THE WORKPLACE AND CARDIOVASCULAR HEALTH: CONCLUSIONS AND THOUGHTS FOR A FUTURE AGENDA

by Karen Belkić, MD, PhD, Peter Schnall, MD, Paul Landsbergis, PhD, and Dean Baker, MD

We argued in the introduction that to adequately address the CVD epidemic, there is a need for a social epidemiologic approach that focuses on the workplace. Here, we briefly review the empirical, theoretical, and biological evidence presented earlier to demonstrate "convergent" validation that the relationship between workplace stressors and CVD risk is causal. The empirical findings are consistent with and predicted by the theoretical models, and the linkage between them is demonstrated to be plausible via biological mechanisms and experimental research. We then elaborate on new strategies, presented in the latter part of this book, for enhanced prevention and clinical management, workplace interventions, and social policy to reduce the impact of CVD.

EMPIRICAL EVIDENCE OF WORKPLACE EFFECTS ON CVD

In Chapter 2, we presented a substantial body of findings concerning the impact of workplace psychosocial, chemical, and physical conditions on CVD. The most consistent evidence is provided by research on sources of psychosocial stress at work, which are also the most prevalent risk factors. The most highly studied of these is work with high psychological demands coupled with low decision latitude, i.e., job strain. On the basis of empirical reviews focused on men and on women, as well as the recent review by the European Heart Network,[34] and notwithstanding some studies with null results, the conclusion of Schnall, Landsbergis, and Baker that "a body of literature has accumulated that strongly suggests a causal association between job strain and cardiovascular disease" has been corroborated and strengthened. The data relating job strain to AmBP and decision latitude to CVD outcomes are particularly compelling.

Besides consistency of association among studies, other evidence supporting causality has emerged. There are now data, albeit limited, suggesting a dose-response relationship between exposure to job strain or its major dimension(s) and both CVD and BP. New job strain cohort studies further confirm that exposure precedes outcome in time. Overall, of ten such studies in men, six show an increased CVD risk due to job strain or its components, and an additional two provide mixed results. Of five cohort studies among women, four demonstrate an elevated CVD risk related to job strain or its components.

Epidemiologic evidence of the plausibility of the relationship between job strain and CVD has expanded. Cross-sectional, as well as some longitudinal data,

linking exposure to job strain with elevated AmBP in men and women suggests one major mediating mechanism for this process. There are now cohort data demonstrating that a change in job strain exposure is associated with a change in BP.[58] Furthermore, some data suggests an association between job strain and/or its major dimensions and other CVD risk factors, primarily smoking intensity in men, and possibly increased coagulation tendencies.

The magnitude of association between job strain and CVD typically range from risk ratios (RR) of about 1.2–2.0 for studies using imputed job characteristics (with resulting nondifferential misclassification bias towards the null), to 1.3–4.0 for studies using self-reported job characteristics. Associations are more consistent and stronger among blue-collar workers, with RR as high as 10. Systolic BP at work (as measured with an ambulatory monitor) among employees facing job strain is typically 4–8 mmHg higher than among those without job strain.

Another model of work stress, the Effort-Reward Imbalance (ERI) Model, also has been studied cross-sectionally and longitudinally, primarily in men. There are significant positive associations between high effort/low reward and elevated lipid levels, hypertension, and CVD, with magnitudes of effect similar to or even greater than in self-report job strain studies. A British study indicates that the effects of job control and ERI are statistically independent of each other in the prediction of CHD,[6] and a currently unpublished Swedish study finds that the combined effects of exposure to job strain and to ERI on CVD are much stronger than the separate effects of each model.[51]

There are also data indicating a relationship between threat-avoidant vigilant work and CVD. For example, in studies comparing occupations, professional driving, particularly urban transportation, emerges as the occupation with the most consistent evidence of elevated risk of CHD and hypertension (see Chapter 2). Such psychosocial factors may help to explain the nine-fold difference between high and low CVD risk occupations in men and a five-fold difference in women.

In addition to psychosocial job stressors, there is some evidence that work schedules and physical and chemical workplace hazards may increase the risk of CVD. Notwithstanding the difficulties involved in researching this area, a substantial body of longitudinal data implicates shift work as an independent CVD risk factor; however, there are also some well-designed cohort studies with null findings. Investigations of long work hours are more sparse, but quite consistently show a relationship to elevations in ambulatory and casual BP, and to CVD. In three fairly recent papers, the effect of long work hours independently of other workplace stressors was demonstrated with respect to increased BP and risk of MI. Finally, some support exists for a significant association between physical factors—most notably cold, heat, noise, and passive smoking—and hypertension and/or CVD. While sedentary jobs have been linked to CVD risk, certain patterns of workplace physical activity (e.g., irregular episodes of heavy physical exertion alternating with sedentary work), also are implicated in risk of MI. As to other physical factors, such as vibration and heavy lifting, physiologic data suggests that these may have an untoward effect on the CV system; however, epidemiologic data is extremely limited. Cardionoxious chemical agents include: carbon disulfide (a well-established risk factor for CAD), nitrate esters (sudden cardiac death), carbon monoxide (myocardial ischemia, MI, sudden death, CHD mortality), lead and arsenic (possible risk factors for hypertension), and solvents (dysrhythmias, with methylene chloride giving a clinical picture similar to carbon monoxide).

Population Attributable Risk, Occupational Factors, and CVD

Psychosocial, chemical, and physical exposures at the workplace, along with sedentary work, represent a major public health burden on working populations. We can calculate the population attributable risk (PAR%)—i.e., the reduction of incidence if the population were entirely unexposed to occupational risk factors for heart disease—to estimate the degree to which work-related factors account for the epidemic of hypertension and CVD. The PAR% calculations depend on two assumptions: (1) the prevalence of exposure, and (2) the strength of association between exposure and the outcome of interest. Thus, the PAR% results will vary greatly among population groups and study results, engendering some difficulty in generalization.

