Review Paper

Masked Hypertension Definition, Impact, Outcomes: A Critical Review

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The phenomenon of masked hypertension (MH) is defined as a clinical condition in which a patient's office blood pressure (BP) level is <140/90 mm Hg but ambulatory or home BP readings are in the hypertensive range. The prevalence in the population is about the same as that of isolated office hypertension; about 1 in 7 or 8 persons with a normal office BP level may fall into this category. The high prevalence of MH would suggest the necessity for measuring out-of-office BP in persons with apparently normal or well-controlled office BP. Reactivity to daily life stressors and behavioral factors such as smoking, alcohol use, contraceptive use in women, and sedentary habits can selectively influence MH. MH should be searched for in individuals who are at increased risk for cardiovascular complications including patients with kidney disease or diabetes. Individuals with MH have been shown to have a greater-than-normal prevalence of organ damage, particularly with an increased prevalence of metabolic risk factors, left ventricular mass index, carotid intima-media thickness, and impaired large artery distensibility compared with patients with a truly normal BP level in and out of the clinic or office. Also, outcome studies have suggested that MH increases cardiovascular risk, which appears to be close to that of in-office and out-of-office hypertension. The aim of this review was to define the entity of MH, to describe its prevalence in the general population, and to discuss its correlation with cardiovascular events. (J Clin Hypertens. 2007;9:956–963) ©2007 Le Jacq

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Arterial hypertension (AH) is the most frequent reason for adults to visit the physician's office.[1] Recent results indicate that there are at least 65 million adults in the United States with the diagnosis of hypertension, while data from European trials demonstrate that AH affects about 25% of the adult population.[1,2] Recent survey findings indicate that while awareness of hypertension is >70% and >80% of patients being treated, a target blood pressure (BP) level of <140/90 mm Hg is achieved in only about 50% of patient.[3,4]

Until recently, the diagnosis of AH was based on measurements performed in the doctor's office. The addition of ambulatory BP monitoring (ABPM) and home BP (HBP) recordings to conventional clinic measurement for defining BP status in clinical practice has added a new complexity to the process; the separation of normotension and hypertension can now be assessed independently by several methods, resulting in 4 potential groups of patients: truly normotensive; hypertensive (true, or sustained, hypertension); hypertensive by clinic measurement and normotensive by ambulatory or HBP measurements (white coat hypertension); and, finally, normotensive by clinic measurement and hypertensive by out-of-office measurement (Table I). The first 2 groups are easy to deal with. The third group has been extensively studied and is generally accepted as being at intermediate risk for cardiovascular (CV) morbidity when compared with normotensive or hypertensive individuals. Until recently, little attention has been paid to the

Table I. Categories of Hypertension According to Office and Ambulatory Blood Pressure Measurement
Category 1: Normotensive both in the office and at home (true normotension or controlled hypertension)
Category 2: Hypertensive both in the office and at home (true hypertension or uncontrolled hypertension)
Category 3: Hypertensive in the office and normotensive at home (isolated office hypertension or white coat hypertension)
Category 4: Normotensive in the office and hypertensive at home (masked hypertension)

fourth category of patients (normotensive by clinic measurement and hypertensive by home or ambulatory measurement).[5,6] The aim of this review was to define the phenomenon of masked hypertension (MH), its prevalence, and its correlation with CV events and target organ damage.

MH DEFINITION AND PATHOPHYSIOLOGY

Definition

In 1992, Pickering[7] described a condition in which patients who have a normal office BP (OBP) level are hypertensive at home. To date, there is no consensus about the nomenclature for this condition. It has been called isolated home hypertension, isolated ambulatory hypertension, reverse white-coat hypertension, MH, white coat normotension, inverse white coat hypertension, and inverse white coat response. Although Pickering originally used the term MH for untreated persons, the term is also used to refer to patients with treated hypertension in later publications.[8]

