Myocardial Infarction and Psychosocial Risks

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Preface

This volume contains contributions presented at the Fourth International Workshop on Psychosocial Stress and Coronary Heart Disease (CHD) held in Hoehenried near Munich in July, 1979.

The workshop was organized in order to continue and enlarge upon recent discussions concerning empirical evidence and convergence in the field of stress and CHD. Proceedings of the three previous workshops were published in German by Springer-Verlag. The main topic of the 1978 conference was the present state of research in the field of type A behavior and CHD. In addition and in a certain contrast, this present volume concentrates on sociological aspects of stress experience and the development of CHD, especially myocardial infarction. It is thought that a heuristic research device should take into account both situational influences and individual predispositions on a somatic and psychological level.

The scope of this book is limited by the research areas represented by the invited participants, and the editors do not claim to give a representative overview, although they include several prominent authorities among the contributors. The final chapter gives a short critical evaluation of the present state of a difficult but promising area of interdisciplinary medical research. For technical reasons discussions held at the workshop could not be included in this volume.

It goes without saying that the editors are very grateful to all authors for their presentations as well as to Springer-Verlag for the publication of the proceedings. Again, we are especially indebted to Pharma Schwarz Sanol, Monheim, Federal Republic of Germany, whose generous funding provided the economic base for an extremely worthwhile enterprise.

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1. Risk Situations: Work Stressors, Life Events, and Loss of Social Support

Life Events, Job Stress, and Coronary Heart Disease

T. Theorell

In recent years a rapid development has taken place in the research conducted on psychosocial factors and coronary heart disease. From an overall perspective one may see two partially conflicting developments.

1. Development and further research on type A behavior. Friedman's and Rosenman's achievement and time-oriented hostile type A behavior (17) has been demonstrated in two independent prospective studies to be associated with the increased risk of developing early coronary heart disease in American men (13, 30). The excess coronary risk associated with type A behavior, furthermore, seems to be partially independent of conventional risk factors. Studies are being conducted in Europe to verify these findings in non-American settings. The observations on "coronary-prone behavior" have stimulated several lines of research.
   a) Type A behavior versus the opposite (type B behavior) in different psychological experiments, such as challenges requiring a great deal of concentration. The surroundings of the experiment have been varied with regard to controllability, noise levels, delay before introducing the challenge, etc. (5, 8, 12). The general conclusion emerging from these studies is that subjects with type A behavior have more difficulty in relaxing during restful conditions and in tolerating delays than other subjects. In addition, they are not tolerating lack of control during psychological challenge as easily as type B subjects. This is also reflected in physiologic reactions during the experiments in acute conditions.

b) Research has been focused on the difficult questions concerning the development of type A behavior. Is it genetic? Twin studies indicate that some components of type A behavior may be partly inherited (29, 36). Behavior observation studies of small children interacting with adults (parents and nonparents), with or without type A behavior, are beginning to increase our understanding of the learned aspects of type A behavior (25).

c) Several researchers have started to analyze correlates of type A behavior in a broad cultural context by utilizing cross-cultural epidemiologic data (2, 14).

d) Some research has been undertaken to use "type A behavior modification techniques" in the primary prevention of coronary heart disease (31).

2) Epidemiologic research in countries of the western hemisphere indicates that lower educational level or lower social class is associated with a higher incidence of coronary heart disease (1, 10, 15, 24).

This has generated research in two different fields:

a) One possible explanation for the increased incidence of coronary heart disease (CHD) in the lower social classes may be lack of social support. A prospective study of a county in California recently showed that various indexes of social support could be considered independent predictors of risk of death during the follow-up (4).

b) Another possible explanation for the social class-CHD incidence association may be that lower social class is associated with poorer job conditions. This has also generated research which will be described below.

Thus, we have two broad concepts, type A behavior and low social class, which are associated with coronary heart disease. Type A behavior, however, has been observed in several studies (18, 22) to be more frequent in upper social classes than in lower ones. This may illustrate the protective effects associated with the upper social class: Although the upper social groups have more type A behavior, they have a lower CHD incidence.

The research field in which I have been involved i.e., life events and job stresses in relation to CHD, is associated with the areas mentioned above. For instance, behavior pattern is associated with psychological and physiologic responses to stressors and life events; in addition, the kinds of life events that people experience may vary greatly between social classes (9). Using the schedule on stressors (stimuli) and physiologic stress reactions introduced by Kagan and Levi (19), one may describe the relationship between life events and physiologic reactions according to Fig. 1.

Rahe (28) has outlined these associations in greater detail. It should be emphasized that life events may lead not only to pathologic changes influencing "real" illness risk, but also to changes in the reporting of illness.

Catecholamines and Life Events

Epinephrine and norepinephrine have been suggested as playing a role, both in the atherogenesis and in the triggering of clinical manifestations of CHD. We therefore performed a longitudinal study to evaluate the life event score for a given week, based upon calculations of total life-change units, based on the definition of Holmes and Rahe (28) and performed follow-up personal interviews, week after week. Urine was collected during work hours (on the last day of the preceding week and always at the same time for a given person) for analysis of daytime epinephrine and norepinephrine excretion. Diet, physical activity, cigarette smoking, and coffee and alcohol consumption were held at levels as constant as possible (34).

The study showed that on an average there was a significant positive intrasubject association (linear regression) between the amount of life change during a given week and the epinephrine output during the same week (see Table 1). Thus, for most subjects it was possible to predict whether the urinary epinephrine excretion would increase or decrease from one week to the next, solely on the basis of a total lifechange calculation.

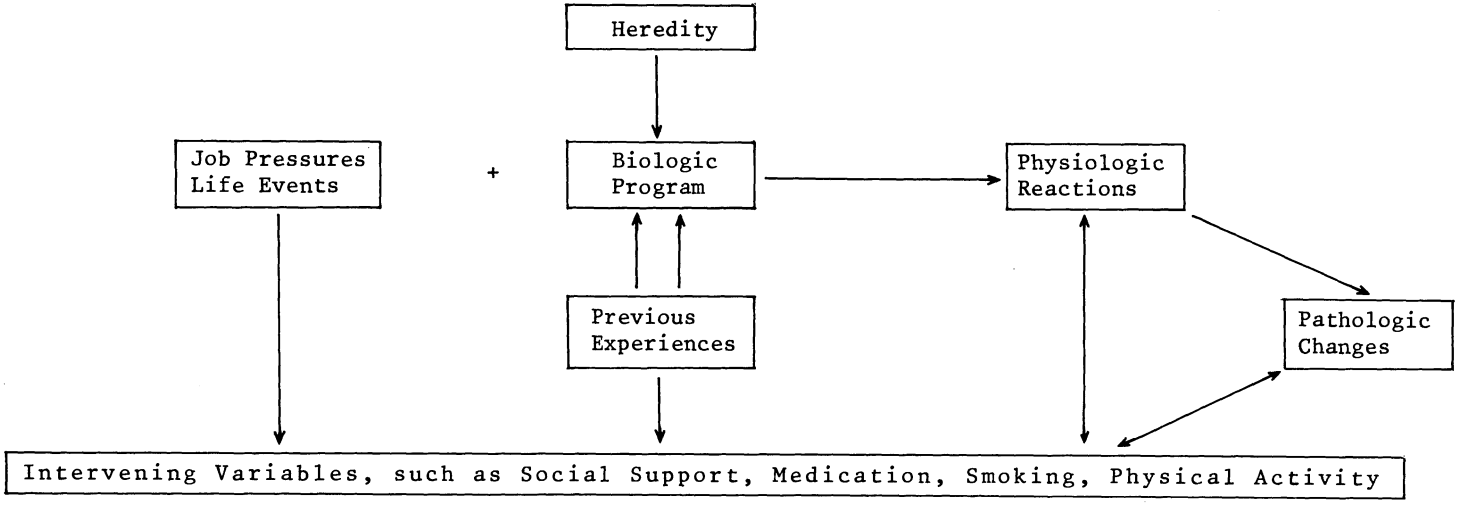

Fig. 1. Interplay between stressors and stress reactions

Table 1. Relationship between life changes (LCU/w) and epinephrine excretion[a]

	Correlation coefficient (r)	Number of accepted observations (N)	Regression coefficient (R)
1	0.120	10	0.037
2	0.073	4	0.077
3	0.709	9	0.344
4	0.185	8	0.060
5	-0.374	9	0.102
6	-0.294	8	0.106
7	0.010	9	0.003
8	0.047	7	0.724
9	0.838	6	0.486
10	-0.415	6	0.191
11	-0.240	3	2.357
12	0.602	7	0.446
13	0.865	9	0.510
14	0.347	8	0.164
15	0.434	7	0.172
16	0.738	6	0.390
17	0.138	14	0.098
18	0.343	7	0.078
19	0.836	7	0.598
20	-[b]	2	0.221
21	0.407	4	0.333

[a] Mean of regressions, 0.320; standard error, 0115; t, 2.778; P, 0.02

[b] n too small (2) to calculate correlation

There were exceptions to these findings however, as shown in Table 1. For instance, a government auditor who had very marked life changes during certain weeks of the study remained at low epinephrine excretion levels throughout the study. The subjects included in this study were male patients below retirement age who had all suffered a myocardial infarction a year before the study. A study performed quite recently by Chadwick et al. (6) on middle-aged male nonpatients employed by a large organization has shown a similar intrasubject relationship between longitudinally observed 2 week life-change total measurements and catecholamine excretion. This association, however, was found only in those subjects who exhibited type A behavior. It is of course possible that our initial study included a relatively large proportion of patients with type A behavior, although that was not recorded. This may illustrate the importance of "biologic programs" in mediating and changing the effects of life events.

What are the effects of life events on CHD risk? A number of studies have evaluated the patients' own reports of important life events that took place during the months preceding the onset of ischemic heart disease.

Connolly (7) studied the prevalence of reported life events in 91 patients and 91 controls during the months preceding a myocardial infarction. The social context of the event was thoroughly analyzed in each case; rigid criteria constructed in advance were used to judge whether a reported item was recorded as a life event. Differences were demonstrated for the prevalence of such objectively defined events during the weeks preceding disease onset, the patients' prevalences exceeding those of the controls. In a retrospective study of middle-aged women with myocardial infarction, Bengtsson et al. (3) found a higher prevalence of such objective events as divorce during the year preceding myorcardial infarction onset than during the control year in the control sample. Parkes et al. (27) demonstrated that bereavement was associated with elevated risk of cardiac death during the 3 months following bereavement.

These studies of objectively defined events have all demonstrated associations between events and risk of myocardial infarction. However, there are also studies, such as those of Hinkle (15), which have failed to demonstrate an association between objectively recorded job changes, such as promotions, demotions, and transferrals on the one hand, and risk of myocardial infarction on the other. Hinkle's study, however, demonstrated a temporal association between periods of night college studies ("double work") and near-future cardiac deaths.

Objectively defined events such as bereavement, emigration, and divorce are unusual. Efforts to screen and intervene in "risk situations" in a population would disregard many life crises that do exist at a given time if objective change were required as a basis for screening classification. The opposite tactic has therefore been to ask for "subjectively perceived periods" in the patients' lives.

Numerous retrospective studies containing all the well-known biases associated with the reporting of periods preceding onset of myocardial infarction or angina pectoris have been published. Elmfeldt et al. (11) reported on the retrospective prevalence of "stress during the 5 years preceding myocardial infarction" and demonstrated that it was significantly higher than among age-matched healthy controls in the population. Orth-Gomér and Ahlbom (26) have reported a study of 50 men with a previous myocardial infarction or angina pectoris, 50 men with excessive risk indicators for ischemic heart disease but without manifestations of illness, and 50 healthy controls without risk indicators, all individually matched in sets of three with regard to age, job position, and kind of job. The interviewer was unaware of each subject's health status. The respondent was asked to describe the "most stressful period in life." The chronology of this period was carefully evaluated. It was demonstrated that "most stressful periods," due mainly to work stressors, were much more frequently reported during the 5 years preceding onset of ischemic heart disease than during the same chronological periods in the two other groups.

Another study by Lundberg et al. (23) of the National Cooperative Chain in Sweden demonstrated that myocardial infarction patients did not report a greater number of events during the year preceding myocardial infarction than did the control group for the same chronological period. However, when the subjects were asked to subjectively rate the amount of upset caused by the events, and when these subjective scores were subsequently added to one another in each individual case, the patient group was demonstrated to have significantly higher total scores than the control group.

A prospective study of nearly 7000 41-61-year-old construction workers in Stockholm has been published (33, 35). These workers replied to a questionnaire comprising approximately 60 items on family and work conditions the year (1972) preceding a 2 year follow-up study. A factor analysis, performed before follow-up data were known, revealed three clusters of items within the questionnaire. One, "psychosocial work load," dealt with items describing the work situation; one described "recent changes in

family structure"; and the last described "long-lasting family troubles."

A 2-year follow-up investigation of the death and hospitalization registers in the greater Stockholm area revealed an incidence of myocardial infarction in the studied sample comparable to that of Swedish males in the same age category. Figure 2 shows the frequency with which each item in the "psychosocial work load" cluster was reported in the entire group studied after exclusion of all those who had long-lasting illness during the year preceding the study. It also shows the frequency with which these items were reported among those who developed a myocardial infarction during the 2-year follow-up period. Most items were reported in excess in the prospective myocardial infarction cases. When all

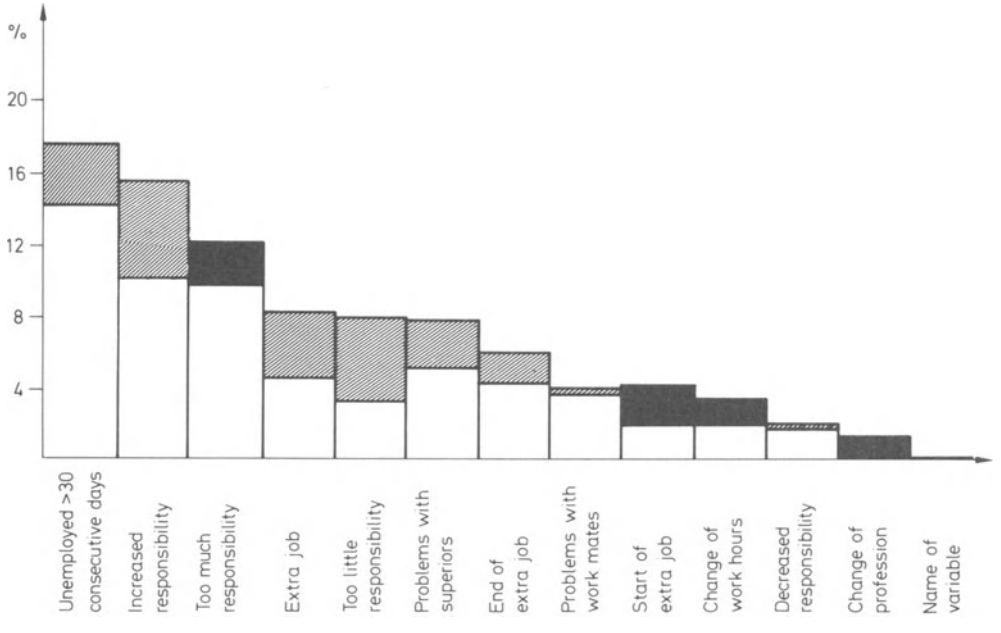

Fig. 2. Prelevance (%) of reported items in the psychosocial work load cluster obtained by means of factor analysis before follow-up data were available. Total group: 5187 41-61-year-old building construction workers who had not had long-lasting (≥ 30 consecutive days) work absenteeism (all diagnoses during year preceding follow-up). MI group: 51 cases of myocardial infarction (deaths included) during 2 years of follow-up.
▨ *Prelevance in MI group exceeds that of total studied group;*
■ *prelevance in total studied group exceeds that of MI group*

the items were combined (see Table 2) by simply assigning those who had responded "yes" to any question among the items to the "psychosocial work load" group and all the others into the "no psychosocial work load" group, it became evident that there was a significant association between "psychosocial work load" and myocardial infarction risk, particularly when cases with a known history of heart disease had been excluded. It should be pointed out that since this analysis was confined to working subjects without any long-lasting illness during the preceding year, all cases with severe and abvious symptoms were excluded from the total myocardial infarction group as well.

A stepwise multiple regression using dichotomized (yes or no) variables is shown in Fig. 3. All the nearly 7000 participants, except those who had been absent from work during the preceding year because of heart disease, hypertension, or diabetes, are included in this analysis. The analysis indicates that it was easier to predict elevated risk of myocardial infarction survival (n, 39) than total myocardial infarction risk (n, 61). The impor-

Table 2. Prospective cardiovascular-cerebrovascular death: Cases and individually matched controls exposed (1) and unexposed (0) to the combination of low freedom in work schedule and high job demand

	Cases	Controls		
1	0	0	0	1
2	0	0	0	0
3	1	0	1	0
4	0	0	0	0
5	1	0	0	0
6	0	0	0	0
7	0	0	0	0
8	0	0	0	0
9	0	0	0	0
10	1	0	0	1
11	0	1	0	0
12	0	0	0	0
13	1	0	0	0
14	0	0	0	0
15	1	0	0	0
16	0	0	0	0
17	0	0	0	0

relative risk; 5.8:1; $p < 0.03$

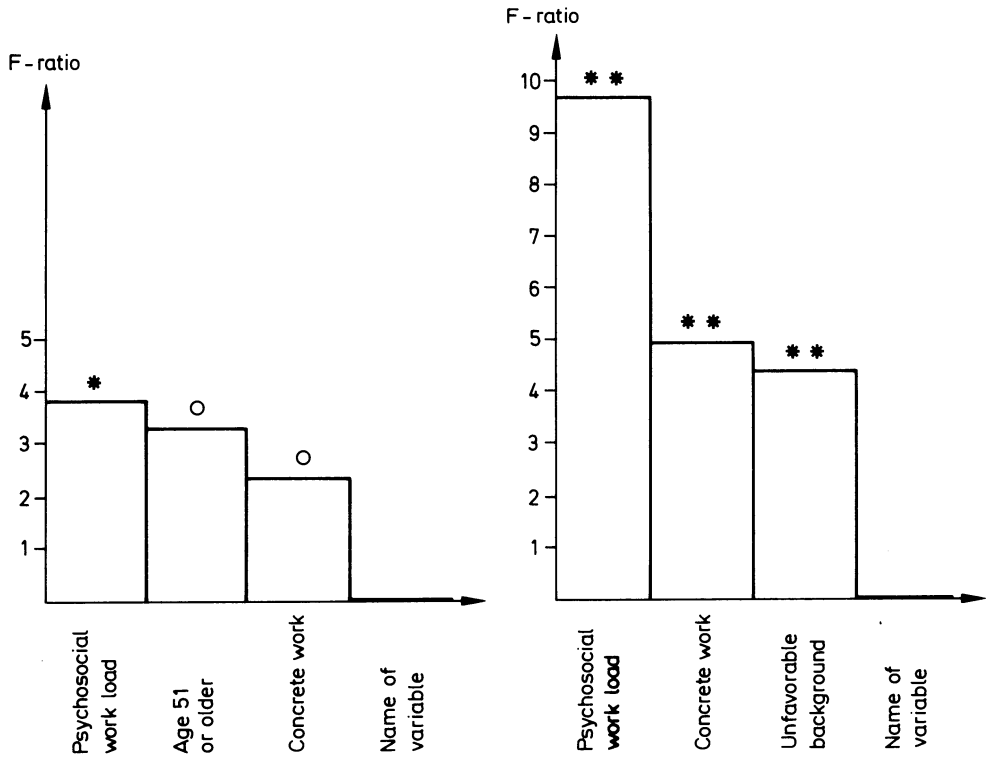

*Fig. 3. Results of step-wise multiple regression analysis of age, profession, present cigarette smoking, and five psychosocial variables in the prediction of myocardial infarction (n, 61; left) and survivals of myocardial infarction after exclusion of deaths (n, 39; right) in 6723 building construction workers after exclusion of those with long-lasting (≥ 30 consecutive days) work absenteeism because of heart disease, hypertension, or diabetes during year preceding follow-up. Three first steps of each analysis. o, independent contribution not significant; *, independent contribution, P < 0.05; **, independent contribution, P < 0.01*

tant variables seemed to be "psychosocial work load" and concrete work, the most heavily demanding work involving many components of static muscle work. Current cigarette consumption and absenteeism exceeding 7 days during the preceding year had no predictive value, whereas "unfavorable background" (growing up as a late child in a large family or in a broken home) was predictive of myocardial infarction survival. Data on blood pressure and serum cholesterol were not available. In the same study we also had the opportunity to evaluate, during the 1st year of follow-

up, all cases recorded in the insurance registers of (a) ulcer or gastritis, (b) low back pain and degenerative joint disease, and (c) severe accidents causing at least 30 consecutive days of work absenteeism. "Psychosocial work load" had no predictive value in relation to these other illnesses. "Long-lasting family troubles" and/or "recent changes in family structure" were not predictive of increased risk of myocardial infarction. This may support the hypothesis that work stress has particular relevance to myocardial infarction. The study of construction workers is not concerned with life events only. However, two of the most important variables in the "psychosocial work load" cluster, namely, "increased work responsibility last year" and "unemployment lasting for more than 30 consecutive days last year," should be regarded as "life events." In this setting, aging leading to a decreasing gap between physical job demands and peak capacity, which is particularly relevant in the concrete worker category, work pressures because of piecework wages, and changes in the structure of the work situation may play roles in the complex web of causation.

It is evident that in the Swedish studies particular emphasis is placed on work in the association between life events and myocardial infarction. This may not necessarily be the case in other countries. Perhaps Swedish men have more inclination to become "addicted" to work than others. In Orth-Gomér's study of middle-aged working men in Stockholm (see above), a comparison was made with employed men in New York who were matched for age, kind of job, and job position. Figure 4 shows marked differences. The Americans frequently blamed their families for stressful periods, whereas the Swedes nearly always blamed their work.

People with different backgrounds rate the importance of events differently. Middle-aged Finnish immigrants in the Stockholm area were compared with native Swedes in the same region (37). The Finnish immigrants reported the same amount of changes during the year preceding examination as the native Swedes living in the same region, but the immigrants estimated these changes to be significantly less important than did the native Swedes.

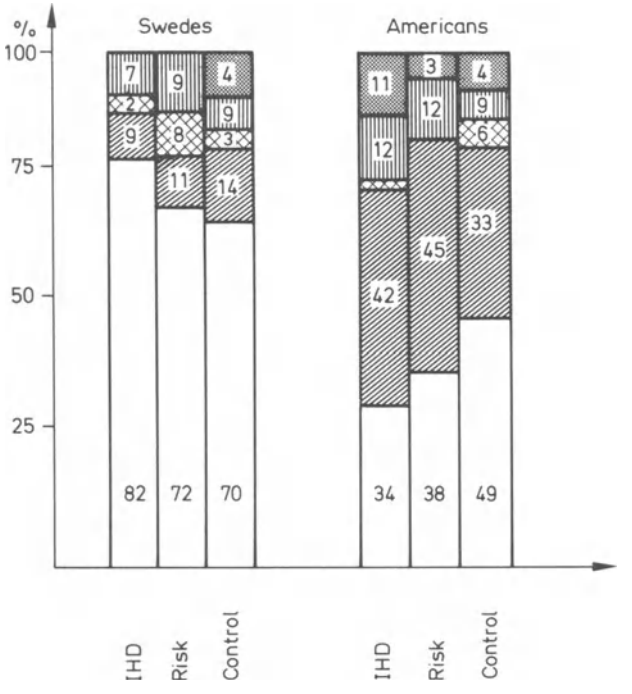

Fig. 4. Proportions of "sources of stress" during "most stressful period in life" in two samples of middle-aged working men in New York and in Stockholm. IHD, ischemic heart disease; risk, elevated risk of developing ischemic heart disease; control, healthy controls. The American and Swedish subgroups are matched in sets of three with regard to age, type of job, and job position. ⊡, disease; ▥ other; ▩ education; ▨ family; □ work

In summary, the available literature on life events gives considerable support to the idea that perceived psychosocial pressures may accelerate the onset of ischemic heart disease. However, there are several conceptual difficulties inherent in this theory. One of these is that it is impossible to know how much the association depends on internal and external processes, respectively. Furthermore, the pathophysiologic mechanisms linking life events and ischemic heart disease manifestations are largely unknown. Catecholamines and corticosteroids may be involved, since these hormones have been linked to both psychosocial processes and to relevant cardiovascular reactions. However, other endocrine systems, such as the renin-angiotensin system, sex hormones, growth hormones, and prolactin may also be involved

(32), as well as behavioral considerations such as changes in smoking habits.

One problem with the "psychological work load" utilized in the study of construction workers is that it contains a mixture of job dimensions. If these dimensions affect the risk of coronary heart disease differently, important associations may be lost. What is it in the work situation that is really important? Much attention has been paid to the fact that one kind of work situation may be perceived differently by different individuals and may have different physiologic effects on different individuals. The work situation itself, on the other hand, has received little attention. As pointed out by Karasek (20) in his analysis of job stress, high psychological demands may have different effects, depending on whether the work situation allows decision making. That is to say, a person who has a very demanding job may not perceive it as stressful if he has the freedom to decide when and how to perform the work.

In a study, of nearly two thousand employed Swedish men, randomly selected according to birth date, during 1968 - 1974, we used the model for job environment analysis constructed by Karasek (20). This model implies that high demands, here operationally defined as a "yes" response to two questions made in a personal interview ("Is your work hectic?" and "Is your work psychologically demanding?"), may be predictive of cardiovascular illness risk only if there is a concomitant lack of control and decision latitude in the job. Consequently, we hypothesized that a measure of limited freedom in the work schedule (revealed by a "No" response to at least two of the three following questions: "Can you place a private telephone call during work time?"; "Can you receive a private visitor at the work place for ten minutes during work hours?"; and "Can you leave the work place for thirty minutes during work hours for a private errand without speaking to your supervisor?"), in combination with "high work demands," would predict increased risk of cardiovascular or cerebrovascular death during the follow-up period.

During the follow-up period from 1968 to 1974, 17 deaths were identified in the sample. Each of these deaths was blindly matched with three living subjects from the same sample with regard to (1) cardiovascular symptoms at the start of the follow-up period, (2) cigarette smoking, (3) age, and (4) education. A paired analysis showed that there was a 5.8 times greater likelihood of death (P, 0.03) in the high-demand low-work schedule freedom category (21) (see Table 2). A related measure, job decision-making latitude, in combination with high work demands, was very significantly associated with the prospective development of self-reported symptoms of heart disease, but not significantly predictive of cardiovascular-cerebrovascular death, after matching with the above-mentioned variables.