Since there is excellent data available for job strain, we can calculate some representative results. Using data from the Cornell Worksite and Ambulatory BP Study[59] with an exposure rate to job strain of 20% and an odds ratio (OR) of 3 between job strain and hypertension, 28.6% of hypertension among working men in New York City could be attributed to job strain.* PAR% also have been calculated for European data on job strain and CVD. The European Heart Network[34] cites Olsen and Kristensen, who used exposure to monotonous, high-paced occupations as a proxy measure for job strain, taking a very conservative estimate, and calculated PAR% for CVD as 6% for men and 14% for women in Denmark.[48] However, when they estimated a total CVD burden for Danish workers due to occupational factors—job strain (but not ERI), sedentary work, physical and chemical exposures, and shift work—the PAR% was greater than 50%. A PAR% of 15.3% for CVD mortality due to isostrain in the Swedish male working population can be calculated based on a reported OR of 1.9 and 20% exposure rate to isostrain.[26] In Europe as a whole the exposure to job strain may be as high as 30%,[9] which would yield a similar or higher PAR% to that calculated for Sweden. A full discussion of the number of cases and the costs of CVD in the U.S. for various estimates of PAR% due to job strain is found in Chapter 11.

While the empirical evidence and PAR% calculations presented above demonstrate the effect of CVD of psychosocial risk factors, and there is additional data for work hours, shift work, and chemical and physical exposures, studies examining the combined or interactive burden of these factors are lacking. There is some evidence of an interaction between psychosocial stressors, such as between job strain and low social support,[11,25] job strain and ERI,[51] and high work demands and low economic rewards.[39] However, we know little about the possible synergistic effects of combinations of various types of risk factors, except for a few studies such as that of Alfredsson, et al. showing an increased SMR for heavy lifting plus hectic work.[2] Nonetheless, even without this knowledge, the evidence to date indicates that workplace risk factors account for an important burden.

THEORETICAL PLAUSIBILITY OF A PSYCHOSOCIAL CONNECTION

Psychosocial Models

The occupational health movements of the latter part of this century raised the concern that the modern work environment caused serious illness and injury. In the

* 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.

1970s, psychosocial researchers began to address this issue with respect to CVD. As posed by Karasek and Theorell: "Did the social organization of work also cause serious physical illness? Without scientific evidence of such associations (evidence of job dissatisfaction would not suffice) the same political will to redress worker hazards could not easily be mustered. This evidence would be much more difficult to accumulate, however. In the case of physical occupational health hazards, such as in coal mining, the cause of injury was often obviously environmental, but for psychosocial risks work-related and nonwork-related factors were interlocked."[29] A critical obstacle was the theoretical conceptualization and modeling of workplace stressors.

A pioneering breakthrough came in 1979 with the publication of the Job Strain Model, based upon the premise that strain occurs when there is excessive psychological workload demands together with low job decision latitude[30] (see Chapter 3). This appears to provoke arousal, as well as distress, activating both the sympathoadrenomedullary and adrenocortical axes, a highly deleterious combination.[13,14]

A third dimension, social support, was added later to the model.[25] It was found that lack of social support at work interacted with job strain to substantially increase the risk of CVD. A variety of investigations, including cross-sectional and longitudinal observational population studies, intervention research, and animal experiments, have shown that social isolation and lack of social support are harmful to CV health.[4,21,49]

More recently, the ERI Model was introduced by Siegrist and colleagues.[60,61] In comparison to the Job Strain Model with its emphasis on moment-to-moment control over the work process (i.e., decision latitude), the ERI model provides an expanded concept, emphasizing macro-level long-term control through rewards such as career opportunities, job security, esteem, and income. The ERI Model posits that work stress results from an imbalance between these rewards and effort. Effort is seen to stem both extrinsically from the demands of the job and intrinsically from the individual's tendency to be overly committed to these work demands.

Key dimensions are shared by the Job Strain and ERI Models: both control as well as challenge (demands) are an integral part of each. However, control varies—from micro (task) level in the former, to macro level in the latter. The nature of the challenge varies from model to model, but there is a challenge of some kind in each.

In addition to these two models, which have been well developed theoretically and empirically confirmed in relation to CVD, other promising formulations are emerging. One is the concept of threat-avoidant-vigilant work, which seems particularly relevant in understanding the stress of certain occupations at high CVD risk. Such work is onerous since it requires continuous maintenance of a high level of attention, in order to avoid the disastrous consequences that could occur with a momentary lapse or a wrong decision.

Social Class, Workplace Factors, and CVD

There is a considerable and consistent body of evidence of an inverse association between socioeconomic status (SES) and incidence and prevalence of CVD, primarily CHD (see Chapter 2). The higher CVD risk among men and women in lower SES groups, e.g., blue-collar workers, began to appear in the 1950s[17,40,79] and has risen progressively over the period 1960–1993.[17]

These changes in CVD mortality rates among the blue-collar workers are paralleled by increasing income inequality, which differs greatly among countries, and is measured by the size of the income gap between the rich and the poor. Income

inequality profoundly affects overall mortality,[27] although only a modest direct effect has been heretofore demonstrated for CVD.[33,42] In the industrialized world, "It is not the richest countries which have the best health, but the most egalitarian."[75] In the U.S., the "earnings distribution among workers has widened greatly and is the most unequal among developed countries."[71] The role of work in relation to income inequality as a potential contributor to adverse health outcomes is an important area for future research. ERI would be a particularly suitable model to investigate these relationships.

As pointed out by Johnson and Hall, not only do "those in the upper levels of the professional and managerial hierarchy enjoy ample financial remuneration, they also have the right to exercise authority over others, to expect obedience and even subservience, and to enjoy prominent social position, the privileges of voluntary action and association, and the many ineffables of an affluent lifestyle."[24] These authors elaborate that work control "varies systematically as a function of social class." SES usually is operationalized by education, income, and occupational status. The latter two factors are features of work. In fact, status at work, variety and scope for use of initiative and skill, and ability to exercise authority and control are some of the main ways by which SES is defined. In the Whitehall study, the distribution of job control was the major factor contributing to the socioeconomic gradient in CHD risk across civil service employment grade.[32,41] In contrast to low job control, job strain has a weaker and, in some studies, null association with SES. However, job strain appears to interact with low SES. Job strain has a stronger association with CVD and with BP in workers of lower SES.

