ASSESSMENT OF THE CARDIOVASCULAR SYSTEM AT THE WORKPLACE

OBTAINING A CVD HISTORY: OBSTACLES AND CHALLENGES

by Peter Schnall, MD, and Karen Belkić, MD, PhD

In the process of ascertaining the cause of an illness (i.e., making the diagnosis) the medical history plays a key role, as the information obtained from the patient informs the analytic process and shapes subsequent questioning. This exchange leads to an ongoing iterative process which, hopefully, results in an intervention intended to correct the problem. The key to the success of this endeavor for the physician is to obtain complete and accurate information from the patient(s) regarding symptoms and other related historical information such as past history of exposures.[45]

There are unfortunately, a number of reasons, many of which are not under the control of the clinician, as to why this is easier said than done. The person being interviewed may be unaware of his or her condition, or may feel anxious over the symptoms or illness and frightened of the possible diagnosis with its implications for future health. Denial may dominate the person's responses if there is a general fear of life-threatening illness, or if there are other possible negative consequences of a diagnosis. All of these factors may interfere with the doctor-patient relationship.

The health professional must be aware that employees may fear losing their jobs under certain circumstances as the result of a visit to a physician. This obstacle is of greatest concern in the work setting. In fact, doctors often function as "gatekeepers," especially in their role of evaluating an individual's fitness for work. Working people usually are well aware of this function and may act in what they perceive to be "their own best interest." The individual's need to continue working may hamper a frank admission of symptoms or conditions which could compromise employability. The physician needs to be acutely aware of the complex social relationships among the patient, clinician, and employer, as well as of societal expectations. The perception that the clinician is acting in the interests of the company can have a chilling effect on the patient. Ethical issues involving confidentiality are raised by this gatekeeping function[141] (see Chapters 8, 9, and 10).

Limitation on clinician's time, especially in primary care settings, represents yet another impediment to obtaining an adequate history. Accurate history-taking requires that the clinician first and foremost establish a rapport with the patient. The interview must be conducted in an environment that is conducive to developing a trusting relationship. The clinician must communicate an interest in the person and a willingness to listen. Time constraints undermine this entire process.

A mix of open-ended and directed questions are needed to get a complete history. The physician's need to collect both qualitative and quantitative data is somewhat akin to the work of an anthropologist. However, too many open-ended questions may waste time and increase the physician's sense of urgency, which can be counterproductive. Questions that are too directed may miss key symptoms, or give the patient the impression that the physician is not truly interested. The latter often is discovered when the physician learns of a key symptom late in the diagnostic process, and the patient comments, "Well, you didn't ask me."

Issues Specific to CVD History-Taking in the Workplace

CV symptoms may be quite prominent and direct the physician quickly in the direction of a relevant diagnosis, e.g., complaint of angina in patients with CAD. There are, of course, standard questions for use by the physician in pursuing a diagnosis of a specific form of CVD. (Chapter 8 outlines an approach to taking a clinical occupational work history as it relates to the CV system.)

However, under- and over-reporting of symptoms is frequent in CV illness—the issue of false positives and false negatives. Employees with chest pain may be quick to conclude they have cardiac disease, requiring the physician to rule out specific heart problems (false positives). As stated by Jennison: "Exaggerated reporting of symptoms might be a way of protesting against a hazardous environment. . . . Under-reporting, which is probably more common, may result from a lack of awareness of symptoms, denial, or fear of being replaced or dismissed from the workplace."[61] The clinician should be alert to the possibility that denial of cardiac symptoms may be especially likely among those very persons whose occupations show high cardiac risk. This denial has been reported, for example, among professional drivers.[12]

In addition, asymptomatic CVD (false negatives) is widespread and, with more sensitive and improved diagnostic techniques, increasingly recognized. Disease processes such as hypertension and myocardial ischemia frequently are asymptomatic or have very subtle symptoms. In high-risk occupations, the clinician should inquire carefully for nonspecific symptoms and maintain a high level of vigilance for complaints of fatigue, malaise, and sleep disturbances. Furthermore, while job strain is implicated in the etiology of hypertension in many studies, subjects frequently do not report psychological or physiologic symptoms even while they report jobs characterized by high demands and little control.[130] Another potential piece of information from the history that should alert the clinician is a job change to avoid work stressors; this could be a clue to the presence of subclinical disease. A notable example is the high cardiac risk seen among persons who have recently switched out of night shiftwork.[95]

The History-Taking and Public Health

An awareness that specific occupations may contribute to the development of certain CV illness can be helpful in both early detection and prevention of serious illness. The presence of symptoms among substantial numbers of people at a workplace may be the first clue to lead the astute clinician to analyze and identify clusters of CV illness related to that workplace (see Chapter 10). Recognition of such clusters of symptoms should motivate the clinician to screen the worksite for the presence of cardionoxious factors, such as job strain. Detection of specific workplace stressors as well as physiologic abnormalities, such as increased prevalence of high blood pressure, aids in the diagnosis of potentially unhealthy worksites.

BLOOD PRESSURE MEASUREMENT: CASUAL, SELF-MEASURED, AND AMBULATORY MONITORING

by Thomas G. Pickering, MD, DPhil

High blood pressure (BP) is one of the major risk factors for CVD, and also contributes to the morbidity of renal disease and diabetes. The relationship between BP and disease is continuous, so that even small elevations can be considered potentially harmful. While it is accepted that the average level of BP over time (often referred to as the true BP) is responsible for much of the damage, it is likely, but unproven, that transient increases of BP also contribute. In addition, there is a pronounced diurnal rhythm of pressure, which is increasingly recognized to be of pathological significance.

Until relatively recently, the vast majority of BP measurements were made using a mercury or aneroid sphygmomanometer in a laboratory or clinic setting, which limited both the number of readings that could be taken and their ecological validity. Thus, measurements made at the worksite were few and far between. The introduction of the techniques of ambulatory monitoring and self-measurement with digital devices has completely revolutionized our ability to measure BP at the workplace and in other settings while subjects go about their normal daily activities. Research using these techniques has shown that BP is highest for many people while they are at work.

