

Work conditions and masked (hidden) hypertension—insights into the global epidemic of hypertension

by Paul A Landsbergis, PhD,¹ Peter L Schnall, MD,² Karen L Belkić, MD,^{3,4} Joseph E Schwartz, PhD,⁵ Dean Baker, MD,² Thomas G Pickering, MD⁶

Landsbergis PA, Schnall PL, Belkić KL, Schwartz JE, Baker D, Pickering TG. Work conditions and masked (hidden) hypertension—insights into the global epidemic of hypertension. *SJWEH Suppl*. 2008;(6):41–51.

Hypertension is the leading cause of cardiovascular disease. There is considerable evidence that work conditions play an important role in the etiology of essential hypertension. Ambulatory blood pressure during waking hours (work and home) is more strongly associated with subsequent cardiovascular disease than the traditional measurement of casual clinical blood pressure. A person with normal clinical blood pressure but elevated awake ambulatory blood pressure is said to have “masked” (or “occult” or “hidden”) hypertension. Masked hypertension is associated with increased cardiovascular risk, and has been observed in 10–30% of adults with normal clinical blood pressure. It has been hypothesized that exposure to workplace stressors first elevates work, but not clinical, blood pressure; after chronic exposure to stressors, both daytime and clinical pressure become elevated. In this manuscript, an algorithm is provided that targets ambulatory monitoring for high-risk groups and helps detect work-related hypertension. A public health approach incorporating clinical guidelines, workplace surveillance, and improved work conditions is recommended for tackling the epidemic of hypertension.

Key terms ambulatory blood pressure; epidemiology; hidden hypertension; job strain; masked hypertension; occult hypertension; review; white-coat hypertension; work stress.

Hypertension is the leading cause of cardiovascular disease (CVD) worldwide (1), and CVD will become the most common cause of death worldwide by 2020 (2). Beginning at 115/75 (systolic/diastolic) mm Hg, the risk of CVD doubles for each increment of 20/10 mm Hg (3).

The worldwide prevalence of hypertension in 2000 was estimated to be 972 million persons or 26.4% of the world’s adult population (4). In developing countries, there has been a rapid increase in its prevalence,

while, in developed countries, the recent trend towards a decrease in the prevalence of hypertension is reversing (5). Nearly two-thirds of the United States (US) population 60 years of age has persistently elevated blood pressure (>140/90 mm Hg) (6), and those with normal blood pressure at the age of 55 years who survive to age 80–85 years have a 90% risk of developing hypertension (7).

CVD and hypertension appear to be epidemics of relatively recent historical origin (8, 9). Hypertension

1 Department of Community and Preventive Medicine, Mount Sinai School of Medicine, New York, New York, United States; and Department of Environmental and Occupational Health Sciences, State University of New York—Downstate Medical Center, Brooklyn, New York, United States.

2 Division of Occupational & Environmental Medicine, Department of Medicine, University of California at Irvine, Irvine, California, United States.

3 Department of Oncology-Pathology, Karolinska Institute, Stockholm, Sweden.

4 Institute for Health Promotion and Disease Prevention Research, University of Southern California School of Medicine, Los Angeles, California, United States.

5 Department of Psychiatry and Behavioral Sciences, State University of New York at Stony Brook, Stony Brook, New York, United States.

6 Behavioral Cardiovascular Health and Hypertension Program, Department of Medicine, Columbia Presbyterian Medical Center, New York, New York, United States.

Reprint requests to: Dr P Landsbergis, Department of Environmental and Occupational Health Sciences, State University of New York—Downstate Medical Center, Box 43, 450 Clarkson Avenue, Brooklyn, NY 11203, USA. [E-mail: paul.landsbergis@downstate.edu]

is primarily a disease of industrial societies, with a very low prevalence in nonmarket agricultural communities (9). The rising prevalence of hypertension in developed countries parallels the transformation of worklife during the past century, away from agricultural work and relatively autonomous craft-based work towards machine-based (including computer-based) labor, characteristic of assembly-lines and mass production (10). The development of hypertension as a global epidemic has occurred in conjunction with urbanization and industrialization and, more recently, economic globalization (2). Lower occupational status, a component of socioeconomic status, is a risk factor for hypertension (11). Key characteristics of the assembly-line approach to job design, whether implemented in blue-collar or white-collar settings, are high workload demands combined with low employee control or autonomy (known as “job strain”) (12), and, during periods of economic growth, long workhours. Job strain (13–15) and long workhours (16–19) have been identified as important risk factors for hypertension. The emerging body of scientific evidence on workplace risk factors for hypertension also implicates work that combines high effort and low rewards (effort–reward imbalance) (20, 21) and work characterized by “threat–avoidant vigilance”. Threat–avoidant vigilance involves maintaining a continuously high level of vigilance in order to avoid disasters, such as loss of human life (14), and it is a feature of several occupations at high risk for hypertension and CVD, including professional drivers (22, 23), air traffic controllers (24), and sea pilots (25). This evidence points to the transformation of work conditions characteristic of industrialization and urbanization as playing a major role in the promotion of the global epidemic of hypertension (10).

The location and techniques of blood pressure measurement provide important information regarding the work-relatedness of hypertension. Blood pressure is higher at work than at home (on a workday) and lower yet on a nonworkday (26). Blood pressure measured during daily life, including work, has shown stronger associations with future CVD than blood pressure measured in a physician’s office, and it has shown strong associations with work stressors. This paper reviews this body of research and discusses implications for clinical work, surveillance, and work stressor reduction.

Location and methods of measuring blood pressure

Measuring casual clinical blood pressure

The traditional method for measuring blood pressure, the use of a sphygmomanometer with a stethoscope by a

physician or other health professional, is convenient and inexpensive, involves low technology, and offers some predictive validity (27). Notwithstanding its wide use, casual clinical blood pressure is unreliable (27). Blood pressure values recorded by different trained personnel vary by as much as 5–10 mm Hg (28) and have relatively poor test-retest reliability (29). Problems with observer measurement due to digit preference (0 or 5) and varying levels of auditory acuity are well recognized. Casual clinical blood pressure measurements, “which are of enormous value on a population basis, often provide a poor estimate of risk in an individual patient for reasons such as observer error, the ‘white-coat’ effect (the transient but variable elevation of blood pressure in a medical setting), and the inherent variability of blood pressure [p 2368]” (30).

