ORIGINAL ARTICLE

Agreement Between Ambulatory and Home Blood Pressure Monitoring in Detecting Nighttime Hypertension and Nondipping Patterns in the General Population

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BACKGROUND
Nighttime blood pressure (BP) and nondipping pattern are strongly associated with hypertensive end-organ damage. However, no previous studies have compared the diagnostic agreement between ambulatory and home monitoring in detecting these BP patterns in the general population.

METHODS
We studied a population-based sample of 180 persons aged 32–80 years. The study protocol included 24-hour ambulatory BP monitoring, home daytime measurements over 7 days, home nighttime measurements (6 measurements over 2 consecutive nights using a timer-equipped home device), and ultrasound measurements for left ventricular mass index (LVMI) and carotid intima-media thickness (IMT). We defined nondipping as a <10% reduction in nighttime BP compared with daytime BP, and nighttime hypertension as BP ≥ 120/70 mm Hg.

RESULTS
The agreement between ambulatory and home monitoring for detecting nighttime hypertension was good (80%, $\kappa = 0.56$, $P < 0.001$). However, their agreement in detecting nondipping status was poor (54%, $\kappa = 0.12$, $P = 0.09$). The magnitude of ambulatory systolic BP dipping percent was 1.7% higher than on home monitoring ($P = 0.004$), whereas no difference was observed for diastolic BP dipping (difference: 0.7%, $P = 0.33$). LVMI and IMT were significantly greater among individuals with nighttime hypertension than in normotensive individuals, irrespective of the measurement method. However, only ambulatory nondippers, but not home nondippers, had more advanced end-organ damage than dippers.

CONCLUSION
We observed a good agreement between ambulatory and home BP monitoring in detecting nighttime hypertension in the general population. Two-night home monitoring could offer an inexpensive and feasible method for the diagnosis of nighttime hypertension.

Keywords: ambulatory blood pressure; blood pressure; carotid intima-media thickness; diagnosis; home blood pressure; hypertension; hypertensive end-organ damage; left ventricular hypertrophy; nighttime hypertension; nondipping.

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Growing evidence indicates that nighttime blood pressure (BP) and blunted or absent decline in BP during sleep, i.e. "nondipping," are more closely related to cardiovascular outcomes than clinic or daytime ambulatory BP.[1,2] Therefore, current guidelines recommend the use of ambulatory monitoring for evaluating patient's BP profile due to its unique advantage for detecting abnormal nighttime BP patterns.[3]

In addition to ambulatory monitors, nighttime BP can now also be measured using novel timer-equipped home monitors.[4–9] According to a recent meta-analysis, these monitors provide similar mean nighttime BP values as ambulatory monitors, with comparable relation to end-organ damage.[10] Furthermore, substantial diagnostic agreement in detecting nondipping and nighttime hypertension statuses between 24-hour ambulatory and 3-night home monitoring has been suggested by 2 previous studies from Greece.[11,12] Furthermore, another Greek study reported that even 2 nights of home monitoring might be sufficient for reliable assessment of nighttime BP profile.[13] However, the results of these studies may not be generalizable as they included only hypertensive patients.

The present study was designed to (i) compare the diagnostic agreement of abnormal BP profiles between ambulatory and home monitoring; (ii) assess the reproducibility of home nondipping and nighttime hypertension statuses using a feasible 2-night measurement protocol; (iii) assess the severity of end-organ damage between abnormal BP profiles detected by the 2 methods in a general population sample.

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METHODS

Participants

The study sample was drawn from the participants of the cardiovascular substudy (n = 493) of DILGOM (DIetary, Lifestyle, and Genetic determinants of Obesity and Metabolic syndrome) study that aimed to assess how nutrition, lifestyle, psychosocial factors, environment and genetics relate with obesity and metabolic syndrome. The DILGOM study was originally carried out in 2007, and of the participating individuals, 493 were also included in the cardiovascular substudy.[14] In 2014, of the 453 still living participants of the cardiovascular substudy, 64% (n = 290) agreed to participate in a reexamination included in the present study. We excluded participants due to one or more of the following criteria: missing covariate (N = 6), or end-organ (N = 5) data, fewer than 20 valid daytime (N = 20) or 7 nighttime ambulatory BP (N = 16), or fewer than 6 nighttime home BP readings (N = 94), resulting in a sample of 180 individuals who were included in all analyses. Each participant provided written informed consent. The study was approved by the Ethics Committee of the Hospital District of Southwest Finland.