Basic Techniques of BP Measurement

THE AUSCULTATORY METHOD

Conventionally, the gold standard in clinical practice has been measurements made with the Korotkoff sound technique by a physician using a mercury sphygmomanometer. However, although hypertension can be identified only by measuring the BP, this popular method is notoriously unreliable. There are three main reasons for this: inaccuracies in the methods, some of which are avoidable; the inherent variability of BP; and the tendency for BP to increase in the presence of a physician (the so-called white coat effect). The Korotkoff sound method tends to give values for systolic pressure (SP) that are lower than the intra-arterial pressure, and diastolic values that are higher, but there is no obvious superiority of phase 5 over phase 4.[22] The official recommendations of organizations such as the American Heart Association is to use the fifth phase.[113]

Some of the major causes of a discrepancy between the conventional clinical measurement of BP and the true BP are listed in Table 1. A number of factors may lead to inaccuracies with the Korotkoff sound technique:

TABLE 1. Comparison of Features of Three Techniques for BP Measurement

Features	Monitor Type *Conventional*	*Electronic (Self-)*	*Ambulatory*
Use for screening	Yes	Possibly	No
Use for BP changes	Limited	Days/weeks	24 hours
No. of subjects	Large	Small	Small
No. of readings	Small	Large	Large
Cost	Low	Low-moderate	High

Cuff Size. The size of the cuff relative to the diameter of the arm is critical. A typical mistake is to use a cuff that is too small, resulting in an overestimation of the pressure.[93] In general, error can be reduced by using a large, adult-sized cuff for all except the skinniest arms. The British Hypertension Society recommends that if the arm circumference exceeds 33 cm, a large adult cuff should be used (width 12.5–13 cm, length 35 cm).[114]

Arm Position. BP measurements also are influenced by the position of the arm.[98] There is a progressive increase in the pressure of about 5–6 mmHg as the arm is moved down from the horizontal to vertical position.

Observer Error and Observer Bias. These are important sources of error when conventional sphygmomanometers are used. Differences in auditory acuity between observers may lead to consistent errors, and digit preference is common, with most observers recording a disproportionate number of readings ending in 5 or 0.[111] The average BP values recorded by trained individual observers have been found to vary by as much as 5–10 mmHg.[44] 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.[94]

Rate of Cuff Inflation and Deflation. The rate of inflation has no significant effect on BP,[72] but very slow rates of deflation (2 mmHg/second or less) diminish the intensity of the Korotkoff sounds, resulting in slightly higher diastolic pressures (DPs). The generally recommended deflation rate is 2–3 mmHg/second.

Technical Sources of Error. There usually are fewer technical errors when a mercury column is used than when a semiautomatic method is applied. The column should be positioned approximately at the level of the heart; the mercury should read zero when no pressure is applied; and the mercury should fall freely when the pressure is reduced.

The Oscillometric Technique

This technique was first demonstrated by Marey in 1876,[89] and it was shown subsequently that when the oscillations of pressure in a sphygmomanometer cuff are recorded during gradual deflation, the point of maximal oscillation corresponds to the mean intra-arterial pressure.[92] The oscillations begin approximately at SP and continue below DP, so that both can be estimated only indirectly, according to some empirically derived algorithm. One advantage of the method is that placement of the cuff is not critical, because a transducer is not used. Other potential advantages are that it is less susceptible to external noise (but not to low-frequency mechanical vibration), and the cuff can be removed and replaced by the patient, for example, to take a shower. The main disadvantage is that such recorders do not work well during physical activity, when there may be considerable movement artifact.

The oscillometric technique has been used successfully in ambulatory (am) BP monitors (such as the U.S. Spacelab recorders) and home monitors. Note that different brands of oscillometric recorders use different algorithms, and there is no generic oscillometric technique. However, comparisons of several different commercial models with intra-arterial and Korotkoff sound measurements have shown generally good agreement.[20,26]

Devices Used for BP Measurement

MERCURY AND ANEROID SPHYGMOMANOMETERS

Many studies of BP in the workplace require screening of subjects, and for this purpose mercury sphygmomanometers remain the gold standard. Aneroid devices commonly are used as a substitute, but they are not as accurate: in one survey, 30% of aneroid dials had errors greater than 4 mmHg.[25] The reliability of clinic pressure for estimating true BP can be improved by increasing the number of readings taken per visit and the number of visits, and by eliminating sources of error, such as digit preference.

ELECTRONIC SELF-MONITORS

In the past few years automatic electronic devices have become increasingly popular. Early versions were mostly inaccurate,[118,151] but currently available ones are often satisfactory.[48,59] Unfortunately, only a few have been subjected to proper validation tests such as the AAMI and BHS protocols.[106] The advantages of electronic monitors have begun to be appreciated by epidemiologists, who have always been greatly concerned about the accuracy of clinical BP measurement.[30] Cooper, et al. have asserted that ease of use and relative insensitivity to who is taking the reading can outweigh any inherent inaccuracy compared to the traditional sphygmomanometer method.[30] Electronic devices are now available that take BP from the upper arm, wrist, or finger. While more distal sites may be more convenient, measurement of BP from the arm (brachial artery) always has been the standard method, and is likely to remain so for the foreseeable future. Neither wrist nor finger monitors can be recommended for worksite studies.

The standard type of monitor for home use is an oscillometric device that records pressure from the brachial artery. Oscillometric monitors are easy to use, since cuff placement is not as critical as with devices that use a Korotkoff sound microphone. One of the limitations of earlier models of electronic monitors was that they required the subject to write down the readings, which is not only inconvenient for worksite studies, but also allows the potential for misreporting.[96] Some devices have a printer attached, which at least avoids observer bias. Others have a memory that can store several hundred readings, from which the data can be downloaded (for example, into the physician's computer, as in the Omron IC).

24-HOUR AMBULATORY MONITORS

First developed more than 30 years ago, amBP monitoring is only now beginning to find acceptance as a clinically useful technique, but it is widely used in research studies, particularly when BP in the natural environment is of interest. Recent technological advances have led to the introduction of monitors that are small, relatively quiet, and take up to 100 readings over 24 hours, while subjects go about their normal activities. They are reasonably accurate while the subject is at rest, but less so during physical activity.

When last systematically surveyed (in 1995) there were 43 different devices on the market. Only 18 had been validated according to the AAMI or BHS criteria, and of these only 9 satisfied the criteria for accuracy.[107] Ambulatory monitors can, in theory, provide information about the three main measures of BP—the average level, the diurnal variation, and short-term variability. Because the currently available monitors take readings intermittently rather than continually and are unreliable during exercise, they give a crude estimate of the short-term variability.

Determining Which Measures Are Clinically Important

In clinical practice, a patient's BP typically is characterized by a single value of the SP and DP, to denote the average or true BP level. Such readings normally are taken in a clinic setting, but there is extensive evidence that in hypertensive patients clinic pressures are consistently higher than the average 24-hour pressures recorded with ambulatory monitors,[80] and in some cases may be within the normal range, leading to a diagnosis of white coat hypertension. Given that there is a discrepancy between the clinic and ambulatory pressures, it is reasonable to suppose that the prediction of risk will be different. Cross-sectional studies have shown that amBP predicts the extent of CV damage better than clinic pressures,[37] and there are now several prospective studies showing that amBP is a better predictor of risk than clinic pressure.

