The prognostic value of isolated office or white coat hypertension (WCH), that is, the condition in which office but not out-of-office blood pressure (BP) is elevated, is still a matter of debate. This is because although in some studies WCH has been associated with a greater prevalence of organ damage and a higher incidence of cardiovascular (CV) morbidity and mortality than those observed in in- and out-of-office normotension (NT),1–3 in other studies no difference (or a nonsignificant increase) in organ damage and a higher incidence of cardiovascular and all-cause mortality (2.76 and 1.58; \(P<0.03\)). Compared with normotensive subjects, the risk of cardiovascular mortality, as adjusted for potential confounders, showed a progressive significant increase in WCH and sustained hypertensive subjects (2.04 and 2.94; \(P=0.006\)). In either group, a significant increase in adjusted risk was also seen for all-cause mortality. However, when the WCH group was subdivided into 2 subgroups, that is, subjects in whom both out-of-office BPs were normal (true WCH, 42%) or one BP was normal whereas the other was elevated (partial WCH, 58%), only the latter showed a significantly greater adjusted risk of cardiovascular and all-cause mortality (2.76 and 1.58; \(P<0.03\)). Compared with normotensive subjects, the partial WCH group also exhibited a marked increase in adjusted risk of developing sustained hypertension over a 10-year time period (2.58; \(P<0.0001\)), but in this case the risk was also increased in true WCH subjects (2.89; \(P<0.0001\)). Thus, WCH includes subjects with a widely different long-term risk of a cardiovascular event. To identify those at higher risk, measurements of both out-of-office BPs are desirable. (Hypertension. 2013;62:168-174.) • Online Data Supplement

Key Words: ambulatory blood pressure monitoring • home blood pressure monitoring • mortality • prognosis • white coat hypertension

The Pressioni Arteriose Monitorate E Loro Associazioni (PAMELA) is a population study in which BP was measured in office, over day and night, and by subjects at home.10 This allowed us to assess the risk of CV and all-cause mortality in individuals in whom an office BP elevation was associated with ambulatory or home BP normality. It further allowed to recalculate the relative risk in 2 WCH subgroups, that is, those in whom both out-of-office BP values were normal and those in whom one was found to be normal but the other was elevated. Mortality was assessed over a 16-year follow-up (ie, the longest follow-up available in longitudinal WCH studies).

Methods

Subjects and Measurements

The methods used in the PAMELA Study have been published in detail elsewhere.10 Briefly, 3200 individuals were randomly selected from the residents in Monza (a town located in the North-East outskirts of Milan, Italy). In the PAMELA Study, BP was measured in office, over day and night, and by subjects at home.10 This allowed us to assess the risk of CV and all-cause mortality in individuals in whom an office BP elevation was associated with ambulatory or home BP normality. It further allowed to recalculate the relative risk in 2 WCH subgroups, that is, those in whom both out-of-office BP values were normal and those in whom one was found to be normal but the other was elevated. Mortality was assessed over a 16-year follow-up (ie, the longest follow-up available in longitudinal WCH studies).

The online-only Data Supplement is available with this article at http://hyper.ahajournals.org lookup/suppl/doi:10.1161/HYPERTENSIONAHA.111.00690/-/DC1.

Correspondence to Giuseppe Mancia, Clinica Medica Ospedale S. Gerardo, Via Pergolesi 33, 20900 Monza (Milan), Italy. E-mail giuseppe.mancia@unimib.it

© 2013 American Heart Association, Inc.