Thus, preliminary studies seem to indicate that combinations of high work demands and limited outlets for tension and self-fulfillment in the work environment may have a pathogenetic significance in the development of coronary heart disease.

The studies I have mentioned should be regarded merely as examples of the work being done in this rapidly growing field. They appear to show that psychosocial job-related factors are associated with the risk of early development of coronary heart disease.

References

1. Antonovsky A (1968) Social class and the major cardiovascular diseases. J Chronic Dis 21:65
2. Appels A (1972) Is cardiac infarct a cultural disease? (in Dutch) Tijdschr Soc Geneeskd 50:446
3. Bengtsson C, Hällström T, Tibblin G (1973) Social factors, stress experience and personality traits in women with ischemic heart disease compared to a population sample of women. Acta Med Scand [Suppl] 549:82
4. Berkman LF, Syme SL (1979) Social networks, host resistance and mortality: A nine-year follow-up study of Alameda county residents. Am J Epidemiol 109:186
5. Carver CS, Coleman AE, Glass DC (1976) The coronary prone behavior pattern and the suppression of fatigue on a treadmill test. J Pers Soc Psychol 33:460

6. Chadwick JH, Chesney MA, Black GW, Rosenman RH, Sevelius G (1979) Psychological job stress and coronary heart disease. Stenciled report, Stanford Research Institute, Stanford

7. Connolly MB (1976) Life events before myocardial infarction. J Human Stress 2:3

8. Dembroski TM, McDougall JM, Shields JL, Petitto J, Lushene R (1978) Components of the Type A coronary-prone behavior pattern and cardiovascular responses to psychomotor performance challenge. J Behav Med 1:159-176

9. Dohrenwend BS (1973) Social status and stressful life events. J Pers Soc Psychol 2:225

10. Dyer AR, Stamler J, Shekelle RB, Schoenberger J (1976) The relationship of education to blood pressure. Findings on 40.000 Chicagoans. Circulation 54:987

11. Elmfeldt D, Wilhelmsen L, Wedel H, Vedin A, Wilhelmsson C, Tibblin G (1976) Primary risk factors in patients with myocardial infarction. Am Heart J 91:412

12. Glass D (1977) Behavior patterns, stress and coronary disease. Wiley New York

13. Haynes S (1979) Type A behavior and coronary heart disease risk - prospective data in the Framingham study. Personal communication

14. Henry JP, Stephens PM (1977) Stress, health and the social environment - a sociobiologic approach to medicine. Thomas, Springfield, Ill.

15. Hinkle LE Jr (1974) The effect of culture change, social change and changes in interpersonal relationships on health. In: Dohrenwend BS, Dohrenwend BP (eds) Stressful life events - their nature and effects. Wiley, New York, pp 9-44

16. Hinkle LE Jr, Whitney HL, Lehman EW, Dunn J, Benjamin B, King R, Plakun A, Flehinger B (1968) Occupation, education and coronary heart disease. Science 161:238

17. Jenkins CD (1976) Recent evidence supporting psychologic and social risk factors for coronary heart disease. N Engl J Med 294:987-994; 1 033-1 038

18. Jenkins CD (1978) A comparative review of the interview and questionnaire methods in the assessment of the coronary-prone behavior pattern. In: Dembroski TM, Weiss SM, Shields JL, Haynes SG, Feinleib M (eds) Coronary-prone behavior. Springer, Berlin, Heidelberg, New York, pp 71-88

19. Kagan A, Levi L (1971) Adaptation of the psychosocial environment to man's abilities and needs. In: Levi L (ed) Society, stress and disease, vol I, The psychosocial environment and psychosomatic disease. Oxford University Press, London, pp 399-404

20. Karasek RA (1979) Job demands, job decision latitude and mental strain: Implication for job redesign. Adm Sci Q 24:285

20a. Karasek RA, Ahlbom A, Baker D, Marxer F, Theorell T (to be published) Job decision latitude, job demands and coronary

heart disease - a cross-sectional and prospective study of Swedish men. Am J Publ Health

21. Karasek RA, Ahlbom A, Marxer F, Theorell T (1979) Perceived work demands, schedule freedom at work and prospective cardiovascular-cerebrovascular death - a matched case-control study. J Human Stress

22. Kornitzer M (1977) In: Tybjaerg Hausen A, Schnor P, Rose G (eds) Ischemic heart disease, the strategy of postponement. FADL, Copenhagen

23. Lundberg U, Theorell T, Lind E (1975) Life changes and myocardial infarction, individual differences in life change scaling. J Psychosom Res 19:27

24. Marmot MG, Rose GA, Shipley M, Hamilton PJS (1978) Employment-grade and coronary heart disease in British civil servants. J Epidemiol Community Health 32:244

25. Matthews K (1977) Caregiver-child interactions and the Type A coronary-prone behavior pattern. Child Dev 48:1 752

26. Orth-Gomér K, Ahlbom A (1979) Impact of psychological stress on ischemic heart disease when controlling for conventional risk indicators. J Human Stress

27. Parkes CM, Benjamin B, Fitzgerald RG (1969) Broken heart, a statistical study of increased mortality among widowers. Br Med J 1:740

28. Rahe RH (1973) Psychosocial characteristics of subjects with myocardial infarction in Stockholm. In: Gunderson EKE, Rahe RH (eds) Life stress and illness. Thomas, Springfield, Ill., pp 90-104

29. Rahe RH, Hervig L, Rosenman RH (1978) Heritability of type A behavior. Psychosom Med 40:478

30. Rosenman RH, Brand RJ, Jenkins CD, Friedman M, Straus R, Wurm M (1975) Coronary heart disease in the Western Collaborative Group Study: Final follow-up experience of 8 1/2 years. JAMA 233:872

31. Roskies E (1979) Considerations in developing a treatment program for the coronary-prone (Type A) behavior pattern. In: Davidson P (ed) Behavioral medicine: Changing health life styles. Bruner/Mazel, New York, pp 94-110

32. Selye H (1970) Experimental cardiovascular diseases. Springer, Berlin, Heidelberg, New York

33. Theorell T, Flodérus-Myrhed B (1977) "Workload" and risk of myocardial infarction - a prospective psychosocial analysis. Int J Epidemiol 6:17

34. Theorell T, Lind E, Fröberg J, Karlsson CG, Levi L (1972) A longitudinal study of 21 coronary subjects - life changes, catecholamines and related biochemical variables. Psychosom Med 34:505

35. Theorell T, Lind E, Flodérus B (1975) The relationship of disturbing life-changes and emotions to the early development of myocardial infarction and other serious illnesses. Int J Epidemiol 4:281

36. Theorell T, de Faire U, Schalling D, Adamson U, Askevold F (1979) Personality traits and psychophysiological reactions to a stressful interview in twins with varying degrees of coronary heart disease. J Psychosom Res 23:89-99

37. Theorell T, Vigborg A, Kallner A, Dehlén G, Wålinder O (to be published) A comparison between Finnish immigrants and native Swedes in the greater Stockholm region - social and medical risk indicators of ischemic heart disease. Scand J Soc Med

Work Load and Coronary Heart Disease

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Introduction

The concept of daily life stress, particularly stress due to work load or "job stress," seems to be embedded in the socio-cultural setting of industrialized populations in such a way that it is self explanatory for all kinds of sustained illness, such as a myocardial infarction. The possible relationship between stress and coronary heart disease is regularly publized by the mass media. On the other hand, prospective studies of coronary heart disease were started more than 25 years ago, but little attention was paid to stress; the possible relationship between stress and coronary heart disease (CHD) has been flatly denied because of the difficulty in defining the term, and on account of two examples, which seemingly negate the relationship: the "stress-free" Finnish lumberjacks having a very high incidence of CHD opposed to the "stressful" Japanese with a rather low CHD incidence. The low epidemiologic interest in job stress as a risk factor for CHD is counterbalanced by the numerous animal and human experimental and clinical studies carried out since Selye developed his three-phased stress concept (58).

The pathogenetic pathways - through the neuroendocrine system, starting in the cortex and ending with the secretion of catecholamines and corticosteroids in the adrenals - which lead to intravascular thrombosis and increased heart rate and blood pressure should stimulate interest in the study of the work load in relation to CHD, as exemplified by the work of Theorell and Flodérus-Myrhed (66).

Our personal interest in the study of work-load arose from a long-term prospective study of two cohorts of middle-aged Belgian males. The 5- and 10-year incidence of CHD was significantly higher in cohort I compared to cohort II, the former employed in a private bank, the latter in a semipublic savings bank (34, 35). These observations were unexpected since the distributions of all classic coronary risk factors were quite similar in the two cohorts.

A job stress questionnaire, sent to all retired subjects of both group 1 year after the 10-year follow-up, disclosed significantly higher self-perceived job stress in the subjects of cohort I during the last 10 years of their professional activity (32).

Based on these results, a method for measuring job stress was further developed and has been used during baseline screening for a prospective study on the relationship between physical activity and CHD in middle-aged males. In the first part of this paper we will present the baseline data from this study, while in the second we will try to develop a working hypothesis concerning the relationship between work setting, job stress, and coronary heart disease.

Materials and Methods

The population sample was selected from employees in industry. All males aged 40-55 years, regularly employed by factories were eligible to participate. In total, 3179 subjects were invited and 74.3% attended the screening examination.

Screening involved the collection of a fasting person's blood sample, the administration of various questionnaires, blood pressure, height, and weight measurement, electro- and vectocardiographic recordings, ankle and humeral systolic blood pressure measurements by means of Doppler ultrasound, and finally, the performance of a submaximal graded exercise test on a bicycle ergometer and a 24-h continuous ECG recording.

Blood samples were drawn in supine position; the subjects had been fasting for at least 12 h. Blood group, serum triglycerides, total cholesterol, and HDL-cholesterol were determined. All samples were analyzed in a central laboratory. Triglycerides were measured according to the method described by Klose et al. (33) (Auto-Analyser, Technicon Instruments Corp., New York). Cholesterol was measured by the Auto-Analyser, according to Boy et al. (3). HDL-cholesterol was measured according to Steele et al. (62). An internal quality control system has been operating for years, and no major fluctuations were observed over the screening period. An external quality control system exists with the WHO Regional Lipid Reference Center, Institute for Clinical and Experimental Medicine, Prague, director Dr. D. Grafnetter.

Some questionnaires were self-administered, while others were given by the interviewer.

A medical questionnaire was designed to gather information on smoking habits and personal and family antecedents of disease; it also included the standardized questionnaire from the London School of Hygiene in its self-administered version with respect to angina, chronic bronchitis, and intermittent claudication (IC) (51).

We further used the Jenkins Activity Survey (27), a self administered questionnaire which defines the coronary-prone type A behavior pattern, characterized in the original study (18) by aggressivity, impatience, self-imposed deadlines, and job involvement. This questionnaire has been validated using another Belgian population (31).

Another self-administered questionnaire, developed by one of the authors, is the "Job Stress Questionnaire," which results in a score of "work load" based on 13 items dealing with dissatisfaction at work, complaints about physical work conditions, changes in work conditions, work pace, responsibility, promotion, salary, professional relationships, and psychosomatic complaints.

All the self-administered questionnaires were completed at home and reviewed at the screening examination by a technician. Two additional questionnaires were administered by the interviewer:

1) The Minnesota Leisure Time Physical Activity Questionnaire, developed at the Laboratory of Physiological Hygiene, School of Public Health, University of Minnesota (65), and validated by objective measures of physical fitness (36). The questionnaire evaluates energy expenditure in leisure time physical activity; energy expenditure is expressed as total activity index (AMI): light AMI, moderate AMI, and heavy AMI.
2) To assess physical activity on the job, a questionnaire was designed by one of the authors. Values for caloric expenditure in various prefessional activities were adapted from Durnin and Passmore (13) and from Spitzer and Hettinger (61). The questionnaire was designed to gather detailed information on attitudes, movements, and postures during a working day. Classification of the subjects into distinct categories of physical activity was based on a booklet published by the Commisariat Général à la Promotion du Travail (1).

Besides the standard 12-lead ECG, we also recorded the X Y Z orthogonal leads on paper and tape. The analysis of the 12-lead ECG was made by hand (Minnesota Code), while the orthogonal leads were analyzed by a Hewlett Packard computer 2110 using the Pipberger Program (49). This analysis enabled us to prospectively compare the prognostic value of Minnesota code items with computer measurements in different lead systems. Statistical procedures include classical univariate and multivariate analyses (60).

Results

On an Individual Basis

The Job-Stress Questionnaire (JSQ) was administered to 2106 subjects. The average job stress score (JSS) was 0.06 and was not significantly age-related (Table 1).

Table 1. Job stress score and age

Age	Job stress score (JSS)		test[a]
40 - 44	0.16		
45 - 49	0.08		2.43
50 - 55	-0.08		N.S.
	$\bar{M}$		s.d.
Total	0.06	±	2.09

[a]Analysis of variance

There is a significant increase of the mean JSS with educational level. White-collar workers show the highest JSS while blue-collar workers have the lowest; executives fall in between. No difference is observed with regard to marital status (Table 2). When socioprofessional class and study level are combined, a JSS gradient persists over the three socioprofessional classes. We observed the highest score in blue-collar workers with a university degree and the lowest in executives with an elementary degree (Table 3).

Table 4 shows Pearson correlation coefficients of JSS with coronary risk factors; except for a low-order positive correlation with HDL-cholesterol and a negative one with body mass index, no significant correlations are observed.

Table 2. JSS and social variables

	JSS	F
Educational Level	$\bar{M}$	
Elementary	-0.63	
Secondary	0.25	51.7[b]
University	0.72	
Socioprofessional level		
Blue-collar workers	-0.58	
White-collar workers	0.48	62.0[b]
Executives	0.24	
Marital status		
Married	0.05	0.16[c]
Alone[a]	0.12	

[a]Alone: bachelors, widowers, or divorced subjects
[b]$P \leq 0.001$
[c]Not significant

Table 3. JSS in relation to socioprofessional class and educational level

	Blue-collar workers	White-collar workers	Executives
Elementary	-0.84	-0.16	[-0.07]
Secondary	-0.32	0.56	0.14
University	[1.25]	0.84	0.54

Table 4. Pearson correlation coefficients (with JSS)

Heart-rate	Systolic blood pressure	Diastolic blood pressure	Chol-esterol	HDL-Chol-esterol	Tri-glycer-ides	Body mass index[a]
-001[b]	0.01[b]	0.03[b]	0.03[b]	0.04[c]	-0.03[b]	-0.05[c]

[a]Boody mass index = Weight/(height)^2
[b]Not significant
[c]$P \leq 0.05$

A significant difference in JSS is observed according to smoking habits; the lowest score is found in smokers while the highest is observed in ex-smokers (Table 5).

We observed a significant negative correlation between the JSS and physical fitness as defined by the load reached on the bicycle ergometer for a heart rate of 150/min. Divided in quartiles, better physical fitness is associated with lower job stress (Table 6).
No significant correlation was observed between total leisure time activity (AMI) and the JSS (Table 6).

Subjects reporting a positive history of current or former asthma had a significantly higher JSS compared to subjects with a negative history (Table 7).

Table 5. JSS and smoking habits

Smokers	Non-smokers	Ex-smokers	F test
-0.08	0.11	0.24	5.16[a]

[a]$P \leq 0.01$

Table 6. JSS, physical fitness, and leisure time activity

A. JSS and physical fitness (work load/150)					
Quartiles	I	II	III	IV	3.4
	0.19	0.13	-0.12	-0.25	
B. JSS and leisure time activity (total AMI)					
Quartiles	I	II	III	IV	1.12
	-0.06	0.06	0.04	0.18	

[a]$p \leq 0.05$
[b]Not significant

Table 7. History of asthma

	JSS	F
Actual		
No	0.05	7.35
Yes	1.62	
Former		
No	0.03	9.44
Yes	0.80	

[a]$p \leq 0.01$

Subjects taking medication had a higher mean JSS compared to those who did not; the highest score was found in those subjects taking tranquilizers (Table 8).

Two multivariate regression analyses of the job stress score were performed. In the first, 16 variables were entered in the equation. Five variables were significantly related to the JSS:

1) Professional status: white-collar workers had the highest and blue-collar workers the lowest JSS.

Table 8. Medication

No	Anti-hypertensives	For heart and blood vessels	Tranquilizers	F test
-0.04	0.16	0.36	1.59	20.2[a]

[a]$p \leq 0.001$

2) Study level: the highly educated showed a higher JSS.
3) Smoking habits: the highest JSS is observed in ex-smokers and the lowest in actual smokers, with nonsmokers falling in between.
4) A correlation with quartiles of scores for leisure time activity AMI.
5) A negative correlation for physical fitness (work-load).

The total variance of JSS explained by these five variables is only 8.1% (Table 9).

In the second analysis, educational level and professional status were excluded; only three variables were significantly related to the JSS: smoking habits, physical fitness (negatively correlated), and age (negatively correlated). The total explained variance is even lower: 2.5% (Table 10). The last table of results on an individual basis shows the relationship of JSS to coronary heart disease: in cases of angina it is significantly positive, while no relationship is found with ECG abnormalities when angina is excluded (Table 11).

Based on Occupational Units

This study is occupationally based and five units were formed: A, B, C, and E with more than 400 subjects on their payroll, with

Table 9. Multiple regression analysis of JSS in 1303 subjects

Variables	Coefficient	R^2	F test
Prof. status (white collar; executive; blue collar)	-0.401	0.049	36.41[a]
Study level (elementary; secondary; university)	0.389	0.063	14.46[a]
Smoking (S; Non; Ex)	0.154	0.067	5.53[b]
Quartiles of total AMI	0.114	0.073	4.25[b]
Work load	-0.006	0.077	4.34[b]

N.S.: BMI; cholesterol, age, heart rate; marital status; quintiles of heavy AMI; Triglycerides; systolic blood pressure; α-cholesterol; diastolic blood pressure

[a] $P \leq 0.001$; [b] $P \leq 0.05$

Table 10. Multiple regression analysis of JSS in 1323 subjects

Variables	Coefficient	R^2	F
Smoking (S; Non; Ex)	0.217	0.007	10.74[a]
Age	-0.027	0.019	4.10[b]
Workload	-0.008	0.022	5.85[b]

N.S.: BMI; cholesterol; quartiles of total AMI; heart rate; marital status; diastolic blood pressure; α-cholesterol; quintiles of heavy AMI; triglycerides; systolic blood pressure.

[a] $p \leq 0.001$; [b] $p \leq 0.05$

unit D being an agglomerate of six small units, each with less than 200 subjects.

Table 12 vertically shows the ranking of the mean job stress score, globally and in each of the three educational level classes: all rankings are quite homogeneous except for one major discrepancy at the university level in unit A.

We have shown a relationship between educational level and JSS on an individual basis; on unit-based means (circled figures) there was also a strong tendency toward an increase in mean JSS with educational level, except for unit C.

Figure 1 shows the ranking of mean JSS and systolic blood pressure, both adjusted for age and educational level: two of the five occupational units show major differences between JSS and systolic blood pressure rankings. The ranking relationship between heart

Table 11. JSS and prevalence of CHD

Angina		JSS	F
No		-0.28	
Suspect		0.73	61.34[a]
Yes		1.07	
ECG (Minnesota code: I_{1-2-3}, IV_{1-2-3}, or V_{1-2-3})			
Angina Excluded	No	0.007	0.0[b]
	Yes	0.010	

[a] $p \leq 0.001$; [b] Not significant

Table 12. Occupational units', JSS and educational level ranking

	Educational level		
Unit	Elementary	Secondary	Universitary
A. 1[a]	1 (1)[b]	1 (2)	4 (3)
B. 2	2 (1)	2 (3)	1 (2)
C. 4	5 (3)	4 (1)	2 (2)
D. 5[c]	4 (1)	5 (2)	5 (3)
E. 3	3 (1)	3 (2)	3 (3)

[a]Lowest mean JSS
[b]Lowest JSS and mean J.S.S. in unit A according to educational level
[c]Highest mean JSS

rate and JSS is quite good: three occupational units show the same rank and two differ only by one rank unit (Fig. 2). Figure 3 shows the ranking of mean JSS and the prevalence of ECG abnormalities (Minnesota code I_{1-2-3}, IV_{1-2-3}, or V_{1-2-3}). For one occupational unit there is a major difference (of three rank

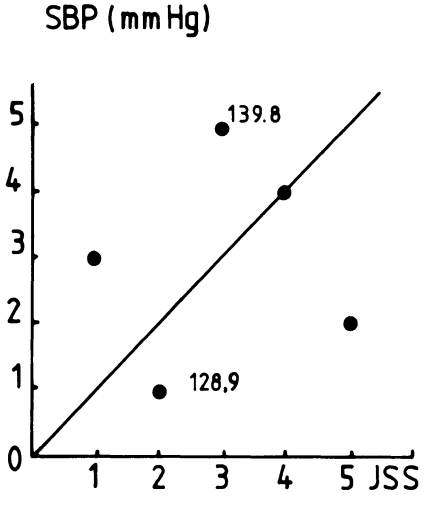


Fig. 1. Ranking of mean occupational unit job stress score (JSS) and systolic blood pressure (SBP) adjusted for age and study level

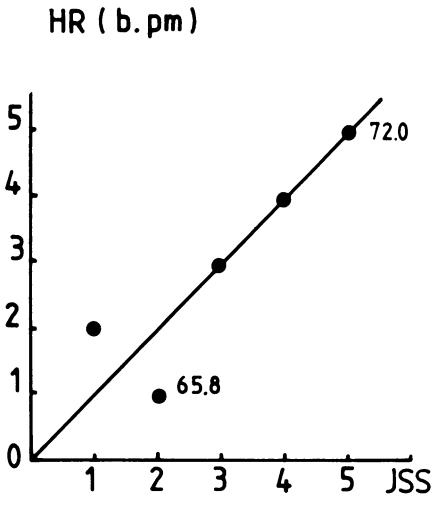


Fig. 2

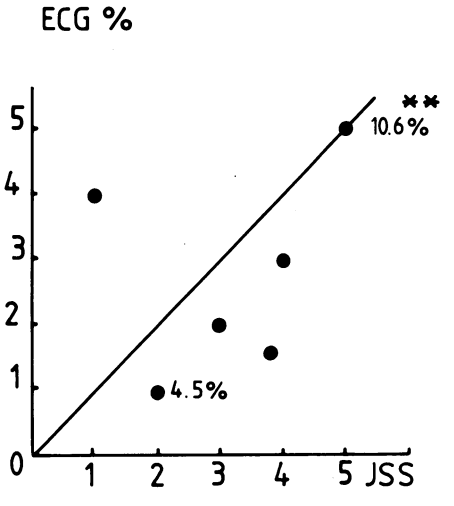


Fig. 3

Fig. 2. Ranking of mean occupational units JSS and heart rate adjusted for age and study level

Fig. 3. Ranking of mean occupational unit JSS and prevalence of ECG abnormalities (Minnesota code: I_{1-2-3}, IV_{1-2-3}, or V_{1-2-3}) adjusted for age and study level

units) between JSS and ECG while for the others, the ranking is quite satisfactory.

Discussion

The road leading from job stress to coronary heart disease is long and not yet paved with overwhelmingly convincing evidence. A given work setting is defined through its physical, cultural, and psychosocial characteristics which can lead to an environmental burden. On the other hand, individuals are defined through their psychological traits and behavior patterns such as type A: traits of neuroticism, anxiety, and depression, all of which have been related to the prevalence or incidence of CHD (4, 28, 41, 48). The work burden (stressor) impinges on the individual's coping mechanisms, resulting in a given amount of strain leading to stress - in this case, job stress. This, in turn, will implicate, according to Selye's stress concept, physiologic neuroendocrine reactions (58) (Fig. 4).

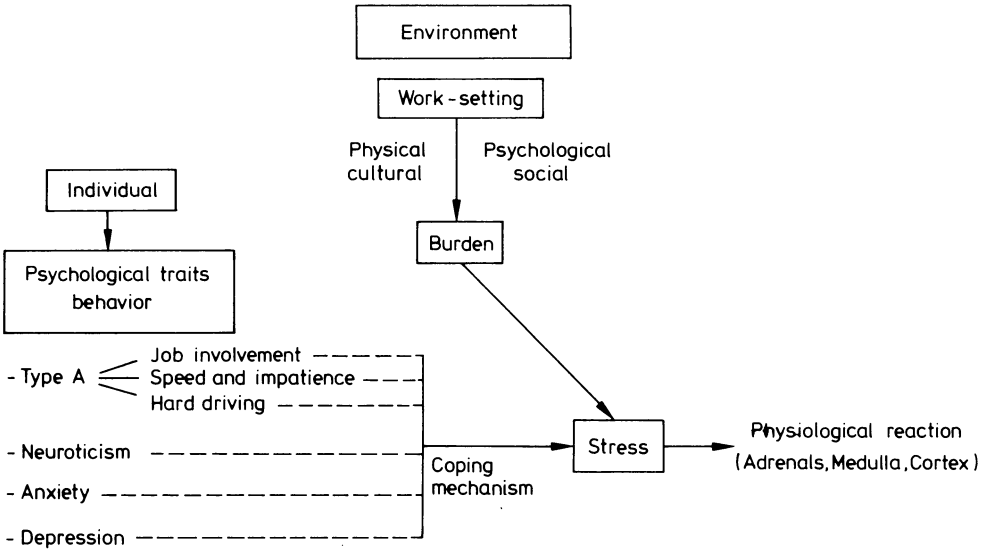


Fig. 4. Schematic representation of the relationship between the environment (work setting) and the individual (psychological traits, behavior) as a stress-inducing system

Objective Work Load

There exist normative work demands which are culture related as well as functions of the kind of industry itself. Those socio-cultural work demands are, in part, redefined at regular time intervals as a result of conventions between management and trade unions. This "objective" work load is related to the organizational structure of the occupational unit, physical working conditions, promotions, working hours, and definition of function or role of each individual.

The objective work load inherent in occupational units has been studied by Caplan et al. among others (8), and Van Dijkhuizen (see Chap. 4).

Methodological problems concern the definition (validity), accuracy (precision), and reproducibility (classification) of the collected data from the occupational units.

Subjective Work Load or Job Stress

According to social-psychologists (10), the subjective perception of the objective working conditions by the individual or group is crucial. If most accept and adapt easily to the sociocultural norms of the working conditions, the prevalence of job stress or, better, "job distress" should remain low. According to Matsumoto (40), the exceptionally low incidence of CHD in Japan, despite its tremendous industrialization, could be attributed to a low level of social stress. Matsumoto points out that, "Japan has not moved from group values toward individualism," that a Japanese employee almost never leaves his company to work for another firm, and that, last but not least, there exist "stress-reducing activities and facilities" such as after-work socializing. Cultural differences in levels of work dissatisfaction were also observed in patients having sustained a myocardial infarction (50).