Not only are psychosocial workplace stressors, most notably low decision-making latitude or control, more prevalent among workers of lower SES, but these workers also are more frequently exposed to physical and chemical hazards that can impact upon the CV system. Shift work generally is more common among blue-collar compared to white-collar workers. Standard cardiac risk factors such as smoking, obesity, and lack of recreational physical activity also are more prevalent among those in the lower SES groups.[28] These risk factors can be affected by an unhealthy workplace.

Thus, low SES is associated with a number of workplace factors that can impact upon CVD risk. These include low job control, exposure to shiftwork, and physical and chemical hazards. Persons in low socioeconomic strata disproportionately receive inadequate wages and salaries, lack promotion prospects, and may face downward mobility. These factors likely contribute to an increased ERI among those in lower SES groups. Exposure to job strain is associated with a greater CVD risk among blue-collar, as compared to white-collar workers. Standard cardiac risk factors, often related to an unhealthy workplace, also are more prevalent in the former. These interrelations, explored in detail in Chapters 2 and 3, render the conclusion of Johnson and Hall that the realities of social class and work are "inextricably linked,"[24] of profound relevance to CV well-being.

Insights from Cognitive Ergonomics and Brain Research

Constructs such as job strain and ERI are based heavily on sociological theory. Cognitive ergonomics and brain research provide insights that complement these models, and provide a deeper understanding of dimensions such as psychological demand, control, and conflict. Thus, for example, when speaking of mentally demanding work, we can go far beyond queries about "working hard" and "working fast." With a more quantitative, objective appraisal of the burden of work processes,

and a better grasp of the possibilities and limitations of the human central nervous system, a more rational approach to work design emerges. By analyzing tasks in terms of allocation of mental resources, we can better determine what is too much (leading to overload), what is too little (leading to underload), what is incoherent or contradictory (leading to conflict), etc. A critical ratio is that between "knowledge-based" labor processes, which require conscious attentional resources, and those that are "skill-based," which can be performed in parallel and feature rapid, smooth, learned, and highly integrated patterns.[15,47] We also can pinpoint how to promote the worker's autonomous control, not only to meet the moment-to-moment exigencies of the situation, but, ideally, to be in harmony with his/her own needs, as well. Simply stated, this knowledge can help humanize the work process. Examples of the practical implementation of this approach are provided in Chapters 3 and 6.

Cognitive ergonomics and brain research also illustrate that emotional dimensions of human labor impact profoundly on mental burden. For survival reasons, our nervous systems are constructed to selectively allocate mental resources to potentially harmful stimuli, even if the threat is purely symbolic. It is essential to take into account the often hidden burden represented by threat-avoidant vigilant activity. Neurophysiologic studies demonstrate that imminent threat of an accident in the symbolically represented traffic milieu is associated with an unusually high level of selective attention. To avoid such situations, compensatory allowance, especially increased time allocation, must be included in the work planning "equation."

There is a need for psychometric tools that account for the total burden of work stressors, using a cognitive-ergonomic approach, and with relevance to CVD risk. The Occupational Stress Index[3] represents one such possible tool. Potential multiplicative interactions and higher-level terms should be explored within that model and more generally. The burden of unpaid labor, which is disproportionately performed by women, also must be considered.

BIOLOGICAL PLAUSIBILITY OF A WORKPLACE–CVD RELATIONSHIP

A large body of evidence indicating that occupational stressors can profoundly impact numerous pathophysiologic processes, resulting in CV dysfunction and disease, has been presented. As described in Chapter 4, experimental animal studies implicate central stress mechanisms in cardiac electrical instability, as well as in hypertension, disorders of heart beat dynamics, and atherogenesis. The reader also is referred to the very recent paper by Rozanski, Blumenthal, and Kaplan, which reviews how psychosocial factors can affect the pathogenesis of CVD.[56]

Stressors most often provoke a defense response, and, in extreme cases, the defeat reaction. These responses, which in the worst situation may both be operative in turn, can activate the sympatho-adrenomedullary and hypophyseal-adrenocortical pathways, respectively. Empirical studies have demonstrated an association between numerous work stressors and elevations in catecholamines and cortisol.

Direct empirical confirmation, based on epidemiologic and field studies at the workplace, is not available for all of the pathways. Of the processes discussed in Chapter 5, the most attention has been paid to exposure to job strain in relation to elevation in BP and development of hypertension. Here, cross-sectional and longitudinal ambulatory BP data clearly show that hypertension can arise as a result of chronic exposure to job strain. Plausible stress mechanisms that can lead from elevations in BP to chronic hypertension include changes in vascular resistance, as well as renal mechanisms. The relationship among chronic exposure to job strain,

elevations in workplace AmBP, and increased left ventricular mass also has been empirically confirmed.

Metabolic changes, including hyperlipidemia and heightened coagulation tendency, together with an increased progression of carotid atherosclerosis, have been linked to aspects of stressful work, especially ERI. A risk for the combined occurrence of hypertension and hyperlipidemia, characteristic of CV metabolic syndrome, has been associated with ERI. CV metabolic syndrome appears to be driven by augmented sympathetic outflow. Further attention is needed to the relation between work stressors and the occurrence of hemodynamic and biochemical abnormalities characteristic of this syndrome.

As to myocardial ischemia, the biological mechanisms generally are well-defined, and many are related to workplace factors (e.g., increased double product [heart rate × SBP], left ventricular hypertrophy, atherosclerosis). Mental stress in the laboratory has been consistently shown to provoke myocardial ischemia in patients with stable ischemic syndromes. However, field studies of myocardial ischemia in relation to workplace stressors are exceedingly sparse.

We also know quite a bit about the stress mechanisms that can lower cardiac electrical stability. Until recently, however, the possibilities for noninvasive ambulatory monitoring to detect the electrically vulnerable myocardium before sudden cardiac death occurred were limited. Neither the quantity nor the pattern of ventricular extrasystolic activity proved sufficiently predictive. With more advanced technologies, it is now feasible to simultaneously follow several ECG parameters that impact on cardiac electrical stability (ST segment, heart rate variability, QT interval), together with ventricular arrhythmias, during work. Furthermore, studies of patients with automatic implantable cardioverter defibrillators (AICD) could examine job-related exposures, providing direct information about the potential for workplace factors to trigger life-threatening tachyarrhythmias. We do know that AICD fire significantly more on Mondays,[52] suggesting a relation to work activity.