Ambulatory blood pressure measurement

Advances in technology over the past 40 years permit blood pressure to be measured repeatedly while people go about their normal daily activities over the course of 24 hours using ambulatory (portable) monitors. An individual reading with an ambulatory monitor is somewhat less accurate than one taken by a human observer using the traditional auscultatory technique (31). However, monitoring ambulatory blood pressure provides a large number of readings that can be averaged to provide a more-reliable measure of blood pressure than casual clinical blood pressure, as well as an estimate that reflects a person’s blood pressure during normal activities (as opposed to sitting in a doctor’s office). Observer bias is also absent because the measurements are taken automatically. Thus ambulatory blood pressure monitoring provides a more-valid measure of a person’s “true” blood pressure, which is defined as the mean level over prolonged periods (30). Monitoring ambulatory blood pressure captures the dynamic blood pressure fluctuations in relation to daily life [ie, to changes in posture and physical activity, to location (eg, work versus home), waking versus sleep, and to mood and psychological state]. Blood pressure varies substantially over the course of 24 hours, and its sensitivity to physical and psychosocial stimuli is clearly evident. For example, the mean ambulatory systolic blood pressure is higher by approximately 4 mm Hg during work than during nonwork (32–35) and the mean 24-hour ambulatory blood pressure is lower on nonwork days than on workdays (26, 36).

A disadvantage of ambulatory blood pressure is that the circumstances of recording are uncontrolled, and any given day may be atypical, for various reasons. Nonetheless, ambulatory blood pressure is a better predictor of target organ damage, such as increases in the size of the

heart's left ventricle, than casual clinical blood pressure is (37–40); it is also a better indicator of future incident CVD (41–46).

Measurement of blood pressure—evidence for the etiology of essential hypertension

Casual clinical blood pressure has been used for research into the etiology of essential hypertension for over 100 years. Age, gender, heredity, obesity, and alcohol intake have been identified as risk factors, and a number of physiological mechanisms, such as salt sensitivity, low as well as high renin, and low calcium play a role. These factors, however, taken together, explain only a part of the risk. Despite enormous research efforts, the definition of essential hypertension, which accounts for about 90–95% of all cases of arterial hypertension (47), remains: “patients with arterial hypertension and no definable cause [p 1381]” (47). One reason why the etiology of essential hypertension remains unclear is that casual clinical blood pressure does not reflect the dynamics of daily life, and it is during the latter that a wealth of information can be gleaned about how essential hypertension develops. Monitoring ambulatory blood pressure offers the opportunity to explore a wide range of hypotheses concerning the etiology of essential hypertension, including work and nonwork stressors. Among work-related stressors, the most intensively investigated has been job strain.

Job Strain. Studies of job strain and casual clinical blood pressure have not consistently shown significant associations (14, 48). However, in 2000, a review of 11 cross-sectional studies of job strain (or its components) and ambulatory blood pressure among men found that the majority reported significant positive associations with work ambulatory blood pressure (14). In the five studies in which blood pressure was also measured outside work, job strain was also associated with increased nonwork systolic ambulatory blood pressure. Of the six cross-sectional studies of job strain and ambulatory blood pressure among women, four reported significant positive associations with work systolic ambulatory blood pressure (15). Work systolic ambulatory blood pressure among workers facing job strain is typically 4–8 mm Hg higher than among those without job strain. Differences between casual clinical blood pressure and ambulatory blood pressure findings may be explained, in part, by the imprecision and biases of taking casual clinical blood pressure measurements and that ambulatory blood pressure, but not casual clinical blood pressure, includes large numbers of readings taken at work. Several ambulatory blood pressure studies with null or mixed findings were single-occupation studies with limited variance in job characteristics

that used conventional generic instruments to assess the components of job strain, namely, high demands and low decision latitude (49–52). The only long-term prospective study of this association between job strain and ambulatory blood pressure found that men exposed to chronic job strain (measured at two points in time separated by 3 years) exhibited an 11–12 mm Hg higher systolic and 6–9 mm Hg higher diastolic work ambulatory blood pressure than men unexposed at both times. In addition, men reporting job strain at baseline but no job strain in the 3-year follow-up exhibited a significant decrease in systolic ambulatory blood pressure of 5.3 mm Hg at work and 4.7 mm Hg at home (13). The cross-sectional association between job strain and elevated ambulatory blood pressure is strong and fairly consistent, with important corroborative longitudinal data, indications of a dose–response relationship (53) and biological plausibility (54). Thus there is substantial evidence supporting the hypothesis that exposure to job strain is causally related to the development of essential hypertension.

Other work stressors. Associations with blood pressure elevation have also been observed for long workhours (16–19), effort–reward imbalance (20, 21), and “threat–avoidant vigilance” (14, 22, 23). Mixed evidence exists for shift work (55). Some empirical evidence implicates chronic exposure to noise (56). For other physical factors, such as cold, heavy lifting, and glare, physiological data show acute blood pressure increases with exposure, but there is no epidemiologic evidence available. Chemicals that may be pressor agents include lead and arsenic (56). In addition, lower occupational status is a risk factor for hypertension (11). More research is needed on the relative contribution of these factors alone and in combination to the etiology of essential hypertension. For example, work stressors have been found to be associated in some studies with overweight and alcohol use (57, 58), and thus health behavior related to these may, in part, mediate the association between work stressors and hypertension.

There is also evidence that work stressors both mediate the association between lower socioeconomic position and CVD risk (59) and interact with socioeconomic position. For example, the cross-sectional association between job strain and blood pressure was substantially greater for men with lower status jobs in the New York City worksite blood pressure study (60), and for both men and women in the Framingham heart study [unpublished data: Landsbergis P, Schnall P, Chace R, Sullivan L, D’Agostino R. Psychosocial job stressors and cardiovascular disease in the Framingham Offspring Study: a prospective analysis [poster]. In: 4th ICOH Conference on Work Environment and Cardiovascular Disease; 2005. Newport Beach (CA): 2005].

Masked (hidden) hypertension

The monitoring of ambulatory blood pressure is beginning to be recognized as having an important role in the diagnosis of hypertension. On one hand, it can be used to rule out “white coat” hypertension (the “false positive” diagnosis). On the other hand, monitoring ambulatory blood pressure allows for the identification of masked (also known as “occult”) hypertension [ie, elevated daytime or 24-hour blood pressure, but normal casual clinical blood pressure (“false negative” diagnosis)]. [Other names used to describe this phenomenon reflect uncertainty or confusion over its etiology: “white-coat normotension”, “reversed white-coat effect”, “isolated ambulatory hypertension”.] Unfortunately, people with normal casual clinical blood pressure are rarely followed up to have their blood pressure re-checked.

Masked hypertension is a much more serious condition than “white-coat” hypertension. People with masked hypertension have levels of target organ damage, such as increased left ventricular mass (40, 61, 62) and carotid plaque (61–63), similar to persons diagnosed with hypertension (“true positives”, people with elevated ambulatory blood pressure and casual clinical blood pressure) and significantly higher than people with normal blood pressure. People with masked hypertension also have a significantly increased risk of developing CVD relative to people with normal blood pressure (64–66) or [with one exception (66)] people exhibiting white-coat hypertension.

