Ambulatory blood pressure monitoring for the management of hypertension

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Abstract

Ambulatory blood pressure monitoring (ABPM) has become indispensable in the current management of hypertension. ABPM is particularly useful in the accurate diagnosis of hypertension. Its diagnostic thresholds had been recently established based on hard clinical outcomes. Cross-classification of patients according to office and ambulatory blood pressure identifies white-coat, masked, and sustained hypertension. ABPM is also useful in cardiovascular (CV) risk assessment. It provides information on daytime and nighttime blood pressure and circadian rhythm, particularly nighttime blood pressure dipping. Nighttime blood pressure is predictive of CV risk independent of office and daytime blood pressure. Isolated nocturnal hypertension is a special form of masked hypertension, with normal daytime but elevated nocturnal blood pressure. It also helps in the evaluation of blood pressure fluctuation and variation, such as morning blood pressure surge and reading-to-reading blood pressure variability. ABPM may derive several other indexes, such as ambulatory blood pressure index and salt sensitivity index, which may be useful in CV evaluations.

Keywords: Ambulatory blood pressure monitoring; Blood pressure control; Hypertension; Antihypertensive treatment

Introduction

Ambulatory blood pressure monitoring (ABPM) makes it possible to measure blood pressure during daily life activities, including mild and moderate physical activity and sleep. It has been recommended in almost all current major hypertension guidelines as an out-of-office technique for blood pressure measurement.[1-4] There is now abundant evidence that ABPM is superior to office blood pressure in the evaluation of blood pressure and in the prediction of cardiovascular (CV) disease risk and events. A series of studies confirmed that ambulatory blood pressure[5-7] especially nighttime blood pressure[8,9] was superior to office blood pressure in predicting total mortality and CV complications.[6,8,9] Cross-classification of patients according to office and ambulatory blood pressure distinguishes those with masked hypertension from normotension, and those with white-coat hypertension from sustained hypertension.[1-3] Masked and sustained hypertension, but not white-coat hypertension, were associated with high CV risk.[6] In addition, ABPM records the reading-to-reading variation in blood pressure and heart rate throughout the day in response to environmental, postural, and emotional changes. ABPM has become an indispensable technique for the diagnosis of hypertension, CV risk assessment, therapeutic monitoring, and guiding individualized treatment. In the present review, we summarized the literature on ABPM in the management of hypertension.

Diagnosis of Hypertension

Diagnostic thresholds

The association between blood pressure and CV outcome is continuous; therefore, there is no blood pressure level above which the CV risk would suddenly rise strikingly.[10] Thresholds of office and ambulatory blood pressure for the diagnosis and management of hypertension are proposed for clinical application. Initially, the thresholds for ambulatory blood pressure were based on the distribution of ambulatory blood pressure in individuals with an office blood pressure in the normotensive range,[11,12] or by regression of the ambulatory on the office blood pressure.[13] Further studies applied a more credible outcome-driven approach. In the Ohasama

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study,[14] the risk of all-cause mortality reached nadir when 24-hour blood pressure increased from 120 mmHg to 133 mmHg systolic and from 65 mmHg to 78 mmHg diastolic. In 2007, the International Database on Ambulatory Blood Pressure in Relation to Cardiovascular Outcome (IDACO) investigators determined ambulatory blood pressure thresholds resulting in multivariable-adjusted 10-year risks similar to those associated with categories of the office blood pressure.[15] In the Jackson Heart Study,[16] the thresholds associated with the risk of the composite of all-cause mortality and CV disease corresponding with an office blood pressure of 140 mmHg were 134 mmHg, 138 mmHg, and 129 mmHg for 24-hour, daytime, and nighttime ambulatory systolic blood pressure, respectively. Diastolic blood pressure was not related to outcome and therefore not analyzed.[16]

Since the 2017 American College of Cardiology/American Heart Association guidelines reclassified office blood pressure, the IDACO investigators derived outcome-driven ambulatory blood pressure thresholds corresponding with the elevated office blood pressure (120–129/<80 mmHg) and stages 1 and 2 office hypertension (130–139/80–89 mmHg and >140/90 mmHg, respectively) in 11,152 participants of 13 populations.[17] Using a composite CV endpoint, the rounded thresholds for elevated 24-hour, daytime, and nighttime ambulatory blood pressure were 120/75 mmHg, 120/80 mmHg, and 105/65 mmHg, respectively, for stage 1 ambulatory hypertension 125/75 mmHg, 130/80 mmHg, and 110/65 mmHg, respectively, and for stage 2 ambulatory hypertension 130/80 mmHg, 135/85 mmHg and 120/70 mmHg, respectively [Table 1].