The diurnal rhythm of BP is pronounced, with a decrease of 10–20 mmHg during sleep and a prompt increase on waking and getting up in the morning. The highest BP levels usually occur between 6 AM and noon, which is also the time at which the prevalence of many CV morbid events tends to be highest.[116] The pattern of BP during the day is, to a large extent, dependent on the pattern of activity, with pressures tending to be higher during the hours of work and lower while at home.[29] In hypertensive patients, the diurnal BP profile is reset at a higher level of pressure, with preservation of the normal pattern in the majority. The short-term BP variability is increased when expressed in absolute terms (mmHg), but the percentage changes are no different. Thus, hypertension can be regarded as a disturbance of the **set point** or **tonic level** of BP with normal short-term regulation. The normal diurnal rhythm of BP is disturbed in some individuals, with loss of the normal nocturnal fall of pressure. This disturbance has been observed in a variety of medical conditions, including renal disease and diabetes, but also occurs as a normal variant, particularly among African-Americans. Subjects whose pressure remains high at night ("nondippers") may have more target-organ damage than those who show the normal pattern ("dippers"), and there is evidence that women nondippers are at greater risk of CV morbidity than dippers,[152] but these findings are not sufficiently well established to be applied to routine clinical practice.

Even less information is available for defining the clinical significance of short-term BP variability, although it may be a risk factor for CV morbidity.

Practical and Technical Issues for Workplace Measurement

The choice of which technique should be used for measuring BP in worksite studies is dictated largely by the study design. Some of the advantages and limitations of the different types of monitors are shown in Table 1. The conventional sphygmomanometer is still the preferred method for screening large numbers of subjects, although electronic monitors are beginning to be used. The disadvantage of the sphygmomanometer is that it gives a restricted view of what happens to BP in different situations; thus, readings may or may not be representative of the subject's true BP.

Electronic (home) monitors are advantageous because they can monitor BP changes over long periods of time. To date, for unknown reasons, they have been little used in research on occupational stress. At this author's facility, electronic monitors were employed to evaluate BP changes associated with the approach of major work deadlines; subjects took their BP daily at the same time of day for many weeks.

Ambulatory monitoring can be performed at the worksite, and is normally done for 24 hours to provide a profile covering work and leisure hours. In most

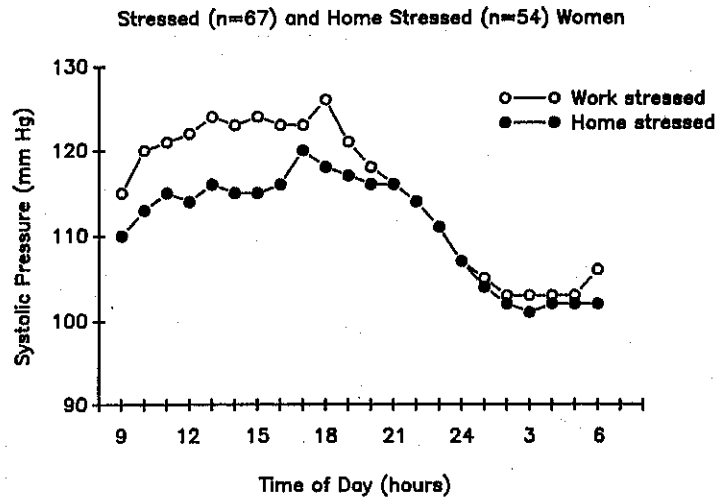

FIGURE 1. Comparison of the blood pressure profiles recorded over 24 hours in two groups of employed women: "work stressed" and "home stressed." (From James GD, Moucha OP, Pickering TG: The normal hourly variation of blood pressure in women: Average patterns and the effects of work stress. J Hum Hypertens 5:505–509, 1991; with permission.)

people, BP is higher at work than at home (Fig. 1); diaries can help determine to what extent this is due to physical activity as opposed to mental stress. It is possible to determine to what extent the effects of work carry over to the evening hours, for example.

There are some special considerations related to worksite measurement:

1. **Effects of posture.** For routine clinical measurement it usually is recommended that the patient be sitting.[113] However, ambulatory monitoring studies of subjects at work may include measurements in the standing position. The BP changes associated with standing are usually modest—little change in systolic pressure and a slight increase in diastolic. However, analyses based on diary entries comparing sitting and standing BP have shown much bigger changes.[49] The most likely explanation is that these differences are due to the activities associated with the different postures, rather than to the postures themselves.

2. **Effects of physical activity.** During dynamic exercise there is an increase of SP but not DP, while isometric exercise increases both. Both types of activity may occur during work, but BP measurement becomes technically difficult even with ambulatory monitors, which may register artifactual readings. Monitoring people with sedentary jobs is more reliable.

3. **Effects of noise and vibration.** The oscillometric method of BP measurement, incorporated by most ambulatory monitors, is relatively immune to the effects of noise, but is affected by mechanical vibration, e.g., driving a taxi.

4. **The use of diaries.** When ambulatory monitoring is used to evaluate BP changes in the workplace, subjects should keep diaries describing their location, postures, moods, and activities, with entries made at the time of each BP reading. This documentation enables correlations to be made between work-related activities and BP.[133] A compromise on quantity of information is advisable, however, since very elaborate diaries tend to result in poor compliance.

Job Strain

The Job Strain Model has been used mainly to study the effects of job strain on the development of CHD, but it also provides a good example of the use of workplace BP monitoring to study the effects of occupational stress on BP.[128] In the Cornell Worksite BP Study, men in high-strain jobs were found to have higher amBP than men in less stressful jobs.[131] Interestingly, this elevation of pressure was seen not only during working hours, but also while at home and during sleep. Furthermore, the association between job strain and BP could not be accounted for by any of the following factors known to influence BP: sodium intake, body mass, race, alcohol intake, education level, smoking, and level of physical activity.

Two other interactive effects were observed: (1) the highest BPs were observed in subjects who were in high-strain jobs and drank regularly; (2) the effects of job strain on BP were much greater in older than in younger subjects.

In a recent analysis of 3-year followup data from the worksite study, a cross-sectional association between job strain and amBP was found to closely parallel the association observed at baseline.[129] Furthermore, longitudinal analyses indicated that after controlling for baseline BP, the followup systolic and diastolic amBPs at work of those classified as having high-strain jobs at both baseline and followup were significantly greater than the BPs of those classified as having low-strain jobs at both baseline and followup. Though not statistically significant, those who switched from low-strain to high-strain jobs showed an increase in amBP, while those who switched from high strain to low strain showed a decrease. These findings have received support from other ambulatory monitoring studies.[85,146,150]

Exposure to job strain also is associated with increased left ventricular mass (LVM),[128] which is consistent with an earlier finding that LVM correlates more closely with the BP measured at work than at other times.[38]

Shift Work

Studies of shift workers also have shown a close linkage between activity and BP. Several studies using ambulatory monitoring have found that BP is highest during the working hours in people who work different shifts, whatever the shift. Furthermore, when the shift ichanges, BP rhythm immediately follows the work shift.[8,28,143] Yamasaki, et al. monitored 24-hour BP in 105 nurses who worked different shifts: day, evening, or night.[157] Awake and sleep times were evaluated from subjects' diaries. The work-time BPs were similar, but the sleep-time pressures were higher in the evening and in nightshift workers, probably because quality of sleep for nightshift workers was not as good as for dayshift workers.