Apparently controlled hypertension

Increased target organ damage (67) and an increased risk of CVD (41, 42) have also been observed among persons with diagnosed hypertension whose blood pressure is being treated and appears to be controlled in clinical settings (casual clinical blood pressure <140/90 mm Hg), but whose daytime ambulatory blood pressure is elevated when compared with that of treated persons with normal casual clinical blood pressure and normal ambulatory blood pressure (67). Persons treated for diagnosed hypertension whose blood pressure appears to be controlled have also been labeled “masked hypertensives”; however, such a label is somewhat misleading since they have already been identified as having hypertension. On the other hand, a high proportion (16–55%) of patients with hypertension who appear to be responding well to treatment (ie, casual clinical blood pressure <140/90) have elevated ambulatory blood pressure (inadequately controlled hypertension) (68). Therefore, monitoring ambulatory blood pressure on a workday is recommended for patients with hypertension, especially those with exposure to workplace stressors.

Prevalence

In general population studies, the prevalence of masked hypertension among people with normal casual clinical blood pressure typically ranges from 10% to 30% (table 1). The variety of definitions of masked hypertension makes prevalence comparisons problematic. The European Society of Hypertension Working Group and the American Heart Association have both recommended that a mean daytime systolic ambulatory blood pressure of 135 mm Hg or a diastolic ambulatory blood pressure of 85 mm Hg (and a casual clinical blood pressure of <140/90 mm Hg) be considered the appropriate ambulatory blood pressure threshold (69, 70).

Risk factors

To help determine the etiology of masked hypertension, some general population studies have identified its risk factors, including age, gender, smoking, alcohol use, coffee consumption, and physical activity (table 1). Masked hypertension was more common among men than women in six studies (40, 61, 62, 65, 66, 71, 72). Masked hypertension was more common among older participants in four studies (61, 62, 65, 66, 71), but was not associated with age in two studies (40, 72). In addition, two studies that used the definition of “ambulatory blood pressure greater than the casual clinical blood pressure” (73, 74) concluded that masked hypertension was associated with younger age. This inconsistency may have resulted, in part, from the inclusion of unemployed persons (eg, retirees, students) in some studies. If employment or work stress is a risk factor for masked hypertension, then the association between age and masked hypertension may be confounded by the length of employment or chronic exposure to work stress. Therefore, future studies should specify whether participants are employed, or they should provide analyses of employed subgroups to help clarify the association between age and masked hypertension.

Only two published studies of masked hypertension have examined work-related risk factors. Gallo et al (75) found that masked hypertension, based on systolic blood pressure, was more common among blue-collar and clerical workers than among higher status white-collar workers. However, such a pattern was not observed for diastolic blood pressure (75). In the longitudinal worksite blood pressure study (60), which measured job strain and ambulatory blood pressure at multiple points in time separated by an average of 3 years, job strain was slightly more common among men with masked hypertension at baseline, using a cutpoint of 85 mm Hg for diastolic blood pressure (table 1), although this association was not statistically significant. In an unpublished analysis of

Table 1. Prevalence of and risk factors for masked (hidden) hypertension determined in general population empirical studies published in English in 1988–2006. (95% CI = 95% confidence interval, ABP = ambulatory blood pressure, DBP = diastolic blood pressure, HTN = hypertension, NS = not significant, SBP = systolic blood pressure, Tx = treatment, WCH = white-coat hypertension, CVD = cardiovascular disease)

| Reference | Clinical blood pressure | | Ambulatory blood pressure | | | Study sample | | | Masked (hidden) hypertension | |
|----------------------------|---|-----|--------------------------------------|-----------------------------------|--------------------|--------------|-----------------------|--|--|--|
| | SBP | DBP | SBP | DBP | Duration | Female (%) | Location | Source, age | Prevalence ^a | Risk factors (versus people with normal clinical blood pressure) |
| Fiedler et al, 1988 (88) | .. | ≤90 | .. | >85 | Daytime, workday | 26 | New Jersey, USA | Worksite; 23–74 years; 36.2 years (mean) | 9.7% (11/113) | .. |
| Imai et al, 1996 (82) | <140 | <90 | Border-line: 134–143; definite: ≥144 | Border-line: 79–84; definite: ≥85 | 24 hours, weekday | 69 | Ohasama, Japan | Population; 20–79 years; 59 ±12 years; 30% HTN Tx | 10.4% borderline; 3.2% definite (untreated sample) | .. |
| Rasmussen et al, 1998 (73) | .. | .. | .. | .. | Daytime, workday | 48 | Glostrup, Denmark | Population; 41–72 years; Tx HTN excluded | .. | Systolic ambulatory blood pressure > clinical blood pressure: 41–42 years: 72% women, 86% men; 71–72 years: 38% women, 51% men |
| Selenta et al, 2000 (71) | <140 | .. | ≥135 | .. | 12 hours, workday | 52 | Vancouver, Canada | 111 community, 208 students; 17–68 years; Tx HTN excluded | 23% | Male, older age, smoking, alcohol use |
| | .. | <90 | .. | ≥85 | .. | .. | .. | 24% | .. | |
| | <140 | <90 | ≥135 | ≥85 | .. | .. | .. | 33.8% ^b (104/308) | .. | |
| Belkic et al, 2001 (80) | <140 | <90 | ≥140 | ≥90 | .. | .. | .. | .. | 16.2% ^b (50/308) | Job strain: prevalence ratio =1.55; unadjusted odds ratio=1.76 (95% CI 0.70–4.40) ^b |
| | .. | <85 | .. | ≥85 | Work | 0 | New York City, USA | Worksites: normotensives + mild HTN (no Tx for 3 weeks); 30–60 years | 19.9% (36/181) | |
| Liu et al, 1999 (61) | <140 | <90 | ≥134 | ≥90 | Daytime, workday | 54 | New York City, USA | 324, worksites; 35 patients; 30–66 years | Men: 31.6% (50/158); women: 8.0% (11/137) | Male, older age, body mass index; smoking (NS) |
| Sega et al, 2001 (40) | <140 | .. | ≥125 | .. | 24 hours, workday | 42 | Milan, Italy (PAMELA) | Population; 25–74 years; Tx HTN excluded | Men: 18.8% (116/617); women: 4.4% (27/616) | Male, older age |
| | .. | <90 | .. | ≥79 | .. | .. | .. | .. | Men: 13.8% (97/701); women: 8.0% (43/535) | |
| Tsai, 2003 (72) | Daytime systolic ABP minus clinical blood pressure ≥5 | | | .. | Daytime, weekday | 59 | Taipei, Taiwan | Volunteers, 38–45 years (mean); no HTN Tx | 48.3% (14/29) (excluding WCH) | Male, lower body mass index; age (NS) |
| Björklund et al, 2003 (64) | <140 | <90 | ≥135 | ≥85 | Daytime | 0 | Uppsala, Sweden | Population; 70 years; CVD; Tx HTN excluded | 30.4% (82/270) | .. |
| Gallo et al, 2004 (75) | <140 | .. | ≥140 | .. | 48 hours, workdays | 100 | Kent (OH) USA | Community; employed; 41 (SD 9) years, CVD; Tx HTN excl. | 27.2% (28/103) | Blue-collar: 39%, clerical: 28%, white-collar: 21% |
| | .. | <90 | .. | ≥90 | .. | .. | .. | .. | 7.8% (8/103) | |
| Ohkubo et al, 2005 (65) | <140 | <90 | ≥135 | ≥85 | Daytime, workdays | 60 | Ohasama, Japan | Population; ≥40 years, 61 years (mean); 30% HTN Tx | Men: 26.5% (80/302); women: 21.4% (141/658) | Older age, HTN Tx (P<0.05); male (P<0.10), smoking (NS) |
| Bombelli et al, 2005 (74) | Daytime ABP minus clinical blood pressure >0 | | | .. | Daytime, workday | 49 | Milan, Italy (PAMELA) | Population; 25–74 years | Men: 39.0% (SBP), 33.6% (DBP); women: 40.1% (SBP), 34.7% (DBP) | Younger age (65.6% if 25–34 years, 10.5% if 65–74 years), gender (NS) |
| Mancia et al, 2006 (66) | <140 | <90 | ≥125 | ≥79 | 24 hours, workday | 49 | Milan, Italy (PAMELA) | Population; 25–74 years | Men: 22.7% (126/556); women: 7.5% (46/617) | Male, older age, smoking, body mass index, cholesterol, glucose |