Subjective job dissatisfaction is related to the type of occupational unit (56, 57), to the job title (14), and to the size of the unit (see Chap. 4).

The methodological problems relate again to the definition (validity), accuracy (precision), and reproducibility (classification) of job stress questionnaires.

Perception of Job Stress and Individual Characteristics

On an individual basis, perception of job stress relates to psychological and behavioral traits.

Type A coronary-prone behavior is related to stressful life events (12, 24) and to job stress. We also observed a correlation coefficient of 0.29 between our job stress score and the Jenkins Activity Survey. It is possible that type A individuals are "job-stress seeking" subjects through their job involvement, speed, and impatience; alternatively, on a collective basis, some industries may preferentially recruit type A individuals, as has been shown by Cafrey (7) in his monastery study. As Mettlin (42) rightly puts it, the type A pattern is integral to the modern occupational career.

Ferguson (14) has shown that in Australia, city telegraphists' traits of neuroticism were positively related to the perception of occupational stress. The same is true for anxiety. From cross-sectional observations, bidirectional hypotheses can be formulated: either neurotic and anxious subjects have lower job-stress perception thresholds, or there exist chronic stress-inducing situations which promote neurotic traits such as anxiety and depression.

Neuroendocrine Changes Due to Job-Stress

J. Cassel (10) wrote that "If we can identify the characteristics or properties of those signals or symbols which generally evoke major neuroendocrine changes in the recipient we will have identified a general class of stressors." The major pathogenetic pathway leading from self-perceived job stress to coronary heart disease must take into account the well-known general adaptation syndrome of Selye (58). This includes the excretion of corticosteroids and catecholamines by the adrenals. Various daily life activities or experimentally induced physical and mental stress have been shown to increase catecholamine excretions in man (9, 37, 64), although the magnitude of the endocrine reaction is related to individual behavioral patterns (20, 44) or personality traits (16).

Again, we are confronted with methodological problems: should these neuroendocrine markers be measured in urine or blood samples? How valid and reproducible are the techniques? What is the intraindividual biologic variation? In other words, how far does one sampling carried out during working hours reflect the subject's day-to-day catecholamine level?

The Neuroendocrine Reaction and Coronary Risk

The crucial test of relating blood or urinary catecholamine levels prospectively to the occurrence of CHD has not yet been performed, to our knowledge, although catecholamines have been shown experimentally to promote atherosclerosis in animals (47). However, catecholamines have been largely implicated in promoting classical

coronary risk factors: tachycardia and blood pressure elevation, although in a transient manner (38). How far this relates to a permanent elevation of both variables is again an open question.

Less disputable is the fact that catecholamines can trigger premature ventricular beats (39). These in turn have been shown to be a risk factor leading to sudden death in patients with proven CHD (53, 67). The link between transient elevation of catecholamines and ventricular fibrillation has been shown in animals and seems possible, but as yet unproven, in man.

A more fundamental approach relates high catecholamine levels to intravascular thrombosis through their effects on platelet aggregation (22, 23, 46, 68). Platelet aggregation could even play a fundamental role in the atherosclerotic process (52). According to Gruchow (21), elevated vanillylmandelic acid levels, a urinary metabolite of catecholamines, are associated with the reporting of chronic nonspecific disease conditions. And finally the neuroendocrine reaction to stress could promote transient serum cholesterol elevation (19).

We have thus tried to show in which way job stress could potentially lead to clinical coronary heart disease. Another concept would involve generally noxious habits promoted by stress, such as smoking, overeating, and lack of physical activity during leisure time.

It is beyond the scope of this paper to review the entire literature concerning the relationship between job stress or work overload, coronary risk factors, and CHD. We have simply tried to summarize some of the available information.

Objective Job Stress

Work overload in tax accountants meeting deadlines induces serum cholesterol elevation as well as a shortened coagulation time (19). An earlier onset of hypertension has been observed among traffic controllers as compared with second-class airmen (11).

Work tasks involving precision and rapidity of execution increase catecholamine excretion (16).

Long working hours are associated with an increased risk of coronary death (6). Upward social mobility has been observed to be related to the incidence of CHD by some (29, 63), but not all, authors (26).

Job stress according to job-title was related to ECG abnormalities, suggestive of CHD in lawyers (17) and in physicians (54, 55).

Subjective Job Stress

Situations perceived by the subjects as potentially stressful accelerate heart rate, which was monitored during working hours through continuous recording (25).

In a so-called ecological study, Sales and House (57) observed a positive correlation between levels of job dissatisfaction in different occupational groups and coronary mortality (standardized mortality ratio). In case-control studies, coronary patients had more complaints about professional problems than did controls (2, 5). Financial and other professional worries are related to the incidence of angina pectoris (15, 41) and myocardial infarction (66) in univariate analysis. Shekelle et al. (59) recently produced evidence that subjective job stress ("tension at work") is related to the occurrence of CHD independent of the major coronary risk factors. Having reviewed and discussed the relevant data concerning the relationship between job stress and CHD as well as the methodological pitfalls, we would like to propose a possible analogy with the controversial relationship between nutrition and CHD.

The sociocultural characteristics of nutrition constitute the environmental "burden" which has been accurately defined when groups ("units") of subjects with strikingly different nutritional patterns are compared in terms of their correlation with serum cholesterol: the correlation coefficient of saturated fat intake with serum cholesterol was 0.89 in the Seven Countries Study (30).

On the other hand, no correlations have been observed on a individual basis between the nutritional patterns of English bank clerks (43) or Americans (45) and their serum cholesterol. In both cases individual coping mechanisms acting against the metabolic "stress" of saturated fats determine in fine the serum cholesterol level (Fig. 5).

In Fig. 6 we compared two hypothetical occupational units with markedly different work loads and two (nonhypothetical) countries, Finland and Italy, which have markedly different nutritional patterns. Both countries and occupational units are represented by three subjects with low, medium, and highly developed coping mechanisms, respectively (subjects A, B, and C). In Finland, a country with a very high saturated fat intake, the mean serum cholesterol is elevated since most subjects (in our case, none) cannot cope with such a high fat intake. On the other hand, in Italy, a country with a low fat intake, only one of the three subjects could not "cope" with the nutritional "stress" so that the mean serum cholesterol was low. Coronary heart disease incidence has effectively been found to be high in Finland and low in Italy.

If we turn to the two hypothetical occupational units and measure catecholamine excretions during working hours, we would expect

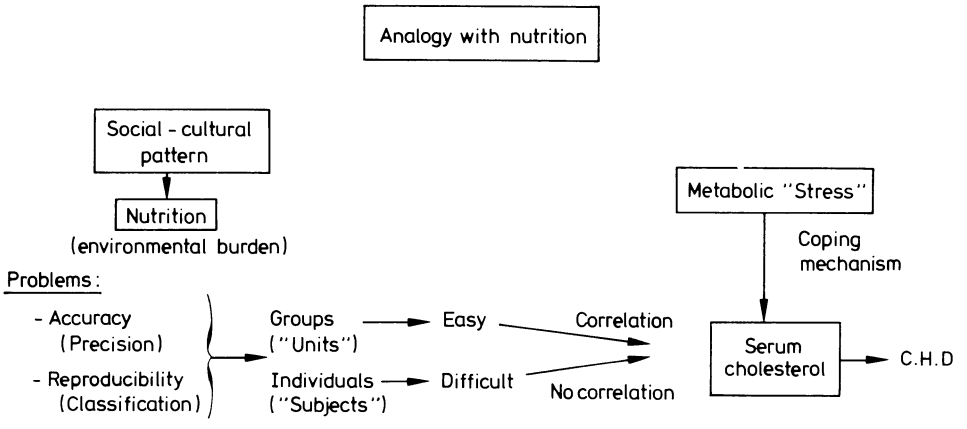


Fig. 5. Schematic representation of the relationship between nutrition and coronary heart disease

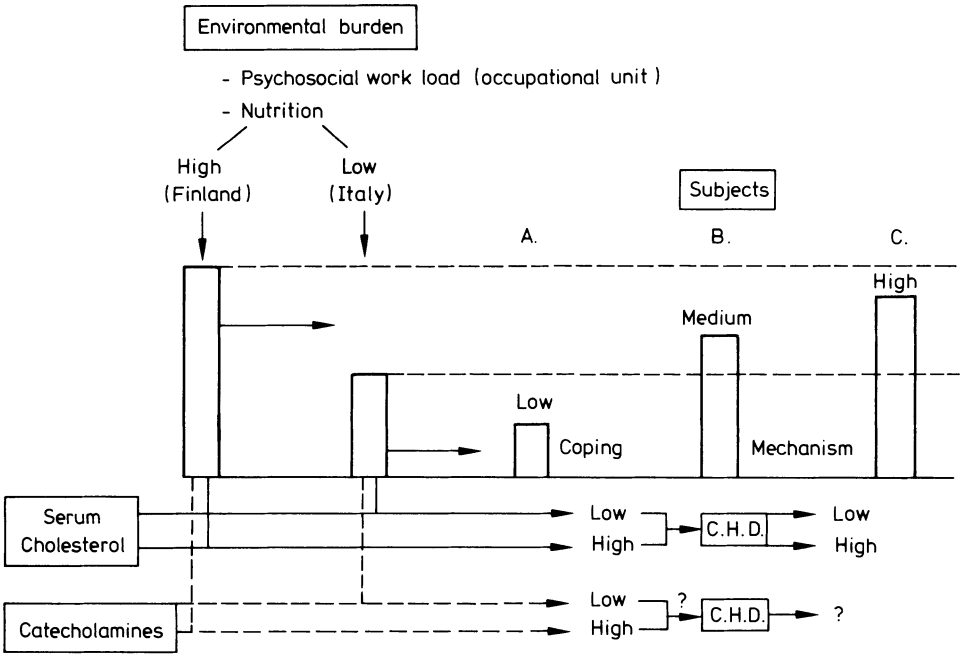


Fig. 6. Schematic representation of a hypothesis concerning the relationship of work load with CHD, based on a possible analogy with the nutritional hypothesis

a high mean level in the unit with subjective work overload and a low level in that unit with no (or almost no) work overload. Indeed, in the former the coping mechanism of almost all subjects (in our case, all three subjects) would be outrun, and in the latter, one of three subjects would fail to cope with the environmental stress. We would like to hypothesize a high incidence of CHD in the highly work-overloaded occupational unit and a low incidence in that unit with a low work load. Although the technical and methodological problems of a prospective epidemiological study, which included catecholamine excretions in the base line examination, are not negligible, we feel that such a study would nonetheless be worthwhile.

References

1. Ambiances des lieux de travail (1976) Commissariat général à la promotion du travail

2. Blöhmke M, Schaefer H, Abel H, Depner R, Gruntzig A, Koschorreck B, Stelzer O (1969) Medizinische und soziale Befunde bei koronaren Herzkrankheiten. MMW 111:701-710

3. Boy J, Bonnafe M, Mazet JB (1960) Determination of total cholesterol by a modification of Pearson's method. Its manual and automatic execution. Ann Biol Clin (Paris) 10-12: 669-675

4. Prozek J, Keys A, Blackburn H (1966) Personality differences between potential coronary and non coronary subjects. Ann NY Acad Sci 134:1057-1064

5. Bruhn JC, McCrady KE, Du Plessis A (1968) Evidence of "emotional drain" preceding death from myocardial infarction. Psychiatr Dig 29:34-40

6. Buell P. Breslow L (1960) Mortality from coronary heart disease in California men who work long hours. J Chronic Dis 11:615-626

7. Cafrey B (1965) Reliability and validity of personality and behavioral measures in a study of coronary heart disease. J Chronic Dis 21:191-204

8. Caplan RD, Cobb S, French FRP, Van Harrison R, Pinneau SR (1975) Job demands and worker health. HEW Publication (NIOSH) 75-160

9. Carruthers ME (1969) Aggression and atheroma. Lancet II: 1170-1171

10. Cassel J (1974) Psychosocial processes and "stress" theoretical formulation. Epidemiology and methods. Int J Health Serv 4/3:471-482

11. Cobb S, Rose RM (1973) Hypertension, peptic ulcer and diabetes in air traffic controllers. JAMA 224:489-492

12. Dimsdale JE, Hackett TP, Block PC, Hutter AM (1978) Emotional correlates of type A behavior pattern. Psychosom Med 40: 580-583

13. Durnin JV, Passmore R (1967) Energy, work and leisure. In: Heineman Studies in Biology No 3. Heineman Educational Books, London

14. Ferguson D (1973) A study of occupational stress and health. Ergonomics 16:649-663

15. Floderus B (1974) Psychosocial factors in relation to coronary heart disease and associated risk factors. Nord Hyg Tidskr [Suppl] 6

16. Frankenhaeuser M, Patkai P (1965) Interindividual differences in catecholamine excretion during stress. Scand J Psychol 6:117-123

17. Friedman EG, Hellerstein HK (1968) Occupational stress, law school hierarchy, and coronary artery disease in Cleveland attorneys. Psychosom Med 30:72-86

18. Friedman M, Rosenman RH (1959) Association of specific overt behavior pattern with blood and cardiovascular findings. JAMA 169:1286-1296

19. Friedman M, Rosenman RH, Carroll V (1958) Changes in the serum cholesterol and blood clotting time in men subjected to cyclic variation of occupational stress. Circulation 17:852-861

20. Friedman M, St George S, Byers SO, Rosenman RH (1960) Excretion of catecholamines, 17-ketosteroids, 17-hydroxycorticoids and 5-hydroxyindole in men exhibiting a particular behavior pattern (A) associated with high incidence of clinical coronary artery disease. J Clin Invest 39:758-764

21. Gruchow HW (1976) Catecholamine activity and reported morbidity. J Chronic Dis 29:773-783

22. Haft JI, Fani K (1973) Stress and the induction of intravascular platelet aggregation in the heart. Circulation 48:164-169

23. Haft JI, Fani K (1973) Intravascular platelet aggregation in the heart induced by stress. Circulation 47:353-358

24. Haynes SG, Levine S, Scotch N, Feinleib M, Kannel WB (1978) The relationship of psychosocial factors to coronary heart disease in the Framingham study. Am J Epidemiol 107:362-381

25. Hennigan JK, Wortham AW (1975) Analysis of workday stresses on industrial managers using heart rate as a criterion. Ergonomics 18:675-681

26. Hinkle LE, Whitney LH, Lehman EW, Dunn J, Benjamin B, King R, Plakun A, Flehinger B (1968) Occupation, education and coronary heart disease. Science 161:238-246

27. Jenkins CD, Rosenman RH, Friedman M (1967) Development of an objective psychological test for the determination of the coronary prone behavior pattern in employed men. J Chronic Dis 20:371-379

28. Jouve A, Sommer A, Avierinos Ch, Fondari J (1973) Premiers résultats d'une enquête prospective sur les maladies cardiovasculaires dans une grande administration Arch Mal Coeur 1:25-33

29. Kaplan BH, Cassel J, Tyroler HA, Cornoni JC, Kleinbaum DG, Hill C, Hames CG (1971) Occupational mobility and coronary heart disease. Arch Intern Med 128:938-942

30. Keys A (1970) Coronary heart disease in seven countries. Am Heart Assoc Monogr 29:170

31. Kittel F, Kornitzer M, Zyzanski SJ, Jenkins CD, Rustin RM, Degré C (1978) Two methods of assessing the Type A coronary-prone behavior pattern in Belgium. J Chronic Dis 31:147-155

32. Kittel F, Kornitzer M, Dramaix M (1980) Coronary heart disease and job-stress in two cohorts of bank clerks. Psychother Psychosom

33. Klose S, Stoltz M, Schlumberger H (1978) New developments for continuous-flow methods which are characterized by very simple handling and are equivalent to reference methods in clinical chemistry. In: Mousson (ed) Organisation des Laboratoires - Biologie Prospective, IVe Colloque International de Pont-à-Mousson, p 472-478

34. Kornitzer M, Thilly C, Vanroux A, Balthazar E (1975) Incidence of ischaemic heart disease in two cohorts of Belgian clerks. Br J Prev Soc Med 29:91-97

35. Kornitzer M, Dramaix M, Gheyssens H (1979) Incidence of ischemic heart disease in two Belgian cohorts followed during 10 year. Eur J Cardiol 9/6:455-472

36. Leon AS, De Backer G, Jacobs D, Taylor HL (1976) Relationship of life habits and physical characteristics to work capacity on treadmill. Circulation [Suppl II] 53:52

37. Levi L (1964) The stress of everyday work as reflected in productiveness, subjective feelings and urinary output of adrenaline and noradrenaline under salaried and piece-work conditions. J Psychosom Res 8:199-202

38. Lorimer AR, McFarlane PW, Provan G, Duffy T, Lawrie TD (1971) Blood pressure and catecholamine responses to "stress" in normotensive and hypertensive subjects. Cardiovasc Res 5: 169-173

39. Lown B, De Silva RA, Lenson R (1978) Roles of psychologic stress and autonomic nervous system changes in provocation of ventricular premature complexes. Am J Cardiol 41:979-985

40. Matsumoto YS (1971) Social stress and coronary heart disease in Japan. Milbank Mem Fund Q 48:9-36

41. Medalie JH, Kahn HA, Neufeld HN, Riss E, Goldbourt U, Perlstein T, Oron D (1973) Myocardial infarction over a five-year period. I. Prevalence, incidence and mortality experience. J Chronic Dis 26:63-84

42. Mettlin C (1976) Occupational careers and the prevention of coronary-prone behavior. Soc Sci Med 10:367-372

43. Morris JN, Marr Jw, Heady JA, Mills GL, Pilkington RT (1963) Diet and plasma cholesterol in 99 bank men. Br Med J 1:571-576

44. Nelson GN, Masuda M, Holmes TH (1966) Correlation of behavior and catecholamine metabolite excretion. Psychosom Med 28: 216-225

45. Nichols AN, Ravenscroft C, Lamphiear DE, Ostrander LD (1976) Independence of serum lipid levels and dietary habits. JAMA 236:1948-1953

46. O'Brien JR (1963) Some effects of adrenaline and anti-adrenaline compounds platelets in vitro and in vivo. Nature 200:763-764

47. Oester YT (1959) Adrenal medullary hormones and arteriosclerosis. Ann NY Acad Sci 72:885-896

48. Ostfeld AM, Lebovits BZ, Shekelle RB, Paul O (1964) A prospective study of the relationship between personality and coronary heart disease. J Chronic Dis 17:265-276

49. Pipberger HV, Stallman FW, Yand K, Draper HW (1963) Digital computer analysis of the normal and abnormal electrocardiogram. Prog Cardiovasc Dis 5:378-392

50. Romo M, Siltanen P, Theorell T (1974) Work behavior, time urgency and life dissatisfactions in subjects with myocardial infarction: a cross-cultural study. J Psychosom Res 18:1-8

51. Rose G, McCartney P, Reid DD (1977) Self-administration of a questionnaire on chest pain and intermittent claudication. Br J Prev Soc Med 31:42-48

52. Ross R, Glomset JA (1976) The pathogenesis of atherosclerosis. N Engl J Med 295:420-425

53. Ruberman W, Weinblatt E, Frank CW, Goldberg JD, Shapiro S, Feldman CL (1976) Prognostic value of one hour of ECG monitoring of men with coronary heart disease. J Chronic Dis 29:497-512

54. Russek HI (1960) Emotional stress and coronary heart disease in American physicians. Am J Med Sci 240:711-721

55. Russek HI (1963) Emotional stress and coronary heart disease in American physicians, dentists and lawyers. Congr Mund Cardiol Mexico Mem IVb:75-85

56. Sales SM (1969) Organizational role as a risk factor in coronary disease. Adm Sci Q 14:325-336

57. Sales SM, House J (1971) Job dissatisfaction as a possible risk factor in coronary heart disease. J Chronic Dis 23: 861-873

58. Selye H (1936) A syndrome produced by diverse noxious agents. Nature 138:32-33

59. Shekelle RB, Schneider AS, Liu SC, Raynor WJ, Garron DC, Ostfeld AM, Paul O (1979) Work tension and risk of coronary heart disease (CHD). Am Heart Assoc CVD Epidemiol Newsletter 26:66

60. Snedecor GW, Cochran WG (1957) Statistical methods, 6th ed. The Iowa State University Press, Ames, Iowa, USA

61. Spitzer H, Hettinger T (1968) Tables donnant la dépense énergétique en calories pour le travail physique. Les Cahiers du B.T.E. (3e série: Psycho-Physiologie) No 302-04

62. Steele BW, Koehler DF, Azar MM, Blaszkowski TP, Kuba K, Dempsey ME (1976) Enzymatic determinations of cholesterol in high density lipoprotein fractions prepared by a precipitation technique. Clin Chem 22:98-101

63. Syme SL, Borhani NO, Buechley RW (1966) Cultural mobility and coronary heart disease in an urban area. Am J Epidemiol 82:334-346

64. Taggart P, Carruthers M (1971) Endogenous hyperlipidaemia induced by emotional stress of racing driving. Lancet I: 363-366

65. Taylor HL, Jacobs DR Jr, Schucker B, Knudsen J, Leon AS, De Backer G (1978) A questionnaire for the assessment of leisure time physical activities. J Chronic Dis 31:741-755

66. Theorell T, Flodérus-Myrhed B (1977) Workload and risk of myocardial infarction. A prospective psychosocial analysis. Int J Epidemiol 6:17-21

67. Van Durme JP (1975) Study of the prognosis and therapy of arrhythmias appearing after the acute phase or a myocardial infarction (in Belgian). Thesis, Gent

68. Yamazaki H, Kobayashi I, Sano T, Shimamdto T (1973) Enhancement of A.D.P.-induced platelet aggregation by adrenaline in vivo and its prevention. Thromb Haemostas 32:490-498

Psychosocial Risk Constellations and First Myocardial Infarction

J. Siegrist, K. Dittmann, K. Rittner, and I. Weber

Introduction

Traditionally, an analysis of isolated characteristics has been employed to separate a myocardial infarction (MI) group from a comparable control group. Thus, the relationship between type A and infarction, between life events and infarction, or between status inconsistency and infarction have been investigated (e.g., 1, 3, 5, 7). In contrast, we would like to demonstrate that a simultaneous analysis of various psychosocial risk factors contributes better to a characterization of infarction groups as compared with control groups. We assume that in the presence of somatic risks, an accumulation of chronic and subacute stressors, that is, the psychosocial risk constellation, will markedly increase the risk of an infarction.

Among the various possible psychosocial risk factors, we emphasize in this paper those stressors which relate to the social context, that is, to stress situations in one's work and private sphere. In other words, we will focus on a sociological analysis. From the above-mentioned global hypothesis, it follows that MI patients are significantly more strongly characterized by chronic and subacute risk situations than persons in the control group. Furthermore, we shall also examine whether a subgroup of MI patients who died of a second infarction after recovering from a first can be distinguished from other MI patients and also from healthy individuals with regard to social risk situations.

Finally, we will present some theoretical considerations in order to construct a modified conceptual framework for a possible

interaction between a coronary-prone behavior pattern and specific work stressors, i.e., for the interaction between risk disposition and risk situation. With this background it is possible to formulate hypotheses concerning the neurohormonal and pathophysiologic precursors of myocardial infarction. In this regard we must caution against simplistic approaches, especially against an exclusive consideration of vascular-atherosclerotic processes.

Materials and Methods

The data to be used were derived from a retrospective study of 380 male patients, aged 30 - 55, with clinically proven first myocardial infarction, who were participating in a rehabilitation program. The control group was composed of 190 healthy males, matched with the sample half of the MI patients on the basis of age and occupational position. The 380 MI patients were studied once more 18 months later catamnestically by means of a questionnaire focusing on occupational, medical, and psychosocial rehabilitation. All investigations were conducted by the Institute for Medical Sociology of the University of Marburg. We whould like to thank Prof. Dr. H. Weidemann, who acted as cardiological consultant.

Although we are quite aware that the evidence provided by retrospective studies is limited, we would like to list a few of the reasons which encouraged us to use this approach. We believe that psychosocial research on myocardial infarction, with regard to both methodology and theory, has not yet advanced to a point where prospective studies, which tend to be extremely costly, can be justified. Furthermore, because of research costs, only short instruments, applicable in written form, can be employed in prospective studies. We consider this to be problematic for reasons of both methodology and content.

We would like to point out, though, that a retrospective study can be justified, first, only if it begins with and attempts to differentiate knowledge accumulated by previous prospective studies; second, if, by utilizing additional measures, it guar-

antees that bias in data collection due to retrospective interpretations is controlled as far as possible; third, if the criterion variable is specified as exactly as possible, leading to the best possible homogeneity of the sample; and finally, if the comparative criteria are applied as stringently as possible to the control group. We have attempted to fulfill all these conditions. Furthermore, we had the advantage of being able to continue this study immediately by means of a catamnestic investigation.

We do not have sufficient time here to deal in detail with possible bias caused by the composition of the samples (cf. 10). We would like to point out, though, that the rate of rejection was below 5% and that we successfully controlled seasonal variations. Furthermore, our sample did correlate, in terms of occupations, with the total population of MI patients undergoing rehabilitation programs in the Federal Republic of Germany, and since there is a high rate of participation in rehabilitation programs in this age and sex group no systematic bias is to be expected.

Because of the unavoidable concentration focused on survivors, one bias factor exists which cannot be controlled, especially since the percentage of sudden deaths is quite high. This creates some difficulties for testing our hypothesis because patients who die a sudden death without manifest prior illness, may have been subjected, while still alive, especially strong to psychosocial stressors (cf. 9).