The overall accepted definition of MH recommended by the European Society of Hypertension is a clinical condition in which a patient's OBP level is <140/90 mm Hg, but ABPM or HBP readings are in the hypertensive range.[9–11] The recently published guidelines from the European Society of Cardiology/European Society of Hypertension described "isolated ambulatory hypertension" or MH, the reverse phenomenon of "white coat hypertension," in individuals with normal OBP levels (<140/90 mm Hg) who may have elevated ambulatory BP or HBP values.[12]

Pathophysiology

Several conventional coronary risk factors, including sex, age, and job strain, have been suggested to be associated with MH, although the exact mechanisms remain to be investigated. Job strain, defined as high psychological demands plus low decision latitude at work, causes an increase in BP and sustained increases in ambulatory BP even at 3-year follow-up. Job stress–induced MH is a subject of increasing interest; job strain was associated with incident hypertension in an 8-year cohort study of 3200 initially normotensive employees[13] and is a risk factor for hypertension, particularly in men with very demanding jobs.[14] Some individuals have been reported to have higher BP readings at work than in the clinic.[15] Job strain causes an increase in ambulatory BP at work, at home, and during sleep[16,17] (ie, individuals who are clinically normotensive may still experience higher ambulatory BP levels at work). Stress-induced MH may develop into sustained hypertension as a result of chronic stress due to job strain. Other factors also play a role in BP increase, including smoking[16,18–20] and excess alcohol intake.[16,19] Patients with MH and sustained hypertension are at equivalent risk for developing CV disease.[19,21,22] Also, stress-induced hypertension may lead to target organ damage.[23–31]

Table II. Categories of Patients in Whom Masked Hypertension Should Be Suspected
Young men
Patients with diabetes
Patients with kidney disease
Patients with transiently elevated blood pressure
Patients with unfavorable health-related lifestyle (smoking, obesity, excess alcohol intake)
Patients with high to normal clinic blood pressure level
Patients at high cardiovascular risk

IDENTIFICATION OF PATIENTS WITH MH

To "unmask" individuals with ambulatory BP–based or HBP-based hypertension presents a problem for the clinician. Clearly, ABPM cannot be performed in all individuals with normal OBP, and the use of self–BP measurement may not be practical for some individuals. Yet, present evidence suggests that MH implies increased CV risk, and efforts should be made to identify individuals with this condition. Young men who are obese and sedentary and persons with increased BP reactions to standing will be optimal candidates for out-of-office BP measurement or ABPM in spite of a normal OBP level. Out-of-office BP readings should also be performed in persons with normal OBP and a high CV risk profile, in diabetic individuals, and in individuals with kidney disease and proteinuria (Table II). The high prevalence of MH among patients who have had elevated OBP at previous examinations suggests that these individuals should also be evaluated to rule out MH.[32,33]

ABPM and HBP monitoring are the most important tools in the diagnosis of MH. Clinically

important disagreement between the 2 methods in the diagnosis of MH is uncommon. Differences in physical activity and emotional challenges during the day might account for the limited reproducibility of out-of-office BP measurements and might explain the large discrepancy between the 2 methods that have been observed in some individuals. In addition, technical differences among the devices used for assessing HBP and ambulatory BP might account, at least in part, for the differences found. Therefore, the 2 methods appear to be interchangeable in the diagnosis of the majority of patients with MH. It might be argued, however, that for long-term follow-up of patients with treated MH, HBP monitoring is more appropriate than ABPM because of its lower cost and greater convenience for repeated measurements.[11,34]

PREVALENCE OF MH IN ADULTS

While there are no definitive data on the prevalence of MH, different BP thresholds have been proposed to define MH ranges.[35] The prevalence of MH in the general population could be as high as 10%,[36,37] while data obtained in several cross-sectional studies have demonstrated large differences, with prevalence rates from a low of 8% to a high of 49%.[32]

Two population-based studies performed in Italy[38] and in Japan[39] report prevalence rates of 9% and 13.4%, respectively. The results from the Pressione Arteriose Monitorate E Loro Associazioni (PAMELA) study[38] of 3200 Italians showed that 9% of the study population had MH, with an average OBP of 129/84 mm Hg, which although still within the normal range was higher than found in the true normotensive participants (112/77 mm Hg). The PAMELA study used a lower upper limit of normal for the 24-hour BP (125/79 mm Hg) compared with most other studies. It could be argued that if a higher level had been used, the number of participants with MH would have been smaller. Data from the Ohasama study,[39] conducted in a small Japanese town, reported that 10.2% of participants with normal OBP levels had ambulatory pressures that were in the "borderline hypertensive" range (>133/78 mm Hg for 24-hour average) and another 3.2% in the "definitely hypertensive" range (24-hour BP >144/85 mm Hg).[38,39]