There is some empirical evidence of a septadian overrepresentation of Mondays vis-à-vis cardiac events.[53,77] The early morning hours, during which several preconditions for plaque rupture and thrombus formation are present, are known to be the period of highest risk for these events.[16,46] In the morning hours after waking, systolic BP increases by about 20–30 mmHg, heart rate and vascular tone rise, platelets are hyperreactive, while fibrinolytic activity is at its low.[67] Sympathetic activation occurs upon assuming the upright position, and in the early morning cortisol is at its peak. This can result in a glucocorticoid-related increase in coronary-artery sensitivity to catecholamine-mediated vasoconstriction.[67,76] The epidemiologic and biological data, taken together, indicate that the stress of work after a weekend of respite may precipitate acute cardiac events among working patients.[38,76] Psychosocial, physical, and chemical factors, along with long and irregular work hours, can chronically promote the underlying pathological processes, as well as act as trigger mechanisms for acute cardiac events.

CONVERGENT VALIDATION OF THE CAUSAL LINK

The theoretical constructs of how workplace factors affect the development of CVD are corroborated by the large body of empirical data confirming this relationship. We have suggested the term "econeurocardiology" (see Chapter 4) to represent the biological paradigm by which social factors, such as work stress, are perceived and processed by the central nervous system, resulting in pathophysiological changes that increase CVD risk. All told, the biological and theoretical plausibility

of this view, coupled with the empirical evidence, provides convergent validation for the conclusion that environmental stressors from the workplace play an important role in the development of CVD.

There is a need for intervention studies as the strongest evidence for causality. These studies also provide practical experience and techniques for implementing changes at the worksite and evaluating their effectiveness. More longitudinal data with assessment of cumulative exposure and changes in exposure is needed, as well. To know where to intervene, the prevalence of both cardionoxious exposures and CVD must be mapped—i.e., **surveillance**. Such a map will facilitate the identification and management of individual exposed workers with varying CVD severity, who may benefit from clinical intervention.

There remain a number of methodologic issues for resolution (see Chapters 6 and 7). These include the need for refined measurement tools of the Job Strain and ERI Models. Improved reliability and validity of exposure assessment would be obtained through "triangulation"—the use of self-report methods complemented by imputation—and by data from observers, whenever possible. Improved outcome assessments with earlier detection at the preclinical level can now be realized by new noninvasive monitoring techniques applicable in field studies at the workplace. More examination also is warranted as to how circumstances of occupational life affect behavior patterns, such as hostility and overcommitment, which can in turn, affect CVD risk.

CURRENT STATUS AND FUTURE DIRECTIONS

Implications for Clinical Practice: Advancing the Discipline of Occupational Cardiology

Unlike several other medical subspecialties (e.g., pulmonology), for cardiology the workplace has yet to become an integral consideration. Consequently, there are few guidelines (with the exception of those related to physical activity levels) to help clinicians make informed recommendations concerning occupational factors, as these pertain to patients with various degrees of CVD severity. In Chapters 8 and 9, we offer physicians and allied health professionals a practical set of tools for the evaluation and management of working people at risk. First, taking an occupational history as it relates to the CV system is imperative, and an approach is outlined to help clinicians accomplish this. Next, a graded, risk-stratified algorithm is proposed for an occupational cardiologic assessment of patients whose jobs could be harmful to the CV system. High-risk, but still preclinical patients are identified, and a set of diagnostic steps is proposed. This work-up can serve to guide clinicians in making specific recommendations concerning working conditions. Ambulatory monitoring is particularly helpful for objectively determining which workplace modifications are most conducive to the patient's CV well-being.

Return to work (RTW) after cardiac events (see Chapter 9) is an especially delicate question. The cardiologic caregiver must evaluate the full clinical picture, including symptoms and morphological and functional status, as well as address complex personal, psychological, social, economic, legal, and ethical issues. The importance of job characteristics is illustrated by the existing, albeit limited, longitudinal data, showing that return to high-strain work is a significant predictor of mortality in young men post-MI, independent of clinical indices.[65] Notwithstanding the need for large-scale clinical investigations of this type, these findings should prompt the clinician to raise the question posed by Theorell and Karasek: "Should

heart attack patients return to stressful jobs?"[66] A similar query could be relevant, as well, to patients with hypertension (see Chapter 10), especially in light of the Cornell Worksite Ambulatory Blood Pressure Study, which indicates that changing from a high- to low-strain job was associated with a sizable fall in AmBP among such patients.[58]

Workers whose cardiac status represents a public safety issue (see Chapter 9) raise another difficult issue, frequently related to RTW. The clinician must render an estimation of risk for the occurrence of cardiac or other events that could lead to impaired consciousness. Airline pilots have received the most stringent evaluation in this regard, but these issues also pertain to operators of ground transport and other heavy machinery and to workers whose jobs entail threat-avoidant vigilant activity. An expanded occupational **public health role of the clinician** could be crucial in this particular realm. Clinicians, together with occupational ergonomists and other specialists, must have greater influence to recommend and implement cardio-protective guidelines about work conditions for these jobs. As it stands now, the clinician is repeatedly faced with the dilemma of making a judgement about the individual's CV work fitness, often knowing full well that the job itself is cardionoxious. A more proactive approach offers the possibility of ameliorating this ethical dilemma.

A public health perspective is vital for the clinician to effectively protect his/her patients exposed to cardionoxious work. The clinician must be on the alert for the occurrence of unexpected patterns or clusters of CVD. Historically, in other medical disciplines, the physician often has been among the first to identify occupationally associated diseases, with resultant major changes in the work environment. However, for a number of reasons (see Chapter 10) clinicians typically have not been the ones to herald the occurrence of clusters of job-related CVD. There is an urgent need to incorporate the concept of an **occupational sentinel health event** into the mainstream of cardiology.

The clinician also can play an important role in evaluating CV health impact of changes in the work environment and worksite health promotion programs, including individual stress management. The physician may be in a unique position to help transform an adversarial situation to a cooperative relationship, and thereby to represent a stabilizing force. Cooperation among the various participants in the work process (e.g., labor, management, occupational hygienists, engineers, economists) can be promoted by the authority of the clinician, whose interest is first and foremost the well-being of his or her patients.

