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Current status of white coat hypertension: where are we?

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Abstract: White coat hypertension (WCH) is characterised by an elevated clinic blood pressure (BP) with normal ambulatory or home BP. It is well recognised in clinical practice and occurs in approximately one-third of untreated patients with elevated clinic BP. Current evidence suggests that WCH is associated with cardiovascular risk factors, including the development of sustained hypertension and the presence of target organ damage. However, its effects on cardiovascular outcomes remain a matter of debate. There is also insufficient evidence from randomised controlled trials to determine whether WCH warrants treatment. This narrative review aims to provide an update on the current understanding of WCH. It focuses on the clinical characteristics and potential implications of WCH, its relationship to cardiovascular risk and the evidence regarding treatment. Gaps in existing research are also highlighted.

Keywords: cardiovascular risk, target organ damage, white coat hypertension

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Introduction

White coat hypertension (WCH) describes a blood pressure (BP) phenotype present in untreated individuals with elevated clinic BP, but normal out-of-office values. There has been a growing recognition of this phenomenon since it was first noted over three decades ago, and it now features in both national and international hypertension guidelines.^{1–4} Despite this, there are considerable gaps in our understanding of WCH. It is still not clear what risk is conferred by WCH and whether it warrants treatment.

WCH is an important phenomenon to understand because it is a proposed risk factor for the development of sustained hypertension (SH), target organ damage (TOD) and possibly the occurrence cardiovascular (CV) events. Recently, WCH has been found to increase the relative risk of sustained hypertension by almost three-fold when compared with patients with normal BP.⁵ Furthermore, WCH can lead to impairment of myocardial function and, compared with normotensive patients, there is an increased risk of carotid atherosclerosis.^{6,7} The association between WCH and CV events is considerably less clear. Some evidence suggests that WCH is

an intermediate risk category positioned somewhere between those with normotension and sustained hypertension.^{8,9} This is in contrast to other studies, which have found that patients with WCH do not have additional CV risk.^{10,11} This apparent lack of clarity deserves further investigation.

The dearth of randomised controlled trial data on WCH has led to inference of results from subgroup analyses performed in patients with numerous forms of hypertension. These types of analyses have yielded controversial results. Some studies are in favour of the treatment of WCH, whilst others suggest that treatment benefits only those with sustained hypertension.^{12,13} Based on this limited evidence, the European Society of Hypertension (ESH) has developed guidelines for the treatment of WCH. They suggest that patients with WCH and no additional CV risk factors should be managed with lifestyle changes and closely followed due to their increased risk of developing SH and TOD. In individuals with WCH and evidence of hypertension-mediated organ damage or elevated CV risk, it may be appropriate to offer recommendations on lifestyle changes in conjunction with drug treatment.³

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Definition

WCH describes an elevated clinic BP in the presence of normal out-of-office BP values. This BP phenotype is also referred to as white coat syndrome. The ESH suggest that patients with an office reading of at least 140/90 mmHg and a mean 24-h BP of less than 130/80 mmHg are deemed to have WCH.¹⁴ This differs from the original definition by Pickering *et al.*, as it uses 24-h rather than day-time ambulatory BP monitoring.¹ This updated definition may be preferable as it includes night-time BP, which has been shown to be a stronger predictor of outcomes than day-time BP.¹⁵ The ESH guidance also states that the term WCH should be reserved for untreated individuals only. This is in contrast to the white coat effect, which describes the difference between an elevated clinic BP and a lower home or ambulatory BP in both untreated and treated patients.¹⁴ The white coat effect is considered clinically significant if the difference between clinic and out-of-office BP exceeds 20/10 mmHg.⁴

There is no universally accepted approach to investigate and define WCH. This is not unrelated to the various definitions of hypertension (Table 1). ESH, American College of Cardiology/American Heart Association (ACC/AHA) and National Institute for Health and Care Excellence (NICE) guidelines all differ from one another in this regard (Table 2). ACC/AHA guidelines define WCH as a clinic BP of 130/80 mmHg or above, in the presence of day-time ambulatory or home BP less than 130/80 mmHg. Ambulatory and home BP monitoring (ABPM and HBPM respectively) are offered only when WCH is suspected and following a 3-month intervention of lifestyle modification.⁴ In the NICE guidelines, ABPM or HBPM is offered to those with a clinic BP of 140/90 mmHg or higher. They are deemed to have WCH if day-time ambulatory or average home BP is below 135/85 mmHg.² The lower threshold values in the ACC/AHA guidelines are due largely to the findings of the Systolic Blood Pressure Intervention Trial (SPRINT) study.¹⁶ This showed that, in patients with systolic BP (SBP) > 130 mmHg and elevated CV risk without diabetes, intensive BP treatment (target SBP < 120 mmHg) reduced CV events and all-cause mortality compared with standard treatment (target SBP < 140 mmHg). Importantly, the participants of this study were unattended during clinic BP measurement.¹⁷ These values are

