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Hypertension at the Workplace – An Occult Disease? The Need for Work Site Surveillance

Karen L. Belkić[a], *Peter L. Schnall*[b], *Paul A. Landsbergis*[c], *Joseph E. Schwartz*[d], *Linda M. Gerber*[f], *Dean Baker*[e], *Thomas G. Pickering*[c]

[a] Institute for Health Promotion and Disease Prevention Research, University of Southern California School of Medicine, Los Angeles, Calif.;
[b] Center for Social Epidemiology, Santa Monica, Calif.;
[c] Zena and Michael A. Wiener Cardiovascular Institute, Mount Sinai School of Medicine, New York, N. Y.;
[d] Department of Psychiatry and Behavioral Sciences, SUNY at Stony Brook, N. Y.;
[e] Division of Occupational and Environmental Medicine, Department of Medicine, University of California at Irvine, College of Medicine, Irvine, Calif.; and
[f] Department of Public Health, Weill Medical College of Cornell University, New York, N. Y., USA

Why the Workplace and Hypertension?

Approximately half of the adult population in industrialized countries has a persistently elevated blood pressure (BP) > 140/90 mm Hg by age 60. The morbidity and mortality associated with essential hypertension (EH) is well established, and the increased risk begins at BP levels substantially lower than the traditional cutoff of 140/90 mm Hg. This major epidemic appears to be socially patterned: as a disease of industrialized societies, with minimal hypertension burden in nonmarket agricultural communities, hunter-gatherers and other nonindustrialized societies [1]. The rising prevalence of hypertension seems to parallel the transformation in working life during the past century, away from agricultural work and relatively autonomous craft-work towards machine-based (including computer-dependent) labor, as is characteristic, e. g., of mass production. An emerging body of scientific evidence implicates specific features of work as important causes of hypertension. As recently argued by Schnall et al. [2] 'The contemporary work environment is the locus in which adults now spend the majority

of their waking hours, performing activities which are increasingly characterized, both by scientists and the workers, as demanding, constraining and highly stressful.' (These characteristics are embodied in the concept of job strain: work which is both psychologically demanding and which offers low decision-making latitude [3].) 'We know that for most people blood pressure is elevated during working hours. We also know that performing demanding, constraining and otherwise mentally stressful activity provokes sharp rises in BP' [2, p. 3].

We can ask the question: Do these observations suggest a way to better tackle the epidemic of EH? Could it be that we are diagnosing some cases of EH too late, by failing to target the role of work in its etiology? As we will elaborate in this paper, it appears that for many people, before BP becomes persistently elevated, there may be a phase of 'occult hypertension at the workplace', in which casual BP remains normal in the clinic setting, but is high during work. We will suggest that reliance upon casual clinic blood pressure determination has contributed to an overemphasis upon those who respond with an acute rise in BP to an atypical psychosocial situation, that of the doctor's (or other health professional's) office. This BP response is very often unrepresentative of the person's usual BP, and this white coat hypertension is of low prognostic value. Meanwhile, people who may be at high risk, those with workplace hypertension, often go undetected.

We will suggest ways to more efficiently use the potential of ambulatory BP (AmBP) monitoring techniques, in conjunction with other, more widely applicable methods to maximize diagnostic yield with respect to workplace hypertension. This will be linked to a targeted approach to surveillance of high-risk workplaces, as a key public health strategy.

Missing the Target: Reliance on Casual Clinic Blood Pressure

How Reliable Is the Standard?

According to conventional practice, the standard for assessing BP has been measurement using a mercury sphygmomanometer by a physician or other health professional [4]. Clinicians have relied on this method to diagnose and treat arterial hypertension for nearly a century.

This method of measuring BP is convenient, inexpensive, low technology, and offers some predictive validity. For these reasons, casual clinic blood pressure (CCBP) has been widely used. Notwithstanding its popularity, CCBP is, in fact, 'notoriously unreliable' [4]. BP values recorded by different trained personnel vary by as much as 5–10 mm Hg [5], and have relatively poor test-retest reliability [6]. Observer measurement problems due to

digit preference (0 or 5) and varying levels of auditory acuity, inter alia, are well recognized. As pointed out by Pickering [4] 'The level of pressure that is recorded also may be profoundly influenced by behavioral factors related to the effects of the observer on the subject, the best known of which is the presence of a physician. Other factors that can influence the pressure that is recorded include the race and sex of the observer' [p. 192]. Another major source of unreliability is 'the inherent variability of BP' [4, p. 191].

Limitations in Predictive Validity

As the conventional method of BP assessment, CCBP has been indispensable in helping to demonstrate the relationship between hypertension and its well-known major sequelae such as cerebrovascular accidents, coronary heart disease (CHD), congestive heart failure, renal failure, and hypertensive retinopathy. In the Framingham Study, for example, among the standard cardiac risk factors, elevated CCBP was found to be 'the most common, most potent and most universal contributor to cardiovascular mortality' [7, p. 5]. More recently, the 6th Report of the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure [8] concluded that 'the positive relationship between SBP and DBP and cardiovascular risk … is strong, continuous, graded, independent, predictive and etiologically significant for those with and without CHD' [p. 2417].