Current Trends in Working Life

As we embark upon the 21st century in the United States, despite a booming economy, much prosperity, and relatively low unemployment rates, there is a large and growing income disparity, and working conditions are deteriorating for many. Working men and women are putting in longer work weeks and are increasingly exposed to job conditions that can undermine CV health.[36a] In Europe, in 1996, 23% of those employed were working more than 45 hours/week.[73] In the U.S., average weekly work hours increased by 3.5 to 47.1 hours from 1977 to 1997.[5] Workers in the U.S. have now surpassed Japanese workers in total number of hours worked per year, and work longer hours than in any other industrialized country.[22]

Substantial changes in job characteristics have occurred over the past generation in industrialized countries. In Europe, surveys indicate an increase in "time constraints" (i.e., workload demands) between 1977 and 1996.[9] Similarly, in the U.S., increases between 1977 and 1997 were reported for "working very fast" (from 55%

to 68%) and "never enough time to get everything done on my job" (from 40% to 60%).[5] Somewhat augmented job decision latitude also has been noted. In Europe, the proportion of workers reporting a measure of autonomy over their pace of work rose from 64% in 1991 to 72% in 1996.[73] In the U.S. "freedom to decide what I do on my job" increased from 56% in 1977 to 74% in 1997, and "my job lets me use my skills and abilities" rose from 77% in 1977 to 92% in 1997.[5] However, at least in Europe, increases in autonomy were not sufficient to compensate for heightened work intensity. The combination of augmented demands and little or no rise in control over the work process results in an increased exposure to job strain. The proportion of high-strain jobs in Europe increased from about 25% in 1991 to about 30% in 1996.[9] Unfortunately, there is no published data on the percentage of the U.S. working force experiencing job strain currently. However, as described above, employed men and women are working harder and longer today than they did 25 years ago.[36a]

Paralleling these trends in working conditions, and in large part responsible for them, new systems of work organization have been introduced by employers throughout the industrialized world to improve productivity, product quality, and profitability. Such efforts have taken a variety of forms and names, including lean production (e.g., Japanese Production Management), total quality management, cellular or modular manufacturing, and high-performance work organizations. These new systems have been extolled as reforms of Taylorism and the traditional assembly-line approach to job design.[36a]

According to a report from the U.S. Departments of Labor and Commerce, over "80% of American workers want a say in decisions affecting their jobs and how their work is performed."[71] The traditional method by which employees have influenced working conditions, including job stressors, is through the establishment of labor unions.[36] This is an example of the exercise of "collective control"[33] a strategy often utilized when prospects for exerting control individually at work are limited. However, in the U.S. the proportion of employees who are members of labor unions has declined sharply in the past 40 years.

Perhaps one explanation for the rapid increase in lean production techniques is the weakened position of labor unions.[36] As a consequence, the labor movement in the U.S. has not been able to greatly influence the enactment of legislation to improve psychosocial working conditions/reduce job strain, such as was accomplished in Scandinavia, nor has it been able to prevent the decline in real income for lower SES employees.[74-80]

Weakened unions also have been unable to prevent employers from implementing aspects of lean production such as downsizing, outsourcing to low-wage suppliers, 24-hour operations, compressed work weeks, increased overtime, contingent work, and workforce flexibility.[36a] Such trends may help explain increases in time constraints and workload demands reported in European and U.S. surveys over the past 20 years. Downsizing[12,54,72] and excessive overtime[8,10,19,68,70] can have dramatic negative effects on employee health. These trends, which result in increased job strain and ERI, contribute to CVD risk differences between upper and lower SES groups[17] and to the minimal or no recent decline in CVD incidence,[18,55,64,78] especially among lower SES workers.[18,69]

One of the consequences of lean production is the progressive disappearance of "passive" and "relaxed" jobs, with the four quadrants of the Job Strain Model collapsed into two: active versus high-strain jobs. Previously passive jobs are now accelerated (e.g., housekeepers in hotels carry phone equipment and upon completion of a task must immediately report to a supervisor for the next assignment; security

workers are routinely assigned to other tasks while simultaneously being on guard). Those who had relaxed jobs, such as some college professors/scientists, now face increasing teaching loads and incessant deadlines for grant proposals. This process of work intensification, if unchecked, may well contribute to a further sharpening of class boundaries: people will tend to be in one of two types of occupations—characterized by high or low levels of decision authority but *all* with high demands. According to the U.S. Departments of Labor and Commerce, "The stagnation of real earnings and increased inequality of earning is bifurcating the U.S. labor market, with an upper tier of high-wage skilled workers and an increasing 'underclass' of low paid labor."[71] At the same time, traditionally autonomous self-employed workers (e.g., physicians and attorneys in private practice, single shop owners) are disappearing. Physicians are working harder and are experiencing progressive loss of their decision-making authority in the setting of corporate managed care.

Leisure time is eroding, and work and home life are blending.[36a] The average U.S. married-couple family worked 247 more hours in 1996 than in 1989.[44] The quality of family life is severely compromised under these circumstances. According to a national U.S. survey in 1997, "Employees with more difficult, more demanding jobs and less supportive workplaces experience substantially higher levels of negative spillover from work into their lives off the job—jeopardizing their personal and family well-being."[5]

Implications for Public Health Policy

The evidence that psychosocial exposures are important in the etiology of hypertension and CVD and that these exposures may well be on the increase has serious implications for public health. These exposures also can affect a range of other health outcomes, including repetitive motion injuries[45]; alterations in the immune system[20]; adverse pregnancy outcomes,[7] including pregnancy-induced hypertension[37]; and negative psychological effects, such as anxiety,[62] burnout,[35] passivity,[29,31] and depression.[30] Increased rates of disorders such as repetitive motion injuries could be the "canary in the coal mine"—a possible warning of future hypertension and CVD.