Hypertension is available at http://hyper.ahajournals.org

DOI: 10.1161/HYPERTENSIONAHA.111.00690
of Milan) to be representative of its population for sex, age decades (25–74 years), and socioeconomic characteristics, according to the criteria used in the World Health Organization–monitoring trends and determinants of cardiovascular diseases (MONICA) study carried out in the same geographical area. The overall participation rate was 64% (n=2051), consistently for each age and sex stratum. The demographic characteristics of nonparticipants were similar to those of participants. This was also the case for CV risk factors based on information collected via phone interviews. Between 1991 and 1993, participants were invited to come to the outpatient sector of the S. Gerardo Hospital of Monza in the morning of a working day, where the following data were collected: (1) 3 office BP measurements, using a mercury sphygmomanometer and making the subject sit for 10 minutes before measurement; (2) a 24-hour ambulatory BP monitoring performed via an oscillometric device (Spacelabs 90207, Spacelabs), with the BP readings set at 20-minute intervals. The subjects were sent home after checking for device accuracy, with the instruction to attend at their home after the first valid readings over the day (95.7%) and night (96.5%). Subjects with >80% valid readings were included in the analysis. The overall participation (72 readings), with a homogeneous distribution (2.9 per hour) throughout the entire recording period and a similar percentage of valid readings over the day (95.7%) and night (96.5%). Subjects with a normal office and a high home or ambulatory BP (masked hypertension) were excluded from analysis, and the remaining data (1589 subjects) were analyzed in 2 steps. One, reflecting what is usual for clinical practice and recommended by guidelines, WCH was diagnosed by an elevation in clinic BP (≥140 mm Hg systolic BP [SBP] or ≥90 mm Hg diastolic BP [DBP]) and a normal 24-hour mean or home BP. Two, the WCH group was divided into 2 subgroups: (1) true WCH, in which both home and 24-hour BP were normal; and (2) partial WCH, in which 1 BP value was elevated. Based on previous analyses, 24-hour BP and home BP normality were defined as <125/79 mm Hg and <132/83 mm Hg, respectively. These values are similar or somewhat lower than the normality values for 24-hour and home BP reported by guidelines. The 2 partial WCH groups in which home or 24-hour BP was elevated were also analyzed. Individuals in whom all BP values were normal or elevated were taken as NT controls and hypertensive (HT) patients, respectively.

Data were expressed as means±SD or percentage. Comparisons between groups were done by ANOVA (for mean values) and the χ² test (for prevalence). The Bonferroni correction was used for multiple comparisons. Trend was assessed by regression model or Cochran–Armitage trend test.

The hazard ratio was calculated by the Cox proportional hazard model, taking the groups with office, 24-hour, and home BP normality as reference. The assumption of the proportionality between BP and event variations was assessed by proper statistical test.