Clinical evaluation and BP measurements

The study protocol has been published previously in detail.[15] All participants provided medical history and information on sociodemographic factors and underwent physical examination and laboratory assessment of serum lipids and blood glucose. Non-high-density lipoprotein cholesterol was calculated as total cholesterol minus high-density lipoprotein cholesterol. Diabetes was defined as fasting plasma glucose level ≥ 7.0 mmol/l or treatment with antidiabetic drugs. Smoking was defined as self-reported daily use of cigarettes. Cardiovascular disease event was defined as history of previous myocardial infarction or stroke reported by the participant.

For the present study, each participant underwent 24-hour ambulatory monitoring, 7-day home measurement protocol and 2-night home BP monitoring. Twenty-four-hour ambulatory monitoring was performed using a validated oscillometric Microlife WatchBP O3 device (Microlife AG, Widnau, Switzerland) with measurements at 20-minute intervals during the day (from 0700 to 2200 h) and 30-minute intervals during the night (from 2200 to 0700 h).[16] Nighttime BP was defined as the mean of all BP values during the reported sleeping period, and daytime BP was defined as the mean of all other BP values of the 24-hour period. Participants were instructed to take home BP measurements with a validated oscillometric Microlife WatchBP Home N device (Microlife AG, Widnau, Switzerland) in sitting position after a 3-minute rest at 1-minute intervals. Daytime home BP was measured twice in the morning (between 0600 and 0900 h) and twice in the evening (between 1800 and 2100 h) on 7 consecutive days. During the last 2 nights of home monitoring, participants activated home nighttime monitoring by taking pre-sleep BP measurement immediately before going to sleep. Thereafter, 3 automated BP measurements were taken at 2, 3, and 4 hours after the initial pre-sleep BP measurement. All daytime and nighttime BP (except for the pre-sleep activation BP measurement) measurements were averaged to determine daytime and nighttime home BP, respectively. We defined nondipping as a reduction in the mean nighttime systolic or diastolic BP less than 10% compared with daytime values, and nighttime hypertension as nighttime systolic or diastolic BP ≥ 120/70 mm Hg. Dipping and hypertension statuses were calculated separately for both methods and separately for systolic and diastolic BP.

Evaluation for end-organ damage

Echocardiographic left ventricular mass measurements were obtained with 2-dimensional guided M-mode with a Vivid E9 ultrasound machine equipped with an M5SD transducer (GE Healthcare, Chicago, IL) according to the recommendations by the American Society of Echocardiography.[17] Left ventricular mass index (LVMI) was calculated by dividing left ventricular mass with body surface area. Left ventricular hypertrophy was defined as LVMI greater than 95 and 115 g/m^2 for women and men, respectively.[17] Carotid intima-media thickness (IMT) measurements were determined from Doppler-guided B-mode with a Vivid E9 device equipped with a 11L-D linear-array transducer (GE Healthcare) according to the American Society of Echocardiography consensus statement.[18] For categorical analyses, we divided the study population in 10-year strata and defined increased IMT as IMT over 75th percentile of each stratum to adjust the high correlation between age and IMT.[19] Details on the assessment of LVMI and IMT have been reported previously.[15]

Statistical analysis

The normality of variables was examined using the Kolmogorov–Smirnov test. LVMI and IMT were log-transformed before analyses to obtain normal distribution. Diagnostic agreement in detecting hypertension phenotypes between ambulatory and home monitoring and between the 2 home monitoring nights was assessed with kappa statistics. In addition, per-participant 2 × 2 contingency tables were constructed to determine sensitivity, specificity, positive predictive value, and negative predictive value for home BP measurements while using ambulatory monitoring as the reference method. Agreement in dipping percentages was tested with a paired t-test and Bland–Altman plots. Depending on equality of variances, a pooled or unequal variance t-test was used to compare severity of end-organ damage between categories by dipping and nighttime hypertension status. We used logistic regression to calculate odds ratios for left ventricular hypertrophy and increased IMT for nondippers vs. dippers and normotensive vs. hypertensive participants. We adjusted the estimates for age, sex, body mass index, diabetes, current smoking, non-high-density lipoprotein cholesterol, antihypertensive medication, and history of a cardiovascular disease event. All analyses were performed with SAS software version 9.4 (SAS Institute, Cary, North Carolina). We considered a 2 sided $P < 0.05$ to be statistically significant.