Results

Before examining the level of psychosocial risk factors in MI patients compared with a healthy sample, we wish to emphasize that despite the exclusive focus on psychosocial risks we still take the well-known standard risk factors into account. As Fig. 1 shows, we have controlled somatic risk factors and have also discovered differences which were only in part significant (data on cholesterol and triglycerides were, unfortunately, collected

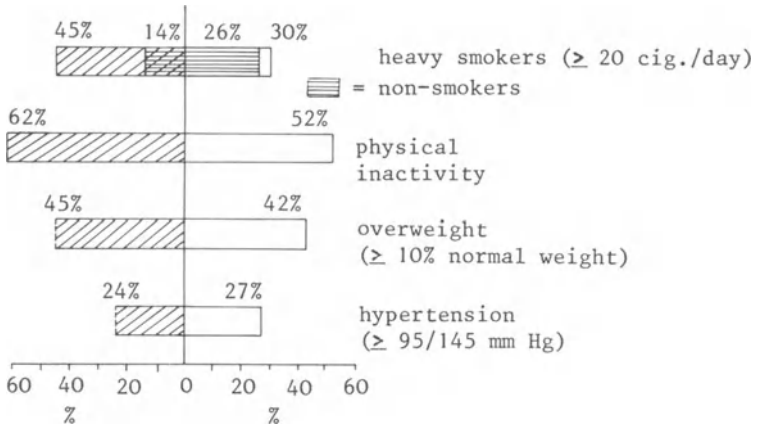


Fig. 1. Standard risk factors. MI patient (N, 190); healthy controls (N, 190)

only from the study sample and not from the control group). This may be in part because some somatic risk factors had, at the time of the study, been reduced by means of therapeutic intervention following an infarction. But even if one takes into account that data on blood pressure and weight may be distorted as a result of therapeutic intervention in the group of MI patients, it is impossible to explain the observed variance on the basis of standard risk factors alone. This had also been an important finding in prospective epidemiologic studies. The many paradoxical cases found in such studies represent, in a way, the focus of our research interest. We have asked ourselves the degree to which psychosocial factors should be regarded as factors complementing and, in some instances, strengthening the impact of somatic factors.

We have distinguished between dispositional and situational psychosocial risk factors. Situational psychosocial risk factors are divided, following suggestions made by Theorell (12, 13), into chronic and subacute factors. We consider a coronary-prone behavior pattern to be the most important disposition [for a survey see, for instance, Dembroski (1)]. On the basis of considerations which will be outlined in more detail below, we have narrowed the contents of the type A concept by placing special emphasis on the characteristic pointed out by David Glass and

collaborators when they wrote: "The coronary-prone behavior may be described as a characteristic style of responding to environmental stressors that threaten an individual's sense of control. Type A's are engaged in a struggle for control" (6, p 181, cf. also 5). Starting with instruments already available and adding new items, we have attempted to formulate a dimension of "ambitions of control."

Work-related stress plays a prominent role in chronic situational psychosocial risks (cf. 12, 13). Special weight has been given to quantitative overload (pressure of time) and to structural limitations of one's work autonomy, such as inconsistent demands which can scarcely be changed and frequent and unforeseen disruptions.

These strains have been combined in an index of "special work load." Traditional strains, e.g., noise, heat, danger of accident, responsibility, and conflicts with supervisors, were investigated. These strains were combined in an index of "general work load." Finally, additional questions dealt with characteristics such as shift work, overtime, rationalization, rate fixing, workplace security, and occupational mobility. Chronic risk situations may also be determined by the private sphere. In an index called "chronic difficulties" we have attempted to include the most important private strains. As is well known, good social support protects against strain in one's work and family. We have assumed that MI patients are not only subject to a greater degree of chronic strain than the control group but, in addition, that they enjoy a lesser degree of social support.

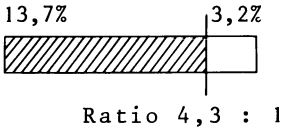
Finally, we have characterized major life-changing events during the 2 years prior to an infarction as subacute strain. For quantitatively determining the degree of subjective strain there exist simple procedures utilizing a score which is either preformulated or determined on an ad hoc basis (cf. 3, 7). In contrast to this method, we have addressed our questions to scaled additional items corresponding to each experienced life event considered by the patients to be a strain. This was done by distinguishing seven dimensions which, from a perspective of stress theory, appeared to be especially significant:

1) Controllability or coercion,
2) predictability,
3) relevance of the event,
4) interruption of everyday routines,
5) situational vulnerability,
6) active coping,
7) social support.

This approach enabled us to calculate both the amount of stressful events per selected subgroup or individual and the respective stress scores or their mean values. Assuming a cumulativeness in the scores, individuals could be assigned from 1 to 44 points per event, with 44 points indicating extreme stress. Accordingly, events could be classified following the degree of subjectively perceived stress [for broader information see (11)].

If we look at the three most important psychosocial indexes separately, i.e., work load, life events, and ambitions of control, we may recognize differences in mean values in each of them. With regard to our hypothesis, these are significant at least on a 1% level, yet they do not suffice to clearly separate the two groups. However, if we combine the three variables in a common unweighted index, a much more obvious difference in mean values can be recognized ($\bar{x}$, 2.42 vs. 1.28; $P < 0.0001$). The same result becomes even more obvious if we compare extreme groups: the high-risk group which in all three respects, i.e., workload, life events, and ambitions of control, is associated with extremely high values, can be found 4.3 times more often in MI patients as in healthy individuals (see Fig. 2). In contrast, the low-risk group, whose values in all three respects are zero or close to zero, is twice as prevalent in healthy individuals compared with infarction patients. It is of special interest that denial, measured by a subscale of MMPI, is significantly higher among low-risk MI persons than among the high-risk group. A highly pronounced presence of at least two of the three indexes is found in 44.8% of MI patients and in only 19.5% of the healthy control group. Combinations of at least two highly pronounced stress indicators can therefore be found 2.3 times more frequently in the MI group than in the healthy control group.

High workload + high life events + high ambitions of control:



Low workload + low life events + low ambitions of control:

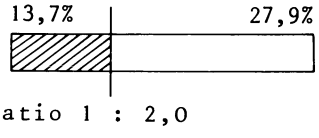


Fig. 2. High risk versus low risk group. N, 190; IG; CG

A stepwise discriminant analysis leads to similar results. However, it should be noted that with this approach healthy individuals can be classified more correctly than the patients because of the latters' larger standard deviations with regard to all relevant variables. The results of a stepwise discriminant analysis of the 190 MI patients and of the 190 healthy individuals are shown in Table 1.

We considered the following variables, listed according to their diminishing discriminatory power: life events, ambitions of control, social support, chronic difficulties, work load, physical activity, blood pressure, weight, and smoking. On the basis of these variables, 70.5% of the patients and 72.6% of the healthy individuals, that is, a mean of 71.6%, could be classified correctly. If one looks only at those under 50 years of age, the percentage of those correctly classified increases to 74. Some

Table 1. Stepwise discriminant analysis of 190 MI subjects and 190 healthy controls[a]

Actual group	Predicted group membership			
	MI subjects		healthy controls	
MI subjects	134	70.5%	56	29.5%
healthy controls	52	27.4%	138	72.6%

[a]Cases correctly classified; 71.6%

of the incorrectly classified MI patients could be associated, however, with typical clusters of infarction populations, as recently conducted cluster analyses have demonstrated. We were able to identify more than half of the MI patients in clusters expected following our hypothesis. Within these clusters, MI patients predominated over individuals from the control group in a ratio of at least 6 to 1.

Let us now portray the complex of situational stress. One might object that the increased stress values of the patients can, to a large extent, be traced to a depressed mood and an increased sensibility following an infarction. To counter this argument, we have controlled both the amount of neuroticism and the tendency toward denial using subscales from personality inventories [FPI (4) respectively MMPI].

Furthermore, we have controlled the time lag between investigation and infarction, which ranged from 6 weeks to 6 months. Although there do exist correlations between ambitions of control and neuroticism (0.59), and, to a lesser degree, between work load and neuroticism (0.29), the amount of explained variance by these psychological variables remains altogether rather small. There is some justification, then, for assigning a certain validity to reported strains, especially since these strains can frequently be located both in terms of time and place, and because they often represent truly biographic turning marks.

With regard to chronic difficulties in one's family, there is, if regarded in isolation no evidence of significantly increased stress in MI patients compared to healthy individuals. However, if the variable "social support" is introduced, it can be demonstrated that 50% of all patients with chronic difficulties had no or very little social support (CC, 0.32; $\underline{P} < 0.01$). This relationship does not exist in the control group. MI patients with chronic private difficulties, for the time prior to the onset of their illness, show higher stress scores with regard to life events than do patients without such chronic difficulties. An analysis of variance demonstrates significant correlations for five central indicators of chronic difficulties (see Table 2).

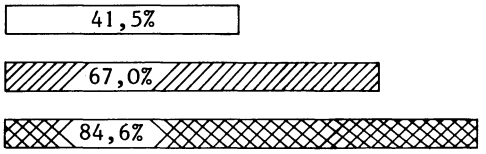
Table 2. Relationship between chronic familial difficulties and life event scores (analysis of variance) in 380 MI subjects

Chronic difficulty	Life events score		
	F	P	t-test:P
Marital Conflicts	8.173	0.001	0.01
Problems with children	19.015	0.001	0.01
Trouble with neighbors	2.873	0.05	0.01
Difficulties with people in general	37.310	0.001	0.01
Bad housing situation	7.434	0.001	0.01

With regard to work-related problems, the following can be shown. The infarction group is characterized, highly significantly, by more general and specific occupational stress than the control group, both groups being matched on the basis of occupational characteristics. Here again, there appears to be evidence for an accumulation of chronic and subacute strains. Both analysis of variance and simple contingency tables show that patients with high chronic occupational stress experience significantly more work-specific acute life events. A similar correlation was discovered in our catamnestic study, which is now being evaluated. So far, 324 of 380 MI patients investigated responded to a mailed questionnaire. In the meantime 13 patients had died of a second infarction. Figure 3 shows the presence of social risk situations in healthy individuals, in surviving MI patients, in and those individuals who died in the meantime. High occupational stress and/or high life-event values are indicated on top and high occupational stress only, below. The percentage of those subjected to high degrees of stress among those who had died in the meantime, in both cases, is more than double the percentage of high stress scores in the healthy individuals. A smaller, but still obvious, difference existed in comparison with surviving MI patients. It is important to mention that only two of the 13 deceased patients were clinically characterized either by a angiographically documented significant (> 50%) narrowing of two or three vessels or by ischemic ST depressions in exercise-ECG (below 50 W).

It is of special interest that we were able to identify, within the group of MI patients, occupational subgroups characterized by an extraordinarily high degree of specific occupational stress.

Percentage of persons with high workload and/or high life event scores:



Percentage of persons with high general or special workload:

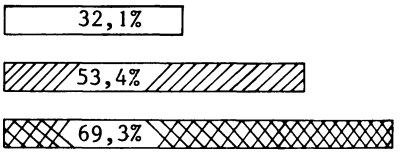


Fig. 3. Social risk situations of healthy controls, MI subjects, and cardiac deaths. ▨ MI patients (N, 190); □ healthy controls (N, 190); ▩ cardiac deaths after 1½ year (N, 13)

These are, first, middle-echelon employees, for instance, supervisors and factory foremen, who can be associated with occupational indicators such as "coordination, organization, and planning," and, second, the group of employed businessmen and salesmen whose status is closely related to their success in turnover. Both subgroups showed high specific occupational stress beyond coincidence (47%, compared with 32% of the total MI group and 16% of the healthy individuals). The percentage of those working many hours of overtime amounted to 70%, compared with 56% of the total MI group and 47% of the healthy individuals. Of the foremen, 69% considered it stressful to be constantly burdened with two kinds of responsibility, compared with 37% of the total MI group and 30% of the control group. Structural limitation of one's work autonomy because of inconsistent demands appears to be a relevant characteristic of unsafe work places. From our observations we conclude that such work places can be found more frequently in smaller and medium-sized firms than in large businesses.

The above-mentioned identification of occupational subgroups with high stress experience is made even more evident by the fact that these groups also differ significantly with regard to the extent

of ambitions of control: A homogeneous occupational subgroup of middle-echelon employees (supervisors and factory foremen) has significantly higher means of ambitions of control than a comparable subgroup of blue-collar workers ($\bar{x}$ = 3.97 vs. 2.90; $P < 0.01$). This is also the case, to a lesser degree, in comparable subgroups of healthy persons. We feel this is a strong argument against the notion of type A as a stable personality trait. It seems that at least the extent of crucial aspects of the coronary-prone behavior pattern may be related to contextual experiences and challenges in the occupational field.

In our catamnestic study we were able to obtain more information on this topic. We found, very unexpectedly, that means of ambitions of control were significantly higher ($P < 0.01$) 18 months after rehabilitation. The percentage of persons with extremely high scores nearly doubled. Interestingly, persons with the greatest amount of actual strain in the field of work and health showed the highest means of ambitions of control (see Fig. 4).

Further research is needed to specify relationships between situational characteristics and psychological predispositions

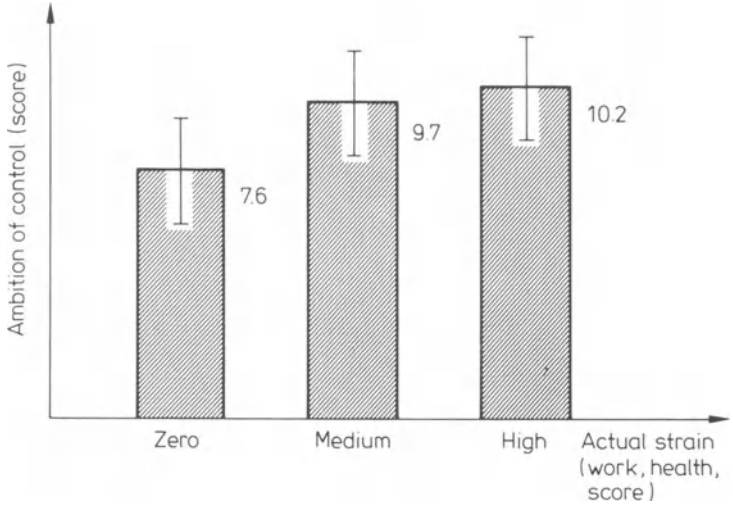


Fig. 4. Relationship between type A (ambitions of control) and perceived actual strain in a post-infarction group (N, 244). C, 0.45; p < 0.001

in the field of stress and CHD. Our final remarks will concentrate on this topic on a theoretical level.

In the last section of our presentation of research results we refer to life events. Figure 5 shows the accumulation of life events regarded as stressful within a 2.year period before the infarction or before the interview. It follows that within this period almost a third of all patients had experienced three or more stressful life events, compared to 13,6% in the healthy population. This accumulation of stress may be regarded, at least as far as a large subgroup is concerned, as precipitating an infarction. If we look at only the prodromal phase we recognize an even more obvious tendency toward accumulation with regard to the relative number of events that happened within a certain time period (see Fig. 6). Even if one could consider bias on the part of the interviewers or causal attributions on the part of the interviewees as an explanation for this strongly documented tendency, there is no possibility, as far as the statistical procedures employed by us are concerned, of leveling these differences. Furthermore, we would like to point out that each stressful event has been exactly identified in terms of both time and context. Differences resulting from varying abilities to recall events should be negligible due to the comparability of the two samples in terms of education, occupation, etc.

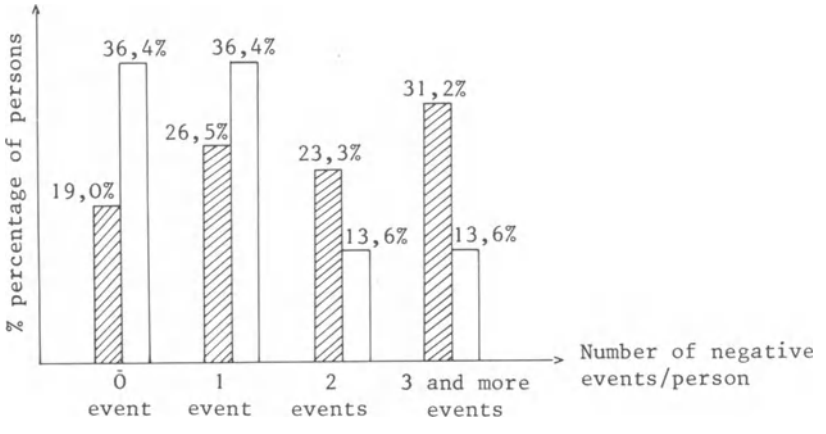


Fig. 5. Accumulation of negative life events. ▨ *MI subjects (N, 190);* □ *healthy controls (N, 190); p, 0.001*

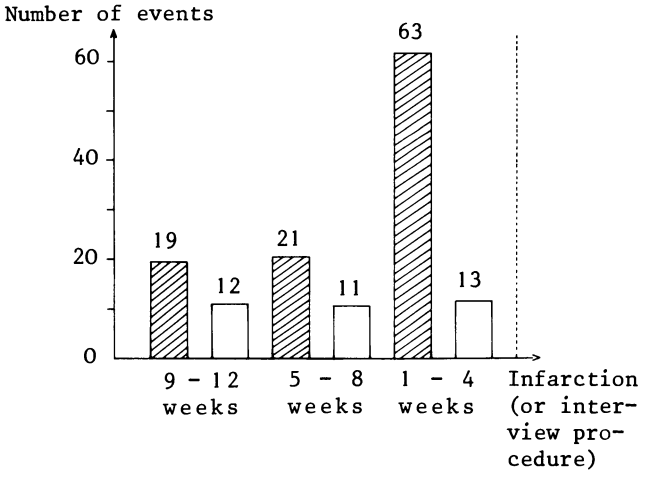


Fig. 6. Negative life events within 3 months before illness onset. ▨ *MI patients (max. N, 190);* ☐ *healthy controls (max. N, 190)*

The group of MI patients is characterized not only by a larger total of events and by a larger number of events per person, but also by a larger number of serious events. We could show that serious events which may jeopardize one's social status appear 2.8 times more often in MI patients than in the healthy population. In addition, infarction patients had been subjected to 2.9 times more events in the occupational sector and to 1.8 times more interpersonal conflicts than the healthy population. Ubiquitous life events, such as illness or death in one's family or among one's friends, were less strongly represented. MI patients do not, to any significant degree, assign a greater importance to comparable events than do healthy individuals if the number of events is kept constant. This applies to both single scales and total scores. However, it may be stated as a general fact that the more events a person experiences, the higher the increase in scores of subjective strain evaluation. The more events within a relatively short period of time, the earlier a person's potential of coping is exhausted; that is, the greater is this person's vulnerability.

Because our results show that MI patients experience a larger number of stressful events and that single events exert more

stress on these individuals because of increased vulnerability, we may have provided additional support for the main argument of life event research.

Discussion

In presenting some of our results [for a broader presentation see (10)], we have intentionally focused on situational conditions.

However, we would like to conclude our presentation with some theoretical considerations related to the interplay of situational and dispositional traits and its significance for a chronicling of stress experience.

It is well known that physiologic stress reactions, according to H.G. Wolff's classic definition (15), appear when a discrepancy exists between demands and coping potential. However, the elements of this definition (external demand, internal coping potential) are insufficient, as we know from the studies of Lazarus and other cognitive psychologists (8). The degree of a physiologically identifiable stress reaction is obviously determined, first, by a subjective appraisal of external demand and one's own capabilities to react, and, second, by the influence exerted by this appraisal on the actual course of coping. To clarify these thoughts, it would be meaningful to depict the four components of the expanded definition of stress in an idealized diagram which combines the most important types of discrepancy between internal demand, available coping potential, and a subjective evaluation of these two factors (see Table 3).

Table 3. Types of subjective stress experience

	I	II	III
External demand	+	+	−
Appraisal of demand	+ } RA	− } UA	+ } OA
Internal coping potential	−	−	+
Appraisal of internal coping potential	− } RA	+ } OA	− } UA

RA, realistic appraisal
UA, underestimation = unrealistic appraisal
OA, overestimation = unrealistic appraisal

To keep the diagram simple we haven mentioned only extreme conditions, with a "plus" indicating a marked presence of the characteristic and a "minus" indicating an insufficient presence of the characteristic.

From this it follows that one may distinguish three types of discrepancy:

Type I parallels the classic definition of stress: a serious external demand is recognized as such while one's own ability to react is considered to be insufficient. Because of the agreement between objective reality and subjective appraisal, we call this type "realistic demand appraisal."

Type II implies misjudgment of the demand situation, i.e., underestimation of the demand while, at the same time, one's own ability to react is overestimated. As a consequence of this misjudgment, the course of actual coping is characterized by a maximal mobilization of, and demand on, internal resources. Given a certain course and accumulation, this situation may result in exhaustion along with feelings of depression and helplessness.

Type III represents an analogous situation, in which minimal external demands are overestimated while one's potential for reacting is underestimated. The resulting reaction is particularly wasteful because too much effort is mobilized to meet the demands. These efforts are not reduced because expectations of failure are associated with a reduction of efforts. We have termed types II and III "unrealistic demand appraisals."

We assume first, that persons with marked ambitions of control bring themselves more often into demanding situations and, second, that they tend, more than others, toward an unrealistic appraisal of these situations. We assume further that chronic psychosocial stress results from a discrepancy between real or perceived demands on the one hand, and real or perceived coping potential on the other. Experimental research is being conducted in our group to clarify these hypotheses and to demonstrate their empirical validity.

Dysfunctional arousal caused by such discrepancies may, in the form of a "bias" (14) influence homeostatic regulations via the sympathetic adrenal-medullary system. Over a longer period of time, this may, therefore, lead to pathogenic changes in the coronary vessels, in the condition of the blood, in the myocardial metabolism, and in the electric stability of the myocardium. We should, however, make it quite clear that, as far as humans are concerned, we are dealing to a large degree only with hypotheses. It would certainly be too simplistic to postulate a linear causality between amount of stress and extent of atherosclerosis (2). Only careful interdisciplinary studies will provide us with further insights. Progress in expanding our knowledge of these relationships would be extremely important not only for medicine in general, but also for preventive public health strategies, in particular.

References

1. Dembroski TM, Weiss SM, Shields JL, Haynes SG, Feinleib M (eds) (1978) Coronary-prone behavior. Springer, Berlin, Heidelberg, New York
2. Dimsdale JE, Hackett ThP, Hutter J AM, Block PC, Catanzano D (1978) Type A personality and extent of coronary atherosclerosis. Am J Cardiol 43:583-586
3. Dohrenwend BS, Dohrenwend BP (eds) (1974) Stressful life events. Their nature and effects. Wiley, New York
4. Fahrenberg J, Selg H, Hampel R (1973) Das Freiburger Persönlichkeitsinventar (FPI). Hogrefe, Göttingen
5. Glass DC (1977) Behavior patterns, stress and coronary disease. Hillsdale, New Jersey, N.J.
6. Glass DC (1977) Stress, behavior patterns and coronary disease. Am Sci 65:177-187
7. Gunderson GK, Rahe RH (eds) (1974) Life stress and illness. Thomas, Springfield, Ill.
8. Lazarus RS (1966) Psychological stress and the coping process. McGraw-Hill, New York
9. Rissanen V, Romo M, Siltanen P (1978) Premonitory symptoms and stress factors preceding sudden death from ischaemic heart disease. Acta Med Scand 204:389-396
10. Siegrist J, Dittmann K, Rittner K, Weber J (1980) Soziale Belastungen und Herzinfarkt. F Enke, Stuttgart
11. Siegrist J, Dittmann K (in preparation) Das Inventar zur Belastungswirkung lebensverändernder Ereignisse (ILE)

12. Theorell T (1976) Selected illnesses and somatic factors in relation to two psychosocial stress indices. J Psychosom Res 20:7-20

13. Theorell T, Flodérus-Myrhed B (1977) "Workload" and risk of myocardial infarction. A prospective psychosocial analysis. Int J Epidemiol 6:17-21

14. Weiner H (1977) Psychobiology and human disease. Elsevier, New York, Amsterdam

15. Wolff H (1953) Stress and disease. Thomas, Springfield, Ill.

Measurement and Impact of Organizational Stress

N. van Dijkhuizen

In a national survey of the problems of middle management in industry in the Netherlands that concluded with the 1978 report "Middenkader en Stress" (6) one of the aims was to measure stress at the middle management level, as well as at the levels surrounding middle management, to be able to make comparisons between functions.

Therefore, we used French and Caplan's stress model (2) and merged it with the roleset model developed by Kahn et al. (3). This gave us the different job levels that were to be investigated: middle managers, their immediate superiors, the supervisors, and the workers, all of whom are in a line relationship. In addition, personnel and maintenance workers in a staff relationship were incorporated as were, in some instances, colleague middle managers.

All subjects answered a stress questionnaire and a questionnaire developed to measure reciprocal ideas about one another's jobs and functions. In addition the middle managers were interviewed, and all subjects had their blood pressure, cholesterol level, and heart rate assessed.

The stress questionnaire (VOS), was adapted from the questionnaire Caplan et al. (1) used in their study of job demands and worker health (4). This questionnaire was translated into Dutch and then modified. Some of the scales were eliminated, and others were replaced by known Dutch scales measuring the same object. The scales were then validated in three pilot studies; after each study the questionnaire was adapted until a definitive

version was ready for use in the main study [this whole process is reported in Van Dijkhuizen and Reiche (5)].

The reciprocal ideas questionnaire, as well as the interview guide, were also tested in one of the pilot studies. This resulted, as far as the VOS is concerned, in a questionnaire comprising three main sections: one for measuring stressors, one for strains, and one for personality characteristics.

Sixteen stressors were included. They are (in parentheses Cronbach's alpha): role ambiguity, environment and person scales, in accordance with PE-fit theory (0.74 and 0.69, respectively); responsibility for person's environment and person scales (0.72 and 0.75, respectively); work load environment and person scales (0.89 and 0.72, respectively); underutilization of skills and abilities (0.55); lack of participation (0.70); role conflict (0.78); job future ambiguity (0.75); lack of social support from immediate superior (0.85), from colleagues (0.78), from others at work (0.72), and from partner, relatives, or friends (0.72); and tensions in contact with other departments (only one item) and in relationships with superior and subordinate (each one item).

The number of strains included in the questionnaire was ten (14 if physiologic strains are counted, as well). They are job dissatisfaction (0.65), loss of self-esteem (0.71), job-related threat (0.74), anxiety (0.80), depression (0.70), irritation (0.77), psychosomatic problems concerning general health (0.66) and the heart in particular (0.73), and smoking and absenteeism (one item each). The physiologic strains were measured by company doctors or organizational medical services according to strictly prescribed methods developed by the Commission for the Detection and Prevention of Ischemic Heart Diseases of the Dutch Association for Organisational Medicine, in cooperation with the Department of Cardiology of Leyden University.

Four personality characteristics were initially included in the questionnaire, but both the scales for measuring "assert good self" and "deny bad self" were eliminated because of their

apparent lack of reliability. The two that were left in the analyses were rigidity (0.81) and AB typology (0.73).

This questionnaire was administered to 578 respondents (including 160 middle managers) of three large (with a staff of over 2000) and 14 medium-sized (staff ranging from 500-2000) companies in various branches of industry spread over the country. Companies and, whenever possible, subjects were selected at random.