AMBULATORY ELECTROCARDIOGRAPHIC MONITORING: STRESS-MEDIATED CLINICALLY RELEVANT ENDPOINTS

by Karen Belkić, MD, PhD

The impressive body of data linking high-strain jobs and other adverse occupational conditions to increased amBP during work has provided empirical corroboration for a mechanistic link between the work environment and risk of CVD.[129] Perhaps most striking is the association between salutogenic changes in the levels of psychological demands and decision latitude on the job and lowered amBP measured at work. Compared to the findings concerning amBP and the work environment, ambulatory electrocardiographic (ECG) endpoints that are clinically relevant

and affected by stress mechanisms and/or other factors in the workplace have been, for the most part, less systematically investigated.

Heart Rate Variability

DEFINITION AND MAJOR PHYSIOLOGIC DETERMINANTS

Heart rate variability (HRV) reflects the beat-to-beat oscillations in the sinus rate. The major determinant of the fluctuations between consecutive heart beats is the respiratory cycle; hence, the term respiratory sinus arrhythmia. Intrapleural pressure changes during the respiratory cycle affect venous pressures; these, in turn, activate reflex mechanisms leading to variations in beat-to-beat intervals. Chemoreceptor feedback also affects sinus rhythm.[76] These phasic, respiratory-related changes in the R-R interval are controlled by parasympathetically-mediated acetylcholine secretion. In contrast, the sympathetic nervous system is mainly responsible for the tonic control of heart rate.[14,64,76]

MEASUREMENT TECHNIQUES

HRV can be assessed in the **time domain**. An estimate of the overall HRV can be obtained by taking the standard deviation (SD) of all the normal sinus (N-N) intervals (SDNN). The long-term components of HRV can be estimated by mean SD of the averages of N-N intervals in all 5-minute segments, while the short-term components can be estimated by several techniques. The root-mean-square successive difference is preferred.[144] Time domain measurement is detailed elsewhere.[74,144]

Measurements in the **frequency domain** (power spectral analysis) quantitate the relative contribution of various frequency bands to total variance. Respiratory sinus arrhythmia has a frequency of about 0.15–0.4 Hz; this component, measured by spectral analysis, is termed the high frequency (HF) component. At baseline, it shows a significant relation to vagal tone.[42] Oscillations in heart rate of a somewhat longer periodicity (0.04–0.15 Hz); the low frequency [LF] component) have been ascribed to sympathetic outflow, possibly together with some parasympathetic modulation. The LF/HF ratio has been said by some investigators to represent sympathetic/parasympathetic balance, such that the greater this ratio, the higher the sympathetic influence and the more parasympathetic tone withdrawn.[144] However, Eckberg and others have called into question some of the latter premises.[42] Particularly controversial is how well the 0.1 Hz RR rhythm reflects sympathovagal balance. Caution must be exercised in relating these spectral parameters to the influence of the limbs of the autonomic nervous system (ANS).

The advantages and disadvantages of the various methods used for power spectral analysis, e.g., autoregression or the nonparametric Fast Fourier Transform, are thoroughly discussed elsewhere.[15,119] There is a growing appreciation for the role of nonlinear, parametric transforms to substantially improve the signal/noise ratio.[9,57,86,124] However, the eventual advantages of these latter techniques have yet to be demonstrated in biomedical investigations.[144] Overall, there is no consensus on optimal approach. However, particularly for autoregression, specific parameters such as choice of order can affect the results. Thus, comparisons among studies require attention to methodologic consistency.[15]

Regardless of the analytic method chosen, meticulous standards concerning ECG equipment, proper choice of sampling rate, and exclusion of artifact and nonsinus beats must be met.[15,144] Manual editing of the R-R data is recommended "to a very high standard, ensuring correct identification and classification of every QRS

complex."[144] HRV cannot be measured in patients with large numbers of ectopics, atrial fibrillation, or sinus node dysfunction.[17] Although there are no formal recommendations, a high percentage (> 95%) of sinus-beats is optimal for reliable HRV analysis. Details on the equipment and other technical requirements for HRV analysis can be found elsewhere.[15,144]

Baseline short-term (5-minute) measures of HRV are stable and reproducible; these should be made using frequency domain methods.[144] Time domain methods are not considered appropriate for psychophysiologic studies.[15] Long-term recordings can be made using time domain analysis (e.g., standard deviation of the normal R-R interval) over a 24-hour period; power spectral analysis also can be performed. In the latter, an ultra-low frequency component (< 0.03 Hz) also appears, and there is a lack of stationarity over lengthy periods. Berntson and colleagues suggest analyzing short epochs at "strategic points" in a 24-hour recording when using frequency domain methods.[15] It is critical to distinguish interpretations of short versus long recordings.[76,144]

Factors Affecting HRV: Covariates and Confounders

Age has a major effect on HRV. O'Brien and colleagues report that the standard deviation of the R-R intervals declines with age, both at rest and during activities such as deep breathing, Valsalva maneuvers, and standing.[108] Normally there is an increased power of the LF component with postural changes such as upright tilt, but this response is attenuated among the elderly.[110] The increased heart rate level associated with physical activity is accompanied by a decrease in HRV.[83] Conversely, respiratory sinus arrhythmia can be pronounced in trained athletes at rest, and is considered a sign of physical fitness.[76]

There also may be gender differences in HRV parameters, although they probably are of lesser importance than age differences.[62] The interaction between HRV and age seems to vary according to gender.[138] Ramaekers and colleagues found that below age 40, men had significantly greater LF power and LF/HF ratio, as well as 24-hour SDNN.[122] Stein and colleagues reported that among younger men all 24-hour time domain indexes of HRV, except those that reflect vagal modulation of heart rate, were significantly higher than among similarly aged women.[138]

Changes in respiratory patterns affect HRV.[15] Relaxation training that included deep breathing exercises was found to be associated with increased time-domain measures of HRV.[147] In contrast, abnormal respiration, for example as seen in sleep apnea, is associated with untoward alterations in HRV.[76] Irregular breathing patterns can diminish the reliability of HRV measures.[99]

Kageyama and colleagues reported that after adjusting for age, the power of the HF component during supine rest with normal breathing among 282 healthy, white-collar, Japanese male workers was significantly decreased among those who were mildly to moderately obese (BMI 21–36).[67] On that basis, the authors concluded that obesity should be considered as a covariate when possible relationships between cardiac parasympathetic activity and other environmental factors are examined.