RESULTS

The characteristics of study participants included in the analyses (n = 180; mean age 57.1 years; 62.2% women) are reported in Table 1. The mean number of daytime ambulatory, nighttime ambulatory, daytime home, and nighttime home BP measurements were 45.7 ± 6.1, 16.8 ± 3.0, 13.3 ± 2.1, and 6 ± 0, respectively.

Results for the sensitivity, specificity, predictive values, and agreement of home monitoring in detecting ambulatory nighttime hypertension and nondipping pattern are presented in Tables 2 and 3. Of the participants, 63 (35.0%) and 61 (33.9%) had nighttime hypertension on ambulatory or home monitoring, respectively (agreement 80%, $\kappa = 0.56$, $P < 0.001$, Table 2). Seventy-one participants (39.4%) were classified as nondippers based on ambulatory monitoring, compared with 107 (59.4%) when home monitoring was used (agreement 54%, $\kappa = 0.12$, $P = 0.09$, Table 3). When analyzed as a continuous variable, mean ambulatory systolic BP dipping was 1.7% higher than on home monitoring (11.5% vs. 9.8%, $P = 0.004$). In contrast, the magnitude of diastolic BP dipping was similar for both methods (difference: 0.7%, $P = 0.33$). Despite the small mean difference in systolic dipping, we found large individual-level differences in dipping percentages between the methods (Figure 1). Participants classified as nondippers had significantly higher nighttime systolic/diastolic ambulatory (118.9 ± 12.9 vs. 107.5 ± 9.5/68.2 ± 7.8 vs. 62.6 ± 6.9 mm Hg) and home BPs (home systolic/diastolic: 115.6 ± 12.1 vs. 107.5 ± 10.9/66.1 ± 7.6 vs. 62.2 ± 6.6 mm Hg) than dippers ($P < 0.001$ for all comparisons).

Individuals with nighttime hypertension had significantly greater LVMI and IMT values than normotensives, regardless of the measurement method (Table 4). Similarly, ambulatory nondippers had significantly greater IMT values than ambulatory dippers ($P < 0.001$ for all, Supplementary Table 1). In contrast, ambulatory nondippers had greater LVMI values than dippers only when systolic and diastolic dipping were analyzed separately (systolic: 94.2 vs. 87.1 g/m^2, $P = 0.046$; diastolic: 95.2 vs. 87.7 g/m^2, $P = 0.04$; systolic or diastolic: 93.0 vs. 87.5 g/m^2, $P = 0.12$, Supplementary Table 1). We found no differences in LVMI or IMT values between dipping categories based on home monitoring (Supplementary Table 1). In logistic regression analyses, only ambulatory systolic and diastolic nondipping were significantly associated with greater odds of left ventricular hypertrophy compared with dipping (ambulatory systolic: odds ratios = 2.19, 95% confidence interval = 1.02–4.71, $P = 0.04$; ambulatory diastolic: odds ratios = 2.55, 95% confidence interval = 1.15–5.62, $P = 0.02$). After adjustment for other covariates, these associations also lost their statistical significance ($P \geq 0.70$ for both, Supplementary Table 2). Furthermore, nondipping pattern was not significantly associated with IMT in our study population irrespective of the measurement method (Supplementary Table 2).

Reproducibility of home nondipping pattern was only moderate between 2 consecutive home monitoring nights (69%, $\kappa = 0.37$, $P < 0.001$). Of the 102 participants who were classified as nondippers based on either systolic or diastolic BP on the first night, only 73 (72%) had the same pattern in the second night. Similarly, of the 78 who demonstrated a dipper pattern on the first night, only 51 (65%) had the same pattern on the second night. In contrast, nighttime hypertension status demonstrated substantial reproducibility between first and second night (83%, $\kappa = 0.62$, $P < 0.001$).

