Twenty-Four–Hour Ambulatory Blood Pressure versus Clinic Blood Pressure Measurements and Risk of Adverse Outcomes in Children with CKD

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Abstract
Background and objectives Our objective was to determine whether clinic BPs (taken at either a single visit or two sequential visits) are inferior to ambulatory BPs in their ability to discriminate risk of adverse outcomes in children with CKD.

Design, setting, participants, & measurements We included 513 participants of the CKD in Children Study who had clinic BPs and 24-hour ambulatory BP monitoring performed during similar timeframes. Predictors of interest were systolic BPs taken at a single visit or two repeated visits within a 1-year period compared with mean wake and sleep systolic ambulatory BPs. Outcomes were left ventricular hypertrophy and ESKD. We determined the ability for each BP parameter to provide risk discrimination using c statistics.

Results During mean follow-up of 3.5 years, 123 participants developed ESKD. In cross-sectional unadjusted analysis, every 0.1 increase in systolic BP index was associated with a 2.0 times higher odds of left ventricular hypertrophy (95% confidence interval, 1.5 to 2.8) by clinic BPs versus 1.8 times higher odds (95% confidence interval, 1.3 to 2.4) by ambulatory wake BP. This statistic was highest for clinic BP (c=0.65; 95% confidence interval, 0.58 to 0.73) but similar to ambulatory wake BP (c=0.64; 95% confidence interval, 0.57 to 0.71) for the discrimination of left ventricular hypertrophy. In longitudinal unadjusted analysis, every 0.1 increase in systolic BP index was associated with a higher risk of ESKD using repeated clinic (hazard ratio, 1.5; 95% confidence interval, 1.3 to 1.8) versus ambulatory wake BP (hazard ratio, 1.6; 95% confidence interval, 1.3 to 2.0). Unadjusted c statistics were the same for wake (c=0.61; 95% confidence interval, 0.56 to 0.67) and clinic systolic BPs (c=0.61; 95% confidence interval, 0.55 to 0.66) for discriminating risk of ESKD.

Conclusions Clinic BPs taken in a protocol-driven setting are not consistently inferior to ambulatory BP in the discrimination of BP-related adverse outcomes in children with CKD.


Introduction
Ambulatory BP monitoring is considered the gold standard metric for the diagnosis of hypertension in children (1). However, the logistic burden of performing ambulatory BP monitoring (availability of equipment and obtaining and returning the device), its associated discomfort (2), and poor reimbursement by insurers (primarily indicated for suspected white coat hypertension) (3) render ambulatory BP monitoring more cumbersome to perform than clinic BP measurements. Realistically, it is challenging to repeat ambulatory BP monitoring with the same frequency as clinic visits to confirm continued BP control in children.

Most prior observational studies comparing ambulatory BP monitoring with clinic-based BP monitoring have primarily used BP readings taken at a single visit as a comparator against ambulatory-based BPs in children and adults (4–16). A few studies in children without CKD have suggested a stronger correlation between ambulatory (versus clinic) BPs and left ventricular hypertrophy (LVH), but most of these studies have been relatively small (17,18). Hence, whether clinic BPs are inferior to ambulatory BPs in the prognostication of outcomes of clinical relevance is unclear, especially given the long duration that it takes for the onset of “hard outcomes” in children.

The objective of this study was to compare clinic BP measurements (at a single visit versus mean of clinic readings at two sequential visits within a 1-year period) against ambulatory-derived BP parameters in terms of the magnitude of their difference and discrimination of the risk for LVH and ESKD in the CKD in Children (CKiD) Study.

Materials and Methods

Study Population
Details of the CKiD Study have been previously described (19). Briefly, the CKiD Study is a prospective, multicenter, observational study of children in North America between ages 1 and 16 years old with eGFR between 30 and 90 ml/min per 1.73 m²

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In this study, we included 513 of 758 participants who had a year 1 visit with clinic BP performed concurrently with echocardiogram and ambulatory BP monitoring and excluded those with missing clinic BP, height, echocardiogram, or ambulatory BP monitoring (n=245). All data were derived from the National Institute of Diabetes and Digestive and Kidney Diseases Central Repository in deidentified form, and patients were censored if they were lost to follow-up or administratively censored as of July of 2014. The University of California Institutional Review Board considers this study exempt from human subjects research.