White-coat and masked hypertension

According to both office and out-of-office blood pressure measurements, normotension (patients with normal office and out-of-office blood pressure), white-coat hypertension (patients with office hypertension but normal out-of-office blood pressure), masked hypertension (patients with normal office blood pressure but out-of-office hypertension), and sustained hypertension (patients with office and out-of-office hypertension) can be defined [Figure 1]. In addition to ABPM, home blood pressure monitoring (HBPM) can also be used for similar definitions. However,

HBPM, compared with ABPM, had lower sensitivity, albeit higher specificity, in the diagnosis of white-coat and masked hypertension.[18]

The prevalence of white-coat hypertension ranged from 13% to 23% in clinical settings,[18,19] and was higher in the elderly with little sex difference.[20] There is limited evidence that treating white-coat hypertension or intensively treating white-coat uncontrolled hypertension with medication is clinically relevant. A meta-analysis that included 27 studies comprising 25,786 subjects with white-coat hypertension or white-coat uncontrolled hypertension and 38,483 with normotension followed up for a mean of 3 to 19 years,[21] compared with normotension, white-coat hypertension, but not white-coat uncontrolled hypertension, was associated with an increased risk of CV events (hazard ratio [HR] 1.36, 95% confidence interval [CI]: 1.03–2.00), all-cause mortality (HR 1.33, 95% CI: 1.07–1.67), and CV mortality (HR 2.09, 95% CI: 1.23–4.48). The risk of white-coat hypertension was attenuated in studies that included stroke in the definition of CV events (HR 1.26, 95% CI: 1.00–1.54),[21] and the risk became non-significant in studies using 24-hour systolic/diastolic blood pressure <130/80 mmHg, instead of daytime <135/85 mmHg, to define ambulatory normotension, with a duration of follow-up <5 years, and with prior CV disease, chronic kidney disease, or diabetes mellitus excluded.[21]

The definition of white-coat hypertension may have affected the risk of white-coat hypertension in these previous studies. Indeed, white-coat hypertension with
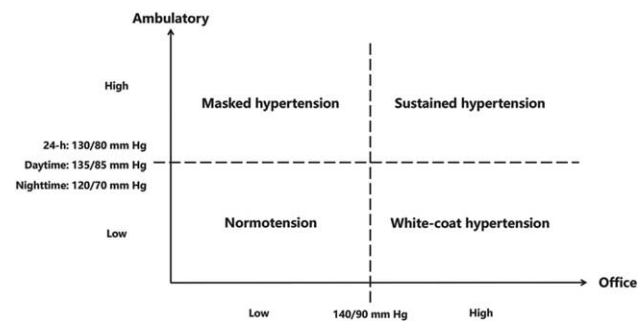
Figure 1: Cross-classification of blood pressure based on office and ambulatory measurements.

Table 1: Proposal for outcome-driven thresholds for ABPM.

Blood pressure category	ACC/AHA 2017 thresholds				IDACO thresholds		
	Office blood pressure	Ambulatory blood pressure			Ambulatory blood pressure		
		24-h	Day	Night	24-h	Day	Night
Elevated blood pressure (mmHg)	120/80	115/75	120/80	100/65	120/75	120/80	105/65
Stage 1 hypertension (mmHg)	130/80	125/75	130/80	110/65	125/75	130/80	110/65
Stage 2 hypertension (mmHg)	140/90	130/80	135/85	120/70	130/80	135/85	120/70
Severe hypertension (mmHg)	160/100	145/90	145/90	140/85	140/85	150/95	130/80

Ambulatory thresholds based on IDACO were obtained by rounding the point estimates for CV events to the nearest integer value ending in zero or five.
ABPM: Ambulatory blood pressure monitoring; ACC/AHA: American College of Cardiology/American Heart Association; CV: Cardiovascular; IDACO: International Database on Ambulatory Blood Pressure in Relation to Cardiovascular Outcome.

normal 24-hour, daytime, and nighttime ambulatory blood pressure did not confer a significantly elevated risk compared to normotension.[22] In addition, the risk of white-coat hypertension may be dependent on the level of CV risk. In an analysis of the IDACO database, 653 untreated subjects with white-coat hypertension were compared with 653 age- and cohort-matched normotensive controls, according to the high and low risk of CV events defined as >3 and <3 risk factors.[23] Over a median follow-up of 10.6 years, the incidence of CV events was higher in 159 high-risk white-coat hypertensive patients than the matched high-risk normotensive people (HR 2.06, 95% CI: 1.10–3.84), but it was similar in the 494 low-risk white-coat hypertensive patients and normotensive controls (HR 1.06, 95% CI: 0.66–1.72).[23] Further subgroup analysis by age showed that the risk of white-coat hypertension was limited to the old (age ≥60 years) and high-risk subjects, and the adjusted HR was 2.19 (1.09–4.37) in the older high-risk group and 0.88 (0.51–1.53) in the older low-risk group (P for interaction = 0.044).[23] In patients with white-coat hypertension, the risk of developing sustained hypertension was significantly higher than those with true normotension,[24] and the current guidelines recommend close follow-up of white-coat hypertension at least once per year.