Among healthy subjects there usually is circadian variation in HRV, with greater power in the LF component during the day and in the HF component at night. The HF component normally increases during nonREM sleep. The nadir of HRV (largest LF, smallest HF power) is in the early morning hours, which is the time of highest incidence of transient myocardial ischemia, MI, and sudden cardiac

death.[110,144] These circadian changes are attenuated or lost after acute MI and in advanced hypertension. Menstrual phase–related changes in HRV have been reported, with LF significantly greater, HF lower, and LF/HF greater during the luteal compared to the follicular phase.[125]

Myocardial dysfunction has been consistently associated with a reduction in time domain measures of HRV, while both HF and low LF components may be lost in advanced heart failure.[110,144] Absolute power of both LF and HF also is lost in autonomic neuropathy. Medications affecting the ANS also influence HRV.

CLINICAL SIGNIFICANCE

Depressed HRV has been found to powerfully predict post-MI mortality and the occurrence of life-threatening ventricular tachyarrhythmias, independently of other post-infarction predictive factors such as left ventricular ejection fraction (LVEF) and the presence of late potentials.[144] The predictive strength of the HRV index (a time domain parameter based on the width of the distribution of N-N intervals, obtained from 24-hour Holter recordings) measured 5–10 days after infarction was found to be equal to or even greater than LVEF among 385 patients who were followed for at least 5 months post-MI. For arrhythmic complications, the sensitivity of the HRV index was 75% and specificity 76%. In comparison, a LVEF < 40% showed a sensitivity of 42% and specificity of 75% for this outcome.[109]

HRV also appears to predict incident CHD. Liao and colleagues performed a population-based case-cohort study in which there were 137 patients with incident CHD and 2252 healthy patients after 3 years.[84] The HF power taken from a baseline supine, resting recording was a significant predictor of CHD, after adjusting for age, race, gender, and other cardiac risk factors (lowest quartile compared with upper three quartiles adjusted relative risk 1.72; 95% CI, 1.17–2.51). Low frequency power, HF/LF ratio, and standard deviation of RR were not significant predictors.

Time and frequency domain analyses of HRV also have been used to investigate autonomic changes associated with various stages of essential hypertension.[86] This information may help indicate mechanisms mediating risk for cardiac events among hypertensives. SNS activation, consistent with the defense reaction, appears to be predominant in the early stages of hypertension.[47,65] Possibly supporting this formulation, the power of the low frequency HRV has been reported to be elevated among patients with mild-moderate hypertension.[115] In contrast, among 40 hypertensive patients with diastolic BP consistently over 95 mmHg compared to age-matched normotensive patients, there was significantly less power of the HF component, together with a greater LF component.[50] Hypertensive patients with left ventricular hypertrophy, but without evidence of coronary artery disease, were found to show not only a 24-hour decrease in time domain–measured HRV, but also a lack of nocturnal rise in occurrence of N-N interval differences > 50 msec.[27] Reviewing these and other studies, Lombardi and Fiorentini conclude that "a reduction in heart rate variability and in particular a marked attenuation of the circadian variation of spectral indices of sympathetic and vagal modifications of heart period seem to characterize the most advanced phases of the disease."[86]

HRV AND ENVIRONMENTAL STRESSORS

A large body of laboratory investigation among healthy subjects demonstrates an association between mental workload and attenuation or disappearance of respiratory sinus arrhythmia.[21,53,69,83,87,105,123,124,126] Kalsbeek ascribed the complete suppression of respiratory sinus arrhythmia to performance at peak capacity with "no

reserve capacity left unoccupied."[69] This contention is corroborated by field studies among pilots, who during the time of landing exhibit a total loss of HRV. Among pilots learning to handle a new type of aircraft, there was a prolonged duration of attenuated HRV during the approach period, prior to touch down.[64]

Exposure to certain physical factors may be associated with stress-mediated and/or autonomic neuropathic alterations in HRV. Male tool operators with vibration white finger disease have been found to have a significantly lower power of the respiratory sinus arrhythmia components compared to healthy controls.[3] Corroborative results concerning vibration exposure and HRV have been reported.[51,52] Occupational exposure to lead has been associated with a dose-related decrease in HRV during deep breathing among 172 male workers.[145]

Assessment of HRV also provides insight into the acute and chronic effects of fatiguing work conditions, as these may relate to untoward CV outcomes. Kageyama and colleagues found that among 223 male, white-collar workers in greater Tokyo, working more than 60 hours of overtime per month was associated with short-term HRV changes while standing at rest.[68] The authors interpreted these findings to be consistent with decreased vagal and increased SNS activity.

Long work hours that include a night shift appear to be associated with profound alterations in the circadian pattern of HRV, with a nearly complete loss of HF surge over 24 hours, reminiscent of the patterns described for post-MI and advanced hypertensive patients. Kobayashi and colleagues examined these effects among 12 healthy nurses who demonstrated normal circadian HRV patterns when working the day shift only.[75] When, after having worked the full day shift, these same nurses went on to work the midnight to 8:30 AM shift, the LF/HF ratio remained at or even above usual daytime levels for all but 3 of the 24 hours. Matsuzaki and coworkers in their study of seven male factory workers reported a significant decrease in power of the HF component with day sleep after night shiftwork compared to night sleep after day work.[91] These findings are particularly intriguing in light of the possible relation between night shiftwork and CVD.

The clinical relevance of HRV renders it a potentially useful endpoint for assessing workplace interventions aimed at promoting CV health. The above-described study of Kobayashi, et al. illustrates this point.[75] These authors also examined the circadian pattern of LF/HF power when, prior to working the night shift, the nurses worked a half- rather than full-day schedule and thereby had a chance to sleep for an average of 4 hours in the late afternoon and early evening, prior to going to work. A distinct drop in LF/HF lasting about 7 hours was observed during this period, although these values were still not as low as during a normal night sleep after day shiftwork. These findings are coherent with the statement of Kristal-Boneh and colleagues that "spectral analysis of HRV may be used to predict optimal worktime under a combination of enhanced mental load and other stressors."[76] More widespread examination of HRV, as a psychophysiologic parameter with importance to cardiovascular well-being, is warranted in various work environments.

Myocardial Ischemia

The definition and physiologic determinants of myocardial ischemia, as well as a review of empirical data linking stress mechanisms and other workplace exposures to myocardial ischemia are presented in Chapter 5. Here, the focus is on ambulatory measurement techniques and the clinical significance of myocardial ischemia detected during Holter monitoring.

