When Office Blood Pressure Is Not Enough: The Case of Masked Hypertension

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An increasing attention has been devoted in the last two decades to masked hypertension (MH), a condition characterized by the fact that classification of a normal blood pressure (BP) status by office measurements is not confirmed by home and/or ambulatory BP monitoring (ABPM). MH definition (i.e., normal office BP, but high out-of-office BP) should be restricted to untreated subjects (true MH) whereas masked uncontrolled hypertension (MUCH) reserved to treated patients previously classified as hypertensives, presenting normal office BP and high ABPM or home values. Both MH and MUCH are associated with metabolic alterations, comorbidities, and hypertension-mediated organ damage (HMOD). Furthermore, the risk of cardiovascular events related to these conditions has been shown to be close or greater than that of sustained hypertension. This review discusses available evidence about MH and MUCH by focusing on its prevalence, clinical correlates, association with HMOD, prognostic significance, and their therapeutic implications.

Keywords: blood pressure; cardiovascular risk; hypertension; masked hypertension; organ damage.

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In the last decades, combined office and out-of-office (i.e., ambulatory or home) blood pressure (BP) measurements have increasingly been applied in clinical practice and research as they provide a comprehensive assessment of cardiovascular risk related to hypertension in a variety of clinical settings. By this approach, four different BP phenotypes may be identified, namely true normotension (normal office and out-of-office BP), sustained hypertension (elevated in-office and out-of-office BP), white-coat hypertension (WCH), or isolated clinic hypertension (elevated office and normal out-of-office BP), and the reverse pattern alternatively defined as masked hypertension (MH), white-coat normotension, and isolated ambulatory or home hypertension (normal office and elevated out-of-office BP). These BP patterns have been reported to differ in terms of prevalence, demographic/clinical correlates and metabolic features, magnitude of hypertension-mediated organ damage (HMOD) as well as risk of nonfatal and fatal cardiovascular events, and all-cause mortality. In the last two decades, increasing attention has been paid to MH, the condition characterized by the fact that classification of a normal BP status by office measurements is not confirmed by home and/or ambulatory BP monitoring (ABPM).

The term MH was used in the early 2000s by Pickering et al. in order to define the hypertensive condition not identified by routine office BP measurements. An increasing number of cross-sectional and longitudinal investigations have provided evidence that patients with MH have an increased frequency of HMOD and, more importantly, a higher risk of cardiovascular complications. This is because out-of-office BP, indeed, either monitored at home or in ambulatory conditions over 24 hours, has been consistently shown to have a closer relationship with morbidity and fatal events, and to be a more sensitive risk predictor of cardiovascular outcomes compared to clinic BP readings.

Several issues concerning MH remain under debate; in particular, elements of uncertainty concern the more reliable method (i.e., home vs. ABPM) for detecting subjects with hypertension out of the office environment, related factors, real prevalence and reproducibility over time, and therapeutic strategies aimed to reduce cardiovascular risk related to this condition.

This review discusses available evidence about MH by focusing on its prevalence, clinical correlates, association with HMOD, prognostic significance, and therapeutic implications.

**DEFINITION OF MH**

Out-of-office BP refers to either home or ABPM measurements, the latter usually performed over 24 hours. Both methods provide a larger number of BP values,
in particular during daily life conditions, compared to conventional office measurement. Current diagnostic thresholds for hypertension are lower for home or ABPM compared to office BP according to the 2018 European Society of Hypertension/European Society of Cardiology (ESH/ESC) guidelines (i.e., average 24-hour BP >130/80 mm Hg and home BP ≥135/85 mm Hg); on the contrary, this is not the case for the 2017 American College of Cardiology/American Heart Association (ACC/AHA) hypertension guidelines (i.e., average daytime BP and home BP ≥130/80 mm Hg). It should be preliminarily noted that MH definition (i.e., normal clinic or office BP, but elevated out of clinic BP) should be restricted to untreated subjects. Treated patients previously classified as hypertensives, presenting normal office BP and elevated ambulatory or home values should not be defined as MH, but rather masked uncontrolled hypertension (MUCH). The distinction is far from being semantic, because these heterogeneous conditions have different clinical, prognostic, and therapeutic implications. Several subtypes of MH have been described in the literature, including morning hypertension and daytime and nighttime hypertension. Daytime MH may be related to unhealthy lifestyle factors such as habitual active smoking, alcohol abuse, and elevated mental or physical stress. Nighttime MH is frequently observed in conditions associated with non-dipping or reverse dipping status, including high salt intake, renal dysfunction, obesity, sleep apnea syndrome, and autonomic failure.