^a Numerator = number of participants with an ambulatory blood pressure above the threshold; denominator = number of participants with casual clinical blood pressure below the threshold.

^b Reanalysis of original data by Landsbergis.

male participants, using the criteria of <140/90 mm Hg for casual clinical blood pressure and 135/85 mm Hg for awake ambulatory blood pressure for masked

hypertension and compared with those with a casual clinical blood pressure of <140/90 mm Hg and an awake ambulatory blood pressure of 135/85 mm Hg,

odds ratios for job strain were 1.54 [95% confidence interval (95% CI) 0.61–3.91] for the baseline and 5.74 (95% CI 1.86–17.72) at the 3-year follow-up, adjusted for age, race, body mass index, education, current smoking, alcohol use, and worksite.

In unpublished findings from a New York City sample (primarily female), comparing health care workers with masked hypertension to those with normal casual clinical blood pressure and ambulatory blood pressure as already defined, odds ratios in the range of 1.5–5.0 were observed for job strain, effort–reward imbalance, evening, night or rotating shiftwork, and having only a secondary education.

Masked workplace hypertension

If masked hypertension occurs because the “at work” component of daytime ambulatory blood pressure is elevated, then it can be considered “masked workplace hypertension”. Since most published studies do not partition the work and home components of daytime ambulatory blood pressure, it is unknown whether the “at work” component is primarily responsible for the high prevalence of masked hypertension, or the increased risk of target organ damage and CVD due to masked hypertension. One study did find a higher correlation between left ventricular mass and work ambulatory blood pressure than home ambulatory blood pressure or clinical blood pressure (76). In addition, because ambulatory blood pressure is generally higher at work than at home (32–34) and work stressors appear to be causally related to the elevation of work ambulatory blood pressure, it is plausible to hypothesize that work stressors are important risk factors for masked hypertension and its sequelae. Therefore, data from the studies in table 1 need to be re-analyzed, and future studies need to examine the role of masked “workplace” hypertension as a cause of target-organ damage and CVD.

Suggested stages in the development of hypertension

On the basis of the presumed mechanisms of neurogenic hypertension (77), we previously hypothesized (56, 78) that there are three stages in the etiology of essential hypertension.

Stage 1. When a person is first exposed to workplace stressors, ambulatory blood pressure (primarily systolic) is elevated at work and casual clinical blood pressure remains normal (a masked hypertension effect). The elevation of blood pressure during waking hours may lead to “early markers” of CVD (increases in left ventricular

mass and atherosclerosis), indicating that the person has more severe hypertension. Evidence of these structural changes would classify the person at stage 2, despite normal casual clinical blood pressure.

Stage 2. With chronic exposure to stressors, casual clinical blood pressure also becomes elevated. Psychosocial factors are likely correlated with both workplace blood pressure and casual clinical blood pressure, and structural changes in the heart and vasculature continue.

Stage 3. Self-sustaining structural processes in the vascular system may lead to a disjuncture between exposures to workplace stressors and blood pressure, since (i) the hypertension process has become autonomous or (ii) exposure to workplace factors that elevate blood pressure may have ceased due to retirement or selection into a less stressful job.

Targeting the monitoring of ambulatory blood pressure towards high-risk groups

The monitoring of ambulatory blood pressure is relatively expensive and skilled-labor intensive. In the United States alone, if monitoring ambulatory blood pressure were to become a routine procedure for the diagnosis and monitoring of hypertension, annual costs could reach USD 6 billion (37). Therefore, guidance is needed for clinicians and worksite cardiovascular screening programs to identify the persons and groups with normal casual clinical blood pressure who would be the most likely to benefit from the monitoring of ambulatory blood pressure. Persons with CVD, a high CVD risk profile, diabetes, or target organ damage would be one indicator. Casual clinical blood pressure above 130/80 mm Hg or risk factors for ambulatory blood pressure elevation, such as smoking, alcohol use, heavy coffee consumption, and lack of physical activity, could be considered. Given the strong associations between work stressors and ambulatory blood pressure, we have presented an algorithm (79, 80) to aid in this goal. Further research is needed to determine the magnitude of associations between job strain, other job stressors, nonwork risk factors, and masked hypertension, which could help refine our recommendations, given in table 2.