Appel and Stason [9] note, however, that office BPs provide 'sub-optimal prediction of cardiovascular risk' [p. 867]. A recent 10-year longitudinal, double-blind placebo-controlled study by Staessen et al. [10] among elderly persons with systolic hypertension indicates that at randomization a 10-mm-Hg higher casual clinic SBP (obtained from six readings at three separate visits) was not associated with increased risk of future cardiovascular and cerebrovascular morbidity and mortality, after adjusting for gender, age, previous cardiovascular complications, smoking and residence in Western Europe. Khattar et al. [11] reported that among patients with uncomplicated hypertension, CCBP was not an independent predictor of two major cardiovascular end-organ sequelae: left ventricular hypertrophy and carotid artery atherosclerosis. Other studies indicate that CCBP shows weak or no correlation with left ventricular mass index (LVMI), both cross-sectionally [12] and longitudinally [13].

The predictive validity of CCBP is particularly limited among those 'whose physician-measured blood pressure straddles the conventional threshold for diagnosis of hypertension and initiation of drug therapy' [9, p. 867]. This point is well illustrated in the Australian National Blood Pressure Study [14] of 3,400 mild hypertensives, half of whom were treated. There were 9 fewer cerebrovascular events per year among the treated (13

events), compared to the untreated (22 events), while the cardiac event rate was identical among the treated and the untreated. Thus, 1,700 persons per year were treated to prevent a total of 9 cerebrovascular (and no cardiac) events. A total of 1,691 received no apparent major benefit from antihypertensive drug treatment, which in itself is not without untoward effects [15]. Similar conclusions are derived from the Multiple Risk Factor Intervention Trial (MRFIT) [16], in which antihypertensive treatment based upon average DBP of at least 90 mm Hg, among middle-aged men with high levels of CHD risk factors revealed 'no favorable association between lowering blood pressure and CHD rate' [p. 1].

Clinical Misclassification: White-Coat Hypertension and Undetected Hypertension of Daily Life

Clinical misclassification is one of the most vexing problems that arises when basing diagnoses upon CCBP. White-coat hypertension – high BP readings due to patient arousal and anxiety in the clinic setting – is well known to most healthcare professionals. This white-coat hypertension or false positive diagnosis often leads to unnecessary treatment, since, according to most studies, this is a relatively benign condition [17–19; for a recent review, see 19, and for an opposing view, see 20]. As recently suggested: 'In the clinic setting there are a number of psychosocial stimuli present that can affect BP. For some individuals the presumably unpleasant experience of the clinic setting can produce an elevation. This may be a unique response (i. e. totally unrelated to the usual universe of BPs during daily life)' [21, p. 205].

Improved reliability and validity can be obtained by measuring clinic BP on several separate occasions. For example, in a study by Jula et al. [22] averaging the results of CCBP taken three times each by a nurse, on four separate clinic visits, resulted in a high correlation with LVMI. Similar conclusions were made by Pearce et al. [23]. Thus, in clinical practice it is generally recommended that elevations in BP be 'persistent', namely that they be recorded on several separate occasions [8, 24]. This entails substantial inconvenience to patients as well as a major burden with regard to healthcare utilization.

While the problem of false positive diagnosis is well recognized and potentially manageable by repeated clinic visits, persons with normal CCBP have conventionally been considered normotensive. Consequently, they are rarely followed up by returning to the clinic to have their BP re-checked. This group has received little attention. As will be discussed, the false negatives: those with normal BP based upon casual clinic measurements, but in whom workplace AmBP is elevated (i. e. those with occult workplace hypertension) may be at high risk, rendering their detection of potential clinical importance.

Limited Etiologic Insight

CCBPs have also been used for etiologic research into EH. Risk factors such as age, heredity, obesity, alcohol intake, inter alia, have been identified, as well as some contributory mechanisms such as salt sensitivity, low as well as high renin, low calcium, etc. These factors, however, explain only a part of the risk. Thus, despite enormous research efforts, the definition of EH remains: 'patients with arterial hypertension and no definable cause' [24, p. 1381]. This accounts for approximately 90–95% of all cases of arterial hypertension [24].

One reason why the etiology of EH remains unclear is that BP measured in the clinic frequently does not reflect the dynamics of daily life, and it is during the latter that a wealth of information can be gleaned about how EH, in its early stages, develops. By measuring BP in the atypical environment of the clinic, we may be missing the target.

We have recently elaborated on this point [21], based upon the presumed mechanisms of neurogenic hypertension [25]. Assuming that psychosocial stressors at work play an important role in the etiology of EH, various stages can be identified: (1) When individuals first are exposed to workplace stressors, BP (primarily systolic) is elevated at work and casual BP remains normal; this is consistent with elicitation of the defense response during work. (2) With chronic exposure to these stressors, both workplace BP as well as casual BP would be elevated. At this stage the psychosocial factors likely are correlated with both workplace and casual BP (structural changes in the heart and vasculature may be occurring). (3) Self-sustaining structural processes in the vascular system may lead to a disjuncture between reported exposures to workplace stressors and BP, since (a) the hypertensive process may now have become autonomous, and (b) exposure to the workplace factors that lead to BP elevations, may have ceased due to retirement or other change in employment status. Thus, the early development of work-related hypertension could not be easily traced with CCBP. Using CCBP an existing relation between exposure to workplace stressors and hypertension could only be detected during stage 2. And even in that stage, the instability of CCBP would still present a clouded picture. In the late (third) stage, retrospective analysis, with its obvious pitfalls, might be the only way to capture how work-related hypertension actually developed. Simply stated, the etiologic role of work stress in the development of EH would be obfuscated by relying solely upon CCBP measurements [21].