MEASUREMENT TECHNIQUES, RELIABILITY, AND VALIDITY

Among patients with documented or a high likelihood of coronary disease, ambulatory recording of ST segment depression of 1 mm or more, lasting for at least 1 minute, has been demonstrated to be a reliable and valid marker of myocardial ischemia. This finding is based on simultaneous investigation using radionuclide or echocardiographic evaluation of ventricular function as well as positron emission tomographic assessment of myocardial perfusion.[4]

Verification of automatically-detected ST segment changes should be made by review of the analog ECG waveform,[81,90] with confirmation of a horizontal or downsloping configuration. At least 1 minute—according to some authors, 5 or 10 minutes—should separate individual episodes of ST segment depression.[112,120,142] Among patients with variant angina, transient elevation of the ST segment can be detected using ambulatory monitoring.[24,79]

Most ambulatory monitoring systems use two bipolar leads, and sites can be chosen to detect likely maximum ischemia based on exercise testing or coronary angiography data.[142] The use of three leads increases the chance of detecting extant ischemia.[77] The diagnostic accuracy of ST analysis may be improved by excluding ST segment depression associated with: an increase in R wave amplitude; PQ and ST segments that run a parallel course; and an abrupt onset and offset of ST segment deviation.[11,154] The coexistent finding on ambulatory monitoring of frank T wave inversion may indicate that horizontal or downsloping ST segment depression is of ischemic origin, since this feature significantly distinguished a large series of women with CAD from healthy controls.[11] Interestingly, cohort data by Daviglus, et al. indicate that incident minor ST-T abnormalities on resting ECG independently predict CV mortality among men.[32]

Many authors exclude patients from analysis who have artifactual postural ST deviation; this can be done by review of an initial recording in supine, sitting, standing, and left and right lateral decubitus positions.[120,142] Patients taking digitalis medications or with left bundle branch block, pre-excitation, or uncorrected hypokalemia also usually are not included in analysis of ST segment deviation.[7,35] LeClercq and Coumel and Sheffield, et al. offer detailed discussion of the technical aspects of ambulatory monitoring of ST changes, including standards of instrumentation.[81,135]

CLINICAL SIGNIFICANCE

A correlation is found among coronary patients between the total duration and number of episodes of ischemic ST segment changes during daily activities and ischemic changes during exercise testing. However, the correlation is of limited magnitude, indicating that each of these tests can provide useful diagnostic information in these patients.[136,142] While there is still some controversy about the precise predictive value of ischemia detected on ambulatory monitoring versus on exercise, it is generally concluded that transient ischemia during daily life independently has an adverse prognostic significance among various subsets of patients with IHD.[18,31,36,41,60,63,73,121,148] At least 75% of myocardial ischemia detected on Holter monitoring is asymptomatic,[4] and the prognostic significance of this finding is not diminished if unaccompanied by symptoms.[5,34,140]

The QT Interval

DEFINITION AND STANDARD MEASUREMENT TECHNIQUES

Left stellate ganglion sympathetic outflow leading to temporal dispersion of ventricular repolarization is manifested on the ECG by a prolonged QT interval.

This is associated with a lowered threshold for ventricular fibrillation and vulnerability to life-threatening ventricular tachycardia.

There are numerous formulations and criteria for defining a long QT interval; dilemmas in this regard have been ongoing for decades.[33,70] The QT corrected for heart rate by Bazett's formula, or the QTc (QT/square root of the R-R interval in seconds) is most frequently used, with a pathologically long QTc being > 0.44–0.46.[88,104] The QT interval ideally should be measured on a 12-lead ECG from the earliest onset of the QRS to the end of the T wave, where it merges with the baseline. An eventual subsequent, discrete U wave should not be included. Leads II, V3, and V4 or V5 are usually the most helpful in distinguishing T and U waves. At least three consecutive QT intervals should be measured and averaged to increase precision.[100,101] Measurement of the QT interval is difficult or impossible with flat T waves. The above-cited criteria are not valid for QRS ≥ 0.12 sec. Further, the correction of QT for heart rate should not be applied in the tachycardic range, especially for heart rate over 125/min.[88] Hypocalcemia, as well as quinidine and several other antiarrhythmics, prolong the QT interval. Women appear to have longer QTc than men, according to normative, age-stratified data among Caucasians.[88]

Clinical Significance

Patients with the hereditary long QT syndrome are known to be at risk for polymorphic ventricular tachycardia, syncope, and sudden cardiac death.[102] Resting QTc is normal in 5–10% of gene carriers for this disorder.[134] Furthermore, the long QT syndrome can occur sporadically without evidence of inheritance.[43] In apparently healthy, population-based samples, approximately 6–8% of men and 3% of women were found to have a QTc > 0.44 on resting ECG.[10,132]

In a population-based cohort study among 1583 men and 1508 women aged 40–65 at baseline, at 15-year followup among the men there was a significantly increased risk of CVD and IHD mortality associated with a QTc > 0.44 (RR 1.8 and 2.1, respectively) after adjusting for age and standard cardiac risk factors.[132] There was no significant relation among the women, however.

Dynamic assessment of QTc using ambulatory monitoring reveals that among post-MI patients the mean 24-hour QTc was significantly greater in those with, compared to those without, subacute or late-occurring life-threatening ventricular arrhythmias. The patients with these arrhythmias also had significantly more peaks of QTc > 0.500.[56] Among 6693 consecutive patients undergoing Holter monitoring, those with a mean QTc > 0.44 over 24 hours had a risk ratio of 2.3 (CI 1.3–4.5) for sudden cardiac death at 2-year followup.[1]

Sympathetic outflow associated with emotional stress, sensory stimuli, and/or physical activity can precipitate ventricular arrhythmias in persons with the long QT syndrome.[82,103,155] Antimony exposure is associated with QT interval prolongation, and an epidemic of sudden death has been described in relation to occupational exposure to antimony trisulfide.[23] Among healthy subjects with normal resting QTc, both the cold pressor and the glare pressor tests elicit significant prolongation of the QTc.[13,153] Recently, validated automatic monitoring techniques suggest that the QTc feasibly could be assessed during work activity.[56]

Bradyarrhythmias and Ventricular Tachyarrhythmias

Notwithstanding medications and structural abnormalities affecting the cardiac conducting system, intense, unopposed vagal outflow can result in various degrees of sinus bradycardia. In extreme, rare cases, Stokes-Adams attacks or even sudden

death[97] can occur from vagally mediated sinus node arrest and asystole. Hypervagotonia in the carotid sinus syndrome and vaso-vagal syncope are seen.[156] It is estimated that about 8% of the population responds to novel or conditional stimuli with extreme vagotonia. Case reports reveal that repeated intense emotional stimuli can trigger multiple Stokes-Adams attacks in extreme vago-dominant individuals.[127] Ambulatory ECG recording is well suited to detect symptomatic or asymptomatic bradyarrhythmias of various degrees of severity in relation to working activity.