Stergiou and colleagues[34] investigated the level of agreement between ambulatory BP and HBP in the diagnosis of MH in 438 patients referred to an outpatient hypertension clinic. Similar proportions of patients with MH were diagnosed by ambulatory BP (14.2%) and HBP (11.9%). Among 132 participants with normal OBP levels, there was disagreement in the diagnosis of MH between the ambulatory BP and the HBP method in 23% of patients for systolic BP and 30% for diastolic BP.[34]

A larger cross-sectional, descriptive study from Spain involving 1400 individuals older than 18 years, randomized and stratified by age and sex, sought to determine the prevalence of MH in the general population by means of HBP measurement.[40] Two BP measurements in the clinic and 12 HBP measurements in 1 week were performed. Pressure was seen as normal when mean OBP levels were <140/90 mm Hg and HBP level was <135/85 mm Hg. Hypertension was defined as an OBP level >140 mm Hg and an HBP level >135/85 mm Hg or if the patient was in treatment for hypertension. MH was diagnosed when the OBP level was <140/90 mm Hg and home BP levels were >135/85 mm Hg. A total of 1153 participants (560 men and 593 women; 82.4% of the sample) were included. The prevalence of MH was 8.9% (CI ±1.6) in the general population and 9.8% (CI ±3.2) in individuals with hypertension.[40] Other researchers found that 36 of 267 men (13.5%) in the Cornell Worksite study had MH, defined as a daytime ambulatory diastolic BP level >85 mm Hg and an OBP level <85 mm Hg.[41] Selenta and associates,[42] in a study of 319 clinically normotensive volunteers, all of whom had 5 clinic and 12-hour daytime ambulatory BP measurements, found that 23% had MH, defined as a daytime BP level >135/85 mm Hg.

The issue with patients who have treated hypertension is somewhat different. By definition, these are patients in whom a diagnosis of hypertension has already been made, so screening is not an issue. MH is of potential importance in these patients, however, because the OBP reading may give a false impression that BP is adequately controlled. The prevalence of MH in treated elderly hypertensive persons was 9.4% when assessed with HBP monitoring in the Self Measurement of Blood Pressure at Home in the Elderly: Assessment and Follow-Up (SHEAF) study[19] and 19% in the Japan Home vs Office Measurement Evaluation (J-HOME) study. The J-HOME study population consisted of 3303 outpatients with essential hypertension who were receiving antihypertensive treatment.[43] In a survey of patients attending a hypertension clinic, all of whom underwent ABPM, Pierdomenico and colleagues[44] reported that one-third of patients whose BP was controlled by clinic criteria (OBP <140/90 mm Hg) had MH (daytime BP <135/85 mm Hg) (Table III).

Table III. Prevalence of MH[a]

Study	No. of Patients	Type of Patients	% of Patients With MH
Sega et al[38]	3200	Adults	9.0
Imai et al[39]	1332	Adults	13.4
Stergiou et al[34]	438	Adults	14.2
Marquez Contreras et al[40]	1400	Adults	8.9
Belkic et al[41]	267	Adults	13.5
Selenta et al[42]	319	Adults	23.0
Matsuoka and Awazu[57]	136	Children	11.0
Lurbe et al[58]	592	Children	7.6
Stabouli et al[59]	85	Children	9.4

[a]Varies greatly according to different definitions. Abbreviation: MH, masked hypertension.