In the analyses it appeared possible to reduce the 16 stressors to three key stress factors, which were interpreted as "ambiguity," "workload," and "poor relations with others."

The first factor, ambiguity, concerns not knowing exactly what the job responsibilities are, what others expect from the respondents, and what their future career possibilities are. Ambiguity is associated with little social support from the immediate superior.

The second factor, work load, concerns the amount of work the respondents have to do. They are often involved in many projects and assignments simultaneously and are expected to do a great deal of work. This factor is connected with role conflict, in which they have to deal with two or more conflicting demands from the same or from several persons.

The third factor, poor relations with others, refers to lack of willingness on the part of their colleagues to support the respondents. They want to participate more with others in making decisions that affect them and their work. These descriptions are based on the situations as they exist for middle management; the other groups of respondents showed basically the same factors, however.

When correlated with the strains, ambiguity appears to have a significant relationship with psychosomatic problems ($r = 0.52$); a higher cholesterol level ($r = 0.48$); anxiety, depression, and irritation ($r = 0.46$); more absenteeism ($r = 0.43$); higher mean arterial pressure ($r = 0.41$; mean arterial pressure is calculated by adding to the systolic pressure twice the diastolic pressure

and dividing the result by three); and with job dissatisfaction (r = 0.32). This means that as more ambiguity is experienced, one will complain more often about insomnia, migraine, stomach aches, shortness of breath, and hard- or fast-beating hearts. One experiences anxiety, depression, or irritation more often. One is more dissatisfied with one's job and thinks of it as boring and dull. One is more often absent from work and experiences higher blood pressure and cholesterol levels. The reader will certainly recognize some well-known risk factors for coronary heart disease in this listing.

The second factor, work load, shows significant correlations with anxiety, depression, and irritation (r = 0.32); higher mean arterial pressure (r = 0.29); and psychosomatic complaints (r = 0.26), but also with less absenteeism (r = 0.27) and more job satisfaction (r = 0.24). Thus, we see that, apart from negative consequences, which were also present with ambiguity, we do get positive consequences as well. It appears, that a heavy work load can make the work more interesting and may yield more opportunities to learn; one is more satisfied with one's job. Besides, the more the work load, the less one is absent. Either the work load raises the threshold, so that one doesnot give oneself time to be ill, even if "something" is being felt, or one is so busy that illness is not even recognized. It may thus seen attractive to increase the work load: if people are more satisfied with their jobs, they try harder and are absent less. However, if the heavy work load lasts too long, negative consequences appear, and then it is questionable whether satisfaction and little absenteeism will survive in the light of high blood pressure.

The third factor, poor relations with others, is significantly associated with job-related threat (r = 0.63), job dissatisfaction (r = 0.28), anxiety, depression, and irritation (r = 0.28) and, less significantly, with psychosomatic problems (r = 0.23), but also with lower mean arterial pressure (r = 0.44) and less absenteeism (r = 0.31). Respondents who have poor relationships with others are more often worried whether they are accepted by

the people they work with, whether they know well what others expect from them, and whether they can meet their contradictory demands. They are more dissatisfied with their work and report more psychosomatic problems and more anxiety, depression and irritation. However, they tend to have lower blood pressure and to be less absent from work. These last two relationships are, in my opinion, hard to explain.

Next, I would like to make some remarks about the influence of personality characteristics on the experiencing of stressors and strains. I will limit myself here to rigidity and AB typology (as measured by Sales).

As far as rigidity is concerned, in our research the workers appear to be the most rigid, followed, in decreasing order of rigidity, by the supervisors, middle managers, and their immediate superiors. Thus, the hierarchy is followed in reverse order. Members of the staff, and notably those in personnel, are least rigid, i.e., most flexible. This observation could have far-reaching consequences for the innovation policy of an organization. It is mainly those flexible staff members who initiate changes that must be accepted by line management and introduced into the daily routine. In general, they will introduce changes far too quickly and the line, because of their personality characteristics, will resist as much as possible. With regard to AB typology, we also notice differences between the various functions: maintenance staff are mostly A-types, followed by middle managers, their immediate superiors, and the workers. Personnell staff and supervisors are most frequently B-types.

In an extreme groups analysis, where we confined ourselves to middle managers, it appeared that the ca. 30% most A-type middle managers have a significantly greater work load ($\underline{P}$, 0.000), greater responsibility for others ($\underline{P}$, 0.005), enjoy a fuller utilization of skills and abilities ($\underline{P}$, 0.01), possess higher self-esteem ($\underline{P}$, <0.003), are more subject to anxiety, depression and irritation ($\underline{P}$, 0.03), and have a higher diastolic blood pressure ($\underline{P}$, <0.09).

We also examined the relationship between stress factors and strains for A- and B-types. I will give you a handful of examples. Regarding poor relations with others, the A-types show a rather strong correlation with psychosomatic problems (r = 0.45); a similar relationship is nonexistent with B-types (r = 0.02). With ambiguity and diastolic blood pressure we see a positive relationship for A-types (r = 0.34) and a negative one for B-types (r = -0.21). Even more striking is the difference with regard to cigarette smoking. The relationship for B-types (r = 0.76) is much stronger than for A-types (r = -0.32). It appears thus that personality characteristics, and especially AB typology, have a clearly conditioning influence on the experience of stressors and strains and on the relationship between these two.

Another variable taken into consideration was "position in the hierarchy." Results of analyses show that the higher one is placed in a hierarchy, the more one is able to report a greater work load (r_s = 0.36), more responsibility for people (r_s = 0.29), more role conflict (r_s = 0.22), less supportive behavior from colleagues (r_s = 0.20), and more role ambiguity (r_s = 0.20), but also less lack of participation (r_s = -0.32), and less job future ambiguity (r_s = -0.25). Strains likewise show differences. The higher the position, the more one is likely to be overweight (r_s = 0.25) and to experience job-related threats (r_s = 0.19), but also more job satisfaction (r_s = -0.31) and less absenteeism (r_s = -0.22).

A third variable taken into account was the size of the company. When dichotomized into large- and medium-sized companies, employees in medium-sized companies score, on an average, higher in work load, disagreements in their relations with other departments, role conflict, job future ambiguity, and lack of support from superiors and colleagues. With regard to strains they report more anxiety, depression, and irritation, higher systolic and diastolic blood pressure, and higher cholesterol levels. In large companies, however, they report lower self-esteem and more absenteeism (all these differences are significant at at least the $P<0.05$ level).

It is worth noting that the differences we found between A- and B-types and between positions in the hierarchy were consistently greater in medium-sized companies.

The preponderance of A-types in certain jobs and organizations may indicate a self-selection process on the part of the persons concerned. A-types seem to strive toward jobs and/or organizations that involve greater challenges and higher demands. It is, in addition, quite possible that a specific job or a specific organization stimulates more A-type behavior because of the very demands made there. This means that, under the pressure of circumstances, one may start to exhibit a certain type of behavior. Our research results indicate that the work situation in medium-sized companies stimulates a greater amount of A-type behavior than in large companies. This is confirmed by comparing the influences of the size of the company, position in the hierarchy, and AB typology: when analyzing these variables together, it appears that the size of the company has the greatest influence, followed by position in the hierarchy, and that AB typology follows some distance behind. Therefore, there is clearly a reciprocal relationship between the individual and the environment and it seems that individuals adjust themselves to this environment.

References

1. Caplan RD, Cobb S. French JRP, Van Harrison R, Pinneau SR (1975) Job demands and worker health. HEW Publication (NIOSH) 75-160
2. French JRP, Caplan RD (1972) Organizational stress and individual strain. In: Marrow AJ (ed) The failure of success. AMACOM, New York
3. Kahn RL, Wolfe DM, Quinn RP, Snoek JD, Rosenthal RA (1964) Organizational stress: studies in role conflict and ambiguity. Wiley, New York
4. Van Bastelaer A, van Beers W (1979) Questionnaire for organizational stress: psychometrical notes comparing data from five samples (in Dutch). University of Nijmegen, Nijmegen (Report 79 AO 01/CG 01)
5. Van Dijkhuizen N, Reiche HMJKI (1976) Measuring organizational stress: on the adaptation of a questionnaire (in Dutch). State University, Leiden (Report A&O 001-76, 002-76 and 003-76)
6. Van Vucht Tijssen J, van den Broecke AAJ, van Dijkhuizen N, Reiche HMJKI, de Wolff CHJ (1978) Middle management and stress (in Dutch). COP-SER, Den Haag

Psychosocial Stress and CHD when Controlling for Traditional Risk Factors

K. Orth-Gomér

Introduction

There is increasing evidence from large-scale retrospective studies (1), but also from prospective studies (5), that emotional stress has an impact on the development of coronary heart disease. There is still confusion, however, concerning how great this impact is and how it operates. In the present study the following questions were asked: Has emotional stress a direct, independent effect on the cardiovascular system? Or is it perhaps mediated by one or more of the standard risk factors, e.g., by a chronic elevation of blood pressure or blood lipids, or by excessive smoking?

To answer these questions, both psychosocial and standard risk factors were studied in a sample of 150 Swedish men.

Material and Methods

In three large companies in Stockholm all men between the ages of 40 and 65 with known CHD, that is, a previous myocardial infarction (MI) or angina pectoris (AP), were examined. Thirty-two men with MI and 18 with AP only were found in this population of 4000 men. The 50 men with CHD were compared to two other groups of 50 men each: (1) 50 men with standard risk factors for CHD but without overt signs of the disease (the latter was verified by a submaximal standard exercise ECG); (2) 50 healthy men who were selected from the payrolls of the companies. Both groups were selected to match the CHD group for age and type of work.

Traditional risk factors (systolic and diastolic blood pressure, serum cholesterol, triglycerides, uric acid, relative weight, pulmonary function, smoking, and alcohol consumption) were measured using standard physical, chemical, and sociological methods.

For the assessment of emotional stress a special psychosocial interview questionnaire (PSI) was developed. This interview procedure takes about 30 min. Twenty-five questions are asked in a defined order and response alternatives are given to each question. In that way the instrument has the advantage of a standardized questionnaire, but allows for personal contact and confidence in the respondent.

The first part focuses on dissatisfaction, changes and conflict in work, education, and family life. In the second part, questions about periods of great emotional stress are asked: If and when did they occur? How long did they last? What was the cause of the stress reactions?

No note is made of previous CHD in the interview. The questionnaire has been described in detail elsewhere (4).

Results

After the interview, for every man with CHD the 5 years prior to onset of symptoms were analyzed for experience of stress, as were the corresponding periods of life in the matched risk and control subjects. Then relative risk ratios associated with the experience of stress were calculated in the following way:

The number of pairs with a stress-negative control but stress-positive CHD subject was divided by the number of pairs with a stress-positive control subject but stress-negative CHD subject. The same procedure was applied in the comparison of the risk in the control group and the risk in the CHD group (Table 1).

The relative risk of developing CHD when exposed to emotional stress was 6 to 1 when comparing the CHD group to the healthy

Table 1. Estimation of relative risk (RR), according to the crude analysis, in each of the three matched comparisons

		Control		
		Stress	No stress	Total
IHD	Stress	4	19	23
	No stress	3	24	27
		7	43	50
RR, 6.33; P, 0.001				
		Risk		
		Stress	No stress	Total
IHD	Stress	4	19	23
	No stress	6	21	27
		10	40	50
RR, 6.17; P, 0.009				
		Control		
		Stress	No stress	Total
Risk	Stress	1	9	10
	No stress	6	34	40
		7	43	50
RR, 1.5; P, 0.44				

group, and around 3 to 1 when comparing the CHD group to the risk group. The difference between the risk and healthy groups, however, was not significant.

These results suggest that emotional stress acts as an independent risk factor rather than via standard risk factors. To test this hypothesis a special multivariate analysis - a summarizing multivariate confounder score - was used to control for the effect of standard risk factors in the association between stress and CHD. The traditional risk factors enumerated above were combined by multivariate analysis to produce a score, and each subject was rank-ordered into one of five strata. The relative risk ratios were then calculated for each stratum and added up (2). Almost exactly the same values were obtained as in the crude analyses: 6.2 for the CHD control comparison and 3.0 for the CHD risk comparison (Tables 2, 3).

Table 2. Reported stress during 5 years prior to acute manifestation of IHD and during the corresponding period in healthy control persons. Classification by confounder-summarizing score. Evaluation of relative risk (RR)

Multivariate score		Stress	No stress	Total
Stratum I	IHD	4	10	14
	Control	0	3	3
		4	13	17
Stratum II	IHD	2	1	3
	Control	1	12	13
		3	13	16
Stratum III	IHD	2	8	10
	Control	0	6	6
		2	14	16
Stratum IV	IHD	3	5	8
	Control	2	7	9
		5	12	17
Stratum V	IHD	5	3	8
	Control	2	7	9
		7	10	17

RR, 6.23; P, 0.004

Discussion

We concluded that the subjective experience of emotional stress may carry an additional risk for the development of CHD, which is independent of standard risk factors. It is, of course, possible that the magnitude of this risk is overestimated, since the men with MI and AP may have retrospectively interpreted their stress reactions as a cause of their disease and therefore exaggerated their importance. Very few men, however, mentioned disease during the interview and in no CHD case was the disease itself the cause of stress. Patients with AP only can be assumed not to have been exposed to as great a threat as MI patients, and thus may have a lesser need to "explain" their disease. Consequently, these two groups were compared for experience of emotional stress, but no significant differences were found.

Table 3. Reported stress during 5 years prior to onset of IHD and during a corresponding period in men with conventional indicators of IHD risk. Classification by confounder-summarizing score. Evaluation of relative risk (RR)

Multivariate score		Stress	No stress	Total
Stratum I	IHD	3	7	10
	Risk	0	6	6
		3	13	16
Stratum II	IHD	1	3	4
	Risk	2	11	13
		3	14	17
Stratum III	IHD	4	5	9
	Risk	0	7	7
		4	12	16
Stratum IV	IHD	5	7	12
	Risk	1	3	4
		6	10	16
Stratum V	IHD	2	5	7
	Risk	3	6	9
		5	11	16

RR, 3.01; P, 0.06

Caution is also needed when analyzing traditional risk factors retrospectively. It is well known that blood pressure, serum lipids, and smoking, for example, may decrease after an acute MI. Had it been possible to examine these patients before the onset of CHD, some confounding by traditional risk factors may have been found.

For a full description of the methods and results of this study the reader is referred to a coming article by Orth-Gomér et al. in the Journal of Human Stress.

A Cross-Cultural Comparison of Emotional Stress and CHD

I have also had the opportunity to examine a similar sample of American men with the same interview questionnaire. This sample consisted of 166 men working in the greater New York area in the same type of companies as the Swedish men. The sample had the

same age distribution and the same composition of men with overt CHD, men with merely risk factors, and healthy men.

When the quantity of reported stress was compared between Swedes and Americans, no significant differences appeared. But when the causes for the feelings of stress were examined, quite substantial differences were found (see Chap. 1, Fig. 4). Work problems were the dominating stressors in the Swedish group, especially in the CHD group, whereas in the American group, family conflicts were a much more common source of stress, particularly in the CHD and risk groups. This suggests that the quality rather than the quantity of stress reactions differs between the two countries.

It may of course be questioned whether the New Yorkers had more objective reasons for family conflicts or the men in Stockholm more objective reasons to feel stress at work. This could not be examined in the present study. For our purposes, emotional stress is the result of the interaction of both environmental stimuli and individual perceptions of and exposure to these stimuli (3). This interaction, not reality itself, seems to be the cause of emotional stress and must be the target of any preventive or therapeutic intervention.

References

1. Elmfeldt D, Wilhelmsen L, Wedel H, Vedin A, Wilhelmsson C, Tibblin C (1976) Primary risk factors in patients with myocardial infarction. Am Heart J 91:412
2. Miettinen OS (1976) Stratification by a multivariate confounder score. Am J Epidemiol 104:609
3. Orth-Gomér K (1974) Ischemic heart disease as a result of psychosocial processes. Soc Sci Med 8:39
4. Orth-Gomér (1979) Psychological stress in Stockholm and New York. J Psychosom Res 23:165
5. Theorell T, Flodérus-Myrhed B (1977) Workload and risk of myocardial infarction. A prospective psychosocial analysis. Int J Epidemiol 6:17

An Approach by Theory of Action to Occupational Stress

L. von Ferber

Introduction

In accordance with the stress research paradigm of House (1), this paper deals with the "individual perception of stress." While House points out the individual's perception of stress, we are mainly concerned with social patterns of stress perception. It is our opinion that the theory of action can solve some problems that arise from the individualistic approach of the stress theory.

Methods

Using the method of intergroup comparison, George Pepping and I interviewed 400 males aged 45 - 55 years, half blue-collar and half white-collar workers. We compared these two different occupational groups and also compared four groups of patients with heart infarction, dyscardia, stroke, back pain.

The diagnoses were certified by clinical records. The perception of stress can be investigated only by interviewing, and the questionnaire ascertained the following dimensions of work load:

physical effort, two questions; environmental influences, five questions; intensity of work, seven questions; mental stress, four questions; and interpersonal problems seven questions.

In this way we investigated the same dimensions as are used in the evaluation of working places. We paid attention to the working places of blue-collar workers as well as of white-collar workers.

Results

We found that these occupational groups differed in patterns of stress perception. This can be demonstrated by ordering the answers by their percentage. In the following tables we distinguish three groups of stress variables: (1) those perceived by blue-collar workers as well as by white-collar workers; (2) those perceived mainly by blue-collar workers; (3) those perceived mainly by white-collar workers.

In summary, we would like to say that patterns of stress perception are related to the work demands of white- and blue-collar workers (see Tables 1, 2).

Occupational patterns of stress perception are formed by collective experiences in the working groups. A correlation between experience at the work place and the occupational pattern of stress perception is evident.

In contrast to this evident correlation, the origin of illness-specific patterns of stress perception is not evident but must be reconstructed by research. Patients suffering from the same disease, as long as they are at their work place, suffer no collective experiences of stress as far as illness-specific stress reactions are concerned. There is no exchange of stress perceptions between patients of the same disease at the work

Table 1. Stress observed by blue-collar and white-collar workers (ubiquitous stress)

Occupational stress	blue-collar workers (n, 200)	white-collar workers (n, 200)
Working in haste	44%	54%
Forced to concentrate at work	42%	46%
Bad memory	40%	27%
Decisions are troublesome	37%	35%
Always sitting and/or standing at work	34%	39%
Shortage of O_2 or lack of air	30%	25%
Restlessness	28%	34%
Overtime work	17%	21%

Table 2. Stress perceived mainly by one occupational group only (occupational stress patterns)

Blue-collar workers	%
Noise	42
Heavy physical effort	31
Heat and perspiration	30
Work in all weather	21
White-collar workers	**%**
Interruptions in the task	53
You can't accomplish all you have to in your work time	38
It's not possible to satisfy all customers	33
Trouble with colleagues and/or boss	22

place. Illness-specific stress perceptions arise from individual coping and defense mechanisms. They change the development of the patient's career. Illness-specific stress perceptions develop through a process of interaction of workers with their work demands and of workers with their premorbid stage.

Certain consequences arise from the interactional status of the illness-specific perception of stress. Terminologically, it is a dependent as well as an independent set of variables.

As Table 3 demonstrates, we can observe an interaction of occupational and illness-specific perceptions of stress; a comparison of groups of white-collar and blue-collar workers with heart infarction: They differ in their occupational stress perceptions, since there are blue collar-specific stress items and white collar-specific stress items. However, HI patients do not differ markedly in their responses to the ubiquitous items.

The rank position and the percentage rate of the item is specific for illness as we can see by comparing stroke patients with HI patients (see Table 4 comparing blue-collar worker infarction patients with blue-collar worker stroke patients). For further information see L.V. Ferber (2).

Table 3. Illness specific perception of stress by patients with heart infarction

Blue-collar workers (n, 50)	%	White-collar workers (n, 50)	%
[c] 1. Working in a haste	54	[c] 1. Working in a haste	61
[a] 2. Noise	38	[c] 2. Forced to concentration at work	58
[c] 3. Forced to concentrate at work	36	[b] 3. Interruptions of the task	56
[c] 4. Always sitting or standing	33	[b] 4. Customers unsatisfied	46
[c] 5. O_2 shortage or lack of air	32	5. Always sitting or standing	38
[c] 6. Restlessness	28	[b] 6. You can't accomplish all you have to	38
[a] 7. Bad memory	28	[c] 7. Restlessness	38
[a] 8. Heavy physical effort	28	[c] 8. O_2 shortage; lack of air	36
[a] 9. Heat and perspiration	26	[c] 9. Decisions are troublesome	31
[a]10. Work in all weathers	24	[c]10. Trouble with colleagues or boss	30

[a]Stress perceived mainly by blue-collar workers
[b]Stress perceived mainly by white collar workers
[c]Stress perceived by blue-collar as well as by white-collar workers

Table 4. Illness-specific perception of stress in blue-collar workers with heart infarction compared to those with stroke

HI patients	n, 50 (%)	Stroke patients	n, 50 (%)
1. Working in haste	54	1. Heavy physical effort	44
2. Noise	38	2. Bad memory	44
3. Forced to concentrate	36	3. Forced to concentrate	38
4. Always sitting or standing	33	4. Heat and perspiration	38
5. O_2 shortage of O_2 or lack of air	32	5. Restlessness	33
6. Restlessness	28	6. Noise	33
7. Bad memory	28	7. Overtime work	29
8. Heavy physical effort	28	8. Working in a haste	29
9. Heat and perspiration	26	9. Always sitting or standing	27
10. Working in all weather	24	10. Working in all weather	25

Summary: Interpretation of the Results by Theory of Action

The results of our investigation show that there are occupation- and illness-specific stress perceptions. In terms of theory of action, we conclude the following:

Work demands belong to the normative category of the work situation. These norms apply to the work place independent of the individual. Accordingly, they differ considerably for blue- and white-collar workers. These social norms only partially determine the orientation of action. In the interaction with work demands every one eventually evolves his own patterns of stress perception to cope with his work demands. The subjective interest in routinizing the daily tasks leads the process of developing individual patterns of coping with work demands. In this way, continuities arise in the orientation of social action.

In his famous work "Zur Psychophysik der industriellen Arbeit" (The Psychophysics of Industrial Work), Max Weber (3) described this kind of orientation of social action. His investigation of industrial work provided the background for the formulation in his theory of action.

From the viewpoint of theory of action it can be expected that patterns of perception influence the development of chronic diseases.

References

1. House JS (1974) Occupational stress and coronary heart disease: A review of theoretical integration. J Healt Soc Behav 15:12-27
2. Von Ferber L (1977) Psychosoziale Hintergründe des Schlaganfalls. In: MMG - Mensch, Medien, Gesellschaft 2. Enke, Stuttgart, pp 36-44
3. Weber M (1908) Zur Psychophysik der industriellen Arbeit. In: Gesammelte Aufsätze zur Soziologie und Sozialpolitik (1924). Mohr, Tübingen, pp 61-255

Type A Behavior: Mechanisms Linking Behavioral and Pathophysiologic Processes

D. C. Glass

Introduction

A large body of data on the role of behavioral factors in the etiology and pathogenesis of coronary heart disease (CHD) has accumulated over the past few decades (e.g., 15). Two promising variables have been identified, namely, psychological stress and what has been called the type A coronary-prone behavior pattern (e.g., 18). Stress may be defined as an internal state of the individual when he is faced with threats to his physical and/or psychic well-being. An individual who shows type A behavior is competitive and hard driving, time urgent, and impatient, hostile, and aggressive. By contrast, type B individuals display these characteristics to a much lesser degree. Type A behavior is the outcome of a person-situation interaction, and it is elicited only in the presence of appropriate environmental circumstances, including the challenge of doing well at a difficult task or the stress of uncontrollable aversive stimulation (9).

Psychological Stress and CHD

Several classes of psychological stressors have been linked to the major cardiovascular disorders, including dissatisfaction with marital relationships and other interpersonal relations. Excessive work and responsibility, which leads to feelings that job demands are beyond the person's control, have also been implicated in the development of coronary disease. There are in addition, some data suggesting that acutely stressful events, such as the death of a close relative or a sudden loss of self-esteem, increase the likelihood of a coronary event. More detailed

discussion of these matters can be found in a number of recent review papers and books (e.g., 9, 15).

The physiologic mechanisms whereby psychological stress may enhance the development of cardiac disorders include repeated increases in serum lipids (e.g., cholesterol) and blood pressure; acceleration of the rate of damage to the coronary arteries over time; facilitation of platelet aggregation; induction of myocardial lesions; and precipitation of cardiac arrhythmias. It is believed, in some quarters, that these effects are mediated by enhanced activity of the sympathetic nervous system and the consequent discharge of catecholamines such as epinephrine (E) and norepinephrine (NE) (e.g., 6, 13).

Type A Behavior and CHD

Perhaps the most thoroughly studied behavioral factor contributing to coronary disease is the type A behavior pattern. Issues concerning type A measurement and classification cannot be discussed within the confines of this paper. Suffice it to note here that the major diagnostic tools are a structured interview developed by Friedman and Rosenman and a self-administered questionnaire called the Jenkins Activity Survey for Health Prediction. More detailed considerations of measurement can be found in Dembroski et al. (4).

The strongest available evidence on the association between type A behavior and CHD comes from the Western Collaborative Group Study (WCGS). The results indicate that type A men experienced about twice the incidence of acute clinical events over an 8.5-year follow-up period, compared to type B men (20). This difference occurred independently of other risk factors, including total serum cholesterol, systolic blood pressure, and daily cigarette smoking. Still other research, using coronary arteriography, has documented more severe occlusion of the coronary arteries in type A compared to type B patients (e.g.).

If we accept the results showing a linkage between behavior pattern A and coronary disease, the next question to be posed

concerns the physiologic mechanisms underlying this association. Clinical studies indicate that lipid and related hormonal differences exist between type A and type B individuals (18). Other research shows increased urinary NE excretion in type A's during an active working day compared to excretion during more sedentary evening activities (2). Also relevant here is a study indicating elevated plasma NE responses to competition and stress among type A compared to type B men (8).

Elevated catecholamine responses are likely to be associated with changes in cardiovascular function which could be crucial in the potentiation of CHD and sudden death. A number of studies have, in fact, shown that type A men display greater episodic increases in systolic blood pressure and heart rate than do type B's in stressful and challenging situations (e.g., 3a, 15a, 17). There is typically little difference between the two types of individuals in basal levels of these cardiovascular variables.