Lower cost alternatives to the monitoring of ambulatory blood pressure

Given the high costs of monitoring ambulatory blood pressure, further research is needed on less expensive

Table 2. Preliminary algorithm to identify high-risk persons and groups most likely to benefit from ambulatory blood pressure (ABP) monitoring and clinical and workplace interventions. (CVD = cardiovascular disease, LVMI = left-ventricular mass index)

| Clinical blood pressure | Exposure to workplace stressors ^a | | | | | | | | | |
|-------------------------|--|--|--|---|----------------------|---|---|----------------------|----|-----|
| | No | Yes: ABP monitoring (awake ABP \geq 135/85 mm Hg) ^b | | | | | | | | |
| | | No | Yes (masked hypertension) | | | | Yes | | | |
| | | | Recommendation | Increased LVMI, carotid atherosclerosis | | Recommendation | Increased LVMI, carotid atherosclerosis | | No | Yes |
| | | | | No | Yes | | No | Yes | | |
| <140/90; >115/75 | Follow routinely | Follow routinely | Reduce exposure to specific stressors; electrocardiography/carotid echo (evaluate early CVD markers) and follow-up | Stage 1 hypertension | Stage 2 hypertension | . | . | . | | |
| \geq 140/90 | Repeat clinical blood pressure (to rule out "white coat" hypertension) | White-coat hypertension | . | . | . | Reduce exposure to specific stressors; electrocardiography/carotid echo (evaluate early CVD markers), clinical evaluation and follow-up | Stage 2 hypertension | Stage 3 hypertension | | |

^a Direct epidemiologic and physiological evidence for job strain, effort–reward imbalance, long workhours, noise, lead, arsenic; mixed evidence for shift work. High-risk occupations: urban transit operators, truck drivers, air traffic controllers, sea pilots. Physiological and indirect epidemiologic evidence for threat–avoidant vigilant work. Only physiological evidence for cold, heavy lifting, glare exposure (56).

^b ABP to assess severity and pattern of elevated blood pressure, to (i) aid in clinical decision making (eg, optimize medication regimen) and (ii) suggest feasible strategies to minimize exposure to work pressors (eg, adjustment of work schedule).

methods, such as “home” or self-monitoring, which would help identify people with elevated blood pressure outside the doctor’s office. Home monitoring of blood pressure, using upper-arm digital monitors, has been recommended for persons diagnosed with hypertension, although only some of the many home blood pressure monitors on the market have satisfied the recommended validation criteria (70). One study found a similar percentage of participants with masked hypertension detected by ambulatory blood pressure (14%) and home blood pressure (11%) (81). Home blood pressure predicts CVD risk better than casual clinical blood pressure for treated patients with hypertension (40, 42, 82).

Wrist blood pressure monitors, which also store readings digitally but are somewhat easier to use than upper-arm cuffs, have also become popular. However, blood pressure can vary substantially in different parts of the arterial tree, and wrist monitors can also be inaccurate if not held at heart level (70). A newer device with a position sensor that keeps the monitor near the level of the heart while recording has also met the validation criteria (83). Another potential limitation is the immediate feedback provided by the devices, which may influence activity or cause anxiety.

A key benefit of monitoring is that measurements can be taken during work and at home with a minimum interruption of ongoing activity. Another advantage of self-monitoring is that home or work blood pressure readings can be collected by self-monitoring over an extended period of time (70), whereas it is not practical to have multiple clinic visits or multiple days of ambu-

latory blood pressure measurements. One disadvantage of self-monitoring is the inability to assess the lack of blood pressure “dipping” during sleep, a suggested CVD risk factor (30).

Concluding remarks

Masked hypertension is a serious individual health problem and a public health concern, for which there has been little research into the role of workplace risk factors. One study found that lower occupational status (blue-collar jobs) is a risk factor, while another found some evidence of a role for job strain. More research is needed to examine the relationships between work organization and masked hypertension.

In addition, the role played by work conditions in the development of hypertension and CVD needs to be brought into the realm of clinical practice. Guidelines are needed for the assessment and management of patients exposed to environmental workplace risk factors (84). Given the availability of inexpensive upper-arm and wrist blood pressure monitors, it should be feasible for all working people to have a sufficient number of blood pressure measurements at work to assess masked workplace hypertension. Working people found to have masked hypertension and exposure to workplace psychosocial stressors could be referred for further assessment of target-organ damage (eg, carotid atherosclerosis and left ventricular mass index). In addition, given the

high proportion of patients whose hypertension appears to be controlled (casual clinical blood pressure <140/90 mm Hg), but who have elevated ambulatory blood pressure, we recommend the monitoring of ambulatory blood pressure on a workday for patients with hypertension, especially those with exposure to workplace stressors.

Unfortunately, given the high prevalence of hypertension and emerging insights concerning work-related hypertension, the individual clinician could become overwhelmed if he or she *alone* attempts to follow the given recommendations. In addition to a role for the clinician, a public health approach is needed, in which blood pressure and workplace risk factors for blood pressure elevation are systematically evaluated on a large scale by appropriately trained allied health workers (eg, industrial hygienists or occupational health nurses). In the Tokyo Declaration (85), occupational health experts from the United States, Europe, and Japan called for a program of “surveillance at individual workplaces and monitoring at national and regional levels in order to identify the extent of work-related stress health problems and to provide baselines against which to evaluate efforts at amelioration [p 5].” Occupations with high prevalence levels of hypertension could be targeted for further evaluation. The clinician can also play an active public health role in this process by identifying clusters of work-related hypertension as potential “occupational sentinel health events” (86).

Surveys in developed countries show increases in stressful work conditions during the period 1990–2005 (87). While comparable temporal trend data are not yet available from developing countries, rapid industrialization suggests that the prevalence of work stressors is increasing in developing countries as well. These trends may well be contributing to the rapid increase in hypertension prevalence in developing countries and the reversal of the previous trend of a decrease in hypertension prevalence in developed countries (5). Reducing work stressors and the prevalence of hypertension will require the worksite surveillance and public health approach described in this paper. In addition, it will require policy efforts at the local, national, and international levels to improve work conditions and promote heart healthy workplaces (85).

Acknowledgments

This manuscript was supported in part by grants HL30605, HL47540, and HL76857 from the National Heart, Lung and Blood Institute, grant OH07577 from the National Institute for Occupational Safety and Health, a grant from the Center for Social Epidemiology,

and by the MacArthur Research Network on SES and Health through a grant from the John D and Catherine T MacArthur Foundation, as well as by a grant from the Signe and Olof Wallenius Foundation.