SURVEILLANCE

According to the authors of the recent Tokyo Declaration, we need to institute 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 effects at amelioration. They recommend that workplaces assess both workplace stressors and health outcomes known to result from such exposures . . . on an annual basis."[1]

Worksite screening should obtain prevalence data on cardionoxious exposures (e.g., job strain) and on work-related CVD. Worksite point estimates of BP (see Chapter 7) would be particularly useful, being inexpensive and relatively simple to obtain, with ambulatory BP monitoring performed whenever possible. Holter monitoring is needed to survey the prevalence of silent myocardial ischemia, and to assess other sensitive, noninvasive parameters such as heart rate variability. Carotid ultrasound is also an invaluable screening tool. The incidence of CVD events and standard cardiac risk factors should be systematically registered. Since many large companies require annual physical exams and collect much of the relevant data, it should be a relatively simple task to enter this information into a database and make it available to those concerned with worker health. Appropriate precautions to protect employee confidentiality must always be observed.[63]

Interventions

Worksites identified as high risk for CVD should be targeted for interventions (see Chapter 13). Primary interventions would focus on creating a healthy workplace. For example, high-strain jobs could be redesigned to provide optimal levels of employee decision-making latitude and skill discretion, and workloads could be realistic, compatible with human capacity. Since the workplace appears to be a "leverage point" with regard to standard CVD risk factors (see Chapter 10), such interventions could have the additional benefit of lowering these risk factors.

A number of worksite intervention studies have specifically focused on reducing stressful features of work organization, and several have measured changes in CVD risk factors. Two Swedish studies exemplify interventions with some successes:

1. Employees of a large government agency participated in an intervention which included worker committees that developed and carried out action plans to reduce sources of workplace stress. A significant decrease in apolipoprotein B/apolipoprotein AI ratio occurred in the intervention group but not in the control group, an effect which could not be explained by smoking, eating, exercise, weight or other lifestyle factors. Stimulation from and autonomy over work significantly increased in the intervention group but remained the same in the control group.[50]

2. Researchers examined a new auto assembly work organization which contained small autonomous work groups having much greater opportunities to influence the pace and content of their work than either traditional assembly work or the Japanese management method of "lean production." Workers in the flexible sociotechnical systems organization did not show increases in systolic BP, heart rate, and adrenaline during their work shift as did workers on a traditional assembly line. In addition, catecholamines showed more rapid "unwinding" (toward non-workday baseline levels) after work in the flexible organization, particularly for female workers.[43]

The workplace is also a good setting for interventions aimed directly at traditional risk factors, e.g., dietary interventions by improved nutrition in cafeterias, exercise programs, and medical treatment (e.g., for hypertension).

Legislation

We will need societal measures to support the above initiatives. Japan and much of Western Europe have taken the lead in passing legislation making certain forms of work stress illegal and mandating healthy work. An example is the *Swedish Work Environment Act (Act No. 677, amended in 1991)* which states:

- Working conditions shall be adapted to people's differing physical and psychological circumstances.
- Employees shall be enabled to participate in the arrangement of their own job situations as well as in work changes and development that affect their jobs.
- Technology, work organization, and job content shall be arranged so that the employee is not exposed to physical or mental loads that may cause ill health or accidents.
- The matters to be considered in this context shall include forms of remuneration and the scheduling of working hours.
- Rigorously controlled or tied work shall be avoided or restricted.
- It shall be the aim of work to afford opportunities for variety, social contacts, and cooperation, as well as continuity between individual tasks.
- It shall further be the aim for working conditions to afford opportunities for personal and occupational development as well as for self-determination and occupational responsibility.

A prerequisite to implementing a "healthy work" policy is the establishment of a system of workplace surveillance to identify high-risk work environments. This, however, remains to be achieved on a broad scale.

Secondly, we may need legislation intended to provide companies with incentives to accomplish these goals. This could include a national tax on companies with excess levels of job-related risk factors and/or CVD outcomes (see Chapter 11). In this way, businesses would be encouraged to reassess their workplaces to lower job strain and other cardionoxious exposures.

Finally, in the U.S. we will need national legislation mandating a healthy workplace, similar to the laws passed in Europe and Japan (see Chapter 12).

We concur with the conclusions of the European Heart Network on Social Factors, Work, Stress, and Cardiovascular Disease in the European Union that "the substantial scientific basis of the association of psychosocial factors and cardiovascular disease risk . . . (should) ensure that social, occupational, and individual factors will not be left off the health agenda."[34] These protective steps are important to reduce the likelihood that working men and women are exposed to cardionoxious risk factors at the workplace. They recognize that today's stressful jobs are the result of human design and thus amenable to change. But taken as a totality the steps outlined above are basically a defensive strategy which fails to address the human need for fulfilling work, work that satisfies human needs for dignity, creativity, and a sense of worth.

We have now reached the point where it is possible to design work that promotes health and well-being. It is not demanding work per se that is harmful, but work without control over how one meets the job demands or uses one's skills. Tomorrow's jobs will be deliberately crafted to allow the full development of the human spirit through work which encourages—not discourages—human potential. This means creating a work environment that is conducive to human mental and physical health. A key characteristic of a "health-liberating" work environment will be the full participation of all working people in the decision-making processes surrounding the organization of work.

REFERENCES

1. The Tokyo Declaration. J Tokyo Med Univ 56:760–767, 1998.
2. Alfredsson L, Spetz C, Theorell T: Type of occupation and near-future hospitalization for myocardial infarction and some other diagnoses. Int J Epidemiol 14:378–388, 1985.
3. Belkic K, Savic C, Theorell T, et al: Mechanisms of cardiac risk among professional drivers. Scand J Work Environ Health 20:73–86, 1994.
4. Berkman L: The role of social relations in health promotion. J Psychosom Res 57:245–254, 1995.
5. Bond JT, Galinsky E, Swanberg JE: The 1997 National Study of the Changing Workforce. New York, Families and Work Institute, 1998.
6. Bosma H, Peter R, Siegrist J, Marmot M: Two alternative job stress models and the risk of coronary heart disease. Am J Pub Health 88:68–74, 1998.
7. Brandt LPA, Nielson CV: Job stress and adverse outcome of pregnancy: A causal link or recall bias? Am J Epidemiol 135:302–311, 1992.
8. Cooper C: Working hours and health. Work Stress 10:1–4, 1996.
9. European Foundation: Time constraints and autonomy at work in the European Union. Dublin, European Foundation for the Improvement of Living and Working Conditions, 1997.
10. Falger PRJ, Schouten EGW: Exhaustion, psychologic stress in the work environment, and acute myocardial infarction in adult men. J Psychosom Res 36:777–786, 1992.
11. Falk A, Hanson BS, Isacsson SO, Ostergren PO: Job strain and mortality in elderly men: Social networks, support, and influence as buffers. Am J Public Health 82:1136–1139, 1992.
12. Ferrie JE, Shipley MJ, Marmot M, et al: The health effects of major organizational change and job insecurity. Soc Sci Med 46:243–254, 1998.