While ventricular tachyarrhythmias are responsible for the vast majority of sudden arrhythmic deaths, the evidence is conflicting as to the value of any of the various forms of ventricular ectopy for independently predicting the occurrence of cardiac events, particularly among those without underlying heart disease.[16,17,71,139] It is increasingly recognized that examination of the interaction among relevant parameters can yield more diagnostically important, predictive information.[55,78,139] For example, Hoberg and colleagues observed that silent myocardial ischemia–related complex ventricular arrhythmias can be triggered by physical activity among a large percentage of patients with clinically stable CAD.[54] These authors propose that "silent myocardial ischemia may be the missing link between the increased risk of cardiac arrest and the lack of premonitory symptoms" during physical exercise. A significant positive association has been found during ambulatory monitoring among women with CHD between the extent of ST segment depression and degree of complexity of ventricular arrhythmias, with a mean 2.2-mm horizontal depression in lead CMV5, when episodes of ventricular tachycardia were recorded.[11] These empirical findings are concordant with clinical-physiologic data demonstrating that myocardial ischemia can act as a promoting substrate for electrical instability of the ventricle.

Dilaveris and colleagues have reported that diminished HRV preceded ST segment depression and was significantly related to the magnitude and duration of myocardial ischemia.[40] Seen in this light, the observations concerning abrupt and total loss of respiratory sinus arrhythmia with work performance at peak capacity acquire a potential clinical relevance. Integrative examination of the interaction among these relevant parameters can be performed in relation to the work environment, using ambulatory ECG monitoring. Because of the intimate interrelation between many of these ECG endpoints and hemodynamic changes, a more complete view of the effects of the occupational milieu on CV well-being would be gained if ECG and BP monitoring were assessed in concert.

POINT ESTIMATES OF BLOOD PRESSURE AT THE WORKSITE *by Peter Schnall, MD, and Karen Belkić, MD, PhD*

There are several major reasons why blood pressure (BP) should be measured at the workplace:

1. **Public health.** Surveillance of the workplace to determine the prevalence of hypertension or elevated BP is important from a public health perspective, especially in light of the evidence that workplace factors play a significant role in the etiology of essential hypertension. Worksite screenings are the only way to identify those groups of individuals with normal clinic BP and elevated worktime BP—the "false negatives"—who are at a high risk for a hypertension-related morbid event.

2. **Improved clinical diagnosis.** Collecting worksite estimates of BP should result in improved diagnosis and treatment of patients. The level of an individual's

worktime BP is important for diagnosis, as a predictor of subsequent illness, and for evaluation of clinical treatment.

3. **Studies of etiology**. To the extent that psychosocial workforce factors play an important etiologic role in the genesis of hypertension, *where* one measures BP matters, because these factors are not static and may or may not be present at any moment. All BP measures are influenced by some set of psychosocial factors. For example, BP in the clinic is subject to the "white coat" effect. BP at home can be affected by home stressors and likely carry-over from work. Sleep BP may be influenced by dreaming (REM sleep), which is associated with elevation of BP and disinhibition of CV control mechanisms.[19] Factors such as work-related stressors, physical activity, and posture are important in workplace BP assessment.

A significant association also has been found between left ventricular mass index and exposure to job strain.[130] Left ventricular enlargement is correlated more highly with average BP at work than at other locations.[6,38,39] These authors propose that the observed associations between job strain, left ventricular hypertrophy, and hypertension suggest a pathophysiologic process that may explain the reported association between job strain and CHD mortality. Measurement of BP at the workplace represents the critical node for further elucidating these inter-relations.

4. **Reliability and validity**. Workplace amBPs are more reliable and have greater validity than casual clinic BPs, and this also may be true for point estimates of worktime BP.

5. **Reduced cost**. It is usually less expensive to collect BP at the workplace than to send individuals to a medical center.

Basal Blood Pressures

The concept of casual BP is derived from the work of Smirk in the 1940s.[137] His idea was that the clinic BP had two independent components—the basal and the supplemental. Basal BPs were those obtained in a seated subject in a comfortable environment after a 30-minute rest by a single observer. The difference between this BP and the initial BP (the casual BP) was the supplemental BP. Smirk found that basal BPs and supplemental readings did not correlate. It was proposed that the basal BP represented the structural or fixed elements of an individual's hypertension, while the supplemental pressure was the elevation attributable to the effects of physical and mental activity.

The fact that casual BP did not correlate with basal BP in Smirk's research requires explication. It is plausible that the psychosocial, mental, and physical factors associated with a visit to the doctor's office reflect a small percentage of actual exposures impacting on an individual's basal tonic BP. In contrast, picture a universe of exposures that influence BP (the frequency and intensity of these exposures determine the ultimate impact on BP). Work stressors, since they occur more frequently (longer hours of the days, more days of the year, more years of a life) and with greater intensity than does the stress of a visit to the doctor's office play a major role in elevating an individual's BP.[129]

The possibility of obtaining a basal BP uninfluenced by psychosocial factors seems unlikely. There is a real question whether 30 minutes or more (rarely achieved in practice) of rest in a comfortable environment is a true reflection of basal BP. Even under such conditions humans are not free of psychosocial influences. In fact, we often have observed increases in BP in patients at rest (anxious subjects may worry increasingly as they "rest"). Even among those persons who manage to fully relax and shut out nearly all psychosocial stimuli, this approximation of basal BP may not be reflective of clinically relevant BP status.

The very concept of basal BP—a BP free of all psychosocial and other stimuli providing a best estimate of tonic true BP—is not compatible with our current notions of the etiology of essential hypertension of neurogenic origin. We now believe that in most cases of essential hypertension, psychosocial factors, especially at work, play an important role in elevation of BP. Basal BP eventually reflects these influences. This process leads to three identifiable stages: (1) When individuals first are exposed to putative causes such as workplace stressors, BPs are elevated at work and basal BP is normal. (2) Chronic exposure to these stressors leads to elevated workplace BPs as well as basal BPs.[46,47] At this stage the psychosocial factors likely are correlated with both measures of BP (structural changes in the cardiovascular system are occurring). (3) In end-stage hypertension, self-sustaining structural processes in the vascular system may lead to disjuncture between reported psychosocial factors and BP (both workplace and basal) since the individual may no longer be exposed to the psychosocial factors (e.g., promotion, retirement) and the BP process is now autonomous.