Examining BP during Working Life: AmBP Monitoring

The possibility of monitoring BP at frequent intervals during daily life was first developed over 30 years ago. With recent major technological advances, this method has become widely applied in research, as well as in the clinical setting. AmBP monitors are now quite small in size, relatively nonperturbative and capable of taking up to 100 readings over a 24-hour period [4]. Thus, a large number of readings are taken in the natural setting of the real world.

Ambulatory monitoring captures the dynamic BP fluctuations in relation to daily life: to changes in posture and physical activity, to location (e. g. work vs. home), waking vs. sleep, and to mood and psychological state. BP varies dramatically during the course of 24 h, and its sensitivity to physical and psychosocial stimuli is clearly evident. It is found, for example, that AmBP is higher (on average by 5 mm Hg or more systolic) during the hours on the job compared to leisure time [26–28]. Furthermore, mean 24-hour AmBP is lower on non-work days compared to work days [29–30].

Advantages and Disadvantages of AmBP

Improved Reliability, Diagnostic and Predictive Validity. While the precision of a single AmBP reading is lower than that of an observer-measured value from a mercury sphygmomanometer, averaged AmBP is more reliable than CCBP. As stated by Pickering et al. [31], 'true blood pressure is best estimated by taking the average of a large number of readings rather than relying on one or two' [p. 12]. Reliability is also improved because the behavioral effect of the observer on the patient, as well as subjective observer errors, are eliminated. There is no observer bias nor white-coat effect.

A large and consistent body of evidence indicates that AmBP is more highly correlated with morbid sequelae compared to CCBP. The data are mainly cross-sectional, and are most abundant with respect to echocardiographically assessed LVMI. In nearly all of the numerous studies reviewed by Appel and Stason [9] and Pickering et al. [31], echocardiographic measures of left ventricular structure were more highly correlated with AmBP (especially SBP) than with CCBP, even when the latter was measured up to 10–12 times. In the study by Devereux et al. [12], work systolic and diastolic AmBP showed the highest correlations with LVMI, compared to home, sleep and 24-hour averages. However, Verdecchia et al. [32] reported a higher correlation between LVMI and nighttime AmBP, with a lack of nocturnal dipping associated with increased LVMI. Recently, Mancia et al. [13] reported a significant longitudinal relation between pharmacological lowering of 24-hour AmBP and regression of LVMI among patients with hyper-

tension and increased LVMI at baseline. Correlations are also generally higher between other signs of hypertension-related end-organ damage such as albuminuria, funduscopic changes and altered arterial pulse wave velocity, and AmBP as compared to CCBP [31].

A series of 5-year longitudinal analyses by Perloff et al. [33–35] reveals that compared to CCBP, AmBP consistently provided superior adjusted prediction of cardiovascular morbid events in patients with and without prior events. Patients whose AmBP was lower than expected based on CCBP were found to be at lower risk of morbid events compared to those with higher AmBP.

Uncontrolled Circumstances of the Natural Setting and Expense of AmBP. A disadvantage of AmBP is that the circumstances of recording are uncontrolled, and any given day may be atypical, for a variety of reasons. Since psychosocial factors vary from day to day, AmBP can be expected to likewise vary, introducing some degree of unreliability. Thus, the clinician would probably want to repeat the recording session if a patient indicated that the recording day turned out to be highly atypical. As elaborated by Steptoe and Vögele [36], a major dilemma for field studies is the complexity of real-life activity and the inability to control concurrent phenomena extraneous to the psychosocial stress effects under study. Keeping a detailed diary can be helpful, but this is burdensome, and may even alter the activity pattern itself.

AmBP monitoring is also relatively expensive – it is skilled-labor intensive and has high technological requirements. Appel and Stason [9] estimate that in the USA alone, if AmBP monitoring were to become a routine procedure for the diagnosis and monitoring of hypertension, the annual costs could be up to USD 6 billion. Thus, it has been emphasized that this method be used judiciously, where it will provide the highest benefit.

Detection of Occult Workplace Hypertension

The role of AmBP monitoring in ruling out white-coat hypertension has been repeatedly emphasized. It is generally assumed that clinic pressures are higher than those during daily activity. The 6th Report of the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure [8] states: 'normal blood pressure values taken by ambulatory measurement are lower than clinic readings while patients are awake' [p. 2418].

At closer inspection, however, it becomes apparent that these sorts of conclusions are not derived from analyses of working population-based samples. In table 1, we present data from the initial case-control sample of 267 men from the Work Site Blood Pressure Study [27, 37, 50]. (The 86 cases were defined as those with casual clinic DBP >85 and ≤105 mm Hg, and the

Table 1. Diagnostic classification of hypertension using casual versus ambulatory BP in a sample of working men from the Worksite Blood Pressure Study [from 27]

Casual DBP taken at the work site clinic	Work site ambulatory DBP		
	>85 mm Hg (n)	≤85 mm Hg (n)	Total (n)
>85 mm Hg	59	27	86
≤85 mm Hg	36	145	181
Total	95	172	267

Positive predictive value of casual DBP >85 mm Hg taken at the work site clinic: 59/86 = 68.6%; negative predictive value of casual DBP ≤85 mm Hg taken at the work site clinic: 145/181 = 80.1%. (Positive predictive value = true positives/(true positives + false positives), where (true positives + false positives) are all those with positive results; negative predictive value = true negatives/(true negatives + false negatives), where (true negatives + false negatives) are all those with negative results [38].)