Predictors of Interest

Clinic BP Ascertainment. All clinic-based BPs were performed by trained and certified personnel by auscultation during the CKiD Study annually using an aneroid sphygmomanometer with an appropriately sized cuff (21). Recertification of personnel obtaining BPs and calibration of BP devices occurred annually. Three consecutive seated readings were obtained at each study visit 30 seconds apart after at least 5 minutes of quiet rest, and the average of these three readings was considered the clinic BP for that visit. The clinic BP from the visit closest in time to ambulatory BP monitoring performance (median time difference between clinic BP and 24-hour ambulatory BP monitoring was 0 days; interquartile range, 0–0) was used as one of the predictors of interest (single clinic BP taken at year 1 visit). All clinic BP readings were indexed to the 95th percentile thresholds for age, sex, and height (to provide comparability with ambulatory BP indices), such that an index of one would represent a clinic systolic BP that was at the 95th percentile for age, sex, and height. This approach is consistent with that of other prior CKiD studies (22,23). Normative clinic BP values used to determine BP indices were derived from the National High BP Education Program Fourth Report as in prior CKiD studies (23,24).

Next, the average of the clinic BP measurements taken at two visits that occurred within a 1-year period after entry into the CKiD Study was included for analysis. The median number of months in between the two clinic visits included for analysis was 7.4 (interquartile range, 5.7–8.7).

Ambulatory BP Monitoring. Ambulatory BP monitoring was performed during the CKiD Study using a SpaceLabs 90217 monitor (SpaceLabs Healthcare), with BPs taken every 20 minutes over a 24-hour period and centrally read as described previously (22,23). All ambulatory BP monitoring performed at year 1 (concurrent with single clinic BP and echocardiogram) were included for analysis. We used mean ambulatory wake and sleep readings as separate predictors of interest. We converted all ambulatory BP measurements into BP indices according to the 95th percentile limits using normative data from Europe as in prior CKiD studies (22,23,25).

BP Parameters of Interest. We were interested in the comparison of four different BP indices in our study, including (1) clinic systolic BPs taken at a single visit, (2) mean of clinic systolic BP readings taken at two study visits within a 1-year period, (3) mean wake ambulatory systolic BPs, and (4) mean sleep ambulatory systolic BPs. Of note, we will focus primarily on systolic BP and its association with adverse outcomes throughout this study given that prior studies have shown the greater importance of systolic BPs as opposed to diastolic BPs in their association with LVH and kidney function decline in children with CKD (26,27).

Outcomes of Interest

LVH. Echocardiograms were performed every 2 years in the CKiD Study, and echocardiogram data concurrent to clinic BP and 24-hour ambulatory BP monitoring at year 1 were included for our primary analysis. M-mode and Doppler echocardiograms were performed by trained technologists using a standardized protocol at the CKiD Study sites (22). We defined LVH using the same definition used in prior CKiD studies, which is a left ventricular mass index greater than or equal to the 95th percentile for normal children and adolescents (22). Our primary analysis of LVH focused on echocardiograms performed at year 1 concurrent to BP measurements, but in secondary analysis, we examined the association between various BP parameters measured at year 1 and presence or absence of LVH at year 3.

Long-Term ESKD Ascertainment. Ascertainment of ESKD onset was performed at annual CKiD Study visits, by phone follow-up, or by provision of information from providers. Patients were administratively censored if they were alive as of July 2014 and had not yet developed ESKD or if they were lost to follow-up (using the last study visit date).

Statistical Analyses

First, we determined the characteristics of participants who had clinic BP measurements, 24-hour ambulatory BP data, and echocardiogram who were included in the study. We then determined the magnitude of the difference between clinic and ambulatory systolic BPs and the Spearman correlation between repeated clinic versus ambulatory systolic BPs.

Second, we used each BP metric in separate regression models to assess their association with two primary outcomes of interest: LVH in cross-sectional analysis (logistic regression) and risk of ESKD in longitudinal analysis (Cox regression). Time to event was determined starting from the date of the year 1 visit (which was the date of echocardiogram performance and clinic BP measurement).

In secondary analysis, among the subset of children who had a second echocardiogram available at year 3, we examined the ability for year 1 clinic and ambulatory BPs to predict LVH at year 3 using logistic regression models.