References

1. Lawes C, Vander Hoorn S, Law M, Elliott P, MacMahon S, Rodgers A. Blood pressure and the burden of coronary heart disease. In: Marmot M, Elliott P, editors. *Coronary heart disease epidemiology*. Oxford (United Kingdom): Oxford University Press; 2005.
2. Graziano J. Global burden of cardiovascular disease. In: Zipes D, Libby P, Bonow R, Braunwald E, editors. *Heart disease*. London: Elsevier; 2004. p 1–19.
3. Lewington S, Clarke R, Qizilbash N, Peto R, Collins R. Age-specific relevance of usual blood pressure to vascular mortality: a meta-analysis of individual data for one million adults in 61 prospective studies. *Lancet*. 2002;360:1903–13.
4. Kearney P, Whelton M, Reynolds K, Muntner P, Whelton P, He J. Global burden of hypertension: analysis of worldwide data. *Lancet*. 2005;365:217–23.
5. Hajjar I, Kotchen J, Kotchen T. Hypertension: trends in prevalence, incidence, and control. *Annu Rev Public Health*. 2006;27:465–90.
6. Hajjar I, Kotchen T. Trends in prevalence, awareness, treatment, and control of hypertension in the United States, 1988–2000. *J Am Med Assoc*. 2003;290(2).
7. Vasan R, Beiser A, Seshadri S, Larson M, Kannel W, D’Agostino R, et al. Residual lifetime risk for developing hypertension in middleaged women and men: the Framingham heart study. *JAMA*. 2002;287:1003–10.
8. Schnall PL, Kern R. Hypertension in American society: an introduction to historical materialist epidemiology. In: Conrad P, Kern R, editors. *The sociology of health and illness: critical perspectives*. New York (NY): St Martin’s Press. 1981. p 97–122.
9. Waldron I, Nowatarski M, Freimer M, Henry JP, Post N, Witten C. Cross-cultural variation in blood pressure: a qualitative analysis of the relationship of blood pressure to cultural characteristics, salt consumption and body weight. *Soc Sci Med*. 1982;16:419–30.
10. Schnall P, Belkic K, Landsbergis PA, Baker D. Why the workplace and cardiovascular disease? In: Belkic K, Landsbergis PA, Schnall P, Baker D, Theorell T, Siegrist J, et al. *The workplace and cardiovascular disease*. Philadelphia (PA): Hanley & Belfus, Inc; 2000. p 1–5. *Occupational Medicine: State of the Art Reviews* vol 15, no 1.
11. Diez Roux A, Chambless L, Merkin S, Arnett D, Eigenbrodt M, Nieto F, et al. Socioeconomic disadvantage and change in blood pressure associated with aging. *Circulation*. 2002;106:703–10.
12. Karasek R, Theorell T. *Healthy work: stress, productivity, and the reconstruction of working life*. New York (NY): Basic Books; 1990.
13. Schnall PL, Landsbergis PA, Schwartz J, Warren K, Pickering TG. A longitudinal study of job strain and ambulatory blood pressure: results from a three-year follow-up. *Psychosom Med*. 1998;60:697–706.
14. Belkic K, Landsbergis PA, Schnall P, Baker D, Theorell T, Siegrist J, et al. Psychosocial factors: review of the empirical

- data among men. In: Schnall P, Belkic K, Landsbergis PA, Baker D, editors. The workplace and cardiovascular disease. Philadelphia (PA): Hanley and Belfus, Inc; 2000. p 24–46. Occupational Medicine: State of the Art Reviews, vol 15, no 1.
15. Brisson C. Women, work and cardiovascular disease. In: Schnall P, Belkic K, Landsbergis PA, Baker D, editors. The workplace and cardiovascular disease. Philadelphia (PA): Hanley and Belfus, Inc; 2000. p 49–57. Occupational Medicine: State of the Art Reviews, vol 15, no 1.
 16. Hayashi T, Kobayashi Y, Yamaoka K, Yano E. Effect of overtime work on 24-hour ambulatory blood pressure. *J Occup Environ Med.* 1996;38(10):1007–11.
 17. Iwasaki K, Sasaki T, Oka T, Hisanaga N. Effect of working hours on biological functions related to cardiovascular system among salesmen in a machinery manufacturing company. *Ind Health.* 1998;36:361–7.
 18. Yang H, Schnall P, Jauregui M, Su T, Baker D. Work hours and self-reported hypertension among working people in California. *Hypertension.* 2006;48(4):744–50.
 19. Fialho G, Cavichio L, Pova R, Pimenta J. Effects of 24-h shift work in the emergency room on ambulatory blood pressure monitoring values of medical residents. *Am J Hypertens.* 2006;19:1005–9.
 20. Vrijkotte TG, van Doornen LJ, de Geus EJ. Effects of work stress on ambulatory blood pressure, heart rate, and heart rate variability. *Hypertension.* 2000;35(4):880–6.
 21. Peter R, Alfredsson L, Hammar N, Siegrist J, Theorell T, Westerholm P. High effort, low reward, and cardiovascular risk factors in employed Swedish men and women: baseline results from the WOLF Study. *J Epidemiol Community Health.* 1998;52(9):540–7.
 22. Belkić K, Savić Č, Theorell T, Rakić L, Ercegovic D, Djordjević M. Mechanisms of cardiac risk among professional drivers [review]. *Scand J Work Environ Health.* 1994;20(2):73–86.
 23. Greiner B, Krause N, Ragland D, Fisher J. Occupational stressors and hypertension: a multi-method study using observer-based job analysis and self-reports in urban transit operators. *Soc Sci Med.* 2004;59:1081–94.
 24. Cobb S, Rose RM. Hypertension, peptic ulcer, and diabetes in air traffic controllers. *JAMA.* 1973;224(4):489–92.
 25. Saarni H, Niemi L, Pentti J, Hartiala J, Kuusela A. Cardiac status and cardiovascular risk factors among Finnish sea pilots. *Int J Occup Med Environ Health.* 1996;9(1):53–8.
 26. Pieper C, Schnall PL, Warren K, Pickering TG. A comparison of ambulatory blood pressure and heart rate at home and work on work and non-work days. *J Hypertens.* 1993;11(2):177–83.
 27. Pickering TG. Blood pressure measurement: casual, self-measured, and ambulatory monitoring. In: Schnall P, Belkic K, Landsbergis PA, Baker D, editors. The workplace and cardiovascular disease. Philadelphia (PA): Hanley and Belfus, Inc; 2000. p 191–6. Occupational Medicine: State of the Art Reviews, vol 15, no 1.
 28. Eilertsen E, Humerfelt S. The observer variation in the measurement of arterial blood pressure. *Acta Med Scand.* 1968;184:145–57.
 29. Garcia-Vera M, Labrador F, Sanz J. Comparison of clinic, home self-measured, and work self-measured blood pressures. *Behav Med.* 1999;25:13–22.
 30. Pickering T, Shimbo D, Haas D. Ambulatory blood pressure monitoring. *N Engl J Med.* 2006;354:2368–74.
 31. Bottini P, Carr A, Prisant L, Rhoades R. Variability and similarity of manual office and automated blood pressures. *J Clin Pharmacol.* 1992;32(7):614–9.
 32. Gerber LM, Schwartz JE, Schnall PL, Devereux RB, Warren K, Pickering TG. Effect of body weight changes on changes in ambulatory and standardized non-physician blood pressures over three years. *Ann Epidemiol.* 1999;9(8):489–97.
 33. Schnall PL, Schwartz JE, Landsbergis PA, Warren K, Pickering TG. Relation between job strain, alcohol, and ambulatory blood pressure. *Hypertension.* 1992;19:488–94.
 34. Schwartz J, Warren K, Pickering T. Mood, location and physical position as predictors of ambulatory blood pressure and heart rate: application of a multi-level random effects model. *Ann Behav Med.* 1994;16(3):210–20.
 35. Pickering T, Harshfield G, Kleinert H, Blank S, Laragh J. Blood pressure during normal daily activities, sleep, and exercise: comparison of values in normal and hypertensive subjects. *JAMA.* 1982;247(7):992–6.
 36. Pickering TG. The effects of environmental and lifestyle factors on blood pressure and the intermediary role of the sympathetic nervous system. *J Hum Hypertension.* 1997;11 suppl 1: S9–S18.
 37. Appel LJ, Stason WB. Ambulatory blood pressure monitoring and blood pressure self-measurement in the diagnosis and management of hypertension. *Ann Intern Med.* 1993;118:867–82.
 38. Pickering TG, Alpert B, de Swiet M, Harshfield G, O'Brien E, Shennan A. Ambulatory blood pressure. Redmond (WA): SpaceLabs Medical, Inc; 1994.
 39. Verdecchia P, Clement D, Fagard R, Palatini P, Parati G. Task force III: target-organ damage, morbidity and mortality. *Blood Press Monit.* 1999;4:303–17.
 40. Sega R, Trocino G, Lanzarotti A, Caruge S, Cesana G, Schiavina R, et al. Alterations of cardiac structure in patients with isolated office, ambulatory, or home hypertension: data from the general population [Pressione Arteriose Monitorate E Loro Associazioni (PAMELA) Study]. *Circulation.* 2001;104(12):1385–92.
 41. Pierdomenico S, Lapenna D, Bucci A, Di Tommaso R, Di Mascio R, Manente B, et al. Cardiovascular outcome in treated hypertensive patients with responder, masked, false resistant, and true resistant hypertension. *Am J Hypertens.* 2005;18:1422–8.
 42. Bobrie G, Chatellier G, Genes N, Clerson P, Vaur L, Vaisse B, et al. Cardiovascular prognosis of “masked hypertension” detected by blood pressure self-measurement in elderly treated hypertensive patients. *JAMA.* 2004;291:1342–9.
 43. Ohkubo T, Imai Y, Tsuji I, Nagai K, Kato J, Kikuchi N, et al. Home blood pressure measurement has a stronger predictive power for mortality than does screening blood pressure measurement: a population-based observation in Ohasama, Japan. *J Hypertens.* 1998;16:971–5.
 44. Perloff D, Sokolow M, Cowan RM, Juster RP. Prognostic value of ambulatory blood pressure measurements: further analyses. *J Hypertens.* 1989;7 suppl 3:S3–S10.
 45. Verdecchia P. Prognostic value of ambulatory blood pressure: current evidence and clinical implications. *Hypertension.* 2000;35(3):844–51.
 46. Clement D, De Buyzere M, De Bacquer D, de Leeuw P, Duprez D, Fagard R, et al. Prognostic value of ambulatory blood-pressure recordings in patients with treated hypertension. *N Engl J Med.* 2003;348:2407–15.
 47. Williams G. Hypertensive vascular disease. In: Fauci A, Braunwald E, Isselbacher K, Wilson J, Martin J, Kasper D, et al, editors. Harrison's principles of internal medicine. 14th ed. New York (NY): McGraw-Hill; 1998. p 1380–94.