ASSOCIATION OF MH WITH TARGET ORGAN DAMAGE IN ADULTS

The first study to look at the issue of target organ damage in patients with MH took place in 1999.[26] A group of patients with MH had greater left ventricular mass and more carotid atherosclerosis than true normotensive persons and thus were similar to true hypertensive persons. The left ventricular mass index was 73 g/m^2 in the true normotensive persons, 86 g/m^2 in the masked hypertensive patients, and 90 g/m^2 in the true hypertensive patients. Carotid plaque was present in 15% of true normotensive participants and in 28% of both the masked and true hypertensive participants. This was the first finding to suggest that patients with MH may be at increased risk for CV morbidity.[26,45] Recently, results from the PAMELA study have shown that left ventricular mass index and hypertrophy were similarly greater in participants found to have normal OBP but elevated HBP or ambulatory BP,[36] while other data published from Japan have demonstrated that the reverse white coat effect is an independent risk factor for left ventricular hypertrophy, especially concentric hypertrophy, in patients with treated hypertension.[46] In another study from Japan, MH was associated with microalbuminuria, a marker of early renal damage, in patients with treated hypertension,[47] while data from the Ohasama study have shown that intima-media thickness of the near and far wall of both common carotid arteries in individuals with sustained hypertension and MH was significantly greater than in those with sustained normal BP and white coat hypertension.[48]

Tomiyama and coworkers[49] investigated how MH modified target organ damage in 332 outpatients with treated hypertension. The participants were classified into 4 groups according to OBP (<140/90 or ≥140/90 mm Hg) and daytime ambulatory BP (<135/85 or ≥135/85 mm Hg) levels (ie, controlled hypertension [low OBP and ambulatory BP level], white coat hypertension [high OBP but low ambulatory BP level], MH [low OBP but high ambulatory BP level], and sustained hypertension [high OBP and ambulatory BP level]). Left ventricular mass index, carotid maximal intima-media thickness, and urinary albumin levels were determined in all patients. Of 332 patients, 51 (15.4%), 65 (19.6%), 74 (22.3%), and 142 (42.8%) patients were identified as having controlled hypertension, white coat hypertension, MH, and sustained hypertension, respectively. Left ventricular mass index, maximal intima-media thickness, and urinary albumin level in MH were significantly higher than in controlled hypertension and white coat hypertension and were similar to those in sustained hypertension. Multivariate regression analyses revealed that the presence of MH was one of the independent determinants of left ventricular hypertrophy, carotid atherosclerosis, and albuminuria.[49]

MH AS A PREDICTOR OF CV EVENTS

Thus, there is a body of evidence that indicates that MH is a significant predictor of CV disease. Data from the PAMELA study[50] revealed that the hazard ratio for CV death showed a progressive increase in those with selective OBP elevations (white coat hypertension), selective 24-hour BP elevations (masked hypertension), and elevations in both OBP and 24-hour BP. This was also true when the above conditions were identified by OBP compared with HBP values. Selective elevation in HBP compared with ambulatory BP or vice versa also carried an increased risk. There was a progressive increase in both CV and all-cause mortality risk in patients in whom OBP, HBP, and ambulatory BP were all normal over those in whom 1, 2, or all 3 BP measurements were elevated, regardless of which BP assessment was considered. The trends remained significant after adjustment for age and sex, as well as, in most instances, after further

adjustment for other CV risk factors. Thus, white coat hypertension and MH, both when identified by OBP and ambulatory BP or by OBP and HBP, are not prognostically innocent.[50]

The prognostic value of MH in CV outcomes was also examined in the Ohasama study,[51] which obtained 24-hour ambulatory BP and "casual" BP values in 1332 participants (872 women, 460 men) aged 40 years or older. Survival and stroke morbidity were then evaluated over a mean duration of 10 years. Composite risk of CV mortality and stroke morbidity examined using a Cox proportional hazards regression model for patients with white coat hypertension (casual BP ≥140/90 mm Hg, daytime BP <135/85 mm Hg; relative hazards [RH], 1.28; 95% confidence interval [CI], 0.76–2.14) was no different from risk in patients with sustained normal BP (casual BP <140/90 mm Hg, daytime BP <135/85 mm Hg). Risk was significantly higher in participants with MH (casual BP <140/90 mm Hg, daytime BP ≥135/85 mm Hg; RH, 2.13; 95% CI, 1.38–3.29) or sustained hypertension (casual BP ≥140/90 mm Hg, daytime BP ≥135/85 mm Hg; RH, 2.26; 95% CI, 1.49–3.41) than in participants with sustained normal BP, however. Similar findings were observed for CV mortality and stroke morbidity among subgroups by sex, use of antihypertensive medication, and risk factor level (all *P* values for heterogeneity >.2).[51]