Table 1. Characteristics of study participants

Characteristics	
Women (%)	62.2
Age (years)	57.1 (12.6)
Body mass index (kg/m^2)	26.8 (5.0)
Serum cholesterol (mmol/l)	5.2 (1.0)
HDL cholesterol (mmol/l)	1.5 (0.4)
Fasting plasma glucose (mmol/l)	5.9 (0.8)
LVMI (g/m^2)	
Women (g/m^2)	84.3 (17.8)
Men (g/m^2)	98.6 (18.0)
IMT (mm)	0.75 (0.16)
Systolic BP (mm Hg)	
Home daytime	124.8 (12.7)
Ambulatory daytime	126.6 (11.3)
Home nighttime	112.3 (12.3)
Ambulatory nighttime	112.0 (12.3)
Diastolic BP (mm Hg)	
Home daytime	75.9 (8.1)
Ambulatory daytime	76.9 (8.0)
Home nighttime	64.5 (7.4)
Ambulatory nighttime	64.8 (7.8)
Dipping (%)	
Home systolic/diastolic	9.8 (6.7)/14.7 (7.8)
Ambulatory systolic/diastolic	11.5 (6.4) /15.4 (8.5)
Hypertension SBP/DBP (N, %)	
Ambulatory daytime/nighttime	49 (27%)/63 (35%)
Home daytime/nighttime	47 (26%)/67 (37%)
Diabetes mellitus (%)	11.1
Current smokers (%)	7.8
History of CVD event (%)	3.3
Antihypertensive medication use (%)	26.1

Data are presented as mean (SD) or as percentage. Abbreviations: BP, blood pressure; HDL, high-density lipoprotein; LVMI, left ventricular mass index; IMT, intima-media thickness; CVD, cardiovascular disease; SBP, systolic blood pressure; DBP, diastolic blood pressure.

DISCUSSION

To the best of our knowledge, this is the first study to assess the agreement of nighttime home and ambulatory monitoring in detecting hypertension and nondipping patterns in the general population. We observed that ambulatory and home monitoring detected nighttime hypertension

Table 2. Diagnostic agreement between home and ambulatory monitoring for detecting nighttime hypertension (*N* = 180)

Home BP variable	No. with home NT/HT	True positive	False negative	False positive	True negative	Agreement, %	Sensitivity, %	Specificity, %	PPV, %	NPV, %	κ (*P* value)
Systolic											
Home first night	130/50	36	13	14	117	85	74	89	72	90	0.64 (<0.001)
Home second night	136/44	33	16	11	120	85	67	92	75	88	0.62 (<0.001)
Home 2-night	133/47	35	14	12	119	86	71	91	75	90	0.64 (<0.001)
Diastolic											
Home first night	132/48	27	14	21	118	81	66	85	56	89	0.50 (<0.001)
Home second night	139/41	24	17	17	122	81	59	88	59	88	0.46 (<0.001)
Home 2-night	136/44	27	14	17	122	83	66	88	61	90	0.52 (<0.001)
Systolic or diastolic[a]											
Home first night	113/67	46	17	21	96	79	73	82	69	85	0.55 (<0.001)
Home second night	120/60	44	19	16	101	81	70	86	73	84	0.58 (<0.001)
Home 2-night	119/61	44	19	17	100	80	70	86	72	84	0.57 (<0.001)

Nighttime systolic/diastolic hypertension defined as nighttime BP ≥ 120/70 mm Hg. Forty-nine, 41, and 63 individuals had systolic, diastolic, and systolic/diastolic nighttime ambulatory hypertension, respectively. Diagnostic agreement was evaluated with kappa statistics. Abbreviations: BP, blood pressure; NT, normotensive; HT, hypertensive; PPV, positive predictive value; NPV, negative predictive value.

[a]Systolic or diastolic hypertension on home monitoring compared with ambulatory systolic or diastolic hypertension.