The prevalence of masked hypertension was 10%–18% in China.[18,19,25] The probability of having masked hypertension increased with office blood pressure, men vs. women, overweight or obesity, diabetes mellitus, chronic kidney disease, and smoking.[26-28] CV risk associated with masked hypertension was almost equivalent to that with sustained hypertension,[6] and was significantly higher than that with normotension,[22] irrespective of isolated daytime[6] or nocturnal[29] hypertension, office normotension or prehypertension,[26] and untreated or treated status.[30,31] In a meta-analysis of 21 studies (n = 130,318),[31] the pooled risk ratio for masked hypertension or masked uncontrolled hypertension vs. normotension or controlled hypertension was 1.67 (1.32–2.13) and 2.19 (1.72–2.78) for all-cause and CV mortality, respectively, and 1.71 (1.53–1.91), 1.95 (1.36–2.80), 1.76 (1.33–2.33), 1.62 (0.27–9.60), and 3.85 (2.03–7.31) for fatal and non-fatal CV, stroke, cardiac, coronary, and renal disease events, respectively.

Whether masked hypertension or masked uncontrolled hypertension should be treated to normalize out-of-office blood pressure remains under investigation. The current guidelines recommend lifestyle modification and antihypertensive therapy in masked hypertension largely on the basis of prospective observational studies.[1-3] One of the concerns in the scientific community is the poor reproducibility of masked hypertension. However, we and several other research groups recently found that masked hypertension had relatively high reproducibility. In 45 Chinese patients with masked hypertension, 28 patients (62.2%) remained masked hypertension after 4 weeks of follow-up, and 13 (28.9%) and 4 (8.9%) converted to normotension and sustained hypertension, respectively.[32] The Improving the Detection of Hypertension study[33] had similar findings in 254 untreated

subjects with office normotension (systolic/diastolic blood pressure <130/80 mmHg) and masked daytime (systolic/diastolic blood pressure ≥130/80 mmHg), 24-hour (systolic/diastolic blood pressure ≥125/75 mmHg), nighttime (systolic/diastolic blood pressure ≥110/65 mmHg), and any masked hypertension (high daytime, 24-h, or nighttime blood pressure). Within a median of 29 days apart, about 2/3 patients with masked daytime hypertension on first monitoring maintained their classification subsequently. The reproducibility was even higher in masked 24-hour, nighttime, and any hypertension. The κ statistic (95% CI) was 0.50 (0.38–0.62), 0.57 (0.46–0.69), 0.57 (0.47–0.68), and 0.58 (0.47–0.68) for masked daytime, 24-hour, nighttime, and any hypertension, respectively.[33] Masked uncontrolled hypertension might be a different scenario, especially in long-term studies.[34] In 1664 hypertensive patients recruited for the European Lacidipine Study on Atherosclerosis study with annual ABPM recordings during 4 years follow-up, only 4.5% masked uncontrolled hypertension persisted throughout the treatment period.[34]

Because of the nature of normal office blood pressure in masked hypertension, patients with this clinical condition have never been included in the previous outcome trials on hypertension. The treatment of masked hypertension is largely unknown. We recently published a randomized placebo-controlled clinical trial in 251 patients with masked hypertension, treated with a classic Chinese herbal formula gastrodia-uncaria granules for 4 weeks.[35] The Chinese herbal formula compared to placebo significantly reduced daytime and 24-hour ambulatory blood pressure by 2.52/1.79 mmHg and 2.33/1.49 mmHg, respectively [Figure 2].[35] A multicenter trial is currently ongoing in China, which investigates whether antihypertensive treatment with an angiotensin receptor blocker would protect target organs in patients with masked hypertension (ClinicalTrials.gov Identifier: NCT02893358). A similar trial in patients with uncontrolled masked hypertension is also ongoing (ClinicalTrials.gov Identifier: NCT02804074).

CV Risk Assessment

Nighttime blood pressure and dipping

Blood pressure follows circadian rhythm, which decreases from awake to asleep period, and gradually increases from asleep to awake period. Under physiological conditions, nighttime systolic and diastolic blood pressures drop by 10% to 20% compared to daytime blood pressure. The classification of dipping status is based on the ratio of night-to-day systolic and diastolic blood pressures ([day-night blood pressure]/day blood pressure × 100%), and the dipping ratio for extreme-dipper, dipper, non-dipper, and riser are ≥20%, 10% to 20%, 0 to 10%, and <0, respectively.

