Longitudinal Study of Left Ventricular Mass Growth
Comparative Study of Clinic and Ambulatory Systolic Blood Pressure in Chronic Kidney Disease

Rajiv Agarwal

Abstract—Left ventricular (LV) hypertrophy is an established cardiovascular risk factor, yet little is known about its trajectory in people with chronic kidney disease. The goal of this prospective research study was to describe the trajectory of LV mass index, its relationship with blood pressure (BP), and specifically to compare the relationship of BP measured in the clinic and 24-hour ambulatory BP monitoring with LV mass index. Among 274 veterans with chronic kidney disease followed for over ≤4 years, the rate of growth of log LV mass index was inversely related to baseline LV mass index; it was rapid in the first 2 years, and plateaued subsequently. Systolic BP also significantly increased, but linearly, 1.7 mmHg/y by clinic measurements and 1.8 mmHg/y by 24-hour ambulatory BP. Cross-sectional and longitudinal associations of both clinic BP and 24-hour ambulatory BP with LV mass index were similar; both BP recording methods were associated with LV mass index and its growth over time. Controlled hypertension, masked uncontrolled hypertension, and uncontrolled hypertension categories had increasing LV mass index when diagnosed by 24-hour ambulatory and awake BP (P<0.05 for linear trend) but not sleep BP. After accounting for clinic BP both at baseline and longitudinally, LV mass index among individuals was additionally predicted by the difference in sleep systolic BP and clinic systolic BP (P=0.032). In conclusion, among people with chronic kidney disease, the growth of LV mass index is rapid. Research-grade clinic BP is useful to assess LV mass index and its growth over time. (Hypertension. 2016;67:710-716. DOI: 10.1161/HYPERTENSIONAHA.115.07052.)

Key Words: ambulatory blood pressure monitoring ■ blood pressure ■ cardiovascular disease ■ hypertension ■ longitudinal studies

Hypertension occurs in at least 70%1 of people with chronic kidney disease (CKD), which is estimated to affect 11% (19.2 million) of the US adult population2; CKD is associated with a large and disproportionate burden of cardiovascular morbidity and mortality.3 CKD rivals diabetes mellitus as a coronary risk equivalent in veterans.4 Established risk factors for cardiovascular disease include left ventricular (LV) mass and blood pressure (BP).5 7 Although both LV mass and BP are powerful cardiovascular risk factors, little information is available on how LV mass evolves among people with CKD.5 8 The cross-sectional relationship between LV mass and BP is well recognized but among those with CKD how LV mass changes over time and how BP predicts this change remains poorly understood.

Accumulating evidence suggests that BP measurements made outside the clinic may provide prognostically superior information.10 13 However, the comparative value of BP obtained in the clinic and that obtained using 24-hour ambulatory BP in assessing LV mass index is unclear. People receiving antihypertensive therapy who have hypertension out-of-office but have normal BP in the clinic are said to have masked uncontrolled hypertension (MUCH).14 An earlier report from my group has reported that the prevalence of MUCH is strongly dependent on the level of clinic BP.15 Thus, those who repeatedly have a low clinic systolic BP (<110 mmHg) are unlikely to have MUCH. However, among those with usual clinic BP of 130 to 139 mmHg, MUCH is prevalent in 2 of 3 and those with usual clinic BP of 120 to 129 mmHg, MUCH is prevalent in 1 of 3. Whether MUCH diagnosed by ambulatory BP monitoring is associated with an increased LV mass is unknown.

In this study, I explored the trajectory and pattern of growth of LV mass, clinic systolic BP, 24-hour ambulatory BP, including sleep and awake BP. Whether ambulatory BP measurement is superior to clinic BP was explored by asking the following questions: (1) compared with clinic BP, is there a stronger association between target organ damage and ambulatory BP; (2) does ambulatory BP-diagnosed MUCH associate with LV mass index and if so by which definition (24-hour, awake or sleep ambulatory BP); and (3) compared with clinic BP, is there an incremental value of ambulatory BP (24-hour, awake, or sleep) in detecting and predicting target organ damage.
Methods
Details of this cohort has been previously published. Briefly, this was a prospective research study to understand the prevalence and mechanisms of MUCH among patients with stages 2 through 4 CKD (estimated glomerular filtration rate defined using the MDRD [Modification of Diet in Renal Disease] equation <90 mL/min per 1.73 m² but >15 mL/min per 1.73 m²). For those with stage 2 CKD, albuminuria (A2 or >300 mg/g creatinine) was required for inclusion in the cohort. Those with an initial clinic BP of ≤140/90 mm Hg were considered eligible and studied further. However, ≤10% of people with a single clinic BP of ≤140/90 mm Hg were found to be hypertensive on further evaluation but they were not excluded.