13. Forsman L: Individual and group differences in psychophysiological responses to stress with emphasis on sympathetic-adrenal medullary and pituitary-adrenal cortical responses. Stockholm, Department of Psychology, University of Stockholm, 1983.
14. Fredriksson M, Sundin O, Frankenhaeuser M: Cortisol excretion during the defense reaction in humans. Psychosom Med 47:313–319, 1985.
15. Gaillard AWK: Comparing the concepts of mental load and stress. Ergonomics 36:991–1005, 1993.
16. Genes N, Vaur L, Renault M, et al: Rythme circadien des infarctus du myocarde en France: resultats de l'etude USIK (Circadian patterns of myocardial infarction in France: Results of the USIK study). La Presse Medicale 26:603–608, 1997.
17. Gonzalez MA, Artalejo FR, Calero JR: Relationship between socioeconomic status and ischaemic heart disease in cohort and case-control studies:1960–1993. Int J Epidemiol 27:350–358, 1998.
18. Hallqvist J, Diderichsen F, Theorell T, et al: Is the effect of job strain on myocardial infarction due to interaction between high psychological demands and low decision latitude? Results from Stockholm Heart Epidemiology Program. Soc Sci Med 46:1405–1415, 1998.
19. Hayashi T, Kobayashi Y, Yamaoka K, Yano E: Effect of overtime work on 24-hour ambulatory blood pressure. J Occup Environ Med 38:1007–1011, 1996.
20. Henningsen GM, Hurrell JJ, Baker F, et al: Measurement of salivary immunoglobulin A as an immunologic biomarker of job stress. Scand J Work Environ Health 18 Suppl 2:133–136, 1992.
21. House JS, Landis KR, Umberson D: Social relations and health. Science 241:540–545, 1988.
22. International Labour Office: Key Indicators of the Labour Market 1999. Geneva, International Labour Office, 1999.
23. Johnson JV: Collective control: Strategies for survival in the workplace. Int J Health Services 19:469–480, 1989.
24. Johnson JV, Hall EM: Class, work, and health. In Amick B, Levine S, Tarlov AR, Walsh DC (eds): Society and Health. New York, Oxford University Press, 1995, pp 247–271.
25. Johnson JV, Hall EM: Job strain, workplace social support, and cardiovascular disease: A cross-sectional study of a random sample of the Swedish working population. Am J Public Health 78:1336–1342, 1988.
26. Johnson JV, Hall EM, Theorell T: Combined effects of job strain and social isolation on cardiovascular disease morbidity and mortality in a random sample of the Swedish male working population. Scand J Work Environ Health 15:271–279, 1989.
27. Kaplan G, Pamuk E, Lynch JW, et al: Inequality in income and mortality in the United States: Analysis of mortality and potential pathways. Br Med J 312:999–1003, 1996.
28. Kaplan GA, Keil JE: Socioeconomic factors and cardiovascular disease: A review of the literature. Circulation 88:1973–1998, 1993.
29. Karasek R, Theorell T: Healthy Work: Stress, Productivity, and the Reconstruction of Working Life. New York, Basic Books, 1990.
30. Karasek RA: Job demands, job decision latitude, and mental strain: Implications for job redesign. Adm Sci Q 24:285–308, 1979.
31. Karasek RA: Job socialization and job strain: The implications of two related psychosocial mechanisms for job design. In Gardell B, Johansson G (eds): Working Life. London, Wiley, 1981.
32. Kawachi I, Marmot M: What can we learn from studies of occupational class and cardiovascular disease? Am J Epidemiol 148:160–163, 1998.
33. Kennedy BP, Kawachi I, Prothrow-Stith D: Income distribution and mortality: Cross-sectional ecological study of the Robin Hood index in the United States. Br Med J 312:1004–1007, 1996.
34. Kristensen TS, Kronitzer M, Alfedsson L: Social factors, work, stress, and cardiovascular disease prevention. Brussels, The European Heart Network, 1998.
35. Landsbergis PA: Occupational stress faced by health care workers: A test of the job demand-control model. J Organiz Behav 9:217–239, 1988.
36. Landsbergis PA, Cahill J: Labor union programs to reduce or prevent occupational stress in the United States. Int J Health Services 24:105–129, 1994.
36a. Landsbergis PA, Cahill J, Schnall P: The impact of lean production and related new systems of work organization on worker health. J Occup Health Psychol 4:108–130, 1999.
37. Landsbergis PA, Hatch MC: Psychosocial work stress and pregnancy-induced hypertension [see comments]. Epidemiology 7:346–351, 1996.
38. Lown B: Sudden cardiac death: Biobehavioral perspective. Circulation 76 Suppl I:I186–I195, 1987.
39. Lynch J, Krause N, Kaplan GA, et al: Workplace demands, economic reward, and progression of carotid atherosclerosis. Circulation 96:302–307, 1997.
40. Marmot M, Rose G, Shipley M, et al: Employment grade and coronary heart disease in British civil servants. J Epid Commun Health 32:244–249, 1978.
41. Marmot MG, Bosma H, Hemingway H, et al: Contribution of job control and other risk factors to social variations in coronary heart disease incidence. Lancet 350:235–239, 1997.
42. McIsaac SJ, Wilkinson RG: Income distribution and cause-specific mortality. Eur J Public Health 7:45–53, 1997.
43. Melin B, Lundberg U, Soderlund J, Granqvist M: Psychophysiological stress reactions of male and female assembly workers: A comparison between two different forms of work organization. J Organiz Behav 20:47–61, 1999.
44. Mishel L, Bernstein J: The state of working America. Washington, DC, Economic Policy Institute, 1998.
45. Moon SD, Sauter SL: Beyond Biomechanics: Psychosocial Aspects of Musculoskeletal Disorders in Office Work. London, Taylor & Francis, 1996.
46. Muller JE, Ludmer PL, Willich SN, et al: Circadian variation in the frequency of sudden cardiac death. Circulation 75:131–138, 1987.