Non-dippers and risers are exposed to more severe target organ damage and a higher risk of CV events than dippers.[8,9] According to a recent participant-level meta-analysis, the sex- and age-adjusted 10-year cumulative incidence of the composite CV outcome significantly
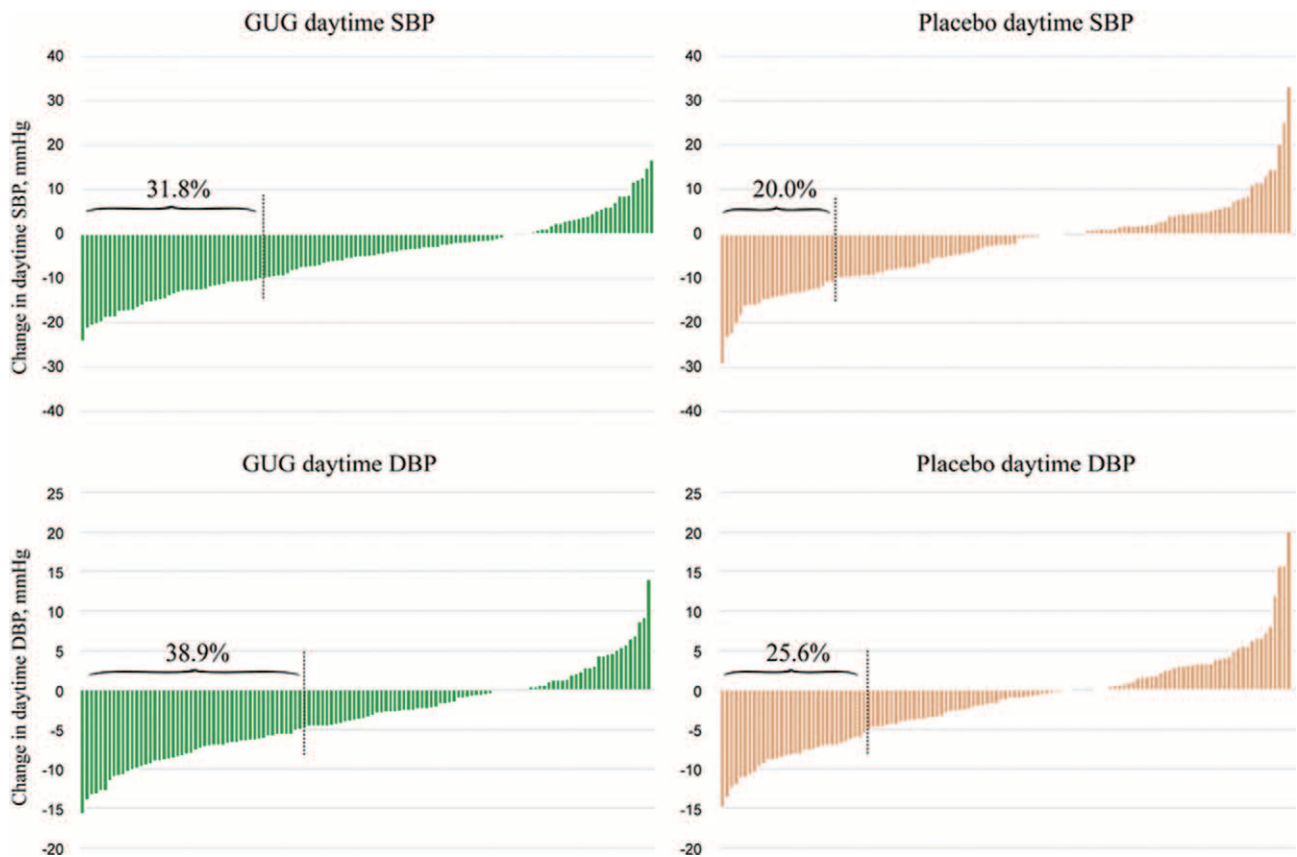
Figure 2: Individual changes in daytime blood pressure after 4-week treatment with the gastrodia-uncaria granules or placebo. The proportions of patients with a daytime SBP reduction of at least 10 mmHg (31.8% *vs.* 20.0%; *P* = 0.034) or a DBP reduction of at least 5 mmHg (38.9% *vs.* 25.6%; *P* = 0.024) were significantly higher in the gastrodia-uncaria granules (GUG) group (left, green bar) than the placebo group (right, orange bar). Reproduced with permission from Zhang *et al.*[35] DBP: Diastolic blood pressure; SBP: Systolic blood pressure.

differed according to the dipping status with the highest risk in reverse dippers (7.77%) and the lowest risk in extreme-dippers (4.76%).[9] However, the prognostic value of dipping status became non-significant after additionally adjusted for nighttime systolic blood pressure.[9] The finding was consistent with the results of the Japan ABPM Prospective Study,[36] which showed a 48% higher overall CV disease risk in risers than dippers. The risk for heart failure was greatest, and independent of nighttime blood pressure.[36]