**PREVALENCE OF MH**

The prevalence of MH, based on studies published before the publication of the 2017 ACC/AHA guidelines, has been estimated between 10% and 25% in population-based surveys and between 14% and 30% in normotensive clinic populations depending on methods and diagnostic criteria as well as on clinical characteristics of study samples (i.e., general population cohorts, subjects with suspected hypertension, diabetes mellitus, chronic kidney disease, metabolic syndrome, obesity, and sleep apnea syndrome).

In most studies providing information on MH, the 24-hour value of 130/80 mm Hg or daytime value of 135/85 mm Hg were the most common cutoffs normal ambulatory BP; corresponding cutoff values for home BP were 135/85 mm Hg. It should be pointed out, however, that Liu et al. in their pioneering study reported that 61 out of 295 clinically normotensive subjects (20%) examined in a hypertension outpatient clinic could be classified as MH based on daytime ambulatory systolic or diastolic BP exceeding 134/90 mm Hg, a diastolic cutoff that tended to underestimate MH prevalence.

Numerous studies investigated MH epidemiology in general population cohorts. In the Pressioni Monitorate e Loro Associazioni (PAMELA), an originally designed research study aimed at assessing normal values, prognostic significance of ambulatory and home BP in a representative sample of a Northern Italian general population (n = 1,860), MH prevalence at entry varied from 9% to 12% of untreated individuals, the variable prevalence depending on whether MH was defined by ambulatory or home BP, diastolic or systolic BP values.

Findings from the Jackson Heart Study, an African American population-based cohort including 909 participants with office systolic/diastolic BP <140/90 mm Hg, reported an MH prevalence of 27.5% (average daytime BP >135/85 mm Hg), a markedly higher figure than the PAMELA population.

In a recent systematic review and meta-analysis including 11 studies conducted in Africa, totaling a pooled sample of 7,789, MH condition (diagnosed by ABPM in 10 out of 11 studies) was present in around 11% of participants. No difference in MH prevalence was observed between studies recruiting participants from the community compared to hospitals; of note, MH was more frequently detected in urban compared to rural areas.

Wang et al. combined data from a moderate-sized community sample (Masked Hypertension Study) with data from a large, national wide sample (National Health
and Nutrition Examination Survey [NHANES]) in order to investigate MH prevalence in the United States. Using multiple imputation to simulate ABPM-hypertension status of NHANES participants, the authors estimated that MH national prevalence of among 139 million US adults with non-elevated clinic BP was around 12%.25

In addition to population-based surveys, single studies performed in subjects with treated, controlled clinic BP, 4,608 of whom (31.1%) had elevated ambulatory BP values (i.e., 24-hour BP equal or higher than 130/80 mm Hg). Finally, in a meta-analysis by Verberk et al.10 including 36 studies for a pooled population of 25,629 participants, average MH prevalence of was 17%. The prevalence was somewhat higher when diagnosis was based on home BP measurement compared to ABPM, although the difference failed to achieve statistical significance (21% vs. 17%, P = 0.42).

The recently released ACC/AHA hypertension guidelines proposed lower diagnostic thresholds of office (BP ≥ 130/80 mm Hg) for defining hypertension. New normal BP criteria have been also extended to ABPM, as values of 125/75 mm Hg, 130/80 mm Hg, and 110/65 mm Hg have been proposed for 24-hour, daytime, and nighttime periods, respectively. At difference, the 2018 ESH/ESC guidelines confirmed the office and ABPM cutoffs recommended in the previous 2013 ESH/ESC guidelines. On the basis of these differences, de la Sierra et al.28 evaluated the prevalence of MH and MUCH, defined by both the ESH/ESC and ACC/AHA criteria, in patients with office BP <130/<80 mm Hg (3,477 untreated and 5,934 treated patients), drawn from the Spanish ABPM Registry. They found that MH and MUCH prevalence ranged from 14% to 66%, being 2-fold higher according to ACC/AHA than ESH/ESC criteria.

Finally, the following points need to be further clarified: MH incidence, reproducibility, and evolution to sustained hypertension. Few studies have examined the incidence of MH and its risk factors. In a cohort study including 1,836 initially office and out-of-office normotensive participants followed up on average for 2.9 years Trudel et al.29 reported that cumulative incidence of MH (office BP <140/90 and daytime ≥ 135/85 mm Hg) was 10.3% and was associated with male gender, older age, higher education level, smoking status, and alcohol intake.