Consider, by way of illustration, an experiment from my own laboratory which studied types A and B men from the work force of the New York City Transit Authority (10). All subjects were free of the major risk factors for CHD, including hypertension, diabetes mellitus, excessive cigarette smoking, and elevated levels of serum cholesterol. The purpose of the study, conducted in collaboration with Dr. Lawrence R. Krakoff of The Mount Sinai Medical School in New York, was to assess the effects upon arterial pressure, heart rate, and plasma catecholamines of simple competition in a game versus competition with a hostile and harassing opponent. The 22 A and 22 B subjects were assigned randomly to either "Harass" or "No Harass" experimental conditions.

The protocol for the study can be summarized as follows. After a 25-min baseline period, subjects played a series of nine games of Pong, a computerized television game similar to tennis, against an opponent who pretended to be another subject. He was actually a member of the research staff who had been trained at the game so that he was virtually unbeatable. Throughout the "tournament," this opponent was either nonharassing, or he exhibited hostility by delivering a series of preprogrammed comments such as: "Come

on, can't you keep your eye on the ball"; "Damn, you're not even trying"; or "I don't understand why you're having so much trouble hitting the ball." The nine-game competition was played for a prize, the winner receiving a $25 gift certificate to a major department store in New York. The certificate was prominently displayed atop the television throughout the contest.

Arterial pressure (SBP and DBP) and heart rate (HR) were monitored every 2 min over the course of the session. Blood pressure determinations were made using a Roche Arteriosonde, which ultrasonically detects arterial wall motion. Heart rate was measured with a photocell plethysmograph, which allowed the recording of digital pulsation. Blood for plasma E and NE was obtained by means of an indwelling venous catheter. Samples were taken at the end of the baseline period and again after the third and sixth games. A final sample was drawn after the ninth and final game, when the subject had lost to the opponent, who received the gift certificate.

Plasma E and NE were measured by radioassay, using catechol O-methyl transferase in the presence of 3H-methyl S-adenosyl-methoinine. Thin-layer chromatography was employed for separation of the reaction products, which were then converted to vanillin for counting by liquid scintillation spectrometry (24). The interassay coefficients of variation were 31% and 24% for E and NE, respectively. Intraassay variation of duplicates was 10% for E and 9% for NE.

The results for each dependent measure were averaged for each of the three experimental periods (i.e., blocks of three-game segments) and compared to their respective baseline values. Statistical analyses of these data revealed the following effects:

1) There were no differences between types A and B, or between the two experimental conditions, in mean baseline values for SBP, DBP, HR, plasma E, and plasma NE ($\underline{P} > 0.20$).
2) All increases in the five dependent measures relative to baseline values were statistically significant ($\underline{P} < 0.05$).
3) The increase in SBP for harassed type A was significantly greater than for Not harassed type A and each of the two B groups ($\underline{P} < 0.05$). The relevant data can be seen in Table 1.

Table 1. Systolic blood pressure: mean changes for each third of the contest (in mmHg)

Condition	First third	Second third	Last third
Type A, harassed	+37.5	+41.9	+38.5
Type A, not harassed	+24.5	+27.9	+27.4
Type B, harassed	+24.2	+26.5	+26.4
Type B, not harassed	+24.1	+25.8	+25.3

4) The effect of harassment on DBP was near-significant at the 0.06 level, but there was no difference between types A and B ($\underline{P} > 0.20$).
5) The increase in HR for type A was significantly greater than for type B ($\underline{P} < 0.05$); this difference was attributable to the HR elevations of the harassed type A's. Table 2 presents these results.
6) The increase in plasma E for the harassed type A's was also significantly greater than for any other experimental group, as can be seen in Table 3.
7) Although the mean change-scores for the plasma NE were in the same direction as those for plasma E, they did not attain acceptable levels of statistical significance. There was simply too much within-group variability.

The conclusions of this experiment can be summarized as follows:

1) Competition elicits significant and similar increases in BP, HR, and plasma catecholamines in types A and B men.
2) The effect of a hostile opponent causes no reliable differences in cardiovascular and plasma catecholamine responses of type B's.

Table 2. Heart rate: mean changes for each third of the contest (in bpm)

Condition	First third	Second third	Last third
Type A, harassed	+26.1[a]	+24.6	+21.4
Type A, not harassed	+15.6[a]	+17.8	+14.9
Type B, harassed	+12.4	+13.4	+12.8
Type B, not harassed	+14.0	+12.1	+10.9

[a]Apparatus failure interfered with the recording of heart rate from one case in this group

Table 3. Plasma epinephrine (E): mean changes during and after the contest (in pg/ml)

Condition	During the contest	After the contest
Type A, harassed	+100.2[a]	+120.5[b]
Type A, not harassed	+ 3.7	+ 19.8
Type B, harassed	+ 16.9[a]	+ 2.0[b]
Type B, not harassed	+ 16.1	+ 20.0[b]

[a]Two cases each were lost from the harassed A and harassed B groups because of technical difficulties connected with assay and blood sampling procedures

[b]Four cases (one harassed A, one harassed B, and two not harassed B's) were eliminated from analysis of E levels in the third (i.e., "after-contest") blood sample. Hemolyzed samples made it virtually impossible to calculate accurate values for these subjects

3) In type A men, however, the harassing opponent elicits greater increases in SBP, HR, and plasma E during competition.
4) It would appear, then, that behavior pattern A is selectively predisposed to ward an enhanced reaction to hostile interactions, but that competition alone does not distinguish between types A and B.

This experiment, as well as others conducted in my laboratory over the past year, suggests some of the cardiovascular and hormonal variables that might account for the greater tendency of type A individuals to develop coronary disease. It is, of course, speculative to argue that excess production of epinephrine and heightened systolic pressor responses serve as the intermediary process by which type A behavior enhances the risk of cardiovascular disease. However, it is probably reasonable to assume that the observed physiologic responses of type A's to environmental stress are mediated via the sympathetic nervous system.

Type A as a Psychological Construct

My remarks have thus far emphasized physiologic mechanisms underlying the type A-coronary disease relationship. There is, however, a more basic issue, namely, whether behavior pattern A is a valid psychological construct.

A large proportion of the population is typically classified as type A, with estimates ranging from 45% to as high as 76% in some populations (e.g., 14, 19). Nevertheless, there is a relatively low incidence of CHD among type A's, albeit significantly greater than in type B's (e.g., 20). Therefore, the causal mechanisms underlying cardiovascular disease may not be distributed evenly throughout the type A group. It is possible that some facets of behavior pattern A have little or no relationship to the disease, since they appear in all type A's rather than in only those who show increased risk. On the other hand, it may be that type A behaviors of any kind always lead to physiologic changes culminating in illness, but that CHD occurs only in some type A's because they lack psychologically and/or physiologically protective mechanisms. In either case, these considerations underscore the importance of understanding the psychological mechanisms giving rise to and sustaining type A behavior.

There are at least three approaches to identifying such mechanisms. The first derives from a factor analysis of the structured interview responses of 186 men from the WCGS (16). Although five primary factors were revealed, only two - competitive drive and impatience - were associated with the later onset of clinical CHD. Subsequent analyses indicated that of the more than 40 interview ratings, only seven items discriminated CHD cases from age-matched healthy controls. Of these seven items, four were directly related to hostility, one was concerned with competitiveness, and the remaining two dealt with vigorousness of voice stylistics. Dembroski (e.g., 5) has developed a component scoring system for the structured interview based on the findings of Matthews et al. The same dimensions that predicted CHD were found to predict experimentally induced elevations in systolic blood pressure and heart rate.

A somewhat different approach to the association between type A behavior and CHD comes from the work of Scherwitz et al. (22), who identified and measured certain speech characteristics that occurred continuously in the structured interview. These characteristics were correlated with simultaneously occurring changes in heart rate, finger pulse amplitude, and blood pressure. Type A

individuals who used many self-references (I, me, my, mine) in answering the interview questions showed the highest levels of systolic blood pressure. By contrast, the type B group had very few significant correlates of self-reference. These results have led to the suggestion that self-involvement might account for both the speech characteristics and the autonomic reactions of type A subjects. Indeed, there is evidence that individuals with acute awareness of themselves behave like type A's. For example, individuals whose attention is focused on themselves are likely to be aggressive when provoked (21). While performing a task, self-aware individuals are more likely to compare their performance to their internal standards of excellence (e.g., 3). To the extent that these standards are high, salient discrepancies between performance and goals may lead to excessive striving, frustration, and helplessness. Thus, Scherwitz et al. (22) suggest that the construct of self-involvement is useful not only because it may explain why type A behaviors arise, but also because correlations with cardiovascular and behavioral variables underscore its potential importance as a key construct in explaining the linkage between type A behavior and CHD.

A third approach to the issue of mechanism comes from my own past work on type A behavior (e.g., 9). In several studies, my students and I have found that type A's work hard to succeed, suppress subjective states (such as fatigue) that might interfere with task performance, conduct their activities at a rapid pace, and express hostility after being frustrated or harassed in their efforts at task completion. It might be argued that these behaviors reflect an attempt by the type A individual to assert and maintain control over stressful aspects of his environment. Type A's engage in a continual struggle for control and, in consequence, appear hard driving and aggressive, easily annoyed, and competitive. Furthermore, this struggle by A's may lead them, when confronted by clear threats to that control, to increase their efforts to assert mastery. On the other hand, if these efforts meet with repeated failure, type A's might be expected to stop responding and act helpless. Stated somewhat differently, initial exposure to threatened loss of control

accelerates control efforts on the part of type A's, whereas prolonged exposure leads to a decrement in these behaviors. This pattern of responding has been described elsewhere as hyper-responsiveness followed by hyporesponsiveness (9).

Experimental results with healthy human subjects reported elsewhere have tended to support the foregoing hypothesis (e.g., 9). However, it should be emphasized that there is no evidence to date which bears on the interactive effects of depression and type A behavior on clinical CHD. Such an association would, of course, be consistent with the hyporesponsiveness aspect of our hypothesis. While it has been suggested that depression is a risk factor for a variety of illnesses and for delayed recovery from such illnesses (e.g., 7, 11, 12), nevertheless there are no data showing that type A's, when depressed, are at greater risk of CHD than their type B counterparts. A test of this notion must await future research.

Central to our approach to type A behavior is the idea that active efforts by type A's to control their environment are accompanied by sympathetic activation and elevated levels of circulating catecholamines. When efforts at control fail - as they will inevitably with an uncontrollable stressor - the theory predicts a shift from sympathetic to parasympathetic dominance. Such abrupt shifts have been implicated in sudden cardiac death (e.g., 7). Still other research, some of which was cited earlier in this paper, indicates that elevated catecholamines are important factors in the pathogenesis of cardiovascular disease and acute clinical events.

The above approaches are but three of many possible avenues for further differentiation of the psychological processes underlying type A behavior and the connection of these processes to physiologic changes that may enhance cardiovascular risk. As these and other models are more fully developed and tested, we may expect the type A concept to be superseded by more differentiated factors that are more closely linked to pathophysiologic mechanisms (cf. 23).

Conclusions

I will conclude this paper with some remarks about directions for future research - at least as I see these directions. As I have already suggested, attention should be given to conceptualizing type A behavior and identifying the psychological processes that produce and sustain it. Most of the existing literature simply describes a behavior pattern and its behavioral and physiologic correlates. We must define more precisely those behaviors in pattern A that are risk inducing. In this connection, serious consideration should be given to the notion that type A behavior is an outgrowth of a person-situation interaction. It follows from this view that efforts need to be directed toward a delineation of the classes of environmental stimuli that elicit the primary facets of the behavior pattern. It is not enough to speak loosely about appropriately challenging and/or stressful events. We need to define these terms with precision. We need to specify the relevant parameters so that we can determine, a priori, which types and levels of stress or challenge are sufficient to produce type A behaviors and the concomitant physiologic responses in both laboratory and field settings.

Another direction for future research should concentrate on linking the behavior pattern or, more appropriately, the underlying dimensions of the pattern, to physiologic processes believed to be routes to atherosclerosis and clinical CHD. Once such correlations are established, subsequent studies (probably with animal models) might be undertaken to elucidate cause and effect; that is, are the physiologic changes observed in type A's under stress the result of behavioral responses, or vice versa? Indeed, both behavioral and physiologic reactions may be consequences of a third, higher-order variable located in the brain. When such causal research is well underway, it will be time for moderate-sized field studies aimed at evaluating whether the principles derived from psychophysiologic experimentation can predict disease endpoints. At that time, too, it might be appropriate to consider the advisability and feasibility of altering type A behavior, or least those facets of the behavior pattern that have been established as enhancing the risk of cardiovascular disease.

References

1. Blumenthal JA, Williams R, Kong, Y, Schanberg SM, Thompson LW (1978) Type A behavior and angiographically documented coronary disease. Circulation 58:634-639

2. Byers SO, Friedman M, Rosenman RH, Freed SC (1962) Excretion of VMA in men exhibiting behavior pattern (A) associated with high incidence of clinical coronary artery disease. Fed Proc 21:99-101

3. Carver CS, Blaney PH, Scheier MF (in press) Focus of attention, chronic expectancy, and responses to a feared stimulus. J Pers Soc Psychol

3a. Dembroski TM, MacDougall JM, Shields JL (1977) Physiologic reactions to social challenge in persons evidencing the Type A coronary-prone behavior pattern. J Human Stress 3:2-10

4. Dembroski TM, Weiss SM, Shields JL, Haynes SG, Feinlieb M (eds) (1978) Coronary-prone behavior. Springer, Berlin, Heidelberg, New York

5. Dembroski TM, MacDougall JM, Shields JI, Petitto J, Lushene R (1978) Components of the Type A coronary-prone behavior pattern and cardiovascular responses to psychomotor performance challenge. J Behav Med 1:159-176

6. Eliot RS (ed) (1979) Stress and the major cardiovascular disorders Futura Mount Kisco, N.Y.

7. Engel GI (1970) Sudden death and the 'medical model' in psychiatry. Can Psychiatr Assoc J 15:527-538

8. Friedman M, Byers SO, Diamant J, Rosenman RH (1975) Plasma catechololamine response of coronary-prone subjects (Type A) to a specific challenge. Metabolism 24:205-210

9. Glass DC (1977) Behavior patterns, stress and coronary disease. Erlbaum, Hillsdale, N.J.

10. Glass DC, Krakoff LR, Contrada R, Hilton WF, Kehoe K, Mannucci, EG, Collins C, Snow B, Elting E (1980) Effect of harassment and competition upon cardiovascular and plasma catecholamine responses in Type A and Type B individuals. Psychophysiology

11. Greene WA, Goldstein S, Moos AJ (1972) Psychosocial aspects of sudden death: A preliminary report. Arch Intern Med 129: 725-731

12. Greene WA, Moss AJ, Goldstein S (1974) Delay, denial and death in coronary heart disease. In: Eliot RS (ed) Stress and the heart. Futura, Mount Kisco, N.Y.

13. Haft JI (1974) Cardiovascular injury induced by sympathetic catecholamines. Prog Cardiovasc Dis 17:73-86

14. Howard JH, Cunningham DA, Rechnitzer PA (1976) Health patterns associated with Type A behavior: A managerial population. J Human Stress 2:24-31

15. Jenkins CD (1976) Recent evidence supporting psychologic and social risk factors for coronary disease. N Engl J Med 294:987-994, 1033-1038

15a. Manuck SB, Craft SA, Gold KJ (1978) Coronary-prone behavior pattern and cardiovascular response. Psychophysiology 15: 403-411

16. Matthews KA, Glass DC, Rosenman RH, Bortner RW (1977) Competitive drive, pattern A, and coronary heart disease: A further analysis of some data from the Western Collaborative Group Study. J Chronic Dis 30:489-498

17. Pittner MS, Houston BK (1980) Response to stress, cognitive coping strategies, and the Type A behavior pattern. J Pers Soc Psychol

18. Rosenman RH, Friedman M (1974) Neurogenic factors in pathogenesis of coronary heart disease. Med Clin North Am 58: 269-279

19. Rosenman RH, Friedman M, Straus R, Wurm M, Kositchek R, Hahn W, Werthessen NT (1964) A predictive study of coronary heart disease: The Western Collaborative Group Study. JAMA 189:15-22

20. Rosenman RH, Brand RJ, Jenkins CD, Friedman M, Straus R, Wurm M (1975) Coronary heart disease in the Western Collaborative Group Study: Final follow-up experience of $8\frac{1}{2}$ years. JAMA 223:872-877

21. Scheier MF (1976) Self-awareness, self-consciousness, and angry aggression. J Pers 44:627-644

22. Scherwitz L, Berton K, Leventhal H (1978) Type A behavior, self-involvement, and cardiovascular response. Psychosomatic Medicine XL:593-609

23. Scherwitz I, Leventhal H, Cleary P, Laman C (1978) Type A behavior: Consideration for risk modification. Health Values: Achieving High Level Wellness 2:291-296

24. Vlachakis ND, Ribeiro AB, Krakoff LR (1978) Effect of saralasin upon plasma catecholamines in hypertensive patients. Am Heart J 95:78-80

Type A, Stress, and Autonomic Reactivity: Considerations for a Study of These Factors in the Work Place

T. M. Dembroski, J. M. MacDougall, J. C. Buell, and R. S. Eliot

The purpose of this chapter is to describe in some detail a proposal for a research project that, in our opinion, is a logical next step in the current efforts to understand the ways in which environmental, behavioral, and physiologic processes interact to contribute to the development of coronary-related diseases. The project described herein is actually already in the initial stage of development and has been submitted to and approved by the Western Electric Company for implementation. The research project is designed to investigate the ways in which personality characteristics, situational factors in the work environment, and individual patterns of autonomic nervous system (ANS) activity are related to stress, illness, and job productivity. The present proposal focuses primarily on the type A coronary-prone behavior pattern and associated patterns of cardiovascular and ANS reactivity. This emphasis reflects that extensive research now links the type A/B behavior dimension to increased risk of coronary heart disease (CHD), and an increasing body of evidence suggests that excessive levels of ANS reactivity shown preferentially by type A individuals may constitute one of the mechanisms for this increased risk. Additional research suggests that nonoptimal ANS responding may also contribute to a variety of other minor and major illnesses which impact negatively on worker efficiency and well-being.

Background and Rationale for the Proposed Research

Where We Have Been

As with many chronic diseases, medical science now recognizes that the etiology of CHD involves a complex interaction of constitutional, environmental, and behavioral factors. In the case of CHD, however, the behavioral component plays a conspicuous and possibly critical role in heightening the progression of normal disease processes. This behavioral involvement appears to take two general forms. The first consists of behaviors which heighten the risk of CHD through ingestion of substances which insult or overburden the body's normal metabolism. These consummatory behaviors include smoking, excessive alcohol consumption, and dietary intake of excessive quantities of such substances as lipids and calorie-dense carbohydrates. The importance of these behavioral factors is underscored by the extensive efforts of governmental and private medical groups to persuade people to eliminate or modify the most damaging of these behaviors (32,34).

However, behavioral factors seem to contribute to the etiology of CHD through a second set of pathways, which globally speaking, all involve the concept of "stress" as a psychological and physiologic response to social and environmental factors. Although the role of stress in coronary disease has been appreciated by practicing physicians for hundreds of years (e.g., William Harvey, John Hunter, William Osler, T. Von Dusch, and others), it is only relatively recently that scientifically based evidence has emerged to support the validity of such processes as contributors to or precipitators of coronary disease (4, 29).

While several behavioral processes have been implicated in the etiology of CHD, including status incongruity and social mobility, inadequate coping with aversive life events, anxiety, neuroticism, and life dissatifaction, the most compelling evidence for a causative behavioral process has emerged from the work of Friedman and Rosenman on the type A coronary-prone behavior pattern (28,29). This behavioral syndrome is characterized by excessive achievement striving, impatience, time urgency, irritability, and aggressive-

ness. Early in their research, Friedman and Rosenman (17) observed that these attributes are often accompanied by vigorous voice stylistics and exaggerated psychomotor mannerisms, which frequently occur in response to verbal challenges or provocations from the social environment. Based on this observation, Friedman and Rosenman developed a structured interview, in which voice and psychomotor activity, as well as the subject's verbal reports concerning the presence or absence of elements of the type A pattern, could be used to designate subjects as type A or the converse pattern called type B. Type A's were identified as those evidencing a preponderance of type A attributes, while type B's were characterized by an absence of type A behaviors. Thus, a coherent definition of a coronary-prone behavior pattern and a means of measuring or operationally defining the pattern were established.

The next step taken by Friedman and Rosenman was to conduct a prevalence study (17, 37) in which they demonstrated that type A attributes were far more characteristic of both male and female patients with CHD than of individuals not showing symptoms of CHD (20). Critics argued, however, that the sample sizes were too small, that the investigators could have biased the results, and that the coronary patients themselves may have incorrectly attributed type A characteristics to themselves in an effort to explain why they had suffered a heart attack. Such criticisms could best be addressed by conducting a study in which type A behaviors were measured before the onset of clinical CHD to determine if the behaviors preceded rather than followed the disease. A study of this sort is called an incidence, or prospective, study, as opposed to a prevalence, or retrospective, study.

Shortly thereafter, Rosenman and Friedman launched the Western Collaborative Group Study (WCGS, 38). More than 3000 middle-aged working male adults, primarily managers who were symptom-free of CHD, participated in the study, which began in the early 1960s. About one-half of these men were designated type A and the other half type B at the start of the study. The final results revealed 8.5 years later that type A's were about twice as likely to

manifest the symptoms of CHD, including angina pectoris, myocardial infarction, and sudden death. Moreover, the type A pattern also predicted second and third coronary events (39, 40) and was shown to be significantly associated with the extent of coronary atherosclerosis at autopsy, regardless of cause of death (19). Since the research team did not know who would develop the disease at the beginning of the study, and since those physicians who judged the type and degree of disease were ignorant of the subjects' type A or B classification, the results were not due to bias. Additional support for the validity of the results was gained when further analyses showed that the standard risk factors of elevated levels of cigarette smoking, serum cholesterol, age, and blood pressure were related to CHD in the same manner as was demonstrated in other investigations such as the classic Framingham study (3, 6, 31). Another very important finding from the WCGS was the demonstration that the type A pattern conferred risk independently of the above-mentioned risk factors, which meant, for example, that type A's were not uniformly higher than type B's in blood pressure, cholesterol levels, age, or number of cigarettes smoked per day (2, 3).

In addition to the WCGS data, a large amount of additional evidence has linked type A attributes with clinical CHD, including data from many other Western countries (28,29). Much of this work in the United States has been conducted by Dr. C. David Jenkins and his associates, who developed a questionnaire method of measuring type A behavior called the Jenkins Activity Survey (JAS, see 29, 30). In support of Dr. Jenkins's findings, Dr. Susan Haynes and her co-workers have shown through reanalyses of data from the Framingham study that Type A attributes, also measured by a questionnaire, were associated with the prevalence and incidence of CHD in both men and women (24). Moreover, additional research has confirmed autopsy findings from the WCGS by documenting a significant association between type A attributes and the arteriographically determined severity of atherosclerosis, again in both sexes (1, 14, 43).

Recently the National Institutes of Health in the United States assembled a panel of more than 50 distinguished scientists to

review and evaluate the research in the type A area. After extensive study and discussion of the available evidence, the review panel concluded that the type A behavior pattern conferred risk "... over and above that imposed by age, systolic blood pressure, serum cholesterol, and smoking and appears to be of the same order of magnitude as the relative risk associated with any of these other factors."

Going Beyond Type A

Even though this is the first time in the history of medicine that a behavior pattern not related to clinical signs and symptoms or consummatory habits has successfully predicted the incidence of morbidity and mortality, these research accomplishments are only a first step in advancing the understanding of the stress-disease relationship (4). Since the type A pattern confers risk independently of the traditional factors, it is reasonable to assume that the behavior pattern translates into CHD by mechanisms other than traditional risk factors and/or by transient fluctuations of these risk factors rather than by static or resting levels. For example, it is well known that in response to stress, cigarette smoking and blood pressure can increase dramatically and that serum cholesterol can rise independently of diet, only to return to normal levels when the stressful situation has waned. This has been demonstrated in accountants as the April 15 tax deadline approached (18) and in medical students as final examinations drew near (25, 35). It is also known that stressful or challenging environmental circumstances induce other, related responses, including enhanced production and release of adrenal corticosteroids and catecholamines (21, 23), all of which, in excess, can have direct toxic effects on the body and can accelerate the progression of cardiovascular and other diseases (for reviews see 5, 7, 8, 13, 15, 25, 41).

These considerations raise the question of whether type A individuals are more prone than their type B counterparts to respond to the challenges of everyday life with greater physiologic responses indicative of defensive arousal. If such is the case,

a plausible means is suggested through which the type A pattern may translate into CHD. A growing body of research supports this hypothesis. In addition to a number of early studies by Friedman and Rosenman (see 15, 16), more recent laboratory research has revealed that many type A persons react to a rather broad range of verbal and performance challenges with exaggerated and inappropriate cardiovascular and catecholamine responses (10, 11, 12, 23). A complete review of the entire type A literature, including this topic, can be found in Dembroski and Halhuber (8).

In addition, research has also revealed that some specific behavioral elements of the type A pattern may be more closely associated with physiologic responses and CHD than other components of the pattern. Recall that the type A pattern is a multidimensional construct containing a number of psychological and behavioral attributes. Dr. Karen Matthews recognized this when she rescored interviews from the WCGS in a manner that allowed ratings of separate components of the type A pattern (33). Here, the results revealed that components reflecting speed of activity, job involvement, and past achievements were not associated with CHD, while attributes reflecting potential for hostility, competitiveness, and impatience were significantly related to CHD. In three recent studies by the present authors, these same attributes were found to be most predictive of ANS arousal in a variety of challenging situations (10, 11, 12).

Moreover, Dr. Redford Williams and his co-workers at Duke University and researchers at Columbia University have shown that the potential for hostility predicted arteriographically documented severity of atherosclerosis (14, 42). It thus appears that high-hostile type A's tend to be at higher risk for CHD than low-hostile type A's and that both of these groups tend to be at higher risk than type B's. However, there is some evidence that high-hostile type B's are at greater risk than low-hostile type B's (42). In any case, both epidemologic and psychophysiologic research raises the issue of whether being hardworking and achievement oriented in and of itself should qualify as coronary-prone, if one behaves in a nonhostile and noncompetitive fashion.

Where We Are Going

The foregoing research strongly suggests the need to measure a variety of personality and behavioral factors in working adults in order to further investigate which of these factors, in conjunction with specific kinds of environmental events in the work place, interact to induce chronic and potentially dangerous levels of physiologic arousal.