48. Schnall PL, Landsbergis PA, Baker D. Job strain and cardiovascular disease. *Annu Rev Public Health*. 1994;15:381–411.
49. Steptoe A, Roy MP, Evans O, Snashall D. Cardiovascular stress reactivity and job strain as determinants of ambulatory blood pressure at work. *J Hypertens*. 1995;13(2):201–10.
50. Goldstein I, Shapiro D, Chicz-DeMet A, Guthrie D. Ambulatory blood pressure, heart rate and neuroendocrine responses in women nurses during work and off work days. *Psychosom Med*. 1999;61:387–96.
51. Brown D, James G, Nordloh L, Jones A. Job strain and physiological stress responses in nurses and nurse's aides: predictors of daily blood pressure variability. *Blood Press Monit*. 2003;8:237–42.
52. Riese H, Van Doornen LJ, Houtman IL, De Geus EJ. Job strain in relation to ambulatory blood pressure, heart rate, and heart rate variability among female nurses. *Scand J Work Environ Health*. 2004;30(6):477–85.
53. Landsbergis PA, Schnall PL, Warren K, Pickering TG, Schwartz JE. Association between ambulatory blood pressure and alternative formulations of job strain. *Scand J Work Environ Health*. 1994;20(5):349–63.
54. Belkic KL, Landsbergis PA, Schnall PL, Baker D. Is job strain a major source of cardiovascular disease risk? [review]. *Scand J Work Environ Health*. 2004;30(2):85–128.
55. Steenland K. Shift work, long hours, and CVD: a review. In: Schnall P, Belkic K, Landsbergis PA, Baker D, editors. *The workplace and cardiovascular disease*. Philadelphia (PA): Hanley & Belfus, Inc; 2000. p 7–17. *Occupational Medicine: State of the Art Reviews*, vol 15, no 1.
56. Schwartz J, Belkic K, Schnall P, Pickering T. Mechanisms leading to hypertension and CV morbidity. In: Schnall P, Belkic K, Landsbergis PA, Baker D, editors. *The workplace and cardiovascular disease*. Philadelphia (PA): Hanley & Belfus, Inc; 2000. p 121–132. *Occupational Medicine: State of the Art Reviews*, vol 15, no 1.
57. Siegrist J, Rödel A. Work stress and health risk behavior [review]. *Scand J Work Environ Health*. 2006;32(6, special issue):473–81.
58. Belkic K, Nedic O. Workplace stressors and lifestyle-related cancer risk factors among female physicians: assessment using the Occupational Stress Index. *J Occup Health*. 2007;49(1):61–71.
59. Marmot MG, Bosma H, Hemingway H, Brunner E, Stansfeld S. Contribution of job control and other risk factors to social variations in coronary heart disease incidence. *Lancet*. 1997;350:235–9.
60. Landsbergis PA, Schnall PL, Pickering TG, Warren K, Schwartz JE. Lower socioeconomic status among men in relation to the association between job strain and blood pressure. *Scand J Work Environ Health*. 2003;29(3):206–15.
61. Liu JE, Roman MJ, Pini R, Schwartz JE, Pickering TG, Devereux RB. Cardiac and arterial target organ damage in adults with elevated ambulatory and normal office blood pressure. *Ann Intern Med* 1999;131(8):564–72.
62. Comment. *Ann Intern Med*. 2000;132(10):842.
63. Hara A, Ohkubo T, Kikuya M, Shintani Y, Obara T, Metoki H, et al. Detection of carotid atherosclerosis in subjects with masked hypertension and whitecoat hypertension by self-measured blood pressure at home: the Ohasama study. *J Hypertens* 2007;25:321–7.
64. Björklund K, Lind L, Zethelius B, Andrén B, Lithell H. Isolated ambulatory hypertension predicts cardiovascular morbidity in elderly men. *Circulation*. 2003;107:1297–302.
65. Ohkubo T, Kikuya M, Metoki H, Asayama K, Obara T, Hashimoto J, et al. Prognosis of masked hypertension and white coat hypertension detected by 24-h ambulatory blood pressure monitoring. *J Am Coll Cardiol*. 2005;46:508–15.
66. Mancia G, Facchetti R, Bombelli M, Grassi G, Sega R. Long-term risk of mortality associated with selective and combined elevation in office, home, and ambulatory blood pressure. *Hypertension*. 2006;47:846–53.
67. Tomiyama M, Horio T, Yoshii M, Takiuchi S, Kamide K, Nakamura S, et al. Masked hypertension and target organ damage in treated hypertensive patients. *Am J Hypertens*. 2006;19:880–6.
68. Pierdomenico S, Cuccurullo F, Mezzetti A. Masked hypertension in treated hypertensive patients. *Am J Hypertens*. 2006;19:873–4.
69. O'Brien E, Asmar R, Beilin L, Imai Y, Mallion J-M, Mancia G, et al. European Society of Hypertension recommendations for conventional, ambulatory and home blood pressure measurement. *J Hypertens*. 2003;21:821–48.
70. Pickering T. Extending the reach of ambulatory blood pressure monitoring. *Am J Hypertens*. 2005;18:1385–7.
71. Selenta C, Hogan BE, Linden W. How often do office blood pressure measurements fail to identify true hypertension?: an exploration of white-coat normotension. *Arch Fam Med*. 2000;9(6):533–40.
72. Tsai P-S. Determinants of the white-coat effect in normotensives and never-treated mild hypertensives. *Clin Exp Hypertens*. 2003;25(7):443–54.
73. Rasmussen S, Torp-Pedersen C, Borch-Johnsen K, Ibsen H. Normal values for ambulatory blood pressure and differences between casual blood pressure and ambulatory blood pressure: results from a Danish population survey. *J Hypertens*. 1998;16:1415–24.
74. Bombelli M, Sega R, Facchetti R, Corrao G, Polo Friz H, Vertemati A, et al. Prevalence and clinical significance of a greater ambulatory versus office blood pressure (reverse white coat condition) in a general population. *J Hypertens*. 2005;23:513–20.
75. Gallo LC, Bogart LM, Vranceanu AM, Walt LC. Job characteristics, occupational status, and ambulatory cardiovascular activity in women. *Ann Behav Med*. 2004;28(1):62–73.
76. Devereux RB, Pickering TG, Harshfield GA, Kleinert HD, Denby L, Clark L, et al. Left ventricular hypertrophy in patients with hypertension: importance of blood pressure response to regularly recurring stress. *Circulation*. 1983;68:476–9.
77. Folkow B. Autonomic nervous system in hypertension. In: Swales JD, editor. *Textbook of hypertension*. London: Blackwell Scientific Publications; 1994. p 427–38.
78. Schnall P, Belkic K. Point estimates of blood pressure at the worksite. In: Schnall P, Belkic K, Landsbergis PA, Baker D, editors. *The workplace and cardiovascular disease*. Philadelphia (PA): Hanley & Belfus; 2000. p 203–8. *Occupational Medicine: State of the Art Reviews*, vol 15, no. 1.
79. Schnall P, Belkic K, Landsbergis P, Schwartz J, Gerber L, Baker D, et al. Hypertension at the workplace—often an occult disease: the relevance and potential in Japan for work site surveillance? *Jpn J Stress Sci*. 2000;15(3):152–74.
80. Belkic KB, Schnall PL, Landsbergis PA, Schwartz JE, Gerber L, Baker D, et al. Hypertension at the workplace—an occult disease?: the need for work site surveillance. *Adv Psychosom Med*. 2001;22:116–38.
81. Stergiou G, Salgami E, Tzamouranis D, Roussias L. Masked hypertension assessed by ambulatory blood pressure versus home blood pressure monitoring: is it the same phenomenon? *Am J Hypertens*. 2005;18:772–8.