Nighttime blood pressure was more closely associated with mortality outcome than daytime blood pressure.[8] Isolated nocturnal hypertension, as a special subtype of masked hypertension, defined as a nighttime hypertension and a daytime normotension, was associated with carotid intima-media thickening, increased urinary albumin-to-creatinine ratio,[37] and higher risk of total mortality and all CV events [Figure 3].[29] In a multiethnic international database on ABPM, the prevalence of isolated nocturnal hypertension was higher in Chinese (10.9%), Japanese (10.2%), and South Africans (10.5%) than in Western (6.0%) and Eastern Europeans (7.9%).[38] Higher prevalence of nocturnal hypertension may be partially attributable to blunted sodium metabolism because of either increased dietary sodium intake or decreased urinary sodium excretion. We recently found a significant interaction between urinary sodium excretion and frac-

tional excretion of lithium, as a measure of proximal tubular sodium reabsorption, in relation to nighttime diastolic blood pressure in 766 untreated Chinese.[9] The individuals with a lower fractional excretion of lithium, which reflects higher proximal tubular sodium reabsorption, had higher nighttime blood pressure (+4.9 mmHg, *P* < 0.001), whereas those individuals with a high fractional excretion of lithium, which reflects lower proximal tubular sodium reabsorption, showed a positive association between blood pressure and dietary sodium intake.[39] Nocturnal hypertension was more prevalent in patients with secondary hypertension,[40,41] obstructive sleep apnea,[40] chronic kidney disease,[41] and the metabolic syndrome,[42] and needs to be screened by ABPM.

Morning blood pressure surge

The CV complications often occur in the morning, and blood pressure usually also peaks in the morning. The parallel observations suggest that an exaggerated morning surge of blood pressure might be associated with adverse CV outcome.[43,44] Sleep-trough or pre-awakening morning blood pressure surge is evaluated by the difference between morning blood pressure after wake up and the nighttime or pre-awakening blood pressure. In the analyses based on the IDACO database,[45] morning blood pressure surge was predictive of CV events and mortality, particularly stroke in Asians and coronary
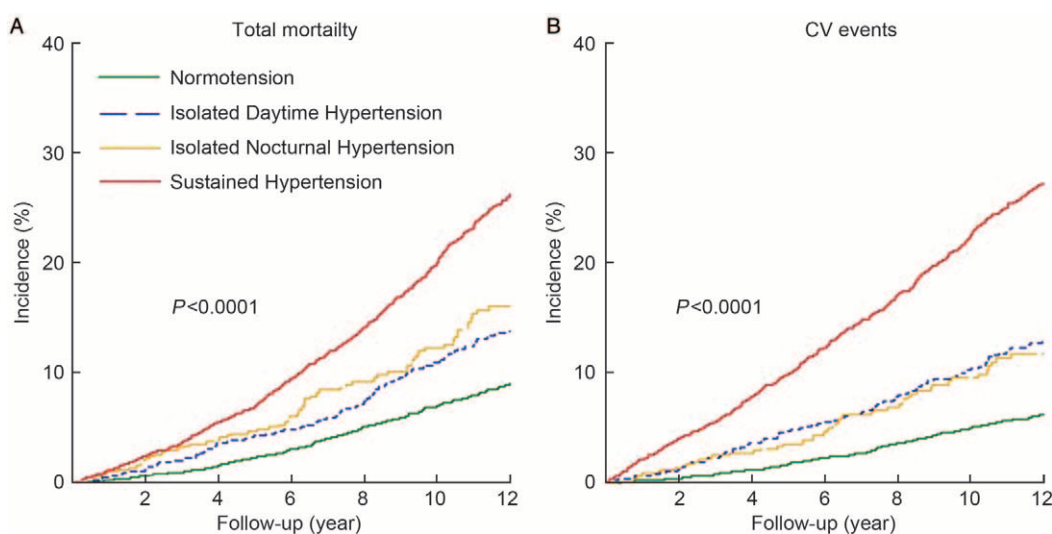
Figure 3: Cumulative incidence of total mortality (A) and all CV events (B) by ambulatory blood pressure status. Probability values are for the differences among the four categories by the log-rank test. Reproduced with permission from Fan *et al.*[29] CV: Cardiovascular.

events in Europeans. The HR of all-cause mortality and all CV events was 1.32 95% CI (1.09–1.59, $P = 0.004$) and 1.30 (1.06–1.60, $P = 0.01$), respectively, in the top decile of the systolic sleep-through morning surge (≥37.0 mmHg) *vs.* the overall population [Figure 4].

In spite of evidence on the usefulness of morning blood pressure surge in CV risk stratification, complexity and low reproducibility limit the clinical use of morning blood pressure surge. Recent studies propose to shift the focus to morning hypertension.[43,44] Morning hypertension refers to high blood pressure in the morning period, regardless of blood pressure during the rest hours of the day, and is defined as a morning blood pressure ≥135/85 mmHg on either ABPM or HBPM. In 1049 untreated Chinese outpatients, both ambulatory and home morning systolic blood pressure were significantly associated with carotid-femoral pulse wave velocity and urinary albumin-to-creatinine.[46] However, in the subgroup who had both HBPM and repeated ABPM within 1 month, only 11.8% to 16.7% of the patients with isolated morning hypertension defined according to various ambuatory definitions remained the status at the second measurement. If defined on home measurements, 42.1% of the patients persisted to be isolated morning hypertension.[46]