### Table 1. Baseline Data From NT, WCH, HT, and True and Partial WCH Subjects

<table>
<thead>
<tr>
<th>Variables</th>
<th>NT</th>
<th>WCH</th>
<th>HT</th>
<th>True WCH</th>
<th>Partial WCH</th>
</tr>
</thead>
<tbody>
<tr>
<td>n, %</td>
<td>825 (51.9)</td>
<td>391 (24.6)</td>
<td>373 (23.5)</td>
<td>164</td>
<td>227</td>
</tr>
<tr>
<td>Men, %</td>
<td>39.9</td>
<td>52.2†</td>
<td>61.1*</td>
<td>50.6†</td>
<td>53.3‡</td>
</tr>
<tr>
<td>Age, y</td>
<td>43.7±11.9</td>
<td>54.7±11.5†</td>
<td>60.1±10.0*</td>
<td>51.6±11.5†</td>
<td>56.9±11.0‡†</td>
</tr>
<tr>
<td>Smokers, %</td>
<td>29.1</td>
<td>22.3†</td>
<td>21.7*</td>
<td>20.7*</td>
<td>23.4</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>23.9±3.6</td>
<td>27±4.3†</td>
<td>27.4±5.2*</td>
<td>26.3±4.3†</td>
<td>27.5±4.2‡</td>
</tr>
<tr>
<td>Office SBP, mm Hg</td>
<td>117.0±10.1</td>
<td>143.1±13.2‡</td>
<td>160.0±18.2*</td>
<td>139.1±11.3†</td>
<td>146.1±13.8†‡</td>
</tr>
<tr>
<td>Office DBP, mm Hg</td>
<td>76.4±6.9</td>
<td>90.4±6.9†</td>
<td>95.0±9.8*</td>
<td>89.7±6.4†</td>
<td>91.0±7.2†</td>
</tr>
<tr>
<td>Home SBP, mm Hg</td>
<td>110.5±10.4</td>
<td>127.2±13.9†</td>
<td>149.0±15.1*</td>
<td>118.6±8.1†</td>
<td>133.4±13.9‡</td>
</tr>
<tr>
<td>Home DBP, mm Hg</td>
<td>69.3±6.9</td>
<td>78.7±8.2†</td>
<td>86.7±9.2*</td>
<td>74.2±5.8†</td>
<td>82.0±8.4‡</td>
</tr>
<tr>
<td>24-h SBP, mm Hg</td>
<td>112.3±6.6</td>
<td>119.4±7.1†</td>
<td>136.6±10.0*</td>
<td>116.7±5.4†</td>
<td>121.4±7.6‡‡</td>
</tr>
<tr>
<td>24-h DBP, mm Hg</td>
<td>69.9±4.8</td>
<td>74.4±5.8*</td>
<td>83.0±7.4*</td>
<td>72.7±4.5*</td>
<td>75.7±6.2‡</td>
</tr>
<tr>
<td>Serum total Ch, mg/dL</td>
<td>213.3±40.9</td>
<td>233.3±42.5†</td>
<td>236.0±41.2*</td>
<td>229.6±41.8*</td>
<td>236.0±43.0*</td>
</tr>
<tr>
<td>Serum glucose, mg/dL</td>
<td>85.5±12.5</td>
<td>93.2±20.9†</td>
<td>98.5±26.4*</td>
<td>93.0±18.9†</td>
<td>93.4±22.2†</td>
</tr>
<tr>
<td>24-h SBP SD, mm Hg</td>
<td>12.5±2.8</td>
<td>14.7±3.5†</td>
<td>15.9±3.8*</td>
<td>13.7±2.9†</td>
<td>15.4±3.6‡</td>
</tr>
<tr>
<td>24-h SBP coefficient of variation, %</td>
<td>11.1±2.5</td>
<td>12.4±3.0†</td>
<td>11.7±2.8*</td>
<td>11.8±2.6*</td>
<td>12.8±2.7†</td>
</tr>
<tr>
<td>Antihypertensive drugs, %</td>
<td>4.3</td>
<td>27.2†</td>
<td>41.8*</td>
<td>22.0†</td>
<td>31.0‡†</td>
</tr>
<tr>
<td>Previous CV event, %</td>
<td>1.8</td>
<td>3.6*</td>
<td>8.0*</td>
<td>3.7</td>
<td>3.5</td>
</tr>
</tbody>
</table>

Data are shown as means±SD or percentage. NT refers to normality of office, 24-h mean BP, and home BP; HT refers to elevation of all 3 BPs; and WCH refers to elevation of office and normality of 24-h or home BP. True WCH refers to individuals in whom both 24-h and home BP were normal, and partial WCH refers to individuals in whom office BP was elevated but only 1 of the 2 out-of-office BPs was elevated. BMI indicates body mass index; BP, blood pressure; Ch, cholesterol; CV, cardiovascular; DBP, diastolic BP; HT, hypertensive; NT, normotensive; SBP, systolic BP; and WCH, white coat hypertension.

*P<0.05 vs NT.
†P<0.05 vs HT.
‡P<0.05 vs true WCH.
Hazard ratios were calculated for unadjusted data, after adjustment for age and sex and after additional adjustment for other possible confounders (ie, smoking [yes or no], history of CV events [yes or no], blood glucose, serum total cholesterol, BMI, and antihypertensive treatment [yes or no]). The fully adjusted hazard ratios were also calculated after exclusion of treated antihypertensive subjects.

All available demographic and clinical variables were entered into a stepwise multivariable analysis to determine the factors independently associated with CV or all-cause mortality in the WCH group. In subjects in whom data were also collected after 10 years from the initial collection, calculations were made of the incidence of new-onset sustained HT, defined as an elevation of all 3 pressures (ie, office, home and 24 hours) in the NT, true WCH, and partial WCH groups. Taking the NT group as reference, the relative risk of developing sustained HT was assessed by a modified Poisson regression model (using robust error variance) before and after adjustment for age, sex, and BMI. Data are expressed as means±SD or 95% confidence intervals (CIs; in brackets) and P values. n refers to number of deaths. For other symbols see text.