Unadjusted models were considered our primary analysis given that the comparisons between BP measurements were made within the same patients within the same timeframe. In additional analysis, we subsequently adjusted these models for age (at the closest clinic visit to time of echocardiogram and ambulatory BP monitoring performance), sex, race, cause of CKD, duration of CKD, body mass index z score, eGFR by bedside Schwartz equation, urine protein-to-creatinine ratio, serum albumin, hemoglobin, and use of antihypertensive medications (all ascertained at year 1).

Next, we included both repeated clinic BPs and wake ambulatory BPs as predictors of all outcomes of interest in nested Cox models. We chose to focus only on mean clinic BP measurements taken at repeated visits (as opposed to
single clinic BPs) given that we found stronger associations between repeated clinic BPs and outcomes of interest. We chose to also use wake systolic BPs in these nested models given our finding of their stronger associations with outcomes of interest compared with sleep systolic BPs.

To provide formal tests of the ability of each BP metric to discriminate risk of outcomes of interest by clinic- versus ambulatory-based BP measurements, c statistics were determined for each logistic or Cox model in unadjusted analyses adjusted for the same covariates as described above. In logistic models, the c statistic was described as the area under the receiver operator curve. In Cox models, Harrell c statistics were used, because c statistics or concordance statistics provide a measure of goodness of fit of a model and the likelihood of a randomly selected person having an event versus not having an event on the basis of the predictor of interest (28); 95% confidence intervals (95% CIs) for c statistics and their differences were determined via bootstrapping technique (using 500 repetitions) to evaluate the fit of each Cox model. We used the c statistic for repeated clinic BP measurements as the reference group when determining whether differences in c statistic were statistically significant.

In sensitivity analyses, we repeated adjusted models using eGFR by the CKiD Study equation (on the basis of serum creatinine and cystatin C) (29) for both outcomes of interest.

Table 1. Characteristics of CKD in Children Study participants included for analysis

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Characteristic, n=513</th>
<th>Mean±SD or N (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median age [IQR], yr</td>
<td>13 [9–16]</td>
<td></td>
</tr>
<tr>
<td>Boys</td>
<td>314 (61)</td>
<td></td>
</tr>
<tr>
<td>Race</td>
<td></td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>351 (68)</td>
<td></td>
</tr>
<tr>
<td>Black</td>
<td>74 (14)</td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td>88 (17)</td>
<td></td>
</tr>
<tr>
<td>Cause of CKD</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Glomerular</td>
<td>138 (27)</td>
<td></td>
</tr>
<tr>
<td>Nonglomerular</td>
<td>375 (73)</td>
<td></td>
</tr>
<tr>
<td>Age- and sex-adjusted BMI z score [IQR]</td>
<td>0.3 [−0.4–1.3]</td>
<td></td>
</tr>
<tr>
<td>Median eGFR by bedside Schwartz [IQR], ml/min per 1.73 m²</td>
<td>51 [34–66]</td>
<td></td>
</tr>
<tr>
<td>Median eGFR by CKiD equation [IQR], ml/min per 1.73 m²</td>
<td>51 [37–65]</td>
<td></td>
</tr>
<tr>
<td>Mean serum albumin,&quot; g/dl</td>
<td>4.3±0.4</td>
<td></td>
</tr>
<tr>
<td>Mean hemoglobin, g/dl</td>
<td>12.7±4.4</td>
<td></td>
</tr>
<tr>
<td>Left ventricular hypertrophy at year 1</td>
<td>59 (12)</td>
<td></td>
</tr>
<tr>
<td>Left ventricular hypertrophy at year 3</td>
<td>32 (12)</td>
<td></td>
</tr>
<tr>
<td>Antihypertensive medication use</td>
<td>352 (69)</td>
<td></td>
</tr>
<tr>
<td>BP parameters, mm Hg</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Clinic systolic BP index at a single visit</td>
<td>0.88±0.10</td>
<td></td>
</tr>
<tr>
<td>Systolic BP index (mean±SD)</td>
<td>0.88 [0.82–0.94]</td>
<td></td>
</tr>
<tr>
<td>Median IQR</td>
<td>0.88±0.08</td>
<td></td>
</tr>
<tr>
<td>Systolic BP indices at two clinic visits</td>
<td>0.91±0.08</td>
<td></td>
</tr>
<tr>
<td>Systolic BP index (mean±SD)</td>
<td>0.91 [0.86–0.97]</td>
<td></td>
</tr>
<tr>
<td>Median IQR</td>
<td>0.91±0.08</td>
<td></td>
</tr>
<tr>
<td>ABP awake systolic BP index</td>
<td>0.92±0.10</td>
<td></td>
</tr>
<tr>
<td>Systolic BP index (mean±SD)</td>
<td>0.92 [0.86–0.98]</td>
<td></td>
</tr>
<tr>
<td>Median IQR</td>
<td>0.92 [0.86–0.98]</td>
<td></td>
</tr>
<tr>
<td>ABP sleep systolic BP index</td>
<td>0.96 (11)</td>
<td></td>
</tr>
<tr>
<td>Systolic BP index (mean±SD)</td>
<td>137 (27)</td>
<td></td>
</tr>
</tbody>
</table>