After obtaining a clinical history, performing a physical examination, and obtaining basic laboratory tests, measurements of BP in the clinic (average of 3 visits), and by 24-hour ambulatory monitoring (24-hour average) were performed as reported earlier. Echocardiogram was performed during 1 of the 3 research visits, all of which were >8 days. BP measurements in triplicate were also obtained independent of the 3 clinic visits at the time of the echocardiogram. BP obtained echocardiogram was called the single visit clinic BP. Specifically, this BP did not specify 5 minutes of seated rest before its measurement.

Classification of Hypertension
The original definition of masked hypertension proposed by Pickering et al16 and one used by the International Database on Ambulatory Blood Pressure in Relation to Cardiovascular Outcomes (IDACO) investigators17 do not take into account BP recordings at night. Accordingly, MUCH was defined as controlled clinic BP (<140/90 mm Hg on average of 3 clinic visits by oscillometric BP measurement) but elevated ambulatory BP.18 Elevated ambulatory BP was defined as elevated day time (≥135/85 mm Hg) BP. To define day time and night time we used patient diaries. Elevated ambulatory BP was also defined in 2 other ways: (1) elevated 24-hour (≥130/80 mm Hg) and (2) either elevated day time as defined above or elevated night time (≥120/70 mm Hg) ambulatory BP.

Echocardiographic Measurements
Two-dimensional–guided M-mode echocardiograms were performed using standard methods20; LV mass was calculated with a previously validated formula.20 For details please see Appendix S1 in the online-only Data Supplement.

Statistical Analysis
Linear mixed models were used to assess the trajectory of LV mass index and BP over time.19 Details of the statistical methods are provided in the Appendix S1.

All statistical analyses were done with Stata 14.0 (Stata Corp, College Station, TX). Nominal level of statistical significance was taken as 2-sided P of 0.05.

Results
Baseline characteristics of participants are shown in Table 1. As expected of a veteran population, participants were older, mostly White men, and two thirds were past smokers. Notable was a high prevalence of diabetes mellitus and cardiovascular disease ascertained by review of medical records. The average estimated glomerular filtration rate was 44 mL/min per 1.73 m² and the median urinary albumin/creatinine ratio was 30 mg/g (interquartile range, 6–230 mg/g). All but 6 participants were receiving antihypertensive drugs for BP control and the average number of antihypertensive drugs used was 3.1. At baseline, BP taken during a single clinic visit at the time of the echocardiogram was 134/72 mm Hg compared with that averaged ≥3 visits, 121/60 mm Hg. On average, the 24-hour ambulatory BP was 127/69 mm Hg (122/65 mm Hg during sleep and 129/72 mm Hg during the wake state). Study flow chart is shown in Figure S1.

Growth Models of LV Mass Index and Ambulatory Systolic BP
Figure 1 shows the individual plots of each participant for the LV mass index, clinic and ambulatory BP measurements. The mean change is shown by the superimposed linear regression line. A linear change seems apparent for BP and an asymptotic change for LV mass index. Figure S2 shows the relationship between slopes and intercepts derived from each individual participant by ordinary least squares regression. Error bars of the slopes are shown where ≥2 measurements of LV mass or BP were available (Figure 2). Slopes of LV mass index showed an inverse log–linear relationship with baseline LV mass index. No participant had a decline in LV mass index if the baseline LV mass index was <125 g/m². Furthermore, notable is that at 4 years few participants had increments in LV mass index. Slopes of BP showed an inverse relationship with baseline BP in each case.