Table 3. Diagnostic agreement between home and ambulatory monitoring for detecting nondipping pattern (*N* = 180)

Home BP variable	No. with home D/ND	True positive	False negative	False positive	True negative	Agreement, %	Sensitivity, %	Specificity, %	PPV, %	NPV, %	κ (*P* value)
Systolic											
Home first night	83/97	44	21	53	62	59	68	54	45	75	0.20 (0.008)
Home second night	89/91	44	21	47	68	62	68	59	48	76	0.25 (<0.001)
Home 2-night	77/103	45	20	58	57	57	69	50	44	74	0.17 (0.02)
Diastolic											
Home first night	127/53	20	27	33	100	67	43	75	38	79	0.17 (0.03)
Home second night	129/51	18	29	33	100	66	38	75	35	78	0.15 (0.09)
Home 2-night	134/46	20	27	26	107	71	43	81	44	80	0.23 (0.003)
Systolic or diastolic[a]											
Home first night	78/102	47	24	55	54	56	66	50	46	69	0.15 (0.046)
Home second night	80/100	49	22	51	58	59	69	53	49	73	0.21 (0.004)
Home 2-night	73/107	48	23	59	50	54	68	46	45	69	0.12 (0.09)

Nondipping was defined as (1 − nighttime BP/daytime BP) × 100 (%) <10%. Sixty-five, 47, and 71 individuals had systolic, diastolic, and systolic/diastolic nondipping pattern on ambulatory monitoring, respectively. Diagnostic agreement was evaluated with kappa statistics. Abbreviations: BP, blood pressure; D, dipper; ND, nondipper; PPV, positive predictive value; NPV, negative predictive value.

[a]Systolic or diastolic nondipping pattern on home monitoring compared with ambulatory systolic or diastolic nondipping pattern.

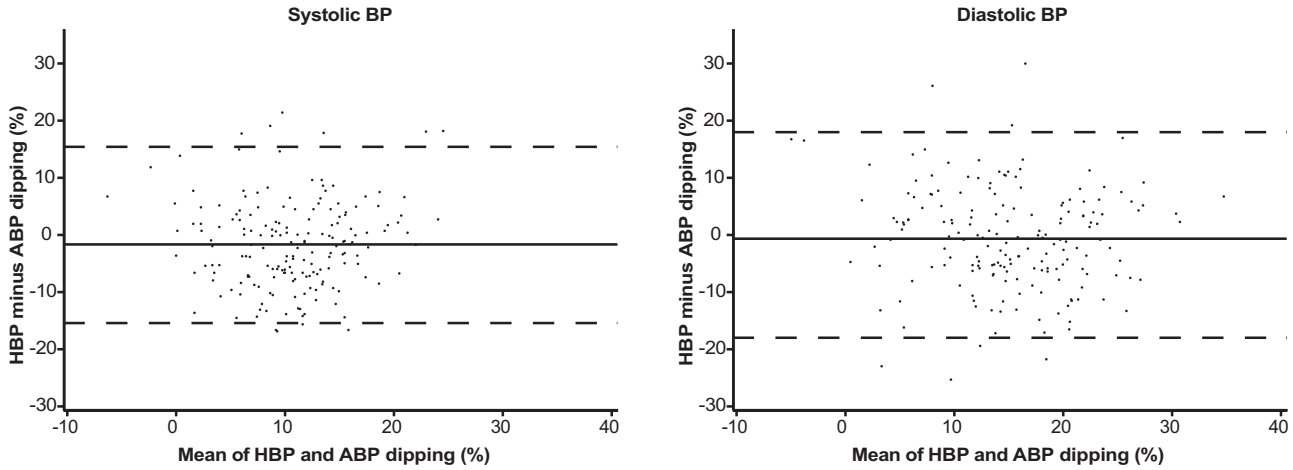

Figure 1. Bland–Altman plots for nighttime home and ambulatory blood pressure dipping (%). Continuous lines represent the mean difference between the methods, and dashed lines represent 95% limits of agreement for the difference (±2 SDs). BP, blood pressure; HBP, home blood pressure; ABP, ambulatory blood pressure.