181 controls as those with clinic DBP ≤85 mm Hg. For a more detailed discussion of how the sample was assembled, see [27, 37]). It can be seen that a total of 27 were in the white-coat hypertension group, with casual DBP >85 mm Hg and average ambulatory work site DBP ≤85. Meanwhile, there were 36 men with occult workplace hypertension defined here as work ambulatory DBP >85 mm Hg, but a normal level of casual work site DBP (≤85 mm Hg).

Since all eligible cases were included, while there was only a random sample of controls, these data do not reflect the correct ratio of cases to controls in the target population, and therefore cannot be used for calculation of prevalence rates, nor false positive and false negative rates. The underrepresentation of those with normal CCBP in this case-control sample suggests that the magnitude of the problem of occult workplace hypertension has been substantially underestimated here, and that it is far more widespread than is white-coat hypertension. A subsequent paper is planned in which these issues will be addressed in detail. It should furthermore be emphasized that the casual BP measurements were made at the work site, albeit in a clinic setting. If a clinic setting far removed from the work environment had been used to measure CCBP, there might have been even more persons with occult workplace hypertension.

The clinical importance of occult workplace hypertension was suggested in a recent paper by Liu et al. [39]. In their sample, persons with normal clinic BP (defined as <140/90 mm Hg) and elevated waking (work and home) AmBP (defined as >134/90) 'the false negatives' (61 of the 295 with normal CCBP), had an LVMI 13 g/m^2 (CI 8–18 g/m^2) greater than those

with normal waking AmBP and clinic BPs. These individuals with occult hypertension during waking life had a similar LVMI and prevalence of discrete atherosclerotic plaques compared to 64 patients with both increased clinic and AmBP. Both groups differed significantly in these respects from those with normal AmBP and normal CCBP, after adjusting for covariates. In other words, the risk of these two major sequelae is similar among those with occult hypertension of waking life ('the false negatives'), compared to those with elevated waking AmBP and casual measurements ('the true positives'). Since the analyses performed by Liu's group combines home and working into the awake category, it may be argued that the evidence is only indirect with regard to the clinical importance of occult workplace hypertension. However, considering that AmBP during work is generally higher than during home wake time, it seems plausible that the relation between occult workplace hypertension and these hypertensive sequelae could be even greater than that reported in the paper of Liu et al. [39]. Further investigation specifically focusing upon the prognostic importance of occult workplace hypertension is warranted.

These findings by Liu et al. [39] are consistent with and may shed some light on the longitudinal findings from the MRFIT study concerning CCBP levels and risk of BP-related CHD. Results from the MRFIT study of 350,000 men indicate that only about 5% of middle-aged men had CCBP exceeding the high-risk threshold (160/95 mm Hg), and less than 1/4 of CHD deaths due to high BP occurred in men with CCBP that high [40]. Many more CHD cases (over 40% of CHD deaths due to high BP) occurred in men with CCBP levels between 140 and 159 mm Hg. Notably, 35% of the BP-related CHD mortality occurred in the large group (50% of middle-aged men) with 'normal' casual clinic SBP, i. e., between 110 and 139 mm Hg [40, 41]. The large number of BP-related CHD deaths outside the 'high risk' group in the MRFIT study is similar to that of other studies such as Framingham [42] and Whitehall [43].

AmBP Monitoring in Etiologic Research of EH

AmBP monitoring offers the chance to explore a wide range of hypotheses concerning the etiology of EH. With careful attention to design issues, especially potential confounders and sufficient power to test the hypothesis, thorough investigation can be made into the role of environmental exposures, various daily activities, psychological factors, as well as the relative and additive burden of work and home responsibilities, inter alia.

Job Strain and AmBP. With respect to workplace factors and AmBP, exposure to job strain (work characterized by high psychological demands and low control [3]) has been the most intensively investigated. A review of the

medical literature demonstrates that among men, studies that measure AmBP during work, as well as other periods, 'show strong, consistent effects of job strain or its major dimension(s) on blood pressure' [44, p. 38]. Most of the reviewed investigations used a cross-sectional design.

One of the largest, however, the above-described Work Site Blood Pressure Study carried out in New York City, includes not only repeated cross-sectional data, but also 6 years of longitudinal follow-up. This investigation began in 1985 as a case-control study of men from eight large work sites. There is now a total of 472 participants (38% women) enrolled from ten work sites. Details of the assembly of the original sample, eligibility criteria and protocol are summarized in several papers [27, 37, 45, 46, 50].

Cross-sectional results at baseline and after 3 years of follow-up are displayed in table 2. (Preliminary data have been analyzed on the 6 years of follow-up, and are consistent with the results presented here). Table 2 shows that after controlling for relevant confounders, mean workplace systolic AmBP was consistently >6 mm Hg higher among the men exposed to job strain (measured by self-report), compared to those not exposed. Moreover, systolic and diastolic AmBP were increased not only at work, but at home and during sleep, as well. Three-year longitudinal results of those chronically exposed to job strain reveal a +11.1/+9.1 mm Hg adjusted difference in work systolic/diastolic AmBP, compared to those unexposed both at baseline and at 3-year follow-up. Furthermore, those whose job strain exposure status changed from 'yes' to 'no' had a mean drop in workplace AmBP of –5.3/–3.2 mm Hg. However, those who changed from nonexposed to exposed did not show a significant change in AmBP [46]. We have previously noted that 'a decrease in blood pressure brought about by a non pharmacological means (such as weight loss) may result in lower risk of heart disease than a comparable fall in BP due to antihypertensive drug treatment. The effect of no longer being exposed to job strain … was comparable in magnitude to losing more than 40 pounds' [47, p. 237].

