A growing body of evidence indicates that office blood pressure (OBP) measurement alone, as performed by the physician, is not sufficient to determine a patient’s true blood pressure (BP) value. Although home blood pressure (HBP) measurement or ambulatory blood pressure monitoring (ABPM) may provide us with a better estimate of a patient’s BP, this may also cause problems if these two types of measurements lead to different treatment conclusions.

Most physicians are familiar with the discrepancy called white coat hypertension (WCH), which is defined as an elevated OBP value in the face of a normal BP outside the office, as determined by either ABPM or HBP. Since several studies have already shown that both ABPM and HBP correlate better with target organ damage than OBP, one may be inclined to think that WCH is rather harmless. However, some studies that evaluated the long-term effect of WCH on cardiovascular prognosis showed that subjects with WCH had an increased risk compared with normotensives, but a lower risk than those with sustained hypertension.1 These data indicate that OBP values should not be completely ignored.

Much less is known about the second discrepancy and, in fact, the opposite of WCH, namely masked hypertension (MH). Although many physicians are unfamiliar with MH, it is not a rare phenomenon as its prevalence ranges from 8 to 33% among different populations.2,3 Some studies even found higher numbers of masked hypertensives when compared with white coat hypertensive subjects. However, it is difficult to determine the real prevalence of MH from the present literature as studies about MH vary widely in definitions, equipment used, populations and measurement procedures. This also holds true for the paper by Aksoy et al.4 in the present issue of the Journal. In this paper the authors dealt with the prevalence of MH among 57 seemingly well-controlled, treated hypertensive patients and 31 untreated normotensive subjects. They found that MH occurred frequently in the hypertensives but not in the normotensives, although in some normotensive subjects there was a clear difference between OBP and HBP. However, the OBP procedure, as performed by Aksoy and co-workers, is rather unusual. The time before the first duplicated measurement was taken and the time between the other duplicated measurements amounted to ten minutes. When such a long time elapses before measurements are taken, the white coat effect will become smaller.5 Consequently, when patients are sitting longer in a chair before the OBP is taken, they will obtain a lower OBP value as compared with their HBP.

Another reason why we still do not know much about MH is related to the large population differences (normotensives, hypertensives, younger subjects, elderly, treated and untreated subjects) among studies that have investigated MH. The study by Aksoy and co-workers shows that there were more patients declared to have masked hypertension in the hypertensive group than in the normotensive group. This may be attributed to at least three factors. Firstly, these patients have BP values that are closer to the upper limits of the definition of MH than normotensive patients. This means that a minor discrepancy between OBP and HBP would more easily lead to a classification of MH in hypertensive patients than in normotensive individuals. Secondly, the hypertensive patients were on antihypertensive drugs that they may have taken in the morning. HBP was performed in the early morning, at the ‘trough’ moment of the antihypertensive activity of the medication. OBP was regularly performed in a range of two to eight hours after drug intake so that it might already have had its influence on the BP. Thirdly, the percentage of men in the studies differs between both groups (47% in the group with treated hypertensives and 71% in the group with healthy volunteers). Indeed, some studies have shown that the prevalence of MH was higher among females than among males; however, other

**Th. Thien was not involved in the handling and review process of this paper.
The explanation for MH has been sought in factors that increase daytime ambulatory blood pressure such as physical activity, stressful conditions, tobacco and coffee, alcohol, sedentary habits or greater reactivity to daily life stressors. However, MH could, of course, also be due to an exceptionally low OBP in the face of increased HBP or ABPM values. Low OBP values can be due to a postprandial dip or previous antihypertensive drug intake. Therefore, when patients take antihypertensive treatment it is absolutely essential to instruct patients carefully on how to take their drugs and verify, at each visit when OBP is performed, if and how their treatment was taken. Despite all speculations, the true identity of MH has not yet been revealed as the reproducibility of MH has not been investigated until now.

Since ABPM and HBP are better risk predictors than OBP, patients with MH, both treated and untreated, exhibit a cardiovascular risk similar to that of sustained hypertensive patients. When subjects have MH and their treatment is based on OBP results, these patients will not receive the treatment they should. For this reason it would be desirable if all patients performed HBP in addition to OBP. However, for practical reasons it is impossible to let all patients perform HBP. Therefore, HBP should be recommended, in particular, for patients with high cardiovascular risk or with symptoms possibly related to their BP level, or when there is a discrepancy between BP values and degree of target organ damage. Eventually, an additional ABPM could be performed to determine the most appropriate treatment if a discrepancy is found between OBP and HBP values.

REFERENCES