Table 2 shows the taxonomy of models for LV mass index, clinic and ambulatory BP measurements. In each case, the unconditional means model is described followed by the growth curve. The unconditional means model accounts for the nested nature of the observations within participants. The natural log of LV mass index was taken as the outcome variable to normalize the data. The unconditional geometric mean LV mass index was 141.6 g/m². The between-subject SD of the log-transformed data may be interpreted as coefficient of variation, or test–retest reliability, which was 16.5%. There was a quadratic relationship of LV mass index with time, with rapid growth in the first 2 years, and subsequent slowing. The negative time² term indicates slowing of growth over time. Single clinic systolic BP showed large amount of variability within participants with intraclass correlation coefficient over years approaching zero. Multiple research-grade clinic BP obtained after 5 minutes of seated rest averaged 122.3 with between-subject SD of 7.5 mm Hg. The magnitude of increase in 24-hour ambulatory BP was similar at 1.8 mm Hg/y; however, the intraclass correlation coefficient was 0.25. On average this research-grade clinic BP increased 1.7 mm Hg/y. The magnitude of increase in 24-hour ambulatory BP was similar at 1.8 mm Hg/y; however, the intraclass correlation coefficient was 0.43. Notably, the growth in sleep ambulatory BP was 2.4 mm Hg/y, which was 60% more than that seen during the awake state. Furthermore, compared with awake ambulatory systolic BP, there was greater test–retest reliability for sleep ambulatory BP (intraclass correlation coefficient for awake 0.36 and sleep 0.48).

LV Mass Index and Its Relationship With Clinic and Ambulatory BP
Figure S2 (Appendix S1) shows the directional changes in systolic BP and LV mass index. Most arrows point in to the right and up suggesting a simultaneous growth in systolic BP and LV mass index. The relationship of systolic BP growth and LV mass index was analyzed formally using mixed models and are summarized in Figure 3. In case of single clinic BP measurement, every 10 mm Hg increase in systolic BP was associated with 2.6% increase in LV mass index (this is shown as intercept). Intraclass increase in 10 mm Hg increase in clinic systolic BP was associated with nonsignificant 2.1% growth in LV mass index. In contrast, multiple research-grade clinic BP measurements were associated with 2.9% greater LV mass at baseline and 4.8% growth over time. These results
were significant as noted by the P values and the 95% confidence intervals not crossing 0. Similar results were seen for 24-hour ambulatory BP (3.9% between and 4.7% within individual change in LV mass index). However, intraindividual growth in sleep ambulatory BP was not associated with increase in LV mass index (P=0.14).

Next, the incremental value of 24-hour, sleep and wake ambulatory BP in predicting LV mass index was assessed (Figure 4). The masked hypertension effect (ambulatory minus clinic systolic BP) was calculated for each individual. The change from baseline in this value over time was then calculated. After accounting for between and within individual change in clinic systolic BP, every 10 mmHg cross-sectional difference in masked hypertension effect was associated with 3.5% increase in LV mass index in case of 24-hour ambulatory BP (P=0.042), 2.8% increase in LV mass index in case of sleep ambulatory BP (P=0.032) but a nonsignificant increase of 3.0% for awake ambulatory BP (P=0.09). Intraindividual longitudinal growth in masked hypertension effect was not associated with LV mass growth (<2% change, P>0.5) for any comparison. Although the mean masked hypertension effect at baseline was 5.5 mmHg (95% confidence interval, 4.1–6.9), there was no change from baseline in masked hypertension effect 0.14 mmHg/y (95% confidence interval, −0.9 to 0.12; Figure S4; Appendix S1).

Finally, the magnitude of change in systolic BP during sleep was related to LV mass; this relationship was seen cross-sectionally and longitudinally. After accounting for daytime systolic BP among individuals and its growth over time, no independent association was found between dipping and LV mass differences cross-sectionally (2.4% change/10 mmHg dipping, P=0.19) or longitudinally (0.002% change/10 mmHg dipping, P=0.94).

**Discussion**

The major findings of this study are as follows: among 274 veterans with CKD followed for over ≤4 years, the rate of growth of log LV mass index was inversely related to baseline LV mass index; it was rapid in the first 2 years, and plateaued subsequently. Systolic BP also significantly increased, not log-linearly but linearly, 1.7 mmHg/y by research-grade clinic measurements, and 1.8 mmHg/y by 24-hour ambulatory BP. The growth in sleep systolic BP was 60% more than awake ambulatory BP. Cross-sectional and longitudinal associations of either clinic BP or 24-hour ambulatory BP with LV mass index were similar. Controlled hypertension, MUCH, and uncontrolled hypertension categories had increasing LV mass index when the diagnosis was made using awake or 24-hour ambulatory BP, but not when using sleep BP. After accounting for research-grade clinic BP both cross-sectionally and longitudinally, LV mass index among individuals was additionally predicted by the difference in sleep systolic BP and...
Dipping was not additionally predictive of LV mass index.