Stata 14 (StataCorp, LLC) was used for the performance of all statistical analyses. P values <0.05 were considered statistically significant for all analyses.

**Results**

Baseline characteristics of the 513 CKiD Study participants included for analysis are shown in Table 1. Median age was 13 years old, 61% were boys, and median eGFR was 51 ml/min per 1.73 m². In general, ambulatory wake and sleep systolic BP measurements were higher than single and repeated clinic systolic BP measurements (Table 1). About 11% of the cohort had BPs that were >95th percentile for age, sex, and height by clinic-based systolic BP, and slightly more than one quarter of the study population had ambulatory BPs that were >95th percentile for sex and height (1).

The absolute mean of repeated systolic BPs was 9 mm Hg lower (SD 11) in the clinic compared with wake ambulatory BP and 4.0 mm Hg higher (SD 12) in the clinic compared with sleep ambulatory BP. The mean difference between clinic and awake systolic BP indices was −0.03 (SD=0.09), with clinic systolic BP being lower, and the mean difference between clinic and sleep systolic BP index was −0.04 (SD=0.10), with clinic systolic BP being lower.

The Spearman correlation between repeated clinic and wake systolic BP indices was 0.44 (P<0.001); the Spearman
correlation between repeated clinic and sleep systolic BP indices was 0.42 (P<0.001).

**BP and LVH**

Approximately 12% of the cohort (n=59) had LVH on their baseline echocardiogram, and this prevalence was unchanged at year 3 (Table 1). All clinic systolic BP measurements (single-visit or repeated visit systolic BPs) and all ambulatory systolic BP parameters (wake and sleep) were statistically significantly associated with the odds of LVH in both unadjusted and adjusted analyses in cross-sectional analysis (Table 2). There was no statistically significant difference between the c statistics of models using ambulatory parameters (24-hour wake: c=0.64; 95% CI, 0.57 to 0.71) and sleep (c=0.63; 95% CI, 0.56 to 0.71) systolic BP) or single-visit BP (c=0.65; 95% CI, 0.56 to 0.73) compared with repeated clinic systolic BPs (c=0.65; 95% CI, 0.58 to 0.73) as a predictor of LVH (Table 2) in unadjusted analyses. Similar findings were noted in adjusted analysis (Table 2).

The c statistic (c=0.67; 95% CI, 0.60 to 0.75) for our unadjusted models that included both repeated clinic systolic BP and wake systolic BP as predictors was slightly higher compared with that for models that only included repeated clinic systolic BPs (c=0.65; 95% CI, 0.58 to 0.73), although this difference did not achieve statistical significance. Addition of wake systolic BP to repeated clinic systolic BP did not improve the c statistic in adjusted models (Table 2). Of note, only repeated clinic systolic BP (odds ratio, 1.8; 95% CI, 1.2 to 2.6) but not wake ambulatory systolic BP (odds ratio, 1.3; 95% CI, 0.9 to 1.9) was statistically significantly associated with LVH in our nested model; similar results were noted in adjusted analyses (Table 2).

In sensitivity analysis, when we repeated our models using the CKiD Study equation-based estimates of GFR, we derived similar results (Supplemental Table 1). In addition, when we repeated our models using year 1 BP parameters to predict LVH at year 3, we again found that clinic BPs were more strongly associated with risk of LVH than wake or sleep BPs and provided higher c statistics (Supplemental Table 2).