In the vast majority of studies, MH was defined by a single ABPM or home BP monitoring performed for short periods of time; consequently, available evidence on the reproducibility of this BP phenotype over time is scanty. Reproducibility of MH pattern examined in a cohort of 503 untreated Japanese workers by repeated home BP monitoring over a 6-month period was approximately 60%.30 Viera et al.31 found that 73% of 31 patients initially classified as having MH, based on office/daytime values, confirmed this BP pattern in the subsequent evaluation 1 week apart.

Trudel et al.32 investigating MH persistence in 1,669 white-collar workers over a 5-year period of follow-up showed that MH was persistent after 3 and 5 years in 38% and 18.5% of subjects, respectively, and progressed to sustained hypertension in 26% and 37% of cases, respectively. In the PAMELA study, clinical variables as well as office, home, and ABPM values were simultaneously measured at baseline and after a 10-year follow-up. The study design allowed to assess long-term variations of office and out-of-office BPs in each participant and documented that approximately 40% of subjects with MH at entry progressed to sustained hypertension at the end of follow-up.33

**CLINICAL CORRELATES OF MH**

Considering that a systematic screening of the general population aimed at unmasking MH is unfeasible, a diagnostic strategy restricted to subjects with a high pretest probability of having MH may be cost-effective. A large body of evidence supports the view that subjects with MH have a worse cardiovascular risk profile than normotensive counterparts. Observational studies focusing on demographic and clinical characteristics of MH reported that high normal office BP, current smoking, habitual alcohol drinking, obesity, metabolic syndrome, job stress, age, and sleep apnea syndrome are major factors affecting out-of-office BP in clinically normotensive individuals.

In the PAMELA population, approximately up to 20% of untreated participants with high-normal office BP exhibited elevated 24-hour ABPM values, at variance from subjects with normal BP (i.e., <120/80 mm Hg) in whom the prevalence of MH was less than 5%.

This strengthens the findings of previous studies reporting that high-normal BP is a strong indicator of the presence of MH and a condition in which out-of-office BP measurements should be considered.

The Masked Hypertension Study, indeed a worksite-based population study including 769 participants with office BP lower than 140/90 mm Hg, documented that MH prevalence was 10-fold higher in prehypertensive subjects (34%) than in subjects with optimal office BP (4%).34

Furthermore, subjects with MH from the PAMELA study had office BP values significantly higher than normotensive subjects (mean systolic BP 126 ± 7 vs. 118 ± 10 mm Hg and diastolic BP 82 ± 4 vs. 77 ± 7 mm Hg, respectively, P < 0.001 for both). Compared to true normotensive subjects, those with MH showed a greater male prevalence (70% vs. 44%, P = 0.002), as well as higher age (48 ± 12 vs. 43 ± 12 years, P = 0.003), body mass index (25 ± 4 vs. 23 ± 3 kg/m², P < 0.001), and fasting blood glucose values (94 ± 28 vs. 86 ± 13 mg/dl, P = 0.03).

In line with the findings provided by the PAMELA population, a number of studies have shown an association of male sex and older age with MH. Recent data collected in 9,550 untreated individuals from 13 population-based cohorts confirmed that MH is more prevalent among men than women (21.1% vs. 11.4%, P < 0.0001).35 The study failed to show a direct correlation between MH and age; the
prevalence rate of MH in men >70 years was even lower by 2-fold than in men aged 40–50 years.

Active and passive smokers have been suggested to have higher ambulatory BP levels than nonsmokers. Likewise, the association between active or passive smoking habit to MH has been reported by some authors but denied by others. The mechanisms by which active smoking increases out-of-office BP include sympathetic activation, endothelial dysfunction, and impairment of arterial compliance. In a case–control study, Verdecchia et al. compared office and 24-hour BP values in 115 heavy smokers (≥20 cigarettes/day; 91 men) and 460 nonsmokers (364 men) with essential hypertension: office BP was nearly identical in smokers and nonsmokers (158/99 vs. 158/98 mm Hg), daytime BP was higher in smokers than nonsmokers (150/97 vs. 143/93 mm Hg), whereas nighttime BP did not differ between the two groups (129/79 vs. 126/78 mm Hg).