Similar results were obtained from an Italian study[44] in 340 patients with responder hypertension (OBP <140/90 mm Hg and daytime BP <135/85 mm Hg), 126 with MH (OBP <140/90 mm Hg and daytime BP >135/85 mm Hg), 146 with false resistant hypertension (OBP ≥140/90 mm Hg and daytime BP <135/85 mm Hg), and 130 with true resistant hypertension (OBP ≥140/90 mm Hg and daytime BP >135/85 mm Hg). During the follow-up period (4.98±2.9 years), the event rates per 100 patient-years were 0.87, 2.42, 1.20, and 4.10 in patients with responder, masked, false resistant, and true resistant hypertension, respectively. After adjustment for several covariates, including OBP (forced into the model), Cox regression analysis showed that CV risk was significantly higher in patients with MH (MH vs responder hypertension, relative risk [RR], 2.28; 95% CI, 1.1–4.7; *P*<.05) and in true resistant hypertension (true resistant vs responder hypertension, RR, 2.94; 95% CI, 1.02–8.41; *P*<.05), whereas there was no significant difference between false resistant and responder hypertension.[44]

MH IN DIABETES AND KIDNEY DISEASE

Although the prevalence of treated or untreated MH in patients with diabetes and nephropathy remains high, the effects on CV and target organ damage have not been clarified. MH is associated with a higher risk of end-stage renal disease in patients with chronic kidney disease.[52,53]

The prevalence and clinical significance of MH in diabetic patients have infrequently been described. A recent study evaluated the impact of MH on microvascular complications and echocardiographic changes in 135 normotensive type 2 diabetes mellitus (DM) patients.[54] Patients underwent urinary albumin excretion rate (UAER) measurement, echocardiography, and 24-hour ABPM. Patients with increased daytime BP levels (≥135/85 mm Hg) were classified as having MH. The prevalence of MH was 30% (n=41). There was no difference between normotensive patients and those with MH based on ABPM, in terms of age, DM duration, smoking status, body mass index, waist circumference, serum creatinine level, or glycemic or lipid profiles. The systolic OBP was higher in the MH group (127.8±7.5 vs 122.9±10.2 mm Hg; *P*=.003) than in the normotensive group. UAER was also increased in the MH group (21.3 [2.5–1223.5] μg/min vs 8.1 [1.0–1143.0] μg/min; *P*=.001), as was interventricular septum (1.01±0.15 cm vs 0.94±0.13 cm; *P*=.015) and posterior wall thickness (0.96±0.12 vs 0.90±0.10 cm; *P*=.006). After adjustments for DM duration, sex, smoking status, and low-density lipoprotein cholesterol and hemoglobin A_{1c} values, all associations were sustained for daytime systolic BP, but not for systolic OBP.[54]

Also, the association of MH with microvascular and macrovascular end organ damage has been studied in 81 clinically normotensive Japanese diabetic persons.[55] The prevalences of silent cerebral infarcts, increased left ventricular mass, and albuminuria were also evaluated. Of 81 patients, 38 (46.9%) were classified as having MH and showed significantly more silent cerebral infarcts (mean ± SE: 2.5±0.5 vs 1.1±0.2; *P*=.017) and more albuminuria (39% vs 16%; *P*=.025), but no increase in left ventricular mass index, over the normotensive persons in the OBP monitoring and ABPM groups. The prevalence of MH in this diabetic population was high (47%). Diabetic patients with MH showed evidence of brain and kidney damage. Hence, out-of-office monitoring of BP may be indicated in diabetics whose BP is normal in the clinic.[55]

CHILDREN AND MH

In children referred because of elevated BP, the MH phenomenon appears to be common.[56] The first MH study in children was published from Japan in 2004.[57] In this study, 136 patients (59 male and