The growth in LV mass index was rapid in the first 2 years and suggests that patients with CKD accumulate cardiovascular risk much before they reach dialysis. That this risk is related to systolic BP is not surprising and is consistent with sparse evidence from 2 studies in patients with CKD not on dialysis. The first study was an 8-center Canadian cohort study of 246 patients with CKD followed for 12 months. It reported that the growth in LV mass was related to both an increase in systolic BP and a lower baseline LV mass. The second study was a multicenter randomized trial of anemia correction in Australia and New Zealand that included 155 patients with CKD allocated equally to target hemoglobin of 9 to 10 or 12 to 13 g/dL. It reported that the change from baseline to 2 years in LV mass index in the 2 groups was 4.5±20 and 2.5±20 g/m², respectively. Those with CKD followed for 12 months. It reported that the growth in LV mass was related to both an increase in systolic BP and a lower baseline LV mass. The second study was a multicenter randomized trial of anemia correction in Australia and New Zealand that included 155 patients with CKD allocated equally to target hemoglobin of 9 to 10 or 12 to 13 g/dL. It reported that the change from baseline to 2 years in LV mass index in the 2 groups was 4.5±20 and 2.5±20 g/m², respectively. Those with

Figure 1. Trajectories of left ventricular mass index and blood pressure (BP) >4 years. Lines represent each individual participant. The number on the x axis just above the time line are the number of participants on each occasion. Circles at time zero are those who had a baseline measurement. After a brief history and physical examination, participants had BP measured in a seated position. The technique of home BP (HBP) monitoring was explained and a self-inflating oscillometric device was dispensed. For 1 week, each participant recorded HBP twice daily. Ambulatory BP monitoring was performed >24 hours. After 1 month hiatus, the study was repeated as in the initial month.

Figure 2. Relationship of ordinary least squares (OLS) slopes and intercepts for individual participants. Error bars represent the SE of the estimate in those participants who had 3 measurements. Baseline left ventricular mass index (LVMI) is plotted on a log scale. All participants who had baseline LVMI of <125 g/m² had growth in LVMI, whereas some with a mass >125 g/m² at baseline had regression of LVMI. SysBP indicates systolic blood pressure.
pre-existing LV hypertrophy had regression of LV mass index and those who had none, had growth in LV mass. The authors reported that there was 3.6% per year change in LV mass index (adjusted for height^2) in the lower hemoglobin group and 0.7% per year in the higher hemoglobin group (P=0.09). Difference in patient characteristics (≈20% diabetes mellitus, >50% women, and age 54 years), participation in randomized trial where BP control was prespecified, and differences in statistical methods may account for slower growth rate in LV mass index in the Australian patients. Besides these 2 sizable longitudinal observations in CKD, the evidence for change in BP and LV mass index is sparse. Specifically, the relationship of ambulatory BP measured BP with echocardiographic LV mass growth is absent.

This study fills some of these knowledge gaps. The study noted that ambulatory BP was similar to research-grade oscillometric clinic systolic BP (identical to the method used in the SPRINT [Systolic Blood Pressure Intervention Trial]) in determining LV mass index at baseline and its growth over time. However, it is to be noted that still most clinical decisions to treat or not to treat hypertension are based on either a single BP measurement made at a clinic visit. A single clinic BP measurement performed at the time of echocardiogram that did not specify 5 minutes of seated rest was unable to detect the growth of LV mass over time. This indicates that multiple visits (3 visits each BP recording in triplicate >1 week after 5 minutes of seated rest) are at least required for the detection of growth of LV mass index over time.

The study illuminates the relationship between awake and sleep ambulatory BP in detecting growth of LV mass over time. Despite the 60% greater longitudinal growth of sleep BP over time, the value of awake ambulatory BP was greater. Wake and sleep BP may reflect different domains of cardiovascular

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Table 2. Taxonomy of Models for LVMI and Systolic Ambulatory BP

BP indicates blood pressure; and LVMI, left ventricular mass index.
disease. Wake BP is influenced by activity; in CKD we have previously reported that activity-induced changes in BP are greater in those who have less physically active.22 Thus, greater wake BP may reflect a more sedentary lifestyle, which by itself is a potent cardiovascular risk factor. Masked hypertension effect (the magnitude of elevation of systolic ambulatory BP over clinic BP) was an independent predictor of LV mass index after accounting for clinic BP taken at multiple visits. Notably, masked effect was significant for sleep BP but not awake BP. Given that there is not much variation in the growth of masked effect, it is not surprising that there is little statistical significance of masked effect growth on LV mass growth. Finally, we did not find an independent value of dipping on LV mass or its growth over time. This is in contrast to an investigation that we did not find an independent value of dipping on LV mass or effect, it is not surprising that there is little statistical significance of masked effect growth on LV mass growth. Finally, we did not find an independent value of dipping on LV mass or its growth over time. This is in contrast to an investigation that found a cross-sectional association of dipping an LV mass.23