For example, in addition to their greater tendency to evidence exaggerated physiologic reactivity, numerous studies have shown that type A's differ in the nature of their behavioral responses to environmental challenges (22). One consequence of this differential pattern of response is the possibility that type A's may induce greater stress into a situation, for example, by heightening conflict or tension or by assuming excessive amounts of work and responsibility (9). Particularly relevant in this regard is research in business and industry which has suggested that A's in comparison with B's report greater levels of work involvement and a greater number of work-related achievements (26, 27). Type A's also have been shown to be promoted to higher-level positions more often than type B's (36), and one study has linked economic growth rates of companies to their proportion of type A personnel (26). However, the same type of research reveals that type A's suffer a greater number of minor illnesses and accidents both on and off the job that resulted in absenteeism (36), and that they are somewhat more prone to alcohol abuse (22, 36). Paradoxically, in one study (36), type B's were more likely than A's to develop high blood pressure.

Unfortunately, however, there was no effort made in the above studies to determine which specific behaviors or personality attributes of types A and B were associated with particular work- or health-related events under specific environmental conditions. For example, it may be that only hostile type B's are prone to develop high blood pressure or perhaps that certain type B's develop hypertension when placed in specific kinds of jobs. By the same token, only some type A's may be at risk, while others show little risk. In our research, for example, type B's generally

perform just as well as type A's on psychomotor and cognitive tasks, although in general they are substantially less physiologically aroused. However, we have created circumstances in the laboratory which resulted in elevations of blood pressure and heart rate in type B's that were equal to those of type A's. Conversely, we have created circumstances that resulted in marked decline of physiologic reactions in A's. Moreover, in our research A's and B's typically do not differ in how they describe their emotions or mood states, which suggests that type A's and B's do not feel any differently despite showing different levels of physiologic response. In other words, we believe it is quite possible to perform well and feel emotion without showing excessive and potentially damaging physiologic changes. These are additional issues that need to be explored in a study of working adults, including the relationships between work and emotional satisfaction, performance on the job, and physiologic arousal. It is therefore important not only to investigate variables that have documented importance in organizational and work-related research, but also to link these factors to physiologic variables critical to health and well-being. Only by directly measuring physiologic reactivity can one investigate the environmental, psychological, and behavioral factors that may be responsible for it.

Finally, although it is true that the type A pattern carries a substantial risk for CHD and possibly other diseases, the specificity of the pattern for predicting illness in particular individuals is quite low. Thus, as we have argued, it is clear that research must go beyond the type A/B behavioral dichotomy to identify individuals who are at risk by virtue of characteristics in addition to, or in conjunction with, the behavior pattern. Much of the research discussed above points to the possibility of using direct clinical and on-the-job measures of physiologic reactivity as predictors of future disease emergence. However, at the present time, this possibility rests on indirect evidence and inference. It must be emphasized that no programmatic research presently exists which unequivocally links these physiologic reactivity differences to cardiovascular pathology

in humans. Until prospective studies can demonstrate that autonomic sympathetic and related neuroendocrine reactivity per se is a risk factor for the development of CHD, this line of research must remain theoretical in nature. Thus, a major objective of such a research project with working adults would be to demonstrate the desirability and feasibility of a subsequent prospective study. This rationale is perfectly consistent with the history of prospective epidemiologic studies, such as the classic Framingham study, which was preceded by smaller-scale studies of smoking, serum cholesterol, and hypertension that documented the need for further prospective research (6).

An Outline of a Research Proposal

The major objectives of the proposed project would be fourfold. The first would be to discover the degree to which various measures of coronary proneness and clinical assessments of static and reactive patterns of physiologic activity are related to excessive ANS and cardiovascular responses during the normal working day. The second objective would be to investigate the relationship between on-the-job levels and patterns of physiologic activity and various measures of job stress, psychological discomfort, minor and major illness, absenteeism, and professional productivity. The third objective would be to investigate possible intervention strategies for helping high ANS reactive individuals to reduce their level of physiologic activity on the job and to observe whether such reductions have beneficial effects on reported levels of stress, productivity, and illness. The fourth objective, perhaps the most important, would be the development of background knowledge and expertise which would permit the future launching of a longer-term prospective study linking the potential for situationally induced excessive physiologic responsivity with chronic disease.

The proposed study would utilize male volunteer participants recruited from management and upper-level supervisory personnel of a large industrial firm. The decision to use exclusively male supervisory and management personnel in such a study would be dictated by the following considerations:

a) Male employees in these job categories have been used preferentially in other recent studies of job stress and disease. Comparability in our sample would permit us to cross-validate important findings from these other studies.
b) Supervisory and management personnel are typically subjected to greater ranges and variability of job stress and satisfaction than are individuals working at lower levels. Focusing on this group tends to maximize the probability of discovering important stress-physiology relationships which might not be detected in samples of persons whose day-to-day work experiences reflect more homogeneous and lower levels of stress.
c) The exclusive use of males in the study would be dictated by the fact that women are at much lower risk for cardiovascular disease and that, at the present time, little background information exists concerning the nature of coronary proneness and cardiovascular reactivity in women.

The proposed study would require at least 140 participants. This figure represents a compromise between the need to maximize sample size to increase the power of the statistical procedures and the desire to minimize both logistical problems inherent in natural-setting research and the extent of disruption of company work schedules. Volunteers would be screened initially for a history of extant CHD and/or hypertension to a degree requiring medication. Individuals with either condition would not be included in the study, since the diseases themselves and their associated therapeutic regimes are likely to alter physiologic activity and reactivity levels.

At the beginning, these participants would be given a complete physical examination including assessment of medical history, pulse rate, blood pressure, resting ECG, blood glucose, cortisol, total cholesterol, high density lipoprotein (HDL) cholesterol, low density lipoprotein (LDL) cholesterol, and triglycerides. These data would be related to other variables measured in the study and used to help assess the baseline health status of the participants. Within a few weeks after the physical examination, participants would be administered the standardized interview

used to assess the type A coronary-prone behavior pattern, a challenging history quiz, and an eye-hand coordination psychomotor test (e.g., reaction time test) while ECG, blood pressure, cardiac output, and peripheral vasoconstriction are monitored. The cardiovascular reactivity observed would be used as a predictor variable for other behavioral and physiologic parameters assessed in the study. In addition, the participants would be administered a battery of standardized questionnaires that have been used to assess personality and behavioral characteristics ob subjects from other industries and agencies.

Upon satisfactory conclusion of initial testing, each of the participants would be randomly assigned to one of 20 seven-person cohorts. The only restriction placed on the assignment procedure would be the requirement that a participant's scheduled week of monitoring be at least 2 weeks removed from any scheduled vacation period. Members in the first cohort would participate in on-the-job physiologic monitoring during the 1st week; those in the second cohort, during the 2nd week; and so forth throughout the 20 cohorts. In the event of illness prior to the scheduled dates of monitoring, necessary adjustments would be made in the cohort composition. Prior to his scheduled week of participation, each person would be notified by mail concerning the monitoring sequence procedures to be followed during the upcoming week. The general test format would require that each member of the cohort report to a specified station (hereafter called the "nurse's station") upon arriving at the plant. There, each participant would be fitted by a nurse trained in the procedures with the portable blood pressure and ECG monitoring units and, on the first of the 5 days, instructed in their use. In addition, each person would be given a small remote paging unit and a pocket-sized pad of activity checklists.

During the course of the day, the paging units would be triggered by a programmed tape reader on an aperiodic schedule, with an average intersignal time of 60 min and a range of 45 - 75 min. This schedule would be varied across the 5 monitoring days so that participants would not alter their normal routines in anticipation

of the signal. Upon hearing the signal, the participant would be instructed to be seated immediately and to trigger both the blood pressure and ECG recorders. Both units would be preprogrammed to complete their recording cycles in approximately 45 s. Thereafter, the participant would complete a brief checklist describing his activities and feelings at the time of the sample point and any necessary deviation from the normal protocol. The entire sequence should require no more than 2 min to complete.

At the end of the working day, each participant would return the recording devices, paging unit, and activities checklist to the nurses' station and provide a urine sample for subsequent analysis. Prior to the next morning, the apparatus would be checked for malfunctions and spot checks made in the participant's blood pressure, ECG tracings, and checklist entries to ensure that adequate records are being obtained. If necessary, additional instructions would be provided to the participant the next morning. It is anticipated that these check-in and check-out procedures would average no more than a total of 30 min for each participant per day. On occasion, a participant might have to return to the nurse's station during the day to correct minor malfunctions, such as slippage of the occlusion cuff or loosening of a recording electrode. At the conclusion of the 5th day, each participant would complete a more extensive day-by-day retrospective description of the week's activities.

The conclusion of the first cycle of 20 weeks of monitoring would be followed immediately by a second cycle in which the cohorts would be tested in the same sequence used in the first cycle. Again, it is recognized that some deviations in scheduling would occur due to vacation and illness, but every effort would be made to minimize the degree of temporal shifting in the test sequence.

Independent of the physiologic monitoring procedures, all study participants would complete a weekly work load report and a brief checklist designed to evaluate the incidence of minor physical illness and psychological distress. Repeated sampling, of these data would permit the investigators to ascertain both the average

levels and variability in participants' work experiences and the longitudinal relationship between work stress and psychological and physical distress. In addition, company records of unit and individual productivity and work lost due to illness would be assembled for each participant.

At the conclusion of the monitoring phase of the study, all participants would receive the same tests used in the intake phase of the study. The basic purpose of this third phase of the study would be to secure retest data for the major physiologic, psychophysiological, health behavior, and psychological profile variables measured during intake. Such information would be used primarily to (a) assess the reliability of the type A pattern and related constructs; (b) assess the stability of individual differences in clinically assessed psychophysiologic responses; and (c) identify significant shifts in such areas as health status, job satisfaction, and personal adjustment and, when possible, ascertain the causal sequence for such shifts. The sequencing of the various tests and specific procedures employed would parallel the procedures used in the first phase of the study as closely as possible. Similarly, the participants would go through the retest items in the same order in which they were initially tested. This would be done to insure that the test-retest interval would be approximately the same for all participants.

Subsequent to completion of the retest phase of the formal study, it would be of interest to determine whether highly reactive individuals could be trained to modify their excessive ANS responsivity. Toward this end, a subsample of highly reactive individuals would be invited to participate in a series of stress management sessions designed to help them reduce their physiologic and emotional reactivity to the pressures of the work place. Thereafter, these participants would receive a third, abbreviated monitoring test to assess the effectiveness of the intervention.

An analysis of the data generated by a study of this magnitude would be quite complex, but in outline form it would include the following: (a) descriptive analyses of the person-demographic,

occupational, physiologic, and psychological characteristics of the participants comprising the study sample, and comparison of the sample with others employed in similar studies; (b) factor analyses of the self-report measures in an effort to resolve these numerous veriables into a smaller subset of more fundamental and nonoverlapping dimensions; (c) univariate and multivariate correlational analyses of the interrelationships among the various physiologic measures obtained at intake, and their relationships to logically antecedent predictor variables such as type A, life stress, and health-related behaviors (e.g., smoking); (d) correlational assessment of the predictive relationship between behavioral, physiologic, and psychophysiologic intake measures and physiologic responses to normal on-the-job work activities; (e) within-subject and across-subject evaluation of the relationships between daily and weekly fluctuations in physiologic arousal and reported levels of stress and physical illness; (f) correlational analyses of the test-retest stability of the various measures obtained at the beginning and termination; and (g) multivariate analyses of factors predictive of levels of job success, satisfaction, stress, and similar considerations across the 10 month period of the monitoring phase of the study.

Significance of the Proposed Research

For the first time in medical history, epidemiologic research has firmly established an association between a style of behavior and the incidence of the major clinical symptoms of CHD. In addition, evidence is accumulating that these behaviors may relate to other less severe illnesses, as well as to accidents and alcohol abuse. It is now time to take the next step, which rests on the assumption that type A behavior leads to disease through physiologic reactions. This step involves the attempt to transcend the error inherent in purely behavioral or psychological assessment procedures by focusing directly on environmentally induced physiologic arousal. The proposed research described above would be designed to explore the variables critical in triggering this differential physiologic arousal and to derive

more precise information concerning the environmental, psychological, and physiologic mechanisms responsible for such arousal. In pursuing this hypothesis, we recognize the possibility that an optimal level of physiologic response might very well be the ideal. In any case, it is plausible to expect that such individual differences, if stable over time, may have important implications for differential susceptibility to various chronic and acute disease processes and perhaps other threats to health and well-being as well. The research project described above would allow us to answer a number of questions of this sort and would be useful in developing potential screening devices that could be used for future prospective research and perhaps for identifying individuals at risk for a variety of insalubrious events in the work place.

References

1. Blumenthal JA, Williams R, Kong Y, Schanberg S, Thompson LW (1978) Type A behavior and angiographically documented coronary disease. Circulation 58:634-639
2. Brand RJ (1978) Coronary prone behavior as an independent risk factor for coronary heart disease. In: Dembroski TM, Weiss SM, Shields JL, Haynes SG, Feinleib M (eds) Coronary prone behavior. Springer, Berlin, Heidelberg, New York, pp 11-24
3. Brand RJ, Rosenman RH, Sholtz RI, Friedman M (1976) Multivariate prediction of coronary heart disease in the Western Collaborative Group Study compared to the findings of the Framingham Study. Circulation 53:348-355
4. Buell JC, Eliot RS (1979) Stress and cardiovascular disease. Mod Concepts Cardiovasc Dis 4:19-24
5. Davis R (1974) Stress and hemostatic mechanisms. In: Eliot RS (ed) Stress and the heart. Futura, Mount Kisco, N.Y., pp 97-122
6. Dawber TR, Meadors GF, Moore FE (1951) Epidemiological approaches to heart disease: The Framingham Study. Am J Public Health 41:279
7. Dembroski TM (tobe published) Zusammenhang zwischen Psychophysiologie und Verhalten bei Typ A-Personen. In: Dembroski TM, Halhuber MJ (eds) Verhalten und koronare Herzkrankheit. Springer, Berlin, Heidelberg, New-York
8. Dembroski TM, Halhuber MJ (eds) (tobe published) Verhalten und koronare Herzkrankheit. Springer, Berlin, Heidelberg, New York
9. Dembroski TM, MacDougall JM (1978) Stress effects on affiliation preferences among subjects possessing the Type A coronary-prone behavior pattern. J Pers Soc Psychol 36:23-33

10. Dembroski TM, MacDougall JM, Shields JL, Petitto JA, Lushene R (1978) Components of the Type A coronary-prone behavior pattern and cardiovascular responses to psychomotor performance challenge. J Behav Med 1:159-176

11. Dembroski TM, MacDougall JM, Herd JA, Shields JL (1979) Effects of level of challenge on pressor and heart rate responses in Type A and B subjects. J Appl Psychol 9:209-228

12. Dembroski TM, MacDougall JM, Lushene R (1979) Interpersonal interaction and cardiovascular response in Type A subjects and coronary patients. J Human Stress 5:28-36

13. Eliot RS (1979) Stress and the major cardiovascular diseases. Futura, Mount Kisco, N.Y.

14. Frank KA, Heller SS, Kornfeld DS (1978) Type A behavior pattern and coronary angiographic findings. JAMA 240:761-763

15. Friedman M (1977) Type A behavior pattern: Some of its pathophysiological components. Bull NY Acad Med 53:593-604

16. Friedman M (to be published) Biochemische Anomalien bei Personen mit extremem Verhaltensmuster vom Typ A. In: Dembroski TM, Halhuber MJ (eds) Verhalten und koronare Herzkrankheit. Springer, Berlin, Heidelberg, New York

17. Friedman M, Rosenman RH (1959) Association of a specific overt behavior pattern with increases in blood cholesterol, blood clotting time, incidence of arcus senilis and clinical coronary artery disease. JAMA 169:1286-1296

18. Friedman M, Rosenman RH, Carroll V (1958) Changes in the serum cholesterol and blood clotting time in men subjected to cyclic variation of occupational stress. Circulation 17: 852-861

19. Friedman M, Rosenman RH, Straus R, Wurm M, Kositchek R (1968) The relationship of behavior pattern A to the state of the coronary vasculature: A study of 51 autopsied subjects. Am J Med 44:525-538

20. Friedman M, Brown AE, Rosenman RH (1969) Voice analysis test for detection of behavior pattern: Responses of normal men and coronary patients. JAMA 208:828-836

21. Friedman M, Byers SO, Diamant J, Rosenman RH (1975) Plasma catecholamine response of coronary-prone subjects (Type A) to a specific challenge. Metabolism 4:205-210

22. Glass DC (1977) Behavior patterns, stress and coronary disease. Erlbaum, Hillsdale, N.J.

23. Glass DC, Krakoff LR, Contrada R, Hilton W, Kehoe K, Mannucci E, Collins C, Snow B, Elting E (to be published) Effect of harassment and competition upon cardiovascular and catecholaminic responses in Type A and Type B individuals. Psychophysiology

24. Haynes SG, Feinleib M, Kannel WB (1978) Prospective study of psychosocial factors and coronary heart disease in Framingham. Am J Epidemiol 108:229

25. Herd JA (1978) Physiological correlates of coronary prone behavior. In: Dembroski TM, Weiss SM, Shields JL, Haynes SG, Feinleib M (eds) Coronary prone behavior. Springer, Berlin, Heidelberg, New York, pp 129-136

26. Howard JH, Cunningham DA, Rechnitzer PA (1976) Health patterns associated with Type A behavior: A managerial population. J Human Stress 2:24-32

27. Howard JH, Cunningham DA, Rechnitzer PA (1977) Work patterns associated with Type A behavior: A managerial population. Human Relations 30:825-836

28. Jenkins CD (1976) Recent evidence supporting psychologic and social risk factors for coronary disease. N Engl J Med 294: 987-994, 1033-1038

29. Jenkins CD (1978) Behavioral risk factors in coronary artery disease. Annu Rev Med 29:543-562

30. Jenkins CD, Rosenman RH, Friedman M (1967) Development of an objective psychological test for the determination of the coronary-prone behavior pattern in employed men. J Chronic Dis 20:371-379

31. Kannel WB, McGee D, Gordon T (1976) A general cardiovascular risk profile: The Framingham Study. Am J Cardiol 38:46-51

32. Keys A, Aravanis C, Blackburn H, van Buchem F, Buzina R, Dsordsevic B, Fiozana F, Karvonen M, Menotti A , Pudov V, Taylor H (1972) Probability of middle-aged men developing coronary heart disease in five years. Circulation 45:815-828

33. Matthews KA, Glass DC, Rosenman RH, Bortner RW (1977) Competitive drive, Pattern A, and coronary heart disease: A further analysis of some data from the Western Collaborative Group Study. J Chronic Dis 30:489-498

34. MRFIT Collaborating Investigators (1976) JAMA 235:825

35. Peterson JE, Keith RA, Wilcox AA (1962) Hourly chances in serum cholesterol concentration: Effects of the anticipation of stress. Circulation 25:798-803

36. Rose RM, Jenkins CD, Hurst MW (1978) Air traffic controller health change study. Report to the Federal Aviation Administration, June 1978

37. Rosenman RH, Friedman M (1961) Association of a specific overt behavior pattern in females with blood and cardiovascular findings. Circulation 24:1173-1184

38. Rosenman RH, Friedman M, Straus R, Wurm M, Kositchek R, Hahn N, Wertaessen N (1964) A predictive study of coronary heart disease: The Western Collaborative Group Study. JAMA 189: 15-22

39. Rosenman RH, Jenkins CD, Brand R, Friedman M, Straus R, Wurm M (1975) Coronary heart disease in the Western Collaborative Group Study: Final follow-up experience of $8\frac{1}{2}$ years. JAMA 223:872-877

40. Rosenman RH, Brand RJ, Sholtz RI, Friedman M (1976) Multivariate prediction of coronary heart disease during 8.5 year follow-up in the Western Collaborative Group Study. Am J Cardiol 37:903-910

41. Williams RB (1978) Psychophysiological processes, the coronary prone behavior pattern, and coronary heart disease. In: Dembroski TM, Weiss SM, Shields JL, Haynes SG, Feinleib M (eds) Coronary prone behavior. Springer, Berlin, Heidelberg, New York, pp 141-146

42. Williams RB, Haney T, Gentry WD, Kong Y (1978) Relation between hostility and arteriographically documented coronary atherosclerosis. Psychosom Med 40:88

43. Zyzanski SJ, Jenkins CD, Ryan TJ, Ryan T, Flessas A, Everest M (1976) Psychological correlates of coronary angiographic findings. Arch Intern Med 136:1234-1237

Pattern A, Psychologic Correlates and Therapeutic Implications

M. Haider, M. Koller, E. Lukesch, and M. Müller

A number of research programs have recently been undertaken correlating psychosocial risk factors in humans with physiologically abnormal reactions and dysregulations to clarify the pathophysiologic mechanisms which lead to an increased risk of coronary heart disease.

In this context our institute was involved in a number of epidemiologic, experimental, and intervention studies.

An Epidemiologic Study on Psychophysiologic Correlates of Pattern A

Method

Fifty patients (white-collar workers, aged 35 - 55), who within the last 2 months had suffered a myocardial infarction and stayed in rehabilitation centers, were compared to matched controls (n, 50) with regard to different psychophysiologic measures. Behavior patterns of both groups were defined by the modified and translated Jenkins Activity Survey (JAS). Different personality factors, life change events, and job attitudes were investigated in personal interviews. Pre- and postinfarction levels of somatic risk factors, such as hypertension, cigarette smoking, obesity, and blood lipids, were registered, and as additional information, the HDL-cholesterol level, which has an inverse correlation to the prevalence of CHD, was analyzed.

Preliminary Results

Compared to the controls, we could find that the distribution of type A and B subjects was significantly shifted toward extreme type A's in MI patients, as indicated in Fig. 1.

With regard to personality variables of validated personality tests, higher scores of dominance (16 PF, $P \leq 0.01$) and aggres-

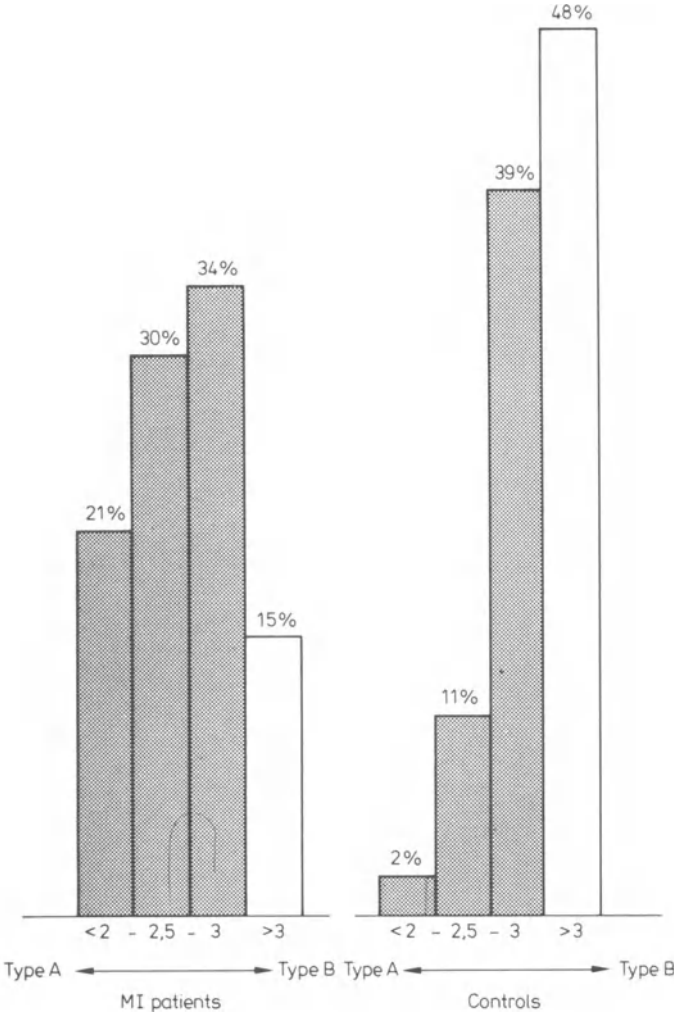


Fig. 1. Distribution of types A and B according to the Jenkins activity scale in MI patients and controls. $P \leq 0.01$, χ^2 *test*

sivity (FPI, n.s.) were calculated for the MI group. The differentiation of both groups according to behavior types revealed that a significantly higher proportion ($P \leq 0.01$) of MI type A's showed excessively high scores in dominance, whereas controls did not differ much on this item. Aggressivity was highest in MI type A's, followed by control type A's.

The total number of recent life changes, especially those related to job alterations, was much higher ($P \leq 0.05$) in MI patients than in the control group. Attitudes toward the job were rated as ambivalent by MI patients and a significantly higher proportion ($P \leq 0.01$) felt very strained by their work and had had an excessive rate of working hours and irregular work times within the last 5 years. Looking at the somatic risk factors, we can report the following results: The percentage of MI's who smoked (especially more than 20 cigarettes per day) and were suffering from considerable overweight was significantly higher than in the controls; however, no significant differences appear within the groups of either type A or B individuals. Blood pressure values differed only slightly between the personality groups. The physiologic values referred to here are confined to cholesterol and HDL-cholesterol. There was a tendency towards higher cholesterol levels in MI patients in spite of treatment and special diet in the rehabilitation clinic with type A's showing slightly higher values.

The most pronounced differences were found in the HDL-cholesterol fraction relating to total cholesterol levels. This lipid subfraction was significantly lower ($P \leq 0.05$) in MI patients. In this parameter, we observed a continuous decrease in values from type B to extreme type A patients. This finding is summarized in Fig. 2.