These stages are consistent with the presumed mechanisms of neurogenic hypertension—an initial reversible stage with predominant elevation of systolic BP as the defense response is elicited. With repeated exposure, structural changes occur in the heart and vasculature.[46,47]

Problems with Clinic Measures

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). It has been called the "white coat" phenomenon and is characterized by normal ambulatory BP and high clinic casual BP. Most studies of individuals who display this unique response appear to show they are at relatively low risk for morbid events compared to those with sustained hypertension.[117a] However, Julius, et al. argue to the contrary, on the basis of point measurements at home versus clinic.[66] They found an increased prevalence of cardiovascular metabolic syndrome (SNS driven) among young adults with white coat hypertension, and postulate that white coat hypertension is mediated by over-activity of the SNS.

According to Pickering: "True blood pressure may be regarded as the average level over a prolonged period of time, which is generally thought to be the most important determinant of target organ damage."[117] The interest in and subsequent research with amBP monitoring arose out of the observation that the typical casual clinic BP is highly variable and does not reliably reflect BP during daily life. A body of evidence that workplace amBP is a better predictor of morbidity and mortality has emerged. BPs obtained with an ambulatory monitor are more reliable and valid than other assessments of BP (see pages 191–196).

Combining information from both clinic BPs and amBPs allows identification of four groups—two in which the clinic and ambulatory measures agree and two crossover groups:

- Individuals with normal amBPs and elevated BPs are the **false positives** (i.e., those with white coat hypertension) if one accepts amBP as the definitive measure of BP.
- Individuals with normal clinic BP but elevated amBP are the **false negatives**. This group is potentially at risk of an untoward event since amBPs are increased, yet these individuals remain undiagnosed. It has been found, for example, that among individuals in stressful professions (e.g., journalists, professional drivers) selected to

have normal clinic BP, worktime DBP averaged to nearly 90 mmHg, and some individuals had sustained increases in work BP that failed to decrease below 150/100 during the entire recording period.[149] Obtaining estimates of BP at the workplace in such individuals is extremely important from a clinical perspective, and alone is justification for worksite BP screenings.

Problems with Ambulatory Measures

First, logistics at the workplace frequently are complicated. Participants wearing a monitor require time to don the equipment, experience frequent interruptions of work—albeit of short duration—when BP is measured and a diary filled out, and need time to remove the monitor at the end of the work period. For some work activities these interruptions may have serious consequences for job performance and productivity, especially when large numbers of participants are involved.

Second, costs are high for several reasons. Equipment, including the monitors themselves and the necessary computers, are expensive. Moreover, amBP measurement is a technologically sophisticated and labor-intensive process requiring highly trained personnel to collect and process data. Trained technicians hook the subject up to the equipment; insure its proper working; explain its use to the wearer, including the completion of an accompanying diary (for location and activity codes); and enter and code the obtained data into a computer.

Point Estimates as an Alternative to AmBP Monitoring

What is needed is a point estimate of BP that approximates a typical amBP reading in the same subject (Table 2). We can imagine a subject wearing an amBP monitor at work and wanting to obtain several readings during the same period, which approximate those that the machine is obtaining when it records BP every 15 minutes or so during the working day.

No current protocol exists for obtaining point estimates of BP at the workplace. Below we outline a new BP protocol for obtaining point estimates of BP while a

TABLE 2. Point Estimates of Workplace Blood Pressure

Advantages	Disadvantages
Cost effective and feasible as means of screening an entire workplace to determine prevalence of hypertension. Less expensive than amBP monitoring.	Fewer samples and therefore lower reliability compared to amBP. However, individual point estimates are likely more reliable than an individual amBP reading (individual machine readings are imprecise).
Comprehensive database development for a variety of public health and research purposes.	Logistics of workplace screenings may be difficult.
Enhanced reliability and validity in comparison to clinic BPs. As compared to office-obtained casual BPs, they better reflect real work life—not contaminated by spurious, clinic-related stimuli. Workplace-obtained, casual BPs correlate well with workplace amBP in contrast to casual clinic BPs and are better correlated with workplace psychosocial risk factors such as job strain.	
Can inform treatment decisions. Identify those subjects who are false positive or false negative on casual clinic BP.	

subject is actually working (contrast with the current American Heart Association protocol for casual clinic BP recording, 1998[2]).

Protocol for Obtaining a Point Estimate of Worktime BP

Part 1. Checklist. Use prior to obtaining point measurements at the workplace—before the workday on which BP is to be measured:

1. Obtain informed consent
2. Obtain arm circumference
3. Provide instructions regarding clothing (e.g., loose shirts, accessible arms)
4. Collect medication and medical history (and other relevant data)
5. Use simplest accurate equipment—a carefully calibrated Anaeroid device (not a mercury column)

Part 2. Protocol for obtaining two sets of point estimates. Conducted while individuals are working.

1. Trained observer can measure (not necessarily health professional)
2. Avoid "clinic atmosphere"—no white coats; don't act like a clinician.
3. Aim for informal interactions and neutral conversations—avoid conversations that are of personal relevance to the participant; do not discuss controversial issues until after BP collection.
4. Obtain two sets of point estimates in one workday while subject is at usual work activity.

   First BP estimate should be obtained near start time at beginning of shift (workday) This helps maximize number of workers examined during each day if part of a large population screening.
   - Record time(s) of BP measurements
   - Record subject's body position
   - BPs to be determined with worker in same position as at work (e.g., standing if stands at work)

   Second BP estimate should occur later in same workday or at same time on second day.
5. Equipment: calibrated aneroid sphygmomanometer (determine proper cuff size)
6. Conducting actual point estimate measurements

   First set
   - Taken at workstation, shortest possible interruption of work process
   - Three readings for each point estimate
   - 1 minute between readings

   Second set
   - Repeat above. *This is probably the best single estimate because subject is desensitized.*
7. Average first and second readings for best estimate; discard third
8. Subject feedback

   Give BP results only after second set of estimates obtained

Part 3. Protocol for obtaining related data. Needed to complement point estimate of BP at the workplace.

1. Diary

   Obtain diary data if possible (elective)

   Include observations about job and individual (requires separate protocol and training)

   Work environment (see Chapters 6 and 8)—record usual and atypical for the day of estimate

Assess job characteristics (see Chapters 6 and 8)—including subjective evaluation of work by subject (home life as well, if desired)

2. Medical history, demographics, and other potential confounders
   Potential confounders include alcohol, body-mass index, age, race, gender, family history of hypertension, medications (antihypertensives and oral contraceptives as well as other medications that may potentially affect BP), smoking, caffeine, body position, and physical activity.
3. Data analysis (depends on purpose of measurement)
4. Some unresolved questions
   Start time of BP Screening—possibilities include at beginning of shift or at fixed intervals during shift
   Heart rate abnormalities
   - Bradycardia (extreme)—bear in mind the need to more slowly deflate the BP cuff in the presence of extreme bradycardia that may occur, for example, in well-trained athletes and those with clinical conduction abnormalities.
   - Tachycardia

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