Table 4. Comparison of the severity of end-organ damage between nighttime normotensive/hypertensive participants (*N* = 180)

		LVMI, g/m²			IMT, mm		
	No. with NT/HT	NT	HT	*P* value	NT	HT	*P* value
Ambulatory BP							
Systolic	131/49	85.4 (16.6)	101.1 (20.8)	<0.001	0.71 (0.14)	0.85 (0.18)	<0.001
Diastolic	139/41	87.2 (17.7)	98.0 (21.6)	0.002	0.73 (0.16)	0.82 (0.17)	0.002
Systolic/diastolic	117/63	84.9 (15.9)	98.6 (21.5)	<0.001	0.71 (0.14)	0.82 (0.17)	<0.001
Home BP							
Systolic	133/47	86.4 (17.3)	99.0 (21.2)	<0.001	0.72 (0.15)	0.83 (0.18)	<0.001
Diastolic	136/44	87.5 (18.0)	96.5 (21.2)	0.008	0.73 (0.16)	0.79 (0.17)	0.03
Systolic/diastolic	119/61	85.5 (16.4)	97.9 (21.5)	<0.001	0.72 (0.15)	0.81 (0.17)	<0.001

P values are for the between-group difference in log-transformed LVMI or IMT. LVMI data are presented as g/m² and IMT as millimeters. Abbreviations: BP, blood pressure; NT, normotensive; HT, hypertensive; LVMI, left ventricular mass index; IMT, intima-media thickness.

comparably, but their agreement in detecting nondipping pattern was poor. Reproducibility of nighttime hypertension diagnosis was good between the first and second home measurement nights, whereas for nondipping pattern, it was only moderate. As expected, more advanced hypertensive end-organ damage was observed in those with nighttime hypertension than with normotension, irrespective of the measurement method. In contrast, nondipping status was associated with more severe end-organ damage only when it was based on ambulatory measurements.

We have previously shown that home nighttime measurement is a well-tolerated method for measuring BP.[20] Furthermore, home nighttime BP values are similar to those observed with ambulatory monitoring, and the 2 methods have comparable associations with LVMI and IMT in general population.[15] We have now extended these results by demonstrating good diagnostic agreement between ambulatory and home monitoring for detecting nighttime hypertension. These findings are in accord with those from a study with a hypertensive patient sample.[12,13] Thus, home monitoring could provide a potential alternative for ambulatory monitoring in detecting nighttime hypertension.

Two previous studies with 81 and 131 Greek hypertensive patients reported substantial diagnostic agreement in detecting nondipping pattern between home and ambulatory monitoring.[11,12] However, the kappa coefficients were quite low (0.20–0.31) in both studies. Furthermore, in a 94-patient subsample of the latter study, Kollias *et al.* observed a slightly better agreement between ambulatory and home monitoring for detecting the nondipping pattern (κ = 0.40). However, agreement did not increase after the fourth measurement when a total of 9 home measurements were performed over 3 consecutive nights.[13] This finding was somewhat surprising as larger number of readings would be expected to produce more accurate estimation of BP profile. In contrast, the diagnostic agreement in detecting nighttime hypertension improved up to 8 measurements before it plateaued.[13] In accordance with our results, Kollias *et al.* found more robust diagnostic agreement for detecting nighttime hypertension than nondipping profile between the 2 methods.

The most evident reasons for the disagreement between ambulatory and home monitoring in detecting nondipping pattern are the differences in daytime measurement conditions. Whereas daytime ambulatory monitoring is carried out during habitual daily activity, home measurements are taken in the sitting position after an adequate resting period. These differences usually affect more prominently systolic than diastolic BP, thus promoting a greater difference between systolic ambulatory daytime and nighttime BP than corresponding home BPs. Indeed, we observed that ambulatory systolic BP dipping was 1.7% greater compared with home systolic BP dipping. Consequently, we observed a larger number of systolic nondippers when the diagnosis was based on home instead of ambulatory monitoring. A similar trend was observed also in the studies by Stergiou *et al.* (22% vs. 16%)[11] and Andreadis *et al.* (24% vs. 12%).[12] By contrast, in the Japan Morning Surge-Target Organ Protection (J-TOP) study with 50 hypertensive patients that was designed to assess the changes in home and ambulatory BP patterns induced by antihypertensive medication, the authors observed no significant difference between systolic dipping percent at baseline. However, after 6 months of treatment, ambulatory systolic dipping exceeded home systolic dipping by 3.6% with no differences in mean daytime or nighttime systolic BPs.[21] An even greater difference was observed in the cross-sectional study by Ushio *et al.*, where average nighttime systolic BP fall was 5.3% and 14.7% when assessed with home and ambulatory monitoring, respectively.[7] In all previous studies, apart from the J-TOP study and the study by Ushio *et al.*, a markedly lower number of home than ambulatory measurements were performed. Although this trade-off most likely renders home nighttime monitoring more user-friendly, it may simultaneously reduce its diagnostic agreement with ambulatory monitoring.