Discussion

The preliminary results of our study indicate that the type A behavior pattern in the development of CHD must be considered an independent risk factor, and that it does not increase the incidence of disease by way of its effects on blood pressure, smoking

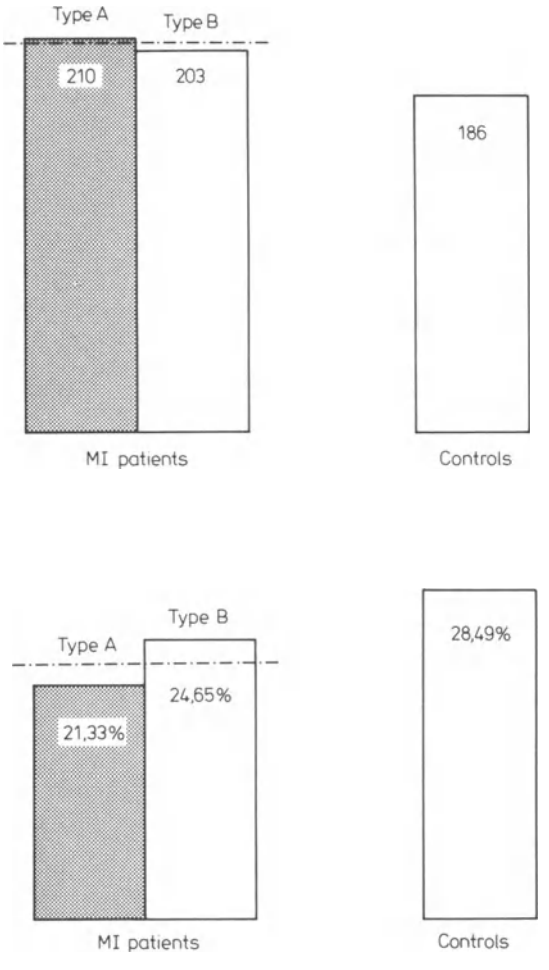


Fig. 2. Blood concentration of cholesterol (upper, mg %) and high density lipoprotein-cholesterol (lower, relative %) in MI patients and controls

habits, or weight. With respect to biochemical parameters, especially to blood lipids, type A behavior may play a mediating role in the development of hyperlipidaemias, especially the relative decrease of HDL cholesterol. One of the mechanisms by which these metabolic changes in blood parameters are induced may be a "conditioned stress reaction."

A Concept of "Conditioned Stress" and Some Experimental Results

Our experiments were designed to investigate stress-induced blood parameter changes. Moreover, we wanted to analyse the importance of conditioned stimuli in the process of metabolic changes. The underlying hypothesis was that neutral stimuli which are presented

several times in connection with stressful stimuli may provoke by themselves a stress reaction and corresponding blood parameter changes. This hypothesis is based on the paradigm of learning (classical conditioning).

Methods

In our first study, animals were exposed to a muscular strain in a modified running wheel (Noble Collip drum). These stress situations were initiated by neutral light and tone signals (unconditioned stimuli, UCS). After a series of combined exposures to this muscular and psychological stress situation and after a one-week break, the UCS was presented alone to test the conditioned physiologic reaction. The second experiment was designed as a simple psychological stress situation. Rats were repeatedly exposed to intermittent white noise of 110 dB for 2 h each time. Again, after a series of combined exposures of the UCS (light signals) and the noise, the UCS was given alone and conditioned alterations of blood parameters were tested (2).

Having succeeded in provoking conditioned blood lipid and protein changes in animals, we then conducted experiments with humans. Subjects were exposed to a complex and realistic stress situation in which they had to perform a rather monotonous task under time pressure and were paid according to their performance. This work situation, additionally aggravated by noise, light flashes, and unsatisfactory illumination, lasted 1 week, 1 h per day. Neutral tone stimuli were administered before each working phase. After a 1 week break, the subjects were divided into two groups. One of them again had to perform for 1 h; the other group was merely exposed to the neutral signals. Over the entire period, blood glucose and blood lipid levels were analyzed (1).

Some Results

Through experiments on stress in rats, it was proven that our chosen stressors influenced immunobiologically important parameters and that these alterations of the immune response of the

organism could be reproduced by conditioning. We demonstrated that after the first stress exposure a relative alpha-1-globulin increase occurred and that this increase could be conditioned. Furthermore, after ten stress exposures the gamma-globulin values were elevated; again, this increase could be conditioned.

Concerning the different blood lipid fractions, alterations which were partially significant were observed. In a comparison of stress and conditioned levels of blood lipids, no uniform response of the organism was found: after the real stress situations, the alpha-lipoproteins were elevated and the values for the beta-fractions were reduced. The chemical analysis of cholesterol and triglycerides also showed decreased values in accordance with the reduced pre-beta- and beta-lipoprotein bands. The total blood lipid content was elevated.

After the conditioning phase, the conditioned increase in alpha-lipoproteins and total lipids was statistically significant; stress and conditioned values were altered uniformly. But it should be noted that the conditioned alpha-lipoprotein level was significantly higher than that of the controls and that, in contrast to the real stress situation, the triglyceride and cholesterol levels were markedly increased, whereas, after stress, marked decreases had been found.

To clarify these differentiated lipid alterations, we analysed the same parameters, and, in addition, the blood glucose levels in our human experiments.

Perhaps it should be mentioned that with regard to stress reactions, blood lipid and glucose changes were different after single and multiple stress exposures, and that the metabolic alterations in repeated stress situations were obviously modified by habituation and adaptive processes. According to our results, stress-induced metabolic mechanisms developed differently for each blood lipid and for the blood glucose fractions. This phenomenon may be explained by the different functions of these blood parameters in the course of the stress exposure. The results of conditioning were consistent with those obtained in the animal experiments.

A clear conditioned effect was found for blood glucose: Both stress and conditioning produced a significant increase in the values after exposure. Cholesterol and beta-lipoprotein reduction, which was especially pronounced after the first stress exposure and after the exposure following a 1 week break, again did not occur in the conditioned situation. Rather, a marked increase in these parameters was found that was significantly different from the values obtained in the first stress exposure, as demonstrated in Fig. 3.

Discussion

Our experimental studies indicate that different pathophysiologic mechanisms are obviously influenced by processes of learning and conditioning. Bringing together the research on type A and the corresponding psychophysiologic correlates, as well as our results on conditioning processes, we may put forward the following hypotheses:

1) Conditioned physiologic stress reactions may be a relevant mechanism in the development of CHD (by shifting the relative content of different blood lipid parameters, such as HDL-cholesterol);

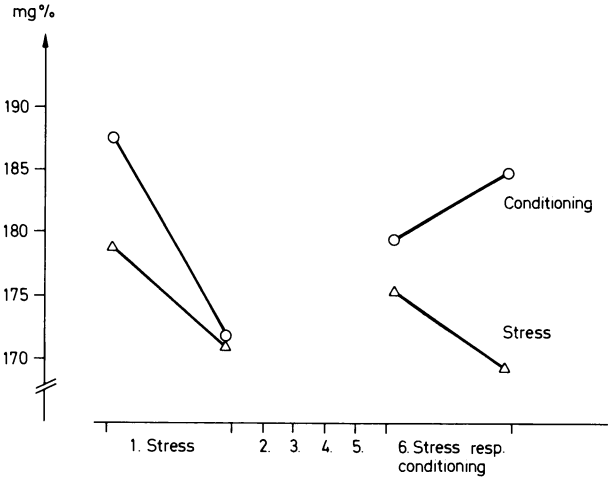


Fig. 3. Mean cholesterol shift as stress response and conditioned stress response. o——o cond. group; ⟷ stress group

2) The probability of type A individuals being exposed to stressful situations, in which conditioning processes are possible, is, by their behavior pattern itself, greater than in type B individuals; and

3) Considering the possibility that type A and B individuals differ in their capacity to be conditioned, as is true for some personality factors, we conclude that through conditioning processes a link between the type A behavior pattern and biochemical alterations, especially with regard to some fractions of blood lipids, may be possible.

We would like to go one step further and propose that if conditioning plays a role in the development of CHD, one of the therapeutic implications should be that deconditioning or desensitization procedures should be included in prevention programs. We are aware of the difficulties connected with individual intervention programs for populations at risk. Nevertheless, we want to offer a suggestion in this regard.

Therapeutic Implications and An Intervention Study of "Deconditioning"

One example of an intervention study designed to deal with the prevention of a second MI was performed in an Austrian rehabilitation center. MI patients were first classified as extreme or moderate A or B types, respectively. The type A individuals were then randomly assigned to two groups. In the intervention group, individual stress situations experienced by each person were ranked in a hierarchy and a systematic desensitization was carried out in which the patients were trained in relaxation techniques. After about ten therapeutic sessions the patients were advised to apply their learned relaxation and deconditioning techniques in real-life stress situations (3).

Looking at the results of this pilot study, we observed in the patients who had participated in our program - in comparison to type A patients with no specific psychotherapeutic intervention and even compared to type B patients - that after 3-, 6-, and

9-months' follow-up; their subjective well being was much greater; they took advantage of their learned (improved) coping mechanisms for their specific stressors; and they kept their somatic risk factors at a lower level for a longer time.

Thus, in discussing therapeutic implications we would first stress that CHD risk prevention programs should include psychotherapeutic intervention directed toward type A behavior and, second, that in this context learning and conditioning effects should be taken into account. Specific psychotherapy will be necessary for reducing type A behavior by making the patients themselves realize their "risk behavior" ("cognitive therapy"), by helping them develop alternative behavior patterns ("problem-solving strategies"), and by teaching them coping mechanisms to prevent extreme stress situations ("stress deconditioning").

Acknowledgments

We would like to thank the directors of the following rehabilitation centers for their cooperation and support:

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Director of the Rehabilitationszentrum Hochegg, Prim. MR Dr. W. Nissel.

References

1. Cervinka R, Koller M, Haider M (1980) Blutlipid- und Blutglukoseveränderungen bei Einfach-, Mehrfach- und Wiederholungsstress. Eur J, Appl Physiol 43:201-212
2. Haider M, Kanz G, Koller N, Schmid H (1977) Stressbedingte Bluteiweiß- und Blutlipidveränderungen und ihre Abhängigkeit von Lern- und Konditionierungsvorgängen. Wien Klin Wochenschr 89:18-24
3. Schoberger R (1979) Verhaltensmodifikation bei Herzinfarktpatienten. Dissertation, Universität Wien

The Syndrome of Vital Exhaustion and Depression and Its Relationship to Coronary Heart Disease

A. Appels

It is well known that many coronary patients report that they felt tired before the onset of myocardial infarction. Those feelings are often accompanied by general malaise (1). To test the structure and predictive meaning of these feelings, a newly developed questionnaire, the Maastricht Questionnaire (MQ), was included in a prospective study. In this study all patients who visited their general practitioner because of complaints of possible cardiac origin were followed during a period of 10 months in an attempt to study which signs or complaints had a predictive meaning as to the occurrence of myocardial infarction or sudden death. Those who experienced a new coronary event (i.e., sudden death, myocardial infarction, serious deterioration of the cardiac status) had a mean MQ score of 133.41 at intakte (n, 37; s.d., 25.15). The mean score of those who experienced no new coronary event was 123.17 (n, 345; s.d., 26.01). The mean score of a healthy control group was 98.42 (n, 317; s.d., 18.42); all differences were significant (2, 3).

A cluster analysis of the items showed that the syndrome measured by the MQ is characterized by feelings of tiredness, loss of vitality, helplessness and hopelessness, depression and hypochondriasis, exhaustion, sleep disturbances, and projection of the powerlessness upon the outer world. The following items illustrate the content of the MQ:

Do you have the feeling that nobody can help you with those problems deep down inside?
Do you have the feeling that you accomplish little lately?
Are you becoming less satisfied with yourself?

Do you lately feel more listless than before?
Have you experienced a feeling of hopelessness recently?
Do you have the feeling that your body is a battery which is losing its power?

In a separate study the MQ and the Jenkins Activity Scale were administered to 238 healthy males, all 39 - 41 years old. It was found that the correlation between JAS and MQ was 0.38, indicating that type A's have more chance of entering into a state of serious exhaustion and depression than type B's.

Those familiar with the book by Dr. Glass on behavior patterns, stress, and coronary disease (5) will have recognized a similarity in the basic assumptions which guided his and our studies.

The notion guiding our research may be explained by a very simple model: People are like airplanes, cruising at different altitudes. Due to his character or to his job demands, the type A person flies at a high cruising altitude. This requires not only a lot of energy (which is reflected in those somatic factors that form a part of the energy household, such as cholesterol or glucose), but at this altitude one meets more stressful events. The work of Glass is like a photographic enlargement of what happens when a type A person meets stressful events which are hard to control. The first reaction is an increase in activities, but when these attempts are unsuccessful, this pattern of hyperresponsiveness is followed by a decrease in activities (hyporesponsiveness) until he regains his cruising altitude.

Repeated cumulative efforts to overcome stressful situations form a disposition to react to these situations with feelings of helplessness and tiredness. Each time a type A person meets a stressful situation this pattern is reinforced and becomes extended. Together with increased atherosclerosis (which is influenced by harmful habits, themselves part of the techniques used to keep flying on high altitudes), feelings of tiredness, depression, and hopelessness form the wear and tear of flying too long at a high altitude. Finally, one becomes unable to mobilize enough energy to cope with a new stressful situation.

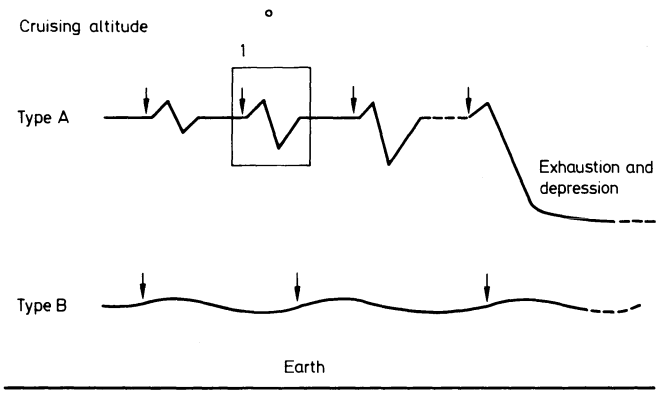

Fig. 1. A simplified model of the pathogenesis of CHD. ↓ Stressfull events; 1 ▭ microsituations studied by Glass

In this period, which has been described by Wolf as "the end of the rope" (7) or as "the decline of the human function curve" (6), one may witness a number of alarm signals: increased sleeping; more visits to the general practitioner (4), an increase in the use of harmful methods to overcome the loss of power (increased smoking, drinking, etc.); an increase in professional activities (the hyperresponsiveness which covers the vital exhaustion and depression), followed by (or simultaneous with) a hyporesponsiveness which manifests itself by increased silence, feelings of listlessness, difficulties in starting one's job, and avoidance of social contacts.

Myocardial infarction generally does not occur as a thunderbolt out of a clear sky. It happens when the sky is clouded by feelings of vital exhaustion and depression.

References

1. Alonzo, AA (1975) Prodromata of myocardial infarction and sudden death. Circulation 52:1056-1062
2. Appels A (1979) Psychological prodromata of myocardial infarction (in Dutch). Ned Tijdschr Psychol 34:213-223
3. Appels A (to be published) Psychic prodromata of myocardial infarction and sudden death. Psychother Psychosom
4. Fulton MB (1969) Sudden death and myocardial infarction. Circulation [Suppl] 4:182-191

5. Glass DC (1977) Behavior patterns, stress and coronary disease. Erlbaum, Hillsdale

6. Nixon PGF (1976) The human function curve. Practitioner 217: 765-770, 935-944

7. Wolf S (1967) The end of the rope: The role of the brain in cardiac death. Can Med Assoc J 97:1022-1025

Psychosocial Stressors in Patients with Premature Myocardial Infarction

W. Langosch

The purpose of this interdisciplinary study was to investigate medical, psychological, and occupational aspects of the long-term history of male patients who are not older than 40 years at the time of their premature myocardial infarction.[1]

Based on a first sample of 35 juvenile MI patients, behavioral and occupational assessment data were subjected to statistical analysis. The results of the occupational assessment are as follows:

1) The analysis of the structure-to-task requirements did not reveal any occupations which could be said to be specific for myocardial infarction.
2) The most frequent stressors were (percentage given in brackets refers to the number of patients who have been confronted with the specified stressor): extraneously determined deadlines (51.4%); extraneously determined interruptions of the work process (45.7%); multifunctional task demands (42.9%); high vigilance (37.1%); holding a second job (34.9%); extraneously determined quantity of work (31.4%); and stressful work hours and other disturbing factors (31.4%).
3) The following factors were experienced as the strongest stressors: a continuously understaffed work situation, extraneously determined deadlines, and extraneously determined interruptions.

1 Special thanks are due to the Bundesministerium für Arbeit und Sozialordnung (Förderungszeichen VI, 6 5-58662-13) for financially supporting this study

The similar distribution of factors of stress and strain across various work environments and demands clearly suggests that a priori they are not associated with specific psychological stressors. The only exceptions are those work environments characterized by an extremely homogeneous structure of task requirements.

4) Compared with the sample of Neuberger and Allerbeck (4), patients' work satisfaction was relatively high, and thus it is obviously of little importance as a coronary risk factor (5).

In regard to their social behavior, patients were found to be relatively unassertive, especially with respect to refusing (94%) or making their own demands (63%). They tended to avoid criticizing others (34%), were scarcely able to accept approval from others (71%), and were inclined toward harsh self-criticism (34%). Furthermore, they had problems in intensifying interpersonal relationships (40%). The above behaviors were largely due to anxiety and behavioral deficits.

Their achievement behavior was characterized by strong competitiveness (89%) and by qualitatively (80%) as well as quantitatively (60%) extreme work habits which were found to be maintained by positive reinforcement, i.e., by appreciation, approval, and social recognition.

The above results are supported by similar results of a related study with 56 post-MI patients aged 40 - 55 years.[1]

Because no control groups were used, no general statement about the specificity of these findings can be made.

In order to investigate the psychophysiologic arousal of the 30 juvenile MI patients, the following study was performed:

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1 This study has been supported by the Bundesministerium für Forschung und Technologie (Förderungszeichen 01 VD 187-AA-TAP 0016)

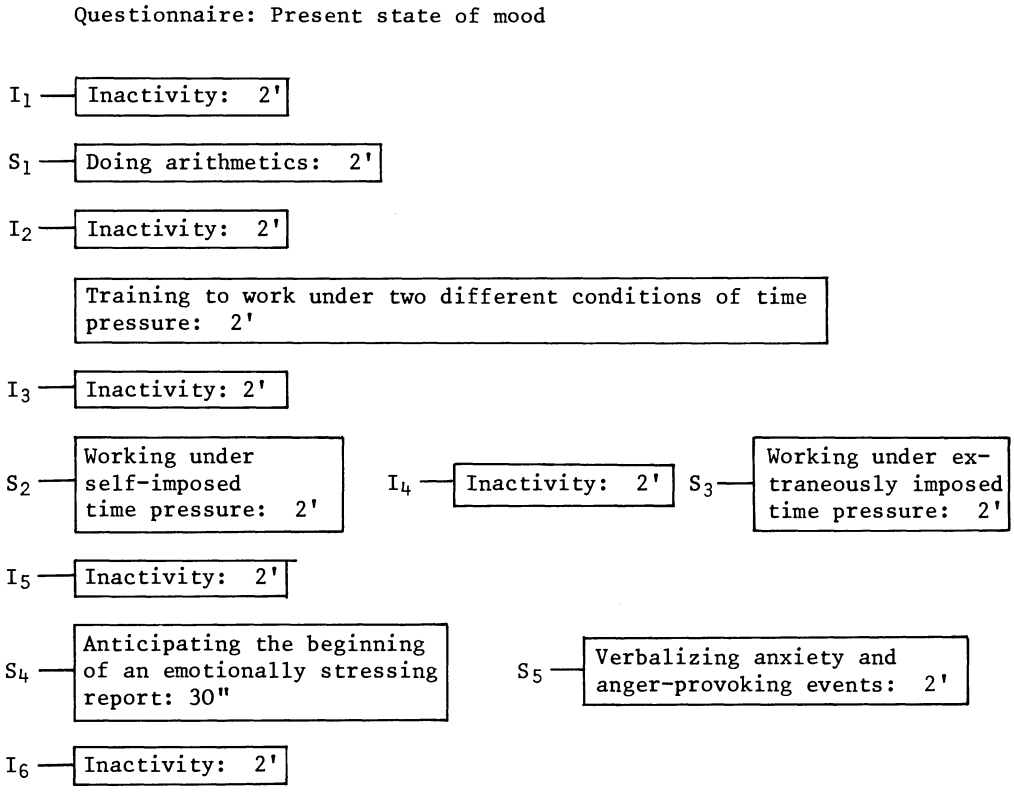

Fig. 1. Design of the psychophysiologic study. Numerals indicate length of each phase

While blood pressure was manually recorded at the beginning and the end of each phase, the ECG, GSR, breathing, and EMG were recorded on a continuous basis. After each phase the patient was asked to rate his subjective state.

The heart rate graph shows a clear rise during the various stress phases, compared to the phase of inactivity. There is no significant difference between stress phases and no significant difference between phases of inactivity.

Similar results were obtained for the other physiologic parameters, i.e., systolic blood pressure, number of GSRs, and the breathing curve.

The patients' ratings of their subjective degree of relaxation at the end of each phase reveal a significant decrease in relaxa-

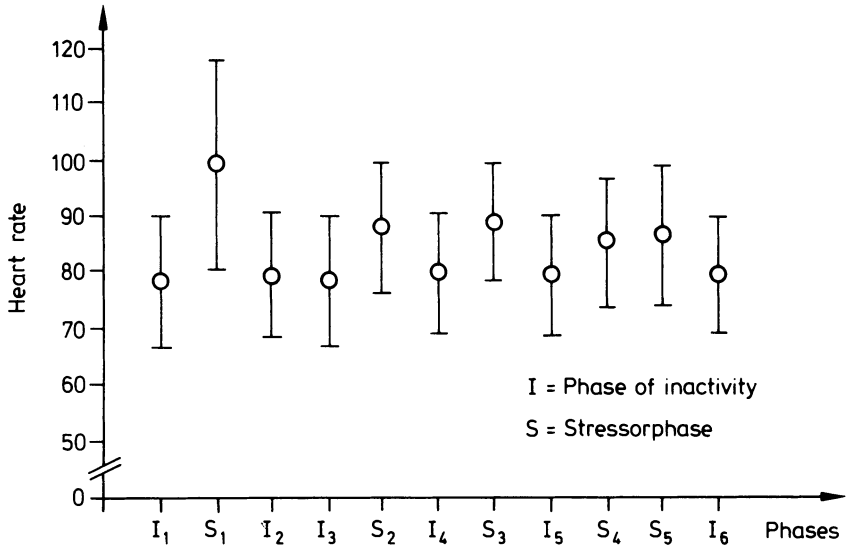

Fig. 2. Changes in heart rate during the 11 phases of the study (I, phase of inactivity; S, stressor phase)

tion during the different stress phases, compared to their corresponding phases of inactivity, with the exception of working under self-imposed time pressure.

The results clearly show that the different physiologic parameters, as well as the subjective variables, are sensitive to the selected psychological stressors and that the stressors used are appropriate stimuli for inducing a state of psychophysiologic arousal.

Table 1. Different coefficients of covariation between selected psychophysiologic parameters

	$\bar{r}_{p\text{-}sit}$	r_{inter}	$\bar{r}_{p\text{-}intra}$
HR[a]/P_s[b]	0.85	0.01	0.58
HR /SCR[c]	0.75	0.15	0.59
P_s /SCR	0.82	0.30	0.54
HR /Rel.[d]	0.64	0.01	0.46
P_s /Rel.	0.50	0.06	0.41

[a] Heart rate
[b] Systolic blood pressure
[c] Number of galvanic skin responses
[d] Subjective degree of relaxation

The various psychophysiologic and psychological parameters were averaged for each of the 11 phases. Based on the data of the averaged patient, the physiologic and psychological parameters were correlated across all 11 phases and found to be consistently high ($\bar{r}_{p\text{-}sit}$). This result shows that the different physiologic and psychological variables change in the same direction when the effect of the differences between patients has been statistically eliminated.

Based on the hypothesis of Schaefer and Blohmke (6) of the pathogenetic dominance of the sympathetic nervous system for the development of coronary heart disease, all patients were examined to determine whether a close relationship could be found between the various psychophysiologic variables.

The calculated correlation coefficients were all found to be insignificant (r_{inter}). This negative result suggests that there is no evidence for any uniform psychophysiologic changes under psychological stress for the whole group of juvenile MI patients. Nevertheless, it is possible that for a selected group of patients a close relationship may exist.

The calculation of the correlation between the different psychophysiologic parameters for one patient at a time and the subsequent calculation of the averaged correlation coefficient for two variables each, i.e., the averaged intraindividual correlation coefficient ($\bar{r}_{p\text{-}intra}$), demonstrated systematic intraindividual positive correlations between psychophysiologic variables.

In summary, these results show that there exists a uniform intraindividual arousal pattern but that it is not possible to trace it for the total group due to evident interindividual variability. The type A behavior concept, which makes frequent reference to the role of sympathetic nervous system activity (1, 2, 3, 7), may help us to better understand this interindividual variability.

A hypothesis was thus formulated that patients who clearly showed characteristics of type A behavior would react to psychological stress with marked psychophysiologic arousal and a largely consistent arousal pattern. It was also hypothesized that they would

display a higher number of those behavioral and personality characteristics suggestive of an increased coronary risk.

Based on the behavioral interview, the sample was divided into high versus low compensatory achievers. Patients who are called "high compensatory achievers" can be characterized as relatively unassertive, scarcely able to accept approval from others, inclined to harsh self-criticism, scarcely able to intensify interpersonal relationships, strongly competitive, markedly careful, strongly conscientious, hard-working, scarcely able to delegate tasks and responsibility, and inclined toward difficult time schedules.

Those who scored high in compensatory achievement behavior were found to be more unsociable, to consider themselves less able to tolerate stress, to have greater difficulties in relaxing in their spare time, to drink less alcohol, and to have lower systolic blood pressure during the psychophysiologic experiment. The "averaged intraindividual correlation coefficients" ($\bar{r}_{p\text{-}intra}$) between the various psychophysiologic parameters indicate marked positive correlations between the psychophysiologic parameters in both patient groups. However, ($\bar{r}_{p\text{-}intra}$) coefficients calculated separately for the two patient groups did not differ significantly.

In summary, these results do not confirm the hypothesis that patients characterized by high compensatory achievement behavior show a more intensive or consistent psychophysiologic arousal pattern.

It appears that dividing the sample according to the degree of compensatory achievement behavior, or a specific cluster of variables which can be regarded as components of the type A behavior pattern, does not further our understanding of the observed interindividual differences in psychophysiologic responding.

Nevertheless, it is possible that the technique of dividing the total group at the median of the selection variable resulted in a leveling of some of the potential group differences. The feasible alternative explanation that the behavioral syndrome of "compensatory achievement behavior" bears little similarity to the type A

behavior pattern, is weakened by the fact that the observed differences in some psychological variables and life-style were in line with the type A behavior concept.

References

1. Dembroski TM, MacDougall JM, Shields JL (1977) Physiologic reactions to social challenge in persons evidencing the Type A coronary prone behavior pattern. J Human Stress 3:2-9
2. Dembroski TM, MacDougall JM, Shields JI, Petitto J, Lushene R (1978) Components of the Type A coronary prone behavior pattern and cardiovascular responses to psychomotor performance challenge. J Behav Med 1:159-176
3. Jenkins CD (1978) Behavioral risk factors in coronary