The relation between passive smoking and MH has been investigated in 154 normotensive subjects exposed to passive smoking and in 10 not-exposed subjects. Passive smokers had similar office BP, but higher daytime BP (+4 mm Hg systolic and +5 mm Hg diastolic, P < 0.05 for both), and higher MH prevalence (23% vs. 8%, respectively) than not exposed subjects. At variance from the aforementioned study, in a large survey carried out in 2,370 white-collar workers, active smoking was unrelated to MH. Finally, neither active, nor former smoking status was related to MH in a population-based study carried out in Japan. Therefore, despite a solid physiopathological basis linking smoking status to increased ambulatory BP, findings in favor of its association with MH remain debated.

It has been suggested that psychosocial factors increase the risk of MH; available evidence on this dangerous association, however, is scarce. Job stress, defined as high psychological demands and low decision attitude, has been reported to be a risk factor for elevated ambulatory BP during work days. Unfortunately, a limited number of studies have specifically investigated the role of job strain as independent determinant of MH. In a recent study aimed to determine whether adverse psychosocial work factors according to the effort–reward imbalance model are associated with the prevalence of MH in a population of 2,369 white-collar workers, Boucher et al. found that exposure to high job stress was associated with MH, after adjusting for sociodemographic and cardiovascular risk factors.

The positive and linear relationship between alcohol consumption, BP, prevalence of hypertension, and cardiovascular risk factors has been established for a long time. Heavy habitual alcohol drinkers have been widely reported to be at high risk of hypertension. A meta-analysis of 56 epidemiological studies including 261,991 individuals provided evidence that reduction of alcohol consumption exerted beneficial effects on cardiovascular health and BP levels even in light-moderate drinkers. As for MH risk related to habitual alcohol intake, some studies have documented a significant association between these two conditions. In a study by Trudel et al., a higher weekly intake was related to increased prevalence of MH in women but not in men. The sex-specific association between alcohol consumption and MH needs to be further investigated.

Obstructive sleep apnea syndrome (OSA) is regarded as an independent risk factor for development and worsening of arterial hypertension, diabetes, heart rhythm disorders, and cardiovascular disease. The prevalence of nocturnal hypertension has been reported to be increased due to the impact of oxygen desaturation in the pathophysiology of BP elevation at night. Although few studies have examined the prevalence of MH in patients with OSA, available evidence indicates that isolated out-of-office hypertension, as assessed by ABPM, may occur in 30%–60% of cases and that continuous positive airway pressure has a favorable effect in reducing nocturnal BP and the prevalence of MH.

HMOD AND MH

HMOD reflects intermediate stages in the disease continuum linking high BP and coexistent risk factors to cardiovascular fatal and nonfatal events. A solid body of evidence supports the view that markers of cardiac and extra-cardiac HMOD are strong predictors of cardiovascular disease over and beyond BP levels and conventional risk factors.

Cardiac HMOD

As for the heart, systemic hypertension adversely affects cardiac structure and function by inducing a wide array of morpho-functional changes such as myocyte hypertrophy and fibrosis resulting in left ventricle (LV) dysfunction and left atrial and aortic root enlargement. Left ventricular hypertrophy (LVH), the pivotal marker of subclinical organ damage, is the consequence of chronic exposure of LV to pressure overload in combination to a variety of unhealthy non-modifiable and modifiable factors.

In their pioneering study, Liu et al. investigated for the first time the association of MH with HMOD in 295 clinically normotensive and 64 sustained hypertensive subjects and showed that LV mass index in 61 MH subjects was higher than in 234 true normotensives (86 ± 16 g/m² vs. 73 ± 14 g/m², respectively). Some years later, our research group showed that in the PAMELA population LVH prevalence in MH subjects (14%) was lower than in sustained hypertensives (26%), but much greater than in true normotensive participants (4%).

A meta-analysis of 12 studies published since 1999 provided a comprehensive information on cardiac HMOD, as assessed by echocardiography, in a pooled population of 776 untreated MH subjects identified by ABPM in different clinical settings as compared to 2,467 true normotensive and 1,641 sustained hypertensive individuals. The principal findings of the meta-analysis can be summarized as follows: (i) pooled LV mass index progressively increased from normotensive (79.2 ± 0.35 g/m²) to MH (91.6 ± 4.0 g/m²) and sustained hypertensive subjects (102.9 ± 3.3 g/m²); Figure 2; (ii) LVH prevalence rates were higher in MH (14%) and sustained hypertensive subjects (11%) than in normotensive controls (4%), without significant difference between the hypertensive groups; (iii) systolic BP either in the office as during daytime ambulatory monitoring showed a direct, significant correlation with LV mass index in both MH and
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sustained hypertensives. Subsequent studies have further shown that MH is more frequently associated to increased LV mass index independently of several confounders across different age strata. In a cross-sectional study of 774 black and white men and women aged 20–30 years, MH subjects exhibited higher odd ratios for increased left ventricular mass index (odds ratio 1.67, \( P = 0.03 \)) compared to normotensive individuals.