Figure 4. Mixed model estimates of percent change in left ventricular (LV) mass index per 10 mmHg change in clinic systolic blood pressure (sys-BP) and 10 mmHg change in masked effect. Masked effect was calculated as ambulatory (24 hours, sleep or wake) minus clinic systolic BP. Sleep and 24-h ambulatory masked effect were predictive of LV mass index in the cross-sectional coefficient. ABPM indicates ambulatory BP monitoring.

There are several strengths of our study: our study used 24-hour ambulatory BP monitoring, the gold standard method to diagnose out-of-office hypertension. Our study was prospective; it carefully collected information on echocardiographic LV mass solely for the purposes of the study. BP pattern in each individual using several BP monitoring methods, used both a single visit at the time of echocardiogram and multiple clinic visits to define clinic hypertension, and had a high completion rate.

The 4 clinical implications of our findings are as follows: (1) among patients with CKD, nearly all of whom were taking antihypertensive medications, LV mass growth is rapid as is increase in both research-grade clinic and ambulatory BP. Given the growth of both BP and LV mass are concordant, attempts to control hypertension may be useful to abrogate the rate of LV mass over time, (2) sleep-time BP may add additional value to detect target organ damage and it remains to be seen whether treatment of sleep BP can mitigate LV mass, (3) MUCH diagnosed by ambulatory BP during the wake state or >24 hours but not during sleep is supported by the study, and (4) the consistency of masked hypertension effect over time suggests a biological basis for MUCH.16

Perspectives

Among people with CKD, the growth of LV mass index is rapid. Clinic BP obtained at multiple visits and one that adheres to the guidelines to measure BP is useful to assess LV mass index and its growth over time. Clinic BP recordings performed exceptionally well in this study, in part, because of multiple measurements in triplicate at each visit that were used as predictors. In the usual day-to-day care of patients, it is unlikely that clinic BP will have such predictive power. Nonetheless, the study illustrates the value of research-grade clinic BP in its ability to predict between people and within individual change in target organ damage. Ambulatory BP monitoring during sleep may be of additional value in detecting variations among individuals in LV mass index. In patients with CKD, this study supports using awake BP in making a diagnosis of MUCH.

Acknowledgments

I thank the participants for their time and effort, and dedicate this work to the memory of J. Michael Bouldin, RDSCS who performed many of the echocardiograms reported in this study.
Sources of Funding
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Disclosures
Dr Agarwal has consulted for several pharmaceutical companies that make antihypertensive drugs, including Merck, Takeda, Novartis, Daiichi Sankyo, Abbvie, Bayer, and Johnson and Johnson.

References

Novelty and Significance
• Ambulatory BP monitoring during sleep may be of additional value in detecting variations among individuals in LV mass index.

Summary
Clinic BP obtained at multiple visits and one that adheres to the guidelines to measure BP is useful to assess LV mass index and its growth over time. Clinic BP recordings performed exceptionally well in this study, in part, because of multiple measurements in triplicate at each visit that were used as predictors. In the usual day-to-day care of patients, it is unlikely that clinic BP will have such predictive power. Nonetheless, the study illustrates the value of research grade clinic BP in its ability to predict between people and within individual change in target organ damage.
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A longitudinal study of left ventricular mass growth: a comparative study of clinic and ambulatory systolic BP in CKD

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Echocardiographic measurements

Two-dimensional guided M-mode echocardiograms were performed by an accredited technician during one of the study visits with a digital cardiac ultrasound machine (Cypress Acuson, Siemens Medical). The protocol specified recording of at least 12 cycles of 2-dimensional parasternal long- and short-axis LV views with optimal orientation of the cursor beam used to derive additional M-mode recordings. Each patient underwent six M-mode measurements of interventricular septal thickness in diastole (IVSTd), LV internal diameter in diastole (LVIDd) and systole (LVIDs), LV posterior wall thickness in diastole (LVPWd) and systole (LVPWs) and left atrial (LA) diameter using standards of the American Society of Echocardiography (1). LV mass was calculated with a previously validated formula (2):

\[
LV\ mass\ (g) = 0.832 \times [(IVSTd+LVIDd+PWTd)^3-(LVIDd)^3]+0.60
\]