Published AmBP studies examining exposure to job strain are less numerous among women. As recently summarized by Brisson [48], there are several studies with nonsignificant results, including one among a large sample (n = 139) of white-collar women without a university degree. There is also a substantial number of studies among women showing a significant positive effect of job strain upon AmBP. The same author emphasizes that the magnitude of effect sizes (up to +8/+6.4 mm Hg) is clinically important. Further, Brisson argues that these are likely underestimates of the true effect sizes, since the studies are cross-sectional and there is evidence that women in high strain jobs tend differentially to move away from such workplaces. Recently, Brisson et al. [49] demonstrated a significant additive effect

Table 2. Summary of statistically significant findings from the work site BP study on job strain[1] and AmBP[2] [data from 27, 46]

Design type	Wave	AmBP effect	Location	Effect size (mm Hg)
Cross-sectional results	Wave 1 (n = 264)	Systolic	Work	+6.8
		Diastolic	Work	+2.8
		Systolic	Home	+6.5
		Systolic	Sleep	+6.2
	Wave 2 (n = 195)	Systolic	Work	+6.4
		Diastolic	Work	+5.0
		Systolic	Home	+6.9
		Diastolic	Home	+4.9
		Systolic	Sleep	+5.0
Longitudinal results	Waves 1 and 2 (n = 195)			
a) Repeated exposure to job strain at times 1 and 2 (vs. those not exposed at times 1 and 2)		Systolic	Work	+11.1
		Diastolic	Work	+9.1
		Systolic	Home	+11.1
		Diastolic	Home	+7.3
		Systolic	Sleep	+10.8
b) Change in exposure[3] (job strain time 1 to no strain at time 2)		Systolic	Work	–5.3
		Diastolic	Work	–3.2
		Systolic	Home	–4.7
		Diastolic	Home	–3.3

All findings presented were statistically significant at a level of at least $p < 0.05$.

[1] Comparing those exposed to job strain (psychological work demands > sample median and job decision latitude < sample median) to the rest of the sample.

[2] Wave 1 cross-sectional analysis was adjusted for age, race, education, BMI, smoking, job physical exertion, urine sodium, type A behavior, work site, alcohol consumption. Wave 2 cross-sectional analysis and the longitudinal analyses were adjusted for age, race, BMI, smoking, alcohol. The other covariates for which adjustment had been made at time 1 were consistently nonsignificant across time and location for both SBP and BP, and were therefore excluded from these latter analyses.

[3] No significant findings were obtained in the group without exposure to job strain at time 1 and with exposure at time 2 at work, at home, nor during sleep.

of exposure to job strain plus high family load: adjusted mean work AmBP was +11/+6.3 mm Hg among those exposed to both of these, compared to those exposed to neither. This differential persisted during the evening hours at a similar magnitude.

Plausibility of Job Strain as a Causal Factor in Hypertension and Population Attributable Risk Estimates. EH is a chronic disease process characterized by persistently elevated (not just acutely elevated) BP, without secondary causes. While the precise mechanisms leading from an acute rise in BP to chronic EH are not fully delineated, the pattern of AmBP findings associated with exposure to job strain seems generally consistent with our present understanding of the phases. As described above, these include adrenergic stimulation in the early stages, and later development of structural changes, eventually leading to fixed hypertension. There is also empirical data linking exposure to job strain with increased LVMI [50]. For an in-depth discussion of these mechanisms as they relate to occupational stressors, see Schwartz et al. [51]. Schwartz et al. [52] note that for work stress to contribute to a tonic elevation in BP, 'the blood pressure of the exposed individuals would have to be elevated not only in the presence of a stressor but also during rest'. Thus, the focus should be on 'exposure to chronic low- or moderate-grade stress rather than on discrete events that are widely acknowledged to produce brief spikes in the blood pressure profile' [p. 299]. Subsequently we note that 'the large, consistent body of data on AmBP and exposure to job strain indicates that these elevations are indeed persistent. They occur not only at work, but also at home and, in some studies, during sleep. Finally, and probably most compelling, are the data indicating that there may be a cumulative effect of chronic exposure to job strain on AmBP' [51, p. 129].

In summary, there is convincing evidence that exposure to job strain is causally related to the development of EH. The association between job strain and elevated AmBP is strong and consistent, with an important body of corroborative longitudinal data and indications of a dose-response relationship [45], and the relationship is biologically plausible. Job strain can be seen as a key paradigm of 20th century working life, with its emphasis on mass production and loss of the autonomy characteristic of craft-work and other earlier modes of labor activity. (See e. g. Karasek and Theorell [53] for an in-depth discussion of this topic). As such, the causal relationship between job strain and EH is plausible in the broader historical and social context, as well.