**Extra-cardiac HMOD**

Consistent, although not univocal, evidence exists about the value of carotid intima-media thickness (CIMT) in predicting incident cardiovascular events.

A meta-analysis of five studies including 2,752 untreated subjects (1,039 normotensive, 497 MH, and 766 hypertensive individuals) showed that mean common CIMT was lowest in normotensive (681 ± 24 µm), intermediate in MH (763 ± 57 µm), and highest (787 ± 58 µm) in sustained hypertensive subjects (\( P < 0.01 \) for the standard difference in means; Figure 3). Prevalence rates of carotid plaque were 15% in normotensive subjects, 28% in MH patients, and 27% in sustained hypertensives.

Additional evidence on the association between carotid atherosclerosis and MH derives from population-based studies including a large fraction of treated hypertensives and from treated hypertensive cohorts. In a cross-sectional investigation of 2,915 community-dwelling Japanese aged ≥40 years, average CIMT was significantly higher among MH subjects (770 µm) than among their normotensive counterparts (670 µm; \( P < 0.001 \)). Furthermore, Tomiyama et al., examining a group of 332 treated hypertensives, reported that maximal CIMT, but not mean IMT, was greater in MUCH (1,930 µm) than in controlled hypertensives (1,610 µm).

Other studies addressing markers of HMOD such as retinal changes, renal dysfunction, microalbuminuria, and arterial stiffness converge in emphasizing the increased frequency of micro- and macrovascular changes in subjects with MH.

The mechanisms underlying the association between MH and HMOD are not fully elucidated. A crucial factor probably relays on persistent out-of-office BP elevation, due to sympathetic over-activation elicited by stress, anxiety, job strain, smoking, alcohol consumption, metabolic alterations, and sleep apnea.

**MH AND CARDIOVASCULAR RISK**

A mounting body of evidence supports the notion that the risk of cardiovascular events is substantially greater in MH compared to normotension and close to or even higher than in sustained hypertension. The first study aimed to investigate the prognostic significance of MH for cardiovascular morbidity was conducted by Björklund et al. in a population of untreated 570 elderly men. During a follow-up of 8.4 years MH, as assessed by ABPM, exhibited a similar incidence of fatal and nonfatal coronary events, stroke, and peripheral vascular deaths as sustained hypertension, the relative risk being approximately 3-fold in MH than in true normotensives. Similar findings were reported by Bobrie et al. in a cohort of 4,939 treated elderly hypertensive patients followed up for a mean of 3.2 years. As compared to patients with controlled hypertension (normal office and home BP), the hazard ratio (HR) of cardiovascular events was 1.96 (95% confidence interval [CI], 1.27–3.02) in patients with uncontrolled BP (high BP with both measurement methods), 2.06 (95% CI, 1.22–3.47) in patients with normal office BP and elevated home BP , and 1.18 (95% CI, 0.67–2.10) in patients with elevated office BP and normal home BP . In the PAMELA population, the risk of cardiovascular death showed a progressive increase from subjects with normal office and 24-hour BP, MH, and sustained hypertension after adjusting for major confounders (Figure 4).

After these seminal reports, in the last years numerous observational, registry-based studies and meta-analyses...
have addressed the prognostic implications of both MH and MUCH.\textsuperscript{63–66} A Spanish-based registry cohort of 63,910 untreated and treated patients managed in primary care (follow-up of 4.7 years) MH, assessed by office/ABPM, was more strongly associated with all-cause mortality (HR, 2.83; 95% CI, 2.12–3.79) than sustained hypertension (HR, 1.80; 95% CI, 1.41–2.31) or WCH (HR, 1.79; 95% CI, 1.38–2.32).\textsuperscript{67} Results for cardiovascular mortality were similar as those for all-cause mortality.