**Longitudinal Analyses of ESKD Risk**

During mean follow-up of 3.5 years, 123 of 513 participants developed ESKD. All clinic systolic BP measurements (single or repeated visits) and all ambulatory systolic BP parameters (wake and sleep) were statistically significantly associated with the risk of ESKD in unadjusted analysis (Table 3). The c statistic for either single-visit (c=0.61; 95% CI, 0.55 to 0.66) or repeated clinic visit systolic BPs (c=0.61; 95% CI, 0.55 to 0.66) was slightly higher than that for sleep (c=0.58; 95% CI, 0.52 to 0.64) and the same as wake (c=0.61; 95% CI, 0.56 to 0.67) systolic BP in unadjusted analyses (Table 3), but these c statistics were not statistically significantly different.

In unadjusted models including both repeated clinic and wake systolic BPs as predictors in the same model, the c statistic (c=0.62; 95% CI, 0.57 to 0.68) was slightly higher than the c statistic for models including only repeated clinic systolic BPs, although again, this difference did not achieve statistical significance (Table 3). Addition of wake systolic BP to repeated clinic systolic BP in adjusted models also did not improve the c statistic (c=0.90; 95% CI, 0.86 to 0.92). In nested models, both repeated clinic (hazard ratio, 1.3; 95% CI, 1.1 to 1.6) and wake systolic BPs (hazard ratio, 1.4; 95% CI, 1.1 to 1.8) were associated with risk of ESKD in unadjusted analysis (Table 3).

In sensitivity analysis, when we repeated our models using the CKiD Study equation to estimate GFR, results were similar in unadjusted and adjusted analyses for the risk of ESKD (Supplemental Table 1).

**Discussion**

The American Heart Association guidelines currently consider ambulatory BP monitoring the gold standard metric for the assessment of BP in children (1,30). The rationale for this guideline is on the basis of the stronger association between ambulatory BP measurements and target organ damage seen in children with and without CKD compared with clinic BPs in multiple observational studies (17,18,22,26,31–34). However, most of the literature has focused on the relative risk (measured
by hazard or odds ratios) of adverse outcomes (17,18,22,26,31–34) but has not focused on the discriminatory information provided by clinic versus ambulatory BPs. In our study, we found that single-visit or repeated clinic systolic BPs were not consistently inferior to wake or sleep ambulatory systolic BPs for the discrimination of outcomes of interest, such as LVH or ESKD.

Prior studies of the association between BPs taken by different techniques and outcomes of interest (especially among children without CKD) have not been able to include “hard outcomes” because of a paucity of long-term follow-up data and the duration of follow-up that would be required for atherosclerotic outcomes to develop (35–42). We believe that the study of this question in a cohort of children with CKD who developed “hard outcomes,” such as ESKD, that can serve as an arbiter of risk discrimination is informative. Furthermore, although BP parameters taken in clinic and by ambulatory BP monitoring were both associated with LVH and ESKD, the strength of the relative risk (measured by odds or hazard ratios) did not always correspond to the discriminatory value of each parameter (measured by c statistics). We believe that both the relative risk and the discriminatory value of clinic and ambulatory BPs should be considered when judging the utility of each approach to BP determination.

Table 3. Risk of ESKD and model discrimination by different systolic BP parameters in unadjusted and adjusted analyses

<table>
<thead>
<tr>
<th>Metric</th>
<th>Unadjusted HRb (95% CI)</th>
<th>Unadjusted c Statistic (95% CI)</th>
<th>Adjusted HRb,c (95% CI)</th>
<th>Adjusted c Statistic (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinic systolic BP index</td>
<td>1.5 (1.3 to 1.8)</td>
<td>0.61 (0.55 to 0.66)</td>
<td>1.2 (1.0 to 1.4)</td>
<td>0.89 (0.86 to 0.91)</td>
</tr>
<tr>
<td>at a single visit</td>
<td>Mean of all clinic systolic BP indices at up to two visits</td>
<td>1.5 (1.3 to 1.8)</td>
<td>0.61 (0.55 to 0.66), Referencea</td>
<td>1.2 (1.0 to 1.4)</td>
</tr>
<tr>
<td>Mean ABP wake systolic BP index</td>
<td>1.6 (1.3 to 2.0)</td>
<td>0.61 (0.56 to 0.67)</td>
<td>1.3 (1.0 to 1.6)</td>
<td>0.90 (0.85 to 0.91)</td>
</tr>
<tr>
<td>Mean ABP sleep systolic BP index</td>
<td>1.4 (1.2 to 1.7)</td>
<td>0.58 (0.52 to 0.64)</td>
<td>1.2 (1.0 to 1.5)</td>
<td>0.89 (0.86 to 0.91)</td>
</tr>
<tr>
<td>Mean of all clinic systolic BP indices at up to two visits + mean ABP wake systolic BP index</td>
<td>Clinic:1.3 (1.1 to 1.6); ABP:1.4 (1.1 to 1.8)</td>
<td>0.62 (0.57 to 0.68)</td>
<td>Clinic:1.0 (0.8 to 1.3); ABP:1.3 (0.9 to 1.7)</td>
<td>0.90 (0.86 to 0.92)</td>
</tr>
</tbody>
</table>

HR, hazard ratio; 95% CI, 95% confidence interval; ABP, ambulatory BP.