Figure 1. A, Kaplan–Meier curves for cardiovascular (CV) and all-cause mortality in normotension (NT), white coat hypertension (WCH), and sustained hypertension (HT). WCH was defined by an office blood pressure (BP) elevation and ambulatory (24 hours) or home BP normality. NT and sustained HT were defined by normality or elevation of all 3 BPs, respectively. Average follow-up was 16 years. B, Hazard ratios (HR) for CV and all-cause mortality in WCH and HT, taking the NT group as reference. Data are shown unadjusted, after adjustment for age and sex (partial), and after additional adjustment (full) for other potential confounders (blood glucose, total serum cholesterol, smoking, previous CV events, antihypertensive treatment, body mass index). HRs are shown with 95% confidence intervals (CIs; in brackets) and P values. n refers to number of deaths. For other symbols see text.

Results

WCH as Diagnosed by Ambulatory or Home BP Normality

Table 1 (first to third column) shows that in the PAMELA population the prevalence of individuals with office BP elevation and 24-hour or home BP normality was 24.6%, that is, almost half of the individuals with an office BP elevation. With the exception of the smoking habits, prevalence of male sex, age, dysmetabolic risk factors, antihypertensive drug use, in- and out-of-office BP values, and history of CV events were all progressively greater from NT to WCH and HT individuals.

The incidence of CV (n=77) and all-cause (n=242) mortality also increased progressively from NT to WCH and HT condition, the 3 curves beginning to show a clear separation after a few years (Figure 1A). As shown in Figure 1B, compared with NT, the risk of CV and all-cause mortality increased significantly in WCH before adjustment, after adjustment for age and sex (partial), and, as far as all-cause mortality was concerned, after further adjustment (full) for other confounders (see Methods). With one exception, HT subjects showed a further increase in the risk of CV and all-cause mortality in the unadjusted, partially adjusted, and fully adjusted data (P for trend always significant). As shown in Table 2, in a multivariable analysis on subjects with WCH, CV mortality was predicted by office SBP plus age, history of CV events, smoking, use of antihypertensive drugs, and BMI. Office SBP was also an independent predictor of all-cause mortality, together with home BP, age, sex, smoking, and history of CV events.
The authors of the document, Mancia et al., discuss the prognostic value of white coat hypertension (WCH) as diagnosed by both ambulatory and home blood pressure (BP) normality. They note that in a noticeable number of subjects, normal 24-hour SBP or DBP values were accompanied by a home SBP or DBP elevation and vice versa, allowing the WCH subjects to be split into those in whom both 24-hour and home BP were normal (true WCH, 41.9%) and those in whom out-of-office BP was normal whereas the other was elevated (partial WCH, 58.1%). As shown in Table 1, clinic BP, 24-hour BP, home BP, and other risk factors were frequently more altered in the latter than in the former group. Compared with normotensive (NT) subjects, only in subjects with partial WCH the incidence of cardiovascular (CV) and all-cause mortality showed a clear-cut increase (Figures 3A and 3B), with a significant increment when data were adjusted for age and sex as well as other confounders (Figures 3A and 3B). An increased risk of CV and all-cause mortality was also seen when in the partial WCH group data were separately calculated for the subgroup with only home or only ambulatory BP elevation, although the latter number of patients was small and the increased risk was not statistically significant (Table S1 in the online-only Data Supplement).

Discussion

In the PAMELA population, individuals in whom WCH was diagnosed by an elevation of office BP with normal 24-hour or home BP values exhibited a greater incidence of CV and all-cause mortality over an average follow-up of 16 years than individuals in whom office and ambulatory or home BP were normal. In the WCH group, the risk of dying showed a 3× to 6× increase when calculated from unadjusted data. It remained almost twice (CV mortality) and 50% (all-cause mortality) when adjusted for age and sex as well as other confounders.
mortality) greater than that of normotensive subjects when data were adjusted for relevant confounders. Thus, WCH as commonly identified in clinical practice (ie, from the information provided by office and 1 out-of-office BP) is not clinically innocent but rather associated with a clear-cut increase in the long-term risk of fatal events.