The population attributable risk (PAR%)[1] of hypertension can be calculated using data from the Work Site Blood Pressure Study [37, 50]. With an exposure rate to job strain of 20% and an odds ratio of 2.7 between job strain and hypertension, then 25.4% of hypertension among working men in New York City could be attributed to job strain. This estimate must be taken cautiously, since PAR% is usually calculated for fairly uncommon outcomes. On

[1] PAR% = Pe (RR-1)/1 + Pe (RR-1), where Pe is the exposure rate in the population as a whole, and RR is the risk ratio. OR may be substituted for RR.

the other hand, this PAR% calculation uses a somewhat conservative estimate of risk, as it is based upon current exposure status, a likely underestimate of lifetime exposure to job strain. Bearing these caveats in mind, the PAR% calculation suggests that about 25% of cases of hypertension among working men could be prevented by eliminating exposure to job strain.

An Occupational Cardiologic Approach to the Workplace and Hypertension

We have recently argued that the knowledge about the role played by the workplace in the development of hypertension and of cardiovascular disease needs to be brought into the realm of clinical practice. In particular, guidelines are needed to assess and manage patients exposed to cardionoxious factors in the work environment [54]. In the forthcoming, we further develop this 'occupational cardiologic' approach as it specifically relates to work-related essential hypertension.

Job Strain Exposure Status: An Important Consideration for Improving the Positive Predictive Value of CCBP among Working Subjects. In table 1, we presented data on the diagnostic classification of hypertension using workplace AmBP versus casual BP measures taken at the work site, from a sample of working men participating in the Work Site Blood Pressure Study [27]. It was found therein that a total of 27 of the 267 men were in the white-coat hypertension category (elevated clinic BP and normal work AmBP) and that 36 showed occult hypertension during work (normal clinic BP and elevated work AmBP).

Given the etiologic importance of job strain in relation to hypertension, the question arises as to whether stratifying subjects on the basis of job strain exposure status could help improve the diagnostic utility of casual BP readings. In table 3, it is seen that among the 59 men with self-reported exposure to job strain, only 3 had white-coat hypertension, and the positive predictive value of CCBP was high (87%). In contrast, among the 208 men who were classified as nonexposed to job strain, there were 24 with white-coat hypertension and a positive predictive value of CCBP of only 61.9%. Thus, stratifying by job strain exposure status helped define the group in whom white-coat hypertension could be expected: those who *are not* in the high job strain group.

Stratifying by job strain was not as informative with respect to occult workplace hypertension. There were 10 men with occult workplace hypertension in the group exposed to job strain, with a negative predictive value of 72.2%. Twenty-six men with occult workplace hypertension were in the group unexposed to job strain, such that the negative predictive value was only

Table 3. Diagnostic classification of hypertension using casual versus ambulatory BP stratifying by current self-reported job strain exposure status[1]

Currently exposed to job strain by self-report using median cutpoints

Casual DBP taken at the work site clinic	Work site ambulatory DBP		
	> 85 mm Hg (n)	≤ 85 mm Hg (n)	Total (n)
> 85 mm Hg	20	3	23
≤ 85 mm Hg	10	26	36
Total	30	29	59

Positive predictive value of casual DBP > 85 mm Hg taken at the work site clinic: 20/23=87%; negative predictive value of casual DBP ≤ 85 mm Hg taken at the work site clinic: 26/36 = 72.2%.

Not currently exposed to job strain by self-report using median cutpoints

Casual DBP taken at the work site clinic	Work site ambulatory DBP		
	> 85 mm Hg (n)	≤ 85 mm Hg (n)	Total (n)
> 85 mm Hg	39	24	63
≤ 85 mm Hg	26	119	145
Total	65	143	208

Positive predictive value of casual DBP > 85 mm Hg taken at the work site clinic: 39/63= 61.9%; negative predictive value of casual DBP ≤ 85 mm Hg taken at the work site clinic: 119/145 = 82.1%.

[1] In a sample of working men from the Work Site Blood Pressure Study [27] comparing those exposed to job strain (psychological work demands > sample median and job decision latitude < sample median) to the rest of the sample.

slightly improved (82.1%). This relatively high percentage of occult workplace hypertension among those unexposed to job strain could be due to other job stressors associated with elevated BP, as explored in the next section.

Other Workplace Factors Affecting BP

While the empirical evidence of an etiologic link between exposure to job strain and risk of hypertension is the most extensive and convincing, and stratification by job strain status is shown to improve the positive predictive value of CCBP, other workplace factors may also give rise to BP elevations and may increase the risk of hypertension [51]. Taking these factors into account may be of further help in diagnosing occult workplace hypertension. For example, exposure to high effort and low rewards at work is demon-

strated to predict the combined presence of hypertension and hyperlipidemia [55–56]. This combination suggests the possibility of increased risk of the cardiovascular metabolic syndrome or insulin resistance syndrome. Recently, a study using AmBP revealed elevated work and leisure time SBP associated with exposure to effort-reward imbalance [57]. There are data implicating long work hours with elevations in casual SBP [58] and ambulatory SBP and DBP [59], and shift work with longitudinal risk of hypertension [60]. Yamasaki et al. [61] found that evening/night shift workers exhibited less nocturnal dipping than those who worked day shifts. As mentioned earlier, lack of nocturnal dipping is strongly associated with increased left ventricular mass [32]. While not entirely consistent, the bulk of the empirical evidence suggests that chronic exposure to noise is associated with an elevated risk of hypertension [51]. For other physical factors, cold, heavy lifting and glare, there are physiologic data showing acute BP increases with exposure, but not epidemiologic evidence. Chemicals that may be pressor agents include lead and arsenic [51]. More research is needed to provide quantitative data on the relative contribution of these factors alone and in combination to EH.