77 female; aged 6–25 years, mean 13.1±4.7 years) were studied. In all patients, office BP measurements with auscultatory technique were less than the 95th percentile for sex and age or <140/90 mm Hg for those older than 18 years. MH was diagnosed when either systolic or diastolic daytime ambulatory BP values were greater than or equal to the 95th percentile for sex and height of reference values or ≥135/85 mm Hg for those older than 15 years. Of 136 patients, 15 (11%) had MH. The prevalence of MH was higher in boys (19%) than in girls (5%) but was not different between younger (15 years or younger) and older (older than 15 years) patients (11% vs 12%). The diagnoses in the group with MH included 3 patients with diabetic nephropathy, 2 with obesity, and 2 with orthostatic dysregulation.[57] This study is not representative of a healthy young population; participants consisted of healthy children and those in a recovery phase after an acute or chronic illness.

Other data from Spain have shown that out of 592 youths enrolled (aged 6–18 years) in the study, 535 were normotensive on OBP monitoring and daytime ABPM (90.4%) and 45 had MH (7.6%).[58] Compared with normotensive controls, participants with MH had a higher ambulatory pulse rate, were more obese, and were 2.5-fold more likely to have a parental history of hypertension. Among 34 patients with MH (median follow-up, 37 months), 18 became normotensive, 13 had persistent MH, and 3 had sustained hypertension only. Patients with persistent MH (n=17) or who progressed from MH to sustained hypertension (n=3) had a higher left ventricular mass index (34.9 vs 29.6 g/m$^{2.7}$; *P*=.023) and a higher percentage with left ventricular mass index above the 95th percentile (30% vs 0%; *P*=.014) than normotensive controls.[58]

A recent study from Greece assessed the prevalence of MH in 85 children who underwent OBP measurements and 24-hour ABPM.[59] Children with both office and ambulatory normotension or hypertension were characterized as confirmed normotensives or hypertensives, respectively. MH, defined as office normotension and ambulatory hypertension, was found in 9.4% of the children and was only present in nonobese participants.[59] The above data suggest that the MH phenomenon seems to be common in children.

CV EVENTS AND TARGET ORGAN DAMAGE IN CHILDREN WITH MH

One of the above-mentioned studies in MH in children provided evidence for target organ damage.

The Greek study[59] compared children with normotension (n=45), confirmed hypertension (n=21), white coat hypertension (n=11), and MH (n=8) and found that those with confirmed hypertension and MH had a significantly higher left ventricular mass index than those with confirmed normotension (34.0±5.8 g/m$^{2.7}$, 31.9±2.9 g/m$^{2.7}$, and 25.3±5.6 g/m$^{2.7}$, respectively; *P*<.05). White coat hypertensives tended to have greater left ventricular mass index than did confirmed normotensive patients, but the difference was not statistically significant (27.8±5.1 g/m$^{2.7}$ vs 25.3±5.6 g/m$^{2.7}$). No significant differences were found in the intima-media thickness of the carotid arteries among confirmed participants with normotension, white coat hypertension, MH, and confirmed hypertension.[59]

These findings are based on small groups of children with MH, and more research in larger samples is definitely required, but these results appear to agree with findings from adult studies that showed MH to be associated with increased left ventricular mass index and greater CV risk compared with normotensive patients.

CONCLUSIONS

MH is a clinical condition that applies to patients whose OBP is in the normal range but ambulatory BP or HBP is elevated. A major issue concerns the prevalence of MH; different BP thresholds have been proposed to define MH limit ranges. It is difficult to compare the results of different studies. How such individuals should be identified is questionable; published guidelines on how to define MH are lacking. If it is accepted that ambulatory BP gives a better prognosis than OBP and that the correlation between the two is only moderate, it is logical to propose that there will be a significant number of persons who are truly hypertensive but in whom the diagnosis is missed by clinic measurement. As noted, HBP value may also be used as a criterion for MH. It is crucial that physicians evaluate children's and young adults' out-of-office BP; MH represents a strong predictor of target organ damage and CV disease.

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