In the Japan Morning Surge-Home Blood Pressure study including 4,261 outpatients treated at 71 primary practices or university hospitals, patients with MH, defined by home BP, showed a greater risk for stroke compared to those with controlled BP (HR, 2.77; 95% CI, 1.20–6.37) during a follow-up of 3–9 years, independent of traditional cardiovascular risk factors, urine albumin to creatinine ratio, and circulating B-type natriuretic peptide levels.\textsuperscript{68} A recent meta-analysis of 9 studies, totaling 14,729 participants evaluated with office, ambulatory, or home BP with a mean follow-up of 9.5 years, documented that individuals with MH had significantly increased rates of cardiovascular events and all-cause mortality than normotensives and white-coat hypertensives and lower rates of cardiovascular events than sustained hypertensives (HR, 0.61; 95% CI, 0.42–0.89).\textsuperscript{69} Notably, among patients on antihypertensive treatment, MH was associated to similar rates of cardiovascular events observed in sustained hypertensives.

The association of MUCH with cardiovascular events and all-cause mortality (composite primary outcome) was addressed by a meta-analysis of Pierdomenico et al.\textsuperscript{70} They selected 6 studies based on ABPM (12,610 patients with 933 events) and 5 based on home BP (17,742 patients with 394 events). The HR was 1.80 (95% CI, 1.57–2.06) for MUCH vs. controlled hypertensives. Subgroup meta-analysis showed that HR was 1.83 (95% CI, 1.52–2.11) in studies using ABPM and 1.75 (95% CI, 1.38–2.20) in those using home BP. The risk was significantly higher in MUCH than in controlled hypertensives independently of follow-up length and ethnic groups.

### TREATMENT OF MH

A large amount of evidence supports the view that MH and MUCH are more frequently associated with dysmetabolic risk factors and subclinical HMOD than true normotension.\textsuperscript{71–74} Consistent evidence, however, is lacking about the favorable effect of treatment in the reduction of cardiovascular outcomes. Lifestyle modifications are the first step in the management of both MH and MUCH. Unhealthy habits contributing to out-of-office elevation such as smoking and alcohol abuse should be strongly discouraged; tailored interventions aimed at treating the modifiable risk factors associated with out-of-office hypertension including obesity, diabetes, stress, and sleep apnea syndrome are recommended.

In addition, lowering ambulatory BP (or home BP) values with antihypertensive drugs, despite the presence of normal office BP, should be also considered in these patients with high cardiovascular risk, although the favorable effect of antihypertensive medications in the MH setting has been tested by few studies. Detection of HMOD in subjects with MH is an important argument favoring antihypertensive drugs combined with non-pharmacological measures.

The Home BP measurement with Olmesartan Naive patients to Establish Standard Target BP study investigated the effectiveness of olmesartan-based treatment on office and morning home BP in 21,341 patients classified as MH, WCH, and sustained hypertensives.\textsuperscript{75} Olmesartan-based therapy was effective in all BP groups, as it reduced office BP in WCH, home BP in MH, and office/home BP in sustained hypertensives. Notably, in the MH group antihypertensive treatment was well tolerated and safe.

In a practical therapeutic perspective, subclassification of MH in isolated daytime and isolated nocturnal MH may usefully guide anti-antihypertensive treatment.\textsuperscript{76–78} In the isolated daytime MH, indeed, morning administration of short-acting antihypertensive medications should be the preferred choice. Conversely, in isolated nocturnal MH, a chronotherapeutic intervention based on bedtime...
administration of BP-lowering drugs may effectively restore a normal circadian BP rhythm. In treated patients with MUCH (i.e., characterized by controlled office BP according to recommended targets, but elevated BP according to out-of-office measurements) considering their high cardiovascular risk, treatment up-titration should be recommended in order to ensure an effective control of both office and out-office BP. Furthermore, as MUCH has been frequently associated to a poorer control of nighttime rather than daytime BP, long-acting antihypertensive drugs with a high smoothness index, introduction, or reinforcement of bedtime therapy should be considered in order to improve BP control.

CONCLUSION

MH and MUCH are frequent BP phenotypes in the general as well as in the hypertensive population, only detected by combined office and out-office BP measurements. Both conditions convey an increased risk of HMOD, cardiovascular morbidity/mortality, and all-cause death, and appear to carry a similar risk as sustained hypertension. In order to reduce the burden of cardiovascular disease related to these conditions at the community level, a wider use of out-of-office BP measurement by ABPM and/or home BP in order to detect MH as well as monitor BP control in treated hypertensives is highly required. In addition to lifestyle measures, antihypertensive drug treatment should be started in MH and an accurate reevaluation of therapeutic scheme should be carried out in MUCH with the aim to obtain an optimal out-of-office BP control.

DISCLOSURE

The authors declared no conflict of interest.

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