No differences were noted in the c statistics of any metric compared with that for the reference group, which includes clinic systolic BPs at up to two sequential clinic visits.

Adjusted for age, sex, race, cause of CKD, body mass index z score, duration of CKD, urine protein-to-creatinine ratio, serum albumin, hemoglobin, antihypertensive use, and baseline eGFR (by bedside Schwartz); n=496 included because of missing covariates.

In conclusion, well performed, research-grade clinic systolic BPs are not consistently inferior to ambulatory systolic BPs and enhance the precision of clinic BP measurements (through the incorporation of a larger number of readings as would be available through ambulatory BP monitoring). Some studies have suggested that the shortcomings of a single clinic BP measurement can potentially be overcome by using repeated clinic BPs over time (43). Our data suggest that, in clinics that do not have the capacity to perform ambulatory BP monitoring for the diagnosis of hypertension or confirmation of BP control, performance of standardized, protocol-driven manual systolic BP measurements may provide similar prognostic information as ambulatory BP monitoring.

To date, only one large European trial testing different ambulatory BP targets has been performed in children with CKD (32). In this trial, repeated ambulatory BP measurements were taken in close proximity to achieve ambulatory mean arterial pressure goals during the trial and confirm continued BP control over several years. However, in our experience, the repetition of ambulatory BP monitoring within short intervals to confirm BP control is challenging in real world clinical practice, because many children and their families are resistant to repeating ambulatory BP monitoring in close proximity because of its associated discomfort (32). We believe that the results of our study reinforce the fact that clinic BP measurements remain meaningful metrics of risk.

The strength of this study includes the use of a well characterized cohort for study and the availability of well performed, research-grade clinic BPs and clinically relevant outcomes of interest. Of note, we chose not to evaluate derived BP parameters on ambulatory BP monitoring (such as nocturnal dipping or BP load) in this study, because the goal was to provide simple comparisons of BP values obtained by either clinic or ambulatory systolic BPs that may be more comparable across different modalities. Limitations include the fact that our results are derived from children enrolled in a research study, and they may not be generalizable to all children with CKD. We also note that the lack of superiority of ambulatory-based BPs could be related to the inclusion of a more racially diverse and ethnic population of children in the CKiD Study, for whom ambulatory normative BP values have not been well established. Finally, we are unable to address the issue of whether oscillometric BPs taken in clinic would provide similar prognostic value as manual clinic BPs.
BPs for the purposes of risk stratification in children with CKD. In facilities where ambulatory BP monitoring is unavailable or for children who are not willing to undergo ambulatory BP monitoring, carefully obtained repeated BP measurements using a standardized protocol by auscultatory technique may provide similar prognostic information. We believe that it is important to invest in the continued improvement and standardization of BP measurements obtained during routine practice in clinic. When using oscillometric clinic BP measurements, it may be prudent to perform manual BP measurements to confirm findings. Traditional manual clinic BP measurements remain meaningful predictors of long-term adverse outcomes.

Acknowledgments
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Disclosures
None.

References

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**Supplementary Table 1:** Risk of LVH or ESRD and model discrimination by different systolic BP index parameters in adjusted analyses using CKiD equation.*