Although diagnostic agreement between nighttime home and ambulatory measurements for detecting nighttime dipping profiles was suboptimal in our study, it does not necessarily imply that the home devices are inaccurate. It might merely reflect the differences between the measurement conditions and limited reproducibility of the dipping phenomenon in itself. Studies on the reproducibility of nondipping pattern on home monitoring are virtually nonexistent. The study by Stergiou *et al.* only briefly mentions that agreement of nondipping pattern between 3 consecutive measurement nights varied from 71% to 73%.[11] Similarly, we found that the reproducibility of nondipping status between the first and second home measurement nights was only moderate (69%). However, it should be taken into account that reproducibility of nondipping pattern between 2 sessions of ambulatory monitoring is also only 65–80%.[22–25] One explanation for the suboptimal agreement could be the changing sleep quality between various measurement nights.[26] Therefore, the possibility to easily repeat nighttime BP measurements with a home monitor could be clinically useful, particularly if sleep quality was poor during the first measurement night. In any case, further studies are needed to verify clinically relevant cutoff values for nondipping pattern for home monitoring.

We demonstrated in the current study that participants with nighttime hypertension had greater LVMI and IMT values than normotensive participants, regardless of the measurement method. Similarly, Andreadis *et al.* found a positive, although statistically insignificant, trend toward greater LVMI and IMT values among patients with nighttime home hypertension compared with their normotensive counterparts.[12] In contrast, nondipping status based on home monitoring was not associated with more severe end-organ damage in our study. This might be attributed to the relatively small study sample or the small observed absolute nighttime drop in systolic home BP. In any case, the BP level rather than dipping status on ambulatory monitoring seems to discriminate also more reliably participants with more severe end-organ damage, a notion also supported by the current guidelines.[3]

Several limitations should be considered when assessing the results of our study. First, ambulatory and home nighttime monitoring were carried out in different days approximately a week apart for feasibility reasons; thus, day-to-day BP variability might confound our results. Second, the order of conducting ambulatory and home monitoring was not randomized, and ambulatory monitoring was persistently measured prior to home monitoring. Third, the quality of sleep during measurement nights might affect the absolute BP values.[27] Furthermore, several factors, such as age[28] and obesity,[12,28] are related to the difference between daytime ambulatory BP and home BP, which complicates comparisons of dipping measurement methods. Moreover, antihypertensive medication and the time of drug administration might affect the dipping status.[29] However, in the present study, both ambulatory and home nighttime BP measurements were taken at a similar time of the day, whereas those participants taking antihypertensive drugs (26.1%) were following the same medication administration times to limit the extend of such pharmacodynamic effects. Finally, because the optimal definition for nighttime home dipping has not yet been determined, we used the same diagnostic threshold for home and ambulatory monitoring. However, as discussed earlier, this might not be adequate due to the differences in measurement conditions.

In conclusion, 2-night home monitoring seems to offer an inexpensive, feasible, and reliable method for the diagnosis of nighttime hypertension in the general population. Instead, only poor diagnostic agreement was observed for the detection of nondipper pattern between nighttime home and ambulatory measurements. Additional research is needed to verify the optimal thresholds for home nondipping pattern in a prospective setting.

SUPPLEMENTARY MATERIAL

Supplementary data are available at *American Journal of Hypertension* online.

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DISCLOSURE

Dr. Kantola has participated in conference trips sponsored by Sanofi-Genzyme and Takeda-Shire and received honorariums from the same sources and Amicus for participating in an advisory board meetings. He also has ongoing research collaboration with Bayer Ltd, Idorsia, Sanofi-Genzyme and Takeda-Shire. (All unrelated to the present study). Dr. Salomaa has participated in a conference trip sponsored by Novo Nordisk and received a honorarium from the same source for participating in an advisory board meeting. He also has ongoing research collaboration with Bayer Ltd. (All unrelated to the present study). The other authors report no concflicts of interest.

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