The most important and entirely new finding of our study, however, is that the incidence and risk of CV and all-cause mortality were not significantly different from those of NT subjects if both ambulatory and home BP values were normal. In contrast, in subjects in whom normality of one out-of-office BP was accompanied by elevation of the other, the incidence of fatal events was markedly increased, with a fully adjusted risk of CV and all-cause mortality that was, respectively, \( >3 \times \) and \( \approx 60\% \) greater than that of NT controls. Thus, measuring both 24-hour and home BP allows to distinguish, among WCH individuals, those in whom the risk of a fatal event is increased from those in whom it is not. This means that information on both out-of-office BP values is clinically important and that a diagnostic approach based on 3 rather than 2 different BPs may represent a valuable procedure to be implemented. This is the case also because the condition in which the risk is increased, and that in which it is not, both represent a noticeable portion (\( \approx 60\% \) and 40\%, respectively) of the overall WCH population.

Because in the partial WCH group several subjects were under antihypertensive drugs, the possibility exists that the increased risk originated from those being sustained hypertensive, in whom treatment had controlled out-of-office but not office BP. However, this can be ruled out because in a meta-analysis of a large number of studies antihypertensive treatment has been shown to reduce office BP much more effectively than ambulatory BP, which suggests that the opposite may be the case. Furthermore, and more importantly, subjects with partial WCH also showed a pronounced increase in the risk of mortality when patients under antihypertensive treatment were excluded.

In line with previous observations, individuals in whom office BP was elevated, while 24-hour or home BP was normal, were characterized by a more frequent history of CV events and a greater BMI, serum total cholesterol, and serum glucose values than NT controls. Furthermore, and most importantly, 24-hour and home BP values, although falling within the normal range, were significantly and not marginally greater (24-hour SBP/DBP, +7.1/+4.5 mm Hg; home SBP/DBP, +16.7/+9.4 mm Hg) in the WCH than in the NT group. We can speculate that all these factors may contribute to the increased risk of this condition as well as to the greater probability of a fatal event exhibited by partial WCH subjects in whom the metabolic and BP-related risk factor profiles were

---

**Figure 3.** A, Kaplan–Meier curves for cardiovascular (CV) and all-cause mortality in normotension (NT), true white coat hypertension (WCH), partial WCH, and sustained hypertension (HT). NT and sustained HT subjects were those in whom office, 24-hour, and home blood pressure (BP) were all normal or elevated. True WCH subjects were those in whom office BP was elevated and 24-hour and home BP were both normal. Partial WCH subjects were those in whom office BP elevation was associated with normality of 24-hour or home BP only. B, Hazard ratio (HR) for CV and all-cause mortality in NT, true WCH, partial WCH, and sustained HT, taking NT as reference. CI indicates confidence interval.
significantly worse than those of WCH subjects in whom both out-of-office BP values were within the normal range. This is supported by the results of the multivariable analysis, which showed that in the WCH group several components of the CV risk profile independently predicted mortality. Interestingly, for both CV and all-cause mortality, one independent predictor was office BP, which suggests that in WCH its elevation is not clinically irrelevant but rather contributes to the overall increase in risk.

Several other results of our study deserve mention. One, the incidence and the adjusted risk of CV and all-cause mortality shown by subjects with partial WCH were only moderately less than those of sustained hypertensive patients. This means that these subjects are not too different from individuals with sustained hypertension, which represents a further reason to identify them via an extended BP measurement approach. Two, the mortality curves of the WCH groups set apart from that of normotensive individuals after a follow-up of a few years, after which the differences became progressively more evident with time. This suggests that the prognostic importance of WCH requires studies with rather long follow-ups. Three, a substudy of the Syst-Eur trial on isolated systolic hypertension has reported that in patients with a normal ambulatory BP the effect of treatment was largely limited to an office BP fall, with a reduction of CV morbid and fatal events that were less than that seen in sustained hypertension and not significantly different from placebo.14 However, WCH represents a large fraction (≤30%–40%) of the population with a clinic BP elevation in which trials have shown treatment to have a major protective effect.15 Based on our present observations, this may even more likely be the case in individuals with a partial WCH because their risk is close to that of hypertensive individuals and because their higher out-of-office BP values (home, +23/13 mm Hg; 24 hours, +9/+6 mm Hg versus NT) make a sizeable out-of-office BP reduction with treatment more likely. The answer will have to be provided by randomized trials. Finally, in the partial WCH group, subjects with a home BP elevation had a more clear increase in risk of fatal events than patients with an ambulatory BP elevation. Although supporting the prognostic importance of home BP (despite the availability, in our study, of only 2 home BP values), this does not allow to conclude for its greater prognostic superiority versus ambulatory BP because the subgroups were small and their size unbalanced, the one with an ambulatory BP elevation being only about half of that with a home BP elevation.