<table>
<thead>
<tr>
<th>Metric</th>
<th>Adjusted OR** for LVH(^1) (95% CI)</th>
<th>Adjusted C-statistic (95% CI)</th>
<th>Adjusted HR** of ESRD(^1) (95% CI)</th>
<th>Adjusted C-statistic (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinic systolic BP index at a single visit</td>
<td>1.8 (1.3-2.5)</td>
<td>0.79 (0.74-0.85)</td>
<td>1.2 (1.0-1.5)</td>
<td>0.89 (0.86-0.91)</td>
</tr>
<tr>
<td>Mean of all systolic BP indices at up to two visits</td>
<td>2.1 (1.4-3.0)</td>
<td>0.80 (0.74-0.86) Reference</td>
<td>1.2 (1.0-1.5)</td>
<td>0.89 (0.86-0.92)</td>
</tr>
<tr>
<td>Mean ABP wake systolic BP index</td>
<td>2.1 (1.4-3.1)</td>
<td>0.80 (0.75-0.86) Reference</td>
<td>1.3 (1.0-1.6)</td>
<td>0.89 (0.85-0.91)</td>
</tr>
<tr>
<td>Mean ABP sleep systolic BP index</td>
<td>1.7 (1.2-2.4)</td>
<td>0.80 (0.74-0.86) Reference</td>
<td>1.2 (1.0-1.5)</td>
<td>0.89 (0.84-0.91)</td>
</tr>
<tr>
<td>Mean of all systolic BP indices at up to two visits + ABP mean wake systolic BP index</td>
<td>Clinic: 1.5 (1.0-2.1) Ambulatory: 1.6 (1.0-2.6)</td>
<td>0.81 (0.76-0.87) Clinic: 1.1 (0.8-1.4) Ambulatory: 1.2 (0.9-1.7)</td>
<td>0.89 (0.86-0.91)</td>
<td></td>
</tr>
</tbody>
</table>

\(^1\)Adjusted for age, sex, race, cause of CKD, BMI z-score, duration of CKD, urine protein/creatinine ratio, serum albumin, hemoglobin, anti-hypertensive use, and baseline eGFR (by bedside Schwartz)

*No differences were noted in the c-statistic of any metric compared to that for the reference group, which is mean of clinic systolic BPs taken at up to two visits

** OR = odds ratio; HR = hazard ratio, all reported per 0.1 increase in SBP index
Supplementary Table 2: Association between BP parameters at Year 1 and risk of LVH at Year 3 with model discrimination in unadjusted and adjusted analyses.

<table>
<thead>
<tr>
<th>Metric</th>
<th>Unadjusted OR** for LVH&lt;sup&gt;1&lt;/sup&gt; (95% CI)</th>
<th>Unadjusted C-statistic (95% CI)</th>
<th>Adjusted OR** of LVH&lt;sup&gt;1&lt;/sup&gt; (95% CI)</th>
<th>Adjusted C-statistic (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinic systolic BP index at a single visit</td>
<td>1.8 (1.2-2.6)</td>
<td>0.66 (0.56-0.77)</td>
<td>1.7 (1.1-2.6)</td>
<td>0.83 (0.76-0.91)</td>
</tr>
<tr>
<td>Mean of all systolic BP indices at up to two visits</td>
<td>1.7 (1.1-2.6)</td>
<td>0.62 (0.51-0.73) Reference</td>
<td>1.5 (0.9-2.5)</td>
<td>0.83 (0.75-0.90)</td>
</tr>
<tr>
<td>Mean ABP wake systolic BP index</td>
<td>0.9 (0.6-1.5)</td>
<td>0.52 (0.42-0.63)</td>
<td>1.0 (0.6-1.8)</td>
<td>0.82 (0.75-0.89)</td>
</tr>
<tr>
<td>Mean ABP sleep systolic BP index</td>
<td>0.9 (0.6-1.4)</td>
<td>0.51 (0.41-0.62)</td>
<td>1.1 (0.7-1.7)</td>
<td>0.82 (0.74-0.89)</td>
</tr>
<tr>
<td>Mean of all systolic BP indices at up to two visits + ABP mean wake systolic BP index</td>
<td>Clinic: 2.1 (1.3-3.4) Ambulatory: 0.6 (0.4-1.1)</td>
<td>0.64 (0.52-0.75)</td>
<td>Clinic: 1.8 (1.0-3.3) Ambulatory: 0.7 (0.4-1.4)</td>
<td>0.83 (0.75-0.91)</td>
</tr>
</tbody>
</table>

<sup>1</sup>Adjusted for age, sex, race, cause of CKD, BMI z-score, duration of CKD, urine protein/creatinine ratio, serum albumin, hemoglobin, anti-hypertensive use, and baseline eGFR (by bedside Schwartz); N=264 included due to missing covariates

*No differences were noted in the c-statistic of any metric compared to that for the reference group, which is mean of clinic systolic BPs taken at up to two visits

** OR = odds ratio; HR = hazard ratio, all reported per 0.1 increase in systolic BP index