Finally, several occupational groups show an increased risk of hypertension. The data are most convincing for professional drivers [62–64], but there is also evidence for air traffic controllers [65] and sea pilots [66]. These jobs entail primarily threat-avoidant vigilant work, with a high total occupational stress burden [67]. Cumulative exposure to this burden is a significant independent predictor of hypertension among urban transit operators (UTO). In San Francisco among UTO aged 45–54 with >20 years on the job, 52.2% had hypertension, defined as CCBP > 140/90 mm Hg. In comparison, the prevalence of hypertension was 42.9% among those of that age strata who had been on the job <10 years, and 48.8% among those with 10–20 years on the job. Prior to employment as an urban transport operator, the prevalence of hypertension among a group of the same age was 36.7%. Using a more stringent definition of hypertension (CCBP > 160/95 mm Hg), the effect of cumulative exposure to UTO work showed an even higher level of statistical significance in this age group [64]. In an AmBP study among 30 male urban mass transit operators with at least 5 years on the job, and with normal CCBP (<140/90 mm Hg), work time AmBP was significantly greater among the UTO, compared to 20 matched clerical workers, especially in the early afternoon (effect size = +7/+5 mm Hg). Several drivers had persistently elevated AmBP (never <140/90 mm Hg) during the entire 24 h. While driving the afternoon rush hour as a split shift, the UTOs' mean DBP was 89.4 mm Hg [68]. These AmBP data, albeit on a fairly small sample, suggest the need for a heightened index of suspicion of occult workplace hypertension among UTO. The evidence that prolonged exposure to this work is associated with progres-

sively greater risk of persistent hypertension, renders this conclusion not only plausible, but also of urgent clinical priority.

A Preliminary Algorithm for an Occupational Cardiologic Work-Up of Suspected Hypertension

Particularly in the current climate of managed care and cost containment, nonselective use of ambulatory blood pressure monitoring is clearly not a realistic option. Given the likely magnitude of occult workplace hypertension, more widely applicable methods that are also diagnostically helpful for this specific purpose are needed.

Alternatives to Ambulatory Monitoring to Evaluate Occult Workplace Hypertension. We have developed a protocol for obtaining work site point estimates of BP, as a cost-effective and feasible alternative to AmBP. The key advantage of this proposed method, in contrast to CCBP, is that the measurements are taken directly during work, with minimum interruption of ongoing activity. Briefly, a trained observer measures the worker's BP while at work according to a standardized procedure aimed at obtaining a best point estimate [21]. Specific instructions in the protocol are aimed at diminishing the effect of the observer on the participant's BP. Ideally, another observer would independently and simultaneously be assessing the work environment. The reliability and validity of this new method is now being tested, with the aim of providing a suitable way to screen large numbers of people at the workplace. This new modality for the diagnosis of work-related hypertension is incorporated into an algorithm (table 4), whose starting point is the patient's casual clinic BP levels viewed in conjunction with his or her exposure to job strain and other potential work-related pressor agents.

Self-monitoring of BP at work could be another, albeit less desirable, alternative. As stated by Pickering [4], currently available electronic devices are often satisfactory with respect to reliability. He recommends forearm measurements for work site evaluations. Those models requiring that the patient write down the readings, can be inconvenient and possibly vulnerable to misreporting [4]. Notwithstanding these deficiencies, self-measurement could provide a means of obtaining BP data, if no other option were feasible. There are some newer devices that record multiple BP readings automatically, which may help solve some of these problems. Self-triggering and self-measurement devices may sometimes be useful for educational purposes, to provide the individual with immediate insight into his or her BP levels in a given situation.

In table 4, we have presented a series of preliminary recommendations for initial evaluation of CCBP findings among working people, according to their exposure to job strain and to other potential work-related pressors.

Table 4. Preliminary recommendations for initial evaluation of CCBP findings in working persons, based upon exposure to job strain and other potential work-related pressors[1]

CCBP	Job strain	Other workplace pressors	Preliminary recommendations for initial evaluation
Elevated	+	+	AmBP to assess severity and pattern of elevated BP
Elevated	+	–	AmBP to assess severity and pattern of elevated BP
Elevated	–	+	AmBP to assess severity and pattern of elevated BP (strive to coincide with time of exposure(s))
Elevated	–	–	Repeated clinic BP
Normal	+	+	Rule out 'occult workplace hypertension' with work site point estimate of BP or AmBP (whichever is more feasible)
Normal	+	–	Rule out 'occult workplace hypertension' with work-site point estimate of BP or AmBP (whichever is more feasible)
Normal	–	+	Rule out 'occult workplace hypertension' with work site point estimate of BP or AmBP (whichever is more feasible – strive to coincide with time of exposure(s))
Normal	–	–	Follow routinely (annual BP check-up)

[1] *Other potential work-related pressors.* There is direct epidemiologic as well as physiologic evidence for: effort-reward imbalance, long work hours, shift work, noise, lead, arsenic; high-risk occupations: urban transit operators, truck drivers, air traffic controllers, sea pilots.

There is physiologic and indirect epidemiologic evidence for: threat-avoidant vigilant activity.

There is only physiologic evidence for: cold, heavy lifting, glare exposure.

See Schwartz et al. [51] for more details concerning the evidence on each of these factors in relation to BP elevations and hypertension. Quantitative data are still needed as to the relative contribution of each of these factors alone and in combination to work-related hypertension.

