>

ABPM = ambulatory blood pressure monitoring; BP = blood pressure; cfPWV = carotid-femoral pulse wave velocity; \(\Delta\) BP = variation of blood pressure.
Table 4. Multiple Linear Regression with BP variables showing association with variation of cfPWV at 12 months follow-up.

<table>
<thead>
<tr>
<th>Variables associated with Δ cfPWV</th>
<th>β - coefficient</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Δ 24-hour systolic BP</td>
<td>0.195</td>
<td>0.012</td>
</tr>
<tr>
<td>Δ Aortic systolic BP</td>
<td>0.185</td>
<td>0.018</td>
</tr>
</tbody>
</table>

BP = blood pressure; cfPWV = carotid-femoral pulse wave velocity

Adjusted $R^2$ of the model 0.101; $p=0.018$