Statistical methods

Line graphs of individual level data were generated and relationships of slopes and intercepts plotted prior to model fitting. Linear mixed models were used to assess the trajectory of LV mass index and BP over time (3). Examination of the plots suggested a greater variation in the slopes among those who had 2 year studies compared to those who had 4 year studies. A random coefficient model was fitted where the coefficient term was restricted to those with two year follow up (4). BP was modeled separately for each measurement type (single clinic, multiple clinic, 24h ambulatory, awake ambulatory, sleep ambulatory). Because all BP measurements were obtained in the same participants, the beta coefficients for LV mass growth were not multiply adjusted for differences in confounders. This was because I was not asking the question, what the independent predictors of LV mass growth are. I was simply evaluating various methods of BP measurement as to their comparative ability to predict LV mass growth. When comparing different BP measurements and outcomes, a similar approach has been adopted by us and others (5;6)

Specifically, the following models were fitted:

\[
\ln\{LVMI\}_{ij} = \beta_0 + u_{0i} + \beta_1 \times years_{ij} + \beta_2 \times years_{ij}^2 + u_{1i} \times years_{ij} \\
\times shortstudy_i + e_{ij}
\]

\[
SysBP_{ij} = \beta_0 + u_{0i} + \beta_1 \times years_{ij} + u_{1i} \times years_{ij} \times shortstudy_i + e_{ij}
\]

To examine the association of change in BP with the change in LV mass index, plots of change in each variable were first generated. Mixed linear models were then used where the baseline BP and its change from baseline were modeled.
To assess the independent relationship of ambulatory BP over and above clinic BP, ambulatory minus clinic BP was calculated for each occasion. Baseline and change from baseline in these differences were then modeled as follows:

$$\ln[LVMI]_{ij} = \beta_0 + u_{0i} + \beta_1 \times years_{ij} + \beta_2 \times years_{ij}^2 + \beta_3 \times baselineSBP_i + \beta_4 \times changeSBP_{ij} + u_{1i} \times years_{ij} \times shortstudy_i + e_{ij}$$

To assess differences in log LV mass index among hypertension categories a mixed model was again used. CH, MUCH, and UCH (coded as 0, 1 and 2 respectively) were tested using orthogonal contrasts for linearity.

Various covariance structures were compared using nested models and the likelihood ratio test. An unstructured covariance matrix where slopes and intercepts are allowed to vary independently of each other was found to have the best model fit and was used for all models. Data were modeled using the maximal likelihood estimation.

Reference List


Figure S1: Study Flow. There were 334 patients with CKD who were consented between July 25, 2007 and March 14, 2014. The blue circle represents those who had ambulatory BP (ABPM) recordings, the yellow circle who had echocardiographic LV mass index measurements, and the overlap denotes those who had both. Both echocardiograms and blood pressure recordings were available at baseline in 274, at year 2 in 73 and at year 4 in 19 participants.
Figure S2: Change in LV mass index as a function of change in systolic BP. The grey arrows show the change from baseline to 2 years in systolic BP in relationship to LV mass index. The maroon arrows lines show the change from baseline to 4 years in the corresponding measurements.
Figure S3: Relationship of left ventricular mass index with controlled hypertension (CH), masked uncontrolled hypertension (MUCH), and uncontrolled hypertension (UCH) diagnosed by daytime ambulatory BP. A linear trend was present between increasing severity of hypertension and LV mass index when the conventional definition (p=0.012) or 24-h ABPM (p=0.019) was used to diagnose MUCH.
Figure S4: Masked hypertension effect and its change over time. Plotted is the difference between 24 hour ambulatory and clinic systolic BP ordered by increasing mean masked hypertension effect. Hollow circles indicate baseline masked hypertension effect in those who had 2 year visits. Crosses indicate baseline masked hypertension effect in those who had 4 year visits in addition. The line connects the mean effect. The arrow indicates the magnitude and direction of change from baseline visit. The mean baseline masked hypertension effect was 5.5 mmHg (95% CI 4.1 to 6.9). The change over time was 0.14 mmHg/year (95% CI –0.9 to 1.2, p>0.9)