This algorithm is designed to improve diagnostic yield, based upon our current knowledge concerning the workplace and hypertension.

It was shown in table 3 that most people with elevated casual BP who are exposed to job strain, also have elevated work AmBP. We recommend ambulatory monitoring for these people, to assess the severity of workplace hypertension, as this is prognostically important information that could im-

pact upon clinical decision-making. Another possibility exists that the patient with elevated CCBP is not in the high job strain group, but is exposed to other potential pressors at the workplace. Using similar logic, we would argue that such patients also undergo ambulatory monitoring. For those pressor agents of an intermittent nature, attempts should be made to schedule the monitoring session to coincide with the actual exposure, e. g. during night shift work or when noise levels are high, etc. Efforts should be made to record AmBP during the conditions of a typical work shift. By providing insight into the severity as well as the pattern of BP over 24 h, ambulatory monitoring could be helpful for optimizing the medication regimen, and might also indicate feasible ways of minimizing exposure to work pressors for a given individual, e. g. adjustment of work schedule.

Among those working people with elevated CCBP who are not exposed to major job-related pressors, a series of clinic BP readings taken at several visits should give a reasonable estimate of usual BP. This should help rule out white-coat hypertension, which, as suggested in table 3, is more likely among those in this category.

This preliminary algorithm also provides a strategy for detection of occult workplace hypertension. Recall that while many of the men with occult workplace hypertension were exposed to job strain, there was a substantial percentage who were not classified as such. Also, recall that UTO with normal clinic BP showed elevated mean levels of work AmBP. Other potential workplace pressors need to be examined in this light. Given the possible clinical importance of occult workplace hypertension, as discussed in relation to the data of Liu et al. [39], we recommend that patients who have normal clinic BP, but who are exposed to job strain and/or other major potential workplace pressors, be further evaluated at the workplace. Based upon feasibility, this could either be via work site point estimates or by ambulatory monitoring. Those patients with normal clinic BP and without exposure to job strain or other major workplace pressors, could be followed routinely (annual BP check-up).

It should be emphasized that these recommendations are still of a preliminary nature. In the analyses performed here, we have relied upon fairly arbitrary cutpoints to define exposure to job strain (the 20% quadrant term) and for elevated BP (>85mm Hg diastolic). Further analysis is planned using a range of definitions both for job strain and for BP elevation. Similar types of analysis are needed for other job-related pressors. This knowledge could help refine our approach to the diagnosis of workplace hypertension.

A Public Health Approach to the Workplace and Hypertension: The Need for Surveillance

Given the high prevalence of hypertension and our emerging insights concerning workplace hypertension, it is clear that the individual clinician will quickly be overwhelmed if he or she *alone* attempts to fulfill the above recommendations. There is a very large percentage of working people for whom assessment of BP at the workplace may be warranted. A public health approach is needed, in which job strain and other workplace factors that can lead to BP elevation are systematically evaluated on a large scale. Consideration should be made not only of current status, but also of length of exposure to these factors.

In the Tokyo Declaration [69], experts concerned with work-related health hazards in three major postindustrial societies (the USA, Europe and Japan) have suggested a series of policy initiatives that address this issue. They call 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. They recommend that workplaces assess both workplace stressors and health outcomes known to result from such exposures … on an annual basis' [p. 5]. Based upon the approach outlined in the Tokyo Declaration, we further suggested that workplace screening be used to obtain prevalence data on cardionoxious exposures such as job strain, together with data on relevant cardiovascular outcomes, especially elevated BP. We noted that work site point estimates of BP would be particularly useful, as an inexpensive and relatively simple surveillance method, with ambulatory monitoring performed when feasible. We also emphasized the need for appropriate precautions to protect employee confidentiality and iatrogenic disqualification [54].

The information gathered from work site BP surveillance could be helpful to the clinician in risk assessment of working people. The clinician could also play an active public health role in this process, by identifying clusters of workplace-related hypertension as potential 'occupational sentinel health events'. In other words, by spotting workplaces in which there is an unusually high prevalence of hypertension, especially among younger workers, the clinician could help target sites for priority surveillance [70].

The feasibility and practical utility of this pro-active approach has been demonstrated. Indeed, it was clinicians' observations during systematic cardiovascular examinations of urban transport operators that helped lay the basis for investigations on an international scale, demonstrating the untowardly high prevalence of hypertension among this occupational group [70].

Furthermore, by suggesting that amelioration of conditions, such as job strain, could have a substantial impact upon work site blood pressure [47], clinicians have heralded the need for intervention strategies that focus upon this important risk factor in EH.

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Peter L. Schnall MD, MPH, Center for Social Epidemiology, 1528 6th St., Suite 202,
Santa Monica, CA 90401 (USA)
Tel. +1 310 319 6595, Fax +1 310 319 6597, E-Mail pschnall@workhealth.org

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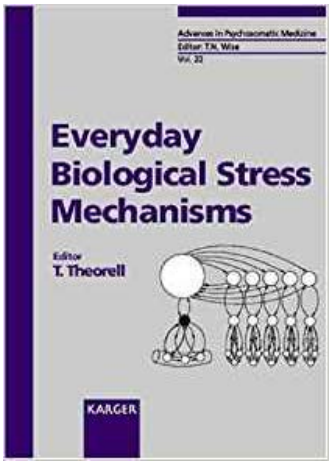

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