Reading-to-reading blood pressure variability

Cumulative evidence suggested that short-term blood pressure fluctuation within a 24-hour period was a significant predictor of CV risk.[47-49] Standard deviation and coefficient of variation of the 24-hour blood pressure are two straightforward evaluations of short-term blood pressure variation. Novel indices, such as average real variability[47] and variance independent of the mean,[50,51] have also been proposed. However, blood pressure variability contributes some but not much stratification over and beyond 24-hour blood pressure.[47] Moreover, the clinical utility of these indices is limited by a lack of accepted thresholds defining normal and pathological

blood pressure variability. The potential benefits from reducing blood pressure variability also remain to be illustrated. Long-acting antihypertensive drugs, such as dihydropyridine calcium-channel blockers and the combination of long-acting compounds, might be able to reduce blood pressure variability independent of mean blood pressure level. Based on these hypotheses, the Reducing Blood Pressure Variability in Essential Hypertension with Ramipril *vs.* Nifedipine gastrointestinal therapeutic system Trial (ClinicalTrials.gov Identifier: NCT02499822) recruited hypertensive patients with home systolic blood pressure standard deviation >7 mmHg and/or daytime ambulatory systolic blood pressure standard deviation >12 mmHg, and aimed to assess whether the degree of treatment-induced changes in blood pressure variability would be related to the degree of regression (or progression) of organ damage, after accounting for mean blood pressure reduction by treatment. This international multi-center randomized clinical trial completed in 2021 may shed some light on the perspectives for the treatment of blood pressure variability.

Guiding Antihypertensive Treatment

There is no evidence yet that ABPM-guided antihypertensive therapy further improves clinical outcomes. However, two randomized controlled trials[52,53] compared ABPM with office blood pressure in guiding antihypertensive treatment for blood pressure control, treatment intensity, and side effects. In a multicenter trial conducted in the 1990 s, 419 patients with an untreated office diastolic blood pressure ≥95 mmHg were randomized to antihypertensive drug treatment adjusted either according to the average of daytime ambulatory ($n = 213$) or three office diastolic blood pressure readings ($n = 206$).[52] During a median of 182 days follow-up, treatment intensity was significantly ($P < 0.001$) less in the ABPM than office blood pressure-guided therapy for the discontinuation rate of antihypertensive drug treatment (26.3% *vs.* 7.3%) and the progression rate of sustained multiple-drug treatment
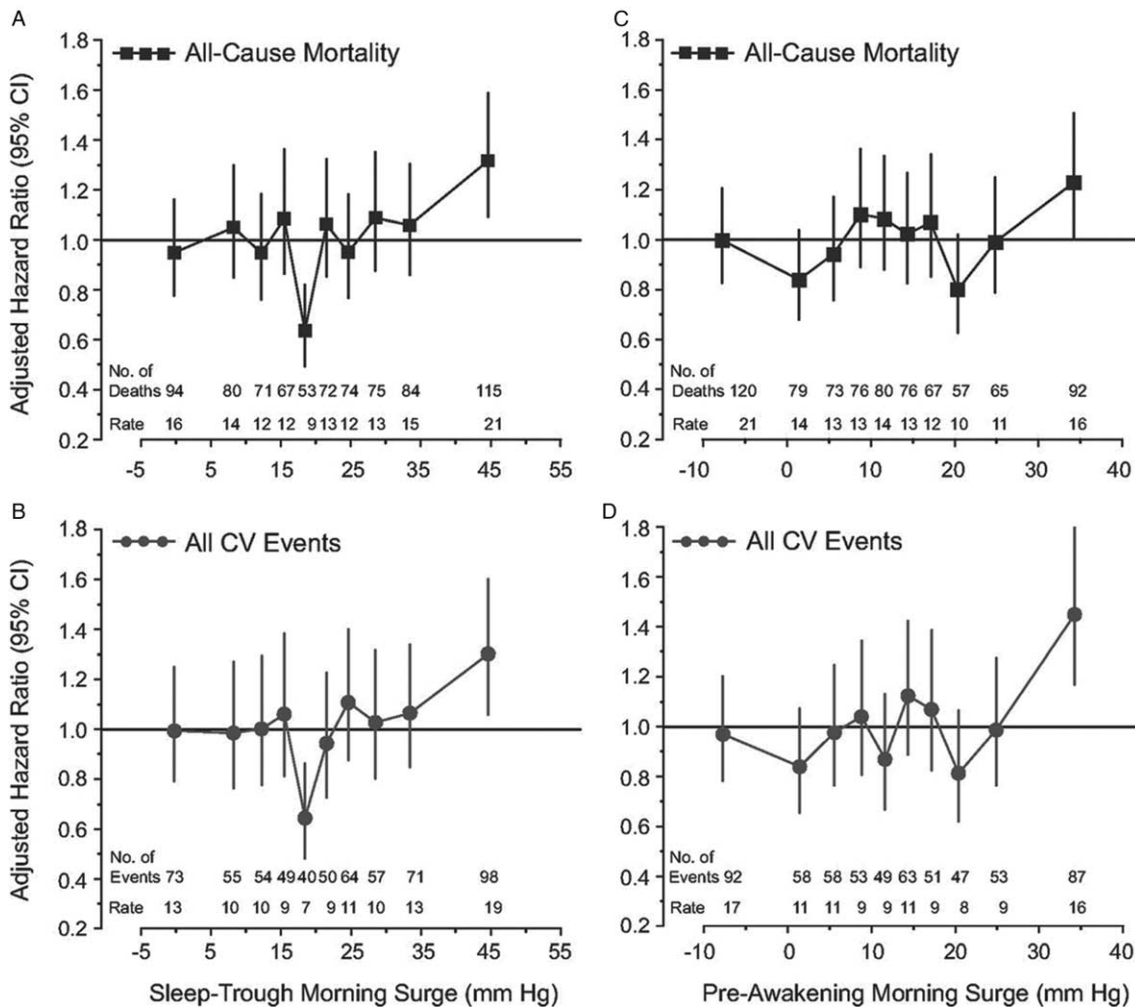
Figure 4: Multivariable-adjusted HRs(95% CI) for all-cause mortality (A and C) and for all fatal combined with non-fatal CV events (B and D) by ethnic- and sex-specific deciles of the sleep-through (A and B) and the pre-awakening (C and D) morning surge in SBP in 5645 subjects. The HRs express the risk in deciles compared with the average risk in the whole study population and were adjusted for cohort, sex, age, body mass index, smoking and drinking, serum cholesterol, history of cardiovascular disease, diabetes mellitus, antihypertensive drug treatment, 24-h SBP, and the systolic night:day blood pressure ratio. The number of events and incidence rates (events per 1000 person-years) are also given for each decile. Reproduced with permission from Li *et al.*[45] CV: cardiovascular; CI: confidence interval; HR: Hazard ratio; SBP: Systolic blood pressure.