significantly lower than those obtained using conventional observed BP measurement due to a reduction in the white coat effect.¹⁸ It has therefore been suggested that the BP values achieved in the SPRINT study, and subsequently reflected in the ACC/AHA guidelines, might be similar to the more conservative thresholds in the ESH and NICE guidelines.¹⁹

There are several explanations for the variation in how WCH is defined. Firstly, some individuals display a slight increase in clinic BP compared with 24-h ambulatory BP.²⁰ It is therefore very difficult to assign a discrete cut-off value at which this elevation in clinic BP becomes pathological, and should therefore be considered WCH. Secondly, multiple comparators can be used when comparing clinic and out-of-clinic readings. These include home BP, day-time ambulatory BP, night-time BP and 24-h recordings. It is not yet clear which of these comparators are preferred for the identification of patients with WCH. Variability in the definition of WCH means that there is some heterogeneity in the subjects included in different studies. It is consequently more difficult to reach conclusions regarding the effects of WCH and its treatment.

Further variability in the identification of patients with WCH occurs due to the methods used to record BP. Poor clinic BP measurement technique such as insufficient patient rest time, crossed legs and small cuff size can lead to spuriously elevated BP measurements that could mimic WCH.²¹ The diagnosis of WCH is also influenced by the number of BP recordings and the regression to the mean phenomenon. In patients classified as having stage 1 hypertension on initial clinic BP measurement (140–159/90–99 mmHg), 35% fall to a lower BP category when a mean of the second and third clinic values is used.²² The same phenomenon occurs when BP is measured on multiple occasions. In a study of patients that initially met the criteria for WCH, mean clinic BP reduced from 146 to 135 mmHg and mean ABPM increased from 120 to 125 mmHg at follow up.²³

Epidemiology

The prevalence of WCH has been estimated at 13% from meta-analysis of studies performed on treated and untreated subjects. This analysis used

Table 1. Current staging of hypertension by NICE, ESH/ESC and ACC/AHA. Adapted from NICE, ESH/ESC and ACC/AHA guidelines.²⁻⁴

| | Systolic BP (mmHg) | Diastolic BP (mmHg) |
|--|--------------------|---------------------|
| NICE (2019) | | |
| Normotension | <120 | <80 |
| Stage 1 Hypertension | ≥140 | ≥90 |
| Stage 2 Hypertension | ≥160 | ≥100 |
| Severe hypertension | ≥180 | or ≥120 |
| ESH/ESC (2018) | | |
| Normotension | <120 | <80 |
| Grade 1 Hypertension | 140–159 | and/or 90–99 |
| Grade 2 Hypertension | 160–179 | and/or 100–109 |
| Grade 3 hypertension | ≥180 | and/or ≥110 |
| ACC/AHA (2017) | | |
| Normotension | <120 | and <80 |
| Elevated BP | 120–129 | and <80 |
| Stage 1 Hypertension | 130–139 | or 80–89 |
| Stage 2 Hypertension | ≥140 | or ≥90 |
| ACC, American College of Cardiology; AHA, American Heart Association; BP, blood pressure; ESC, European Society of Cardiology; ESH, European Society of Hypertension; NICE, National Institute for Health and Care Excellence. | | |

cut-off values of 140/90 mmHg for clinic BP and 135/85 mmHg for out of clinic BP (83 mm Hg in one study) measured by either day-time ABPM or home BP.²⁴ It should be noted that these cut-off values and comparators are the same as those in the NICE guidelines, but differ from those in ESH and ACC/AHA guidelines. The Spanish Ambulatory BP Monitoring Registry has been used to assess the prevalence of WCH according to the ESH definition. Amongst patients with elevated clinic BP, 35% of untreated patients can be classified as having WCH.²⁵ Prevalence estimated in other studies is highly variable, ranging from 10% to 50% depending on the definition of WCH and the cohort studied.²⁶ When we consider these values, it is important to appreciate the low reproducibility of clinic BP measurements. When clinic and ambulatory BP recordings are taken at different timepoints, only 55% of individuals meet the criteria for WCH on both occasions.²⁷ The prevalence of WCH is not distributed

equally across the population. Compared with normotensive patients, there is a preponderance of older age, male sex, obesity and elevated blood lipids in patients with WCH. This is the case regardless of the WCH definition used.²⁵