82. Imai Y, Tsuji I, Nagai K, Sakuma M, Ohkubo T, Watanabe N, et al. Ambulatory blood pressure monitoring in evaluating the prevalence of hypertension in adults in Ohasama, a rural Japanese community. *Hypertens Res.* 1996;19(3):207–12.
83. Altunkan S, Iliman N, Altunkan E. Accuracy of the new wrist blood pressure monitor (OMRON 637 IT) for blood pressure measurement. *J Hypertens.* 2003;21 suppl 4:S22.
84. Belkic K, Schnall P, Ugljesic M. Cardiovascular evaluation of the worker and workplace: a practical guide for clinicians. In: Schnall P, Belkic K, Landsbergis PA, Baker D, editors. *The workplace and cardiovascular disease.* Philadelphia (PA): Hanley & Belfus; 2000. p 213–22. *Occupational Medicine: State of the Art Reviews*, vol 15, no 1.
85. The Tokyo Declaration. *J Tokyo Med Univ.* 1998;56(6):760–7.
86. Fisher J, Belkic K. A public health approach in clinical practice. In: Schnall P, Belkic K, Landsbergis PA, Baker D, editors. *The workplace and cardiovascular disease.* Philadelphia (PA): Hanley & Belfus; 2000. p 245–56. *Occupational Medicine: State of the Art Reviews*, vol 15, no 1.
87. European Foundation. *Fifteen years of working conditions in the EU: charting the trends.* Dublin: European Foundation for the Improvement of Living and Working Conditions; 2006.
88. Fiedler N, Favata E, Goldstein B, Gochfeld M. Utility of occupational blood pressure screening for the detection of potential hypertension. *J Occup Med.* 1988;30(12):943–8.