(27.2% *vs.* 42.7%). Office (144.1/89.9 mmHg *vs.* 140.3/89.6 mmHg) and ambulatory systolic/diastolic blood pressure (129.4/79.5 mmHg *vs.* 128.0/79.1 mmHg) were similarly controlled in the two groups. Left ventricular mass and reported symptoms were also similar in the two groups.[52] A similar study was conducted in 136 patients with sustained hypertension (office systolic/diastolic blood pressure ≥140/90 mmHg and 24-hour systolic/diastolic blood pressure ≥130/80 mmHg), randomly assigned to a treatment strategy guided by 24-hour (target <130/80 mmHg) or office blood pressure (target <140/90 mmHg).[53] After 1 year of follow-up, the change in 24-hour systolic blood pressure was significantly greater in the APBM than office blood pressure group (mean difference –3.6 mmHg, 95% CI –7.0 to –0.3 mmHg), with even smaller number of antihypertensive drugs per participant in the former than latter group (1.76 *vs.* 1.95, *P* = 0.049).[53]

There is an ongoing study that investigates whether ABPM-guided antihypertensive therapy would improve clinical outcomes in patients with masked uncontrolled hypertension, namely, the MASked-unconTrolled hypERtension management based on office blood pressure or on ambulatory blood pressure measurement (MASTER) study (ClinicalTrials.gov Identifier: NCT02804074). In this 4-year trial, 1240 treated hypertensive patients with masked uncontrolled hypertension (repeated on treatment office blood pressure <140/90 mmHg, and at least one of the following: daytime, nighttime, and 24-hour ambulatory blood pressure ≥135/85 mmHg, ≥120/70 mmHg, and ≥130/80 mmHg, respectively) will be randomized to a management strategy based on office or ABPM. Clinical outcomes included CV and renal intermediate measurements (change in left ventricular mass and microalbuminuria) at 1 year and CV events at 4 years. This trial might provide some evidence on the possible benefit of ABPM-guided antihypertensive drug treatment.

There was also a randomized study that compared ABPM with HBPM in guiding antihypertensive therapy in 98 patients with an untreated daytime ambulatory diastolic

blood pressure ≥85 mmHg.[54] At the end of the 24-week follow-up, blood pressure was controlled similarly in the ABPM- and HBPM-guided treatment groups for both home and 24-hour, daytime, and nighttime ambulatory blood pressure changes.