Etiology

There are several proposed triggers for the onset of WCH and these occur throughout the process of recording clinic BP. Patients with WCH have a discrete elevation in BP on arrival at the doctor's office and during manual BP recordings with a cuff and manometer. These elevations in BP exceed those recorded by ABPM during episodes of anxiety or aggravation.²⁸ Public speaking is a frequently cited trigger for episodes of elevated BP. This has been tested by dividing a cohort of hypertensive patients into two groups based on whether their BP response to a doctor's visit was above or below the median value. Individuals in

Table 2. Summary of current status of WCH.

| | |
|---------------------|---|
| Definition | <ul style="list-style-type: none"> • ESH: clinic BP of 140/90 mmHg or above and a mean 24-hour BP of less than 130/80 mmHg • ACC/AHA: clinic BP of 130/80 mmHg or above and day-time ambulatory or home BP of less than 130/80 mmHg • NICE: clinic BP of 140/90 mmHg or above and day-time ambulatory or home BP below 135/85 mmHg |
| Aetiology | <ul style="list-style-type: none"> • Psychological factors (stress, anxiety) |
| Pathophysiology | <ul style="list-style-type: none"> • Poorly understood • Sympathetic and endocrine systems have been implicated |
| Significance in CVD | <ul style="list-style-type: none"> • Increased risk of transitioning to sustained hypertension compared with normotensives • Worse target organ damage compared with normotensives • Some studies have found significantly higher rates of CVD compared with normotensives |
| Treatment of WCH | <ul style="list-style-type: none"> • Syst-Eur and HYVET trials suggest that treating WCH in over 60s and over 80s, respectively, might confer some protection to future CV events |

ACC, American College of Cardiology; AHA, American Heart Association; BP, blood pressure; CV, cardiovascular; CVD, cardiovascular disease; ESC, European Society of Cardiology; ESH, European Society of Hypertension; NICE, National Institute for Health and Care Excellence; WCH, white coat hypertension.

the former group showed greater BP reactivity to public speaking. Whilst this might demonstrate that BP in this patient group is susceptible to psychosocial stressors, the trigger investigated is of little relevance in the real world.²⁹ A greater emphasis on recognising triggers for spikes in BP could provide a means to attenuate WCH, and thus an individual's need for treatment.

Psychological characteristics, particularly anxiety, might be responsible for the presence of WCH. Anxiety is an emerging risk factor for numerous CV diseases, but its role in WCH is less clear.³⁰ Levels of anxiety and other traits can be quantified using a personality inventory. In patients treated with anti-hypertensives, high levels of anxiety have been shown to increase the risk of pseudo-resistant hypertension due to the white coat effect.³¹ These findings are in contrast to earlier work showing that psychological characteristics do not differ between those with WCH and sustained hypertension.³² Clearly, this remains an area of contention. Others have investigated the role of anxiety during a visit to the doctor's office, rather than as a personality trait. This has been done by using two different scales to measure patient anxiety and expectations concerning their BP outcomes. Both of these variables are positively associated with elevated clinic BP. This might suggest that a patient's expectation of high

BP provokes anxiety during the consultation, which causes a transient elevation in BP.³³ This hypothesis is supported by the finding that simply placing a cuff around a patient's arm and not inflating it or recording BP is sufficient to cause a peak in BP of the same magnitude as if a full manual recording had been taken.²⁸ Whilst these studies provide interesting insight into the psychological determinants of WCH, more work is required to fully understand this relationship.

Pathophysiology

The sympathetic and endocrine systems have both been implicated in the genesis of WCH. This has been investigated by simultaneously measuring arterial BP, heart rate and postganglionic muscle and skin sympathetic nerve activity during a doctor's visit. Subjects displayed an elevation in both BP and heart rate during the visit. This was accompanied by an increase in skin sympathetic nerve traffic and a corresponding reduction in muscle sympathetic nerve traffic. With the exception of skin sympathetic nerve traffic, these changes endured for several minutes after the end of the visit.³⁴ This early work laid the foundations for our current understanding of the pathophysiology of WCH, but it must be stressed that it is based on just 10 patients with essential hypertension. The same experiments have since been

repeated, confirming that a doctor's visit changes these four parameters as previously noted. Interestingly, these repeat experiments show that a nurse's visit provoked a significantly attenuated response compared with a doctor's visit.³⁵ The endocrine system may have an important role in the recovery of BP in the hours rather than minutes following a clinic visit. Hospital-initiated ABPM shows that BP takes 2–3 h to reach the usual day-time values. This could be explained by a prolonged sympathetic response or by some endocrine contribution.²⁸ This hypothesis has yet to be investigated.