47. Neerincx MA, Griffoen E: Cognitive task analysis: Harmonizing tasks to human capacities. Ergonomics 39:543–561, 1996.
48. Olsen O, Kristensen TS: Impact of work environment on cardiovascular diseases in Denmark. J Epidemiol Community Health 45:4–10, 1991.
49. Orth-Gomer K: International epidemiological evidence for a relationship between social support and CVD. In Shumaker SA, Czajowski SM (eds): Social Support and Cardiovascular Disease. New York, Plenum Press, 1994, pp 97–117.
50. Orth-Gomer K, Eriksson I, Moser V, et al: Lipid lowering through work stress reduction. Int J Behav Med 1:204–214, 1994.
51. Peter R, Hallqvist J, Reuterwall C, et al: Psychosocial work environment and myocardial infarction: Improving risk prediction by combining two alternative job stress models in the SHEEP Study. (Submitted), 1999.
52. Peter RW, McQuillan S, Resnick SK, Gold MR: Increased Monday incidence of life-threatening ventricular arrhythmias: Experience with a third-generation implantable defibrillator. Circulation 94:1346–1349, 1996.
53. Rabkin SW, Mathewson FAL, Tate RB: Chronobiology of cardiac sudden death in men. JAMA 44:1357–1358, 1980.
54. Richardson D, Loomis D: Trends in fatal occupational injuries and industrial restructuring in North Carolina in the 1980s. Am J Public Health 87:1041–1043, 1997.
55. Rosamond WD, Chanbless LE, Folsom AR, et al: Trends in the incidence of myocardial infarction and in mortality due to coronary heart disease, 1987 to 1994 [abstract]. New Engl J Med 339:863, 1998.
56. Rozanski A, Blumenthal JA, Kaplan J: Impact of psychological factors on the pathogenesis of cardiovascular disease and implications for therapy. Circulation 99:2192–2217, 1999.
57. Schnall PL, Landsbergis PA, Baker D: Job strain and cardiovascular disease. Annu Rev Public Health 15:381-411, 1994.
58. Schnall PL, Landsbergis PA, Schwartz J, et al: A longitudinal study of job strain and ambulatory blood pressure: Results from a 3-year follow-up. Psychosom Med 60:697–706, 1998.
59. Schnall PL, Pieper C, Schwartz JE, et al: The relationship between job strain, workplace diastolic blood pressure, and left ventricular mass index. Results of a case-control study [published erratum appears in JAMA 1992 Mar 4:267(9):1209]. JAMA 263:1929–1935, 1990.
60. Siegrist J: Threat to social status and cardiovascular risk. Psychother Psychosom 42:90–96, 1984.
61. Siegrist J, Peter R: Measuring effort-reward imbalance at work: Guidelines. Dusseldorf, University of Dusseldorf, 1996.
62. Stansfeld SA, North FM, White I, Marmot MG: Work characteristics and psychiatric disorder in civil servants in London. J Epidemiol Commun Health 49:48–53, 1995.
63. Stokols D, Pelletier KR, Fielding JE: Integration of medical care and worksite health promotion. JAMA 273:1136–1142, 1995.
64. Sytkowski PA, D'Agostino RB, Belanger A, Kannel WB: Sex and time trends in cardiovascular disease incidence and mortality: The Framingham Heart Study, 1950–1989. Am J Epidemiol 143:338–350, 1996.
65. Theorell T, de Faire U, Johnson J, et al: Job strain and ambulatory blood pressure profiles. Scand J Work Environ Health 17:380–385, 1991.
66. Theorell T, Karasek R: Should heart attack patients return to stressful jobs? Stress Med 11:219–220, 1995.
67. Tofler GH: Triggering and the pathophysiology of acute coronary syndromes. Am Heart J 134:S55–S61, 1997.
68. Tuchsen F: Working hours and ischaemic heart disease in Danish men: A 4-year cohort study of hospitalization. Int J Epidemiol 22:215–221, 1993.
69. Tuchsen F, Endahl LA: Increasing inequality in ischaemic heart disease morbidity among employed men in Denmark 1981–1993: The need for a new preventive policy. Int J Epidemiol 28:640–644, 1999.
70. Uehata T: Long working hours and occupational stress-related cardiovascular attacks among middle-aged workers in Japan. J Human Ergol 20:147–153, 1991.
71. U.S. Departments of Labor and Commerce: Fact Finding Report. Commission on the Future of Worker-Management Relations. Washington, DC, U.S. Departments of Labor and Commerce, 1994.
72. Vahtera J, Kivimaki M, Pentti J: Effect of organizational downsizing on health employees. Lancet 350:1124–1128, 1997.
73. Walters D: Health and safety strategies in a changing Europe. Int J Health Services 28:305–331, 1998.
74. Weinberg D: A brief look at postwar U.S. income inequality. Washington, DC, U.S. Census Bureau, Current Population Reports, 1996.
75. Wilkinson RG: Unhealthy Societies: The Afflictions of Inequality. London, Routledge, 1996.
76. Willich SN, Lewis M, Lowel H, et al: Physical exertion as a trigger of myocardial infarction. Triggers and mechanisms of myocardial infarction study group. N Engl J Med 329:1684–1690, 1993.
77. Willich SN, Lowel H, Lewis M, et al: Weekly variation of acute myocardial infarction: Increased Monday risk in the working population. Circulation 90:87–93, 1994.
78. Wilson PWF, D'Aostino RB, Levy D, et al: Trends in coronary heart disease: A comparison of the original (1956–1968) and offspring Framingham Study cohorts. Anaheim, CA, American Heart Association, 1991.
79. Wing S, Dargent-Molina P, Casper M, et al: Changing association between community structure and ischaemic heart disease mortality in the United States. Lancet 2:1067–1070, 1987.
80. Wolff E: Top heavy: A study of wealth inequality in America. New York, Twentieth Century Fund Press, 1995.