Our study has several limitations: (1) BP and other relevant data were available only at baseline; (2) information did not extend to nonfatal CV events; and (3) although confirmed by the much larger database on all-cause mortality, the number of CV fatal events was small. More CV outcome data will thus be desirable to confirm, in particular, whether the CV risk of individuals with true WCH is indeed indistinguishable from the NT population. In this context, it should be emphasized that (1) in true WCH, antihypertensive treatment, with its possible protective effect, was much more frequent than in normotensive individuals; (2) WCH was defined by somewhat lower ambulatory and home BP values than those suggested by guidelines, which may have reduced the associated CV risk, and (3) not only partial but true WCH individuals exhibited a significantly greater risk of developing sustained HT than normotensive subjects. This did not result in a greater number of fatal events, but it may have caused an increased risk of less severe CV complications, which we could not address because our data did not include nonfatal events. Overall, it seems possible to suggest that also in true WCH subjects special attention might be desirable.

**Perspective**

In the Pamela population study, the long-term risk of CV and all-cause mortality exhibited by individuals with a WCH as usually diagnosed in clinical practice (high office and normal ambulatory or home BP) was markedly elevated compared with normotensive individuals, although it has been less than in subjects with sustained HT (elevation of in- and out-of-office BP). However, within the WCH group, no significant increase in risk of mortality was exhibited by individuals in whom both home and ambulatory BP were normal (true WCH), thereby being limited to those in whom one BP was normal but the other was elevated (partial WCH). Thus, as far as the risk of mortality is concerned, WCH represents an inhomogeneous population. To identify those with higher and lower risk among WCH individuals, information on both ambulatory and home BP is necessary.

**Disclosures**

None.
References


Novelty and Significance

What Is New?

• This is the first study to assess, over the longest follow-up ever done (16 years), the prognostic value of white coat hypertension (WCH). It is also the first study that assesses cardiovascular risk in 2 WCH subgroups, that is, those in whom both out-of-office blood pressure (BP) values were normal (true WCH) and those in whom one was found to be normal but the other was elevated (partial WCH).

What Is Relevant?

• Compared with normotensives, the risk of cardiovascular and all-cause mortality was markedly increased in WCH diagnosed as done in clinical practice, that is, by means of office and 1 out-of-office BP only. The increased risk, however, was because of individuals with partial WCH, those with true WCH exhibiting a rate of fatal events not different from the normotensive population.

• The risk of developing hypertension, however, is increased in both conditions.

Summary

As diagnosed in clinical practice, WCH is associated with a clear-cut long-term increase of cardiovascular risk. However, within this condition, individuals with a marked or little increase in risk coexist. Their identification can be obtained by distinguishing those with both home and ambulatory BP normality from those in whom normality is limited to 1 out-of-office BP only. This suggests that a diagnostic approach based on both these BP values is desirable.
Long-Term Prognostic Value of White Coat Hypertension: An Insight From Diagnostic Use of Both Ambulatory and Home Blood Pressure Measurements
Giuseppe Mancia, Michele Bombelli, Gianmaria Brambilla, Rita Facchetti, Roberto Sega, Elena Toso and Guido Grassi

Hypertension. 2013;62:168-174; originally published online May 28, 2013;
doi: 10.1161/HYPERTENSIONAHA.111.00690

Hypertension is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2013 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/62/1/168

Data Supplement (unedited) at:
http://hyper.ahajournals.org/content/suppl/2013/05/28/HYPERTENSIONAHA.111.00690.DC1

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/
LONG-TERM PROGNOSTIC VALUE OF WHITE COAT HYPERTENSION – AN INSIGHT FROM DIAGNOSTIC USE OF BOTH AMBULATORY AND HOME BLOOD PRESSURE MEASUREMENTS.