WCH and its significance in cardiovascular disease

WCH is well characterized in the medical literature, but there is not yet a consensus on its prognostic significance in cardiovascular disease (CVD). However, growing evidence has started to establish a link between WCH and risk factors associated with CVD, namely the development of SH and the presence of TOD. Both are important and independent predictors of CV risk.^{36,37}

Sustained hypertension

In the Pressioni Arteriose Monitorate e Loro Associazioni (PAMELA) population, clinic BP and 24-h ABPM was used to stratify subjects into different blood pressure phenotypes.³⁸ At baseline, 758 were defined as true normotensives (normal clinic BP and ABPM), 225 had WCH (elevated clinic BP but normal ABPM) and 124 were found to have masked hypertension (normal clinic BP but elevated ABPM). When these individuals were re-evaluated 10 years later, 136 previously normotensive (18.2%) and 95 previously white coat hypertensives (42.6%) had progressed to SH. The risk of transitioning to SH was 2.5-fold greater for white coat hypertensives (HR 2.51, CI 1.79–3.54, $p < 0.0001$) compared with normotensives. The same trend was observed when HBPM was used at baseline and follow up (HR 3.81, CI 2.57–5.64, $p < 0.0001$). The investigators found that the main predictor of progressing to SH was SBP at baseline. Interestingly, the rise in diastolic BP (DBP) was attenuated, leading to a more pronounced increase in pulse pressure. One may therefore postulate that the progression to SH may be due, in part, to stiffening of the larger arteries.³⁸

An observational study of inhabitants of Ohasama (Japan) yielded similar results to the PAMELA population. At baseline, a total of 777 participants were recruited, of which 649 had sustained normotension (NT). The remaining 128 participants had WCH diagnosed with the use of HBPM. After an 8-year follow up, 22.2% of subjects with sustained NT and 46.9% with WCH developed SH. The odds ratio (OR) for progressing to SH was significantly higher in white coat hypertensives than normotensives (OR 2.86, $p < 0.001$).³⁹ More recently, the results from the Finn-Home study further evaluated the risk of progressing from WCH to SH in the general population. Among the 528 normotensive participants at baseline, 96 (18.2%) developed SH after 11 years. Of the 142 white coat hypertensives at baseline, 74 (52.1%) progressed to SH. The relative risk (RR) for progression from WCH to SH was 2.8 (95% CI 2.2–3.6, $p < 0.0001$).⁵

On the basis of these observations, it could be argued that WCH should be classified as a 'pre-hypertensive' state, as individuals with WCH have increased risk of developing SH when compared with the truly normotensive population.

Target organ damage

Myocardial infarction (MI) and ischaemic stroke are the main clinical manifestations of hypertensive disease. However, in the sub-clinical phases, there is evidence of pre-existing organ damage. One of the most well described forms of TOD in relation to hypertension is left ventricular hypertrophy (LVH). Early findings from the Framingham Heart Study implicated LVH as a harbinger of CVD morbidity and mortality. Over a 4-year period, researchers found the incidence of CVD to be almost 2-fold greater in hypertensive adults with LVH than without LVH. When adjusted for multivariate analysis, the presence of LVH increased the RR of CVD by 1.49 (95% CI 1.20–1.85) in men and 1.57 (95% CI 1.20–2.04) in women.³⁷ With this in mind, a recent meta-analysis aimed to assess the impact of WCH on cardiac structure and function. A total of 7382 individuals were included in the analysis from 25 studies: 2493 were normotensive, 1705 had WCH and 3184 had SH. Clinic BP and ABPM was used to define each phenotype, with echocardiography being used to assess LV mass and function. Three major findings were reported: (i) left

ventricular mass index showed a graded, significant increase from normotensive, white coat hypertensive to sustained hypertensive subjects. (ii) Pooled data from eight studies showed left ventricular diastolic function, as assessed by E/A ratio, to be highest in normotensives (1.17 ± 0.07 , $n=337$), lower in WCH (1.07 ± 0.07 , $n=471$) and lower still in SH (0.99 ± 0.11 , $n=852$). (iii) Five studies documented left atrial diameter in normotensives (3.26 ± 0.03 cm, $n=161$), WCH (3.31 ± 0.11 cm, $n=193$) and SH (3.44 ± 0.13 cm, $n=261$). Left atrial diameter was significantly higher in WCH than in normotensive patients and in SH.⁴⁰