Other Clinical Applications

Evaluation of arterial property

In individuals with elastic arteries, systolic and diastolic blood pressures change in parallel with the variation in mean arterial pressure throughout the blood pressure range. In individuals with stiff arteries, systolic pressure sharply rises with the increase in mean arterial pressure, whereas diastolic pressure may even decline. The dynamic relation between diastolic and systolic blood pressure provides insights into the stiffness of the arterial wall. From a 24-hour ambulatory recording, the regression slope of diastolic on systolic blood pressure could be calculated. Ambulatory arterial stiffness index (AASI) was defined as 1 minus the regression slope [Figure 5].[55,56] The stiffer the arterial tree, the closer the regression slope and AASI were to 0 and 1, respectively. AASI increased with age and mean arterial pressure, and correlated more closely with central and peripheral augmentation indexes than 24-hour pulse pressure.[56] Among normotensive subjects, the 95th percentile of AASI was 0.55 in Chinese and 0.57 in Europeans.[56] AASI ≥0.55 predicted CV and stroke mortality, even in subjects with normal daytime ambulatory blood pressure (<135/85 mmHg).[55] Similar to AASI, pulse pressure, the difference between systolic and diastolic blood pressure, also reflects the response of the arterial system to the intermittent ventricular ejection from the heart. Gavish and Bursztyn[57] developed a model-based method for splitting pulse pressure into
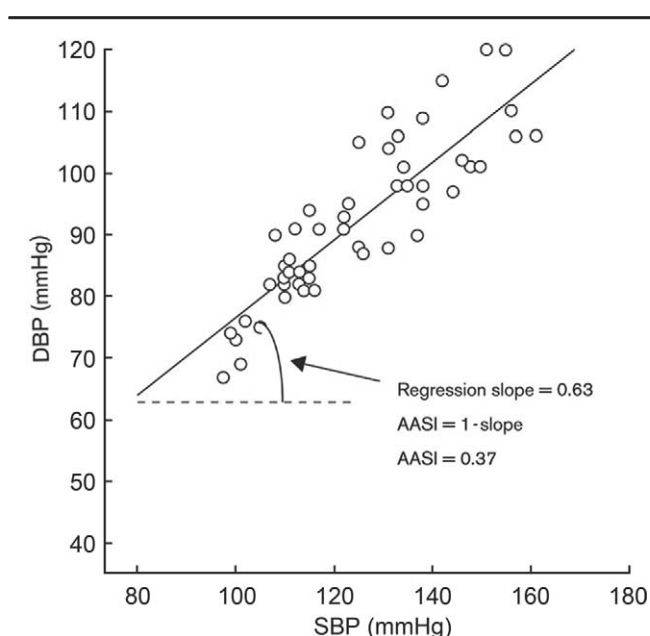
Figure 5: Derivation of the AASI from a 24-hour ambulatory blood pressure recording in one participant, whose 24-hour blood pressure was 129 mmHg for SBP and 95 mmHg for DBP. Reproduced with permission from Dolan et al.[65] AASI: Ambulatory arterial stiffness index; DBP: Diastolic blood pressure; SBP: Systolic blood pressure.

pressure-independent stiffness ("elastic," elPP) and the pressure-dependent stiffness ("stiffening," stPP). The predictive power of elPP[58] and stPP[57] for CV and total mortality was found in Israel and Japanese populations. Future studies evaluating the clinical relevance of these stiffness indices in the Chinese population are warranted.

Evaluation of salt-sensitivity

Reduced nocturnal dipping in blood pressure and increase in 24-hour heart rate were observed in salt-sensitive hypertensive patients during a high-sodium diet. Based on the combination of dipping status and 24-hour heart rate, patients could be divided into three groups of salt sensitivity:[59] low risk if dipping and a 24-hour heart rate ≤70 beats per minute; high risk if non-dipping and a 24-hour heart rate of >70 beats per minute; intermediate risk with the remaining combinations. The sodium sensitivity index evaluated by a formal salt sensitivity test and the prevalence of sodium-sensitive patients increased significantly from the low-risk class (25%) to the intermediate-risk (40%) and high-risk (70%) classes.[59] The performance of 24-hour ABPM may be an alternative assessment of sodium sensitivity in an easier manner than the traditional sodium sensitivity testing approach.

Conclusions and Perspectives

ABPM is clinically useful in the detection of white-coat and masked hypertension, especially isolated nocturnal hypertension. It may also help in CV risk assessment and stratification via evaluating circadian rhythm, blood pressure variability, and properties of the arterial system.

Several national and international hypertension guidelines, therefore, have clearly indicated the superiority of ABPM in the management of hypertension.[2,60,61] However, whether ABPM-guided antihypertensive treatment improves clinical outcomes still remains under investigation.

HBPM is increasingly available and usable in the management of hypertension.[62] In fact, the two out-of-office blood pressure measuring techniques are complementary in almost all aspects of hypertension management. ABPM substantially improves the diagnostic accuracy of hypertension by providing a 24-hour blood pressure profile, whereas HBPM is of particular importance in blood pressure control by improving treatment adherence.[62,63] However, the new technology of wearable devices might combine these two out-of-office techniques and provide accurate and comprehensive blood pressure evaluations in the future.[64]

Conflicts of interest

Jiguang Wang reports receiving lecture and consulting fees from Novartis, Omron, Servier and Viatris. The other authors declared no conflicts of interest.

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