1,2 Giuseppe Mancia; 3 Michele Bombelli; 3 Gianmaria Brambilla;
3 Rita Facchetti; 1 Roberto Sega; 3 Elena Toso; 3,4 Guido Grassi.

1 Department of Health Sciences, University of Milano-Bicocca; 2 IRCCS Istituto Auxologico Italiano, Milan; 3 Clinica Medica, University of Milano-Bicocca, Monza (Milan); 4 IRCCS Multimedica, Sesto San Giovanni (Milan), Italy

Running title: Prognostic value of white coat hypertension

Corresponding Author:
Prof. Giuseppe Mancia
Clinica Medica Ospedale S. Gerardo
Via Pergolesi 33,
20090 Monza Italy
tel.: ¬0039 039/233 3357
dfax.: 0039 039 322274
e-mail: giuseppe.mancia@unimib.it
**Supplemental Table S1.** Hazard ratios (HR) with 95% confidence intervals (95%CI) for cardiovascular (CV) and all cause mortality.

The NT group is taken as reference. Data are shown unadjusted and after adjustment per age, gender, blood glucose, total serum cholesterol, smoking, previous CV events, antihypertensive treatment, body mass index. “↑” indicates BP elevation.

### CV mortality

<table>
<thead>
<tr>
<th>Groups</th>
<th>N</th>
<th>Deaths</th>
<th>not adjusted</th>
<th>p-value</th>
<th>fully adjusted</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>NT</td>
<td>825</td>
<td>8</td>
<td>1 (ref)</td>
<td></td>
<td>1 (ref)</td>
<td></td>
</tr>
<tr>
<td>True</td>
<td>164</td>
<td>2</td>
<td>1.32 (0.28-6.21)</td>
<td>0.2415</td>
<td>0.67 (0.14-3.19)</td>
<td>0.6157</td>
</tr>
<tr>
<td>Partial WCH (Home ↑)</td>
<td>150</td>
<td>15</td>
<td>12.01 (5.09-28.33)</td>
<td>0.0134</td>
<td>2.83 (1.13-7.09)</td>
<td>0.0261</td>
</tr>
<tr>
<td>Partial WCH (24h ↑)</td>
<td>77</td>
<td>4</td>
<td>5.60 (1.69-18.60)</td>
<td>0.5608</td>
<td>2.57 (0.76-8.69)</td>
<td>0.1294</td>
</tr>
<tr>
<td>HT</td>
<td>373</td>
<td>48</td>
<td>15.59 (7.37-32.97)</td>
<td>0.0350</td>
<td>3.01 (1.35-6.73)</td>
<td>0.0073</td>
</tr>
</tbody>
</table>

### All-cause mortality

<table>
<thead>
<tr>
<th>Groups</th>
<th>N</th>
<th>Deaths</th>
<th>not adjusted</th>
<th>p-value</th>
<th>fully adjusted</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>NT</td>
<td>825</td>
<td>53</td>
<td>1 (ref)</td>
<td></td>
<td>1 (ref)</td>
<td></td>
</tr>
<tr>
<td>True</td>
<td>164</td>
<td>22</td>
<td>2.19 (1.33-3.60)</td>
<td>0.0020</td>
<td>1.35 (0.82-2.24)</td>
<td>0.2415</td>
</tr>
<tr>
<td>Partial WCH (Home ↑)</td>
<td>150</td>
<td>44</td>
<td>5.30 (3.55-7.91)</td>
<td>&lt;0.0001</td>
<td>1.74 (1.12-2.69)</td>
<td>0.0134</td>
</tr>
<tr>
<td>Partial WCH (24h ↑)</td>
<td>77</td>
<td>11</td>
<td>2.31 (1.21-4.43)</td>
<td>0.0114</td>
<td>1.22 (0.63-2.36)</td>
<td>0.5608</td>
</tr>
<tr>
<td>HT</td>
<td>373</td>
<td>112</td>
<td>5.44 (3.92-7.55)</td>
<td>&lt;0.0001</td>
<td>1.50 (1.03-2.18)</td>
<td>0.0350</td>
</tr>
</tbody>
</table>