Another index for assessing TOD is measuring the carotid intima-media thickness (CIMT). For many years, CIMT has been used in clinical practice as a surrogate marker for CVD. This is based largely on observational data that show a positive association between CIMT and the incidence of CVD. For example, in the Cardiovascular Health Study of 4476 individuals over the age of 65 and without pre-existing CVD, ultrasound sonography was used to classify individuals into quintiles based on their CIMT. It was found that the incidence of stroke or MI increased across quintiles.⁴¹ Other studies show that CIMT is an independent predictor of future CV events.^{42,43} In the context of sustained hypertension, it is well known that CIMT is greater amongst hypertensives when compared with normotensive controls; however, much less is known about the relationship between WCH and CIMT.⁴⁴⁻⁴⁶ To date, only one meta-analysis has sought to address this issue. Of the 3478 untreated individuals included in this meta-analysis, 940 were normotensive, 666 had WCH and 1872 had SH. CIMT showed a progressive increase from normotensive (718 ± 36 μ m) to WCH (763 ± 47 μ m, $p < 0.01$) and to hypertensive patients (817 ± 47 μ m, $p < 0.01$).⁴⁷ More recently, it has been found that patients with WCH had significantly higher mean CIMT than those with normotension ($p < 0.05$) and significantly lower than individuals with SH ($p < 0.05$). These results remained significant after adjusting for age and traditional CV risk factors such as smoking, diabetes mellitus and hypercholesterolaemia.⁴⁸ The overriding theme from the studies linking TOD and WCH seems to suggest that WCH is an intermediate phenotype between NT and SH on the continuum of CV risk.

CV outcomes

Meta-analyses investigating untreated WCH and CV outcomes have yielded inconclusive results. In a recent study from Cohen *et al.*, untreated WCH was associated with an increased risk of CV events (HR 1.36, 95% CI 1.03–2.00) and CV mortality (HR 2.09, 95% CI 1.23–4.48) compared with NT.⁴⁹ No differences were observed when individuals with white coat uncontrolled hypertension (WCUH), also referred to as treated WCH or WCE, were compared with normotensive subjects. This suggests that untreated WCH may confer higher CV risk than WCUH.⁴⁹ In another meta-analysis by Huang *et al.* ($n=20,445$, mean follow up 9.6 years), CVD was found to be significantly higher amongst untreated white coat hypertensives *versus* normotensives (RR 1.38, 95% CI 1.15–1.65).¹² This supports earlier findings from an analysis of 29,100 participants (NT=1538, WCH=4806 and SH=10,756) from across 14 studies, which concluded that white coat hypertensives had higher rates of CV morbidity and mortality *versus* those with NT (OR 1.73, 95% CI 1.27–2.36, $p=0.006$).⁹

This view, however, is not unequivocal and is challenged by Pierdoemnico *et al.*, who pooled data from eight studies comprising 3050 normotensives, 1279 white coat hypertensives and 3953 sustained hypertensives. They found no difference in CV risk between WCH and NT, with an adjusted HR of 0.96 (95% CI 0.65–1.42) for WCH *versus* NT ($p=0.85$) and 2.59 (95% CI 2.0–3.35) for SH *versus* NT ($p=0.0001$).¹¹ Another viewpoint which may clarify the disagreement in the literature has been presented by Franklin *et al.*⁵⁰ In their study of the IDACO database, 653 individuals with WCH were matched to a normotensive participant on the basis of age and CV risk profile. Low CV risk was defined as 0–2 risk factors, and high risk was defined as ≥ 3 to 5 risk factors, diabetes and/or history of prior CVD events. After a median follow up of 10.6 years, the incidence of CV events was comparable between low risk cohorts with NT and WCH (HR: 1.06; 95% CI: 0.66–1.72; $p=0.80$). In contrast, the incidence of CV events was significantly higher when participants with high risk NT and WCH were compared (HR: 2.06; 95% CI: 1.10–3.84; $p=0.023$). This would suggest that age is not implicated in the association between WCH and CV outcomes. Interestingly, when the data was analysed further, the

excess in the number of CV events amongst WCH were confined to individuals above 60 years of age at baseline with concurrent high CV risk. This small group represented only 3.4% of the WCH population, leading the authors to hypothesise that these individuals may have had isolated systolic hypertension with WCE rather than WCH.⁵⁰ Discrepancies in how WCH was diagnosed, as well as the values used, could offer one explanation to why there is a disagreement between analyses.

Treatment of WCH?

There is scarcity of large, prospective, randomised controlled trials (RCT) evaluating the effect of anti-hypertensive therapy on the incidence of cerebrovascular and CV events in patients with WCH. Currently, there are only two RCTs, and each is a subgroup analysis of a larger trial. The first is a subgroup analysis of 695 participants from the Syst-Eur trial aged >60, with a clinic systolic central blood pressure (CBP) 160–219 mmHg and a diastolic CBP <90 mmHg.¹³ Participants were divided into three groups based on their Systolic ABPM into: non-sustained hypertension (<140 mmHg), mild sustained hypertension (140–159 mmHg) or moderate sustained hypertension (>160 mmHg). They were then randomised to receive anti-hypertensive therapy or a matching placebo. Active treatment significantly reduced clinic and ambulatory BP in patients with sustained hypertension, but reduced clinic BP only in those with non-sustained hypertension. The most important finding from this study was that the incidence of stroke and CV events was lower in both sustained and non-sustained treated hypertensive patients.

The second study was a subgroup analysis of the Hypertension in the Very Elderly Trial (HYVET), a double-blind randomised trial evaluating the efficacy of sustained release indapamide (1.5 mg) ± perindopril (2–4 mg) in elderly hypertensives who were over 80 years old and had a systolic BP between 160–199 mmHg. After a mean follow up period of 13 months, the difference in ABPM between the treatment and placebo arms was –8/–5 mmHg. This translated into a reduction of 21% in total mortality and 34% in all CV events within the treatment arm. In a *post hoc* analysis, 50% (56, 95% CI 40–60%) of participants satisfied the criteria for WCH based on daytime ABPM.⁵¹

The findings of the Syst-Eur and HYVET trials suggest that treating WCH in the over 60s and over 80s might confer some protection to future CV events, but they also need to be considered in the context of their limitations. Both are retrospective analyses of trials assessing anti-hypertensive therapy in sustained hypertension. It would be preferable to draw upon data from trials assessing anti-hypertensive therapy in WCH. In addition, the participants recruited were elderly, and are not representative of the whole population. Most importantly, the numbers of fatal and non-fatal events are low, which limits statistical power and brings into question the reliability of their results.

Research gaps

WCH is an under researched phenotype of sustained hypertension and there is still much to be discovered. Firstly, whether WCH is a benign phenomenon is unclear. This is due to a lack of observational data comparing CV morbidity and mortality between WCH and NT. The number of patients studied in the existing evidence base is currently too low, which would explain the conflicting results. Secondly, if we were to establish there was a need to treat WCH, we must also consider the economic and social implications. Amongst patients with sustained hypertension, approximately 80 patients would need to be treated with a first-line anti-hypertensive agent in order to prevent one death.⁵² Would it have the same benefits to treat that number for WCH? Do we need to treat those with white coat effect? What would be the best prescribed dose? The long-term sequelae of hypertension costs the UK health economy £2 billion per year.⁵³ Would treating those with WCH prove to be cost effective? These are all important questions that need to be answered before 13% of the general population is potentially medicalised.²⁴ Thirdly, the existing definition of WCH is too rigid. It does not take into consideration the fact that WCH is not a static phenomenon and can fluctuate over time. For example, only 55% of individuals who are diagnosed with WCH fulfil the criteria over two visits.²⁷ This is a problem as it brings into question who should be included for observational studies. Furthermore, a recent study suggests that reproducibility over time is poor and this might present a challenge in the design of long-term prognostic studies.⁵⁴ Finally, WCH

must be considered in the wider context of hypertension itself. The recent SPRINT results demonstrated that high-risk individuals who received intensive BP lowering of SBP < 120 mmHg, had significantly lower rates of primary composite outcomes (MI, acute coronary syndromes, heart failure), than those who received standard lowering of SBP < 140 mmHg (1.65% versus 2.19% per year).¹⁶ These results have challenged our traditional view of treating hypertension and has led the ACC/AHA to call for more aggressive targets in BP control.⁴ If treatment thresholds and targets for hypertension change, this might have a ripple effect on the approach to WCH.

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Conflict of interest statement

The authors declare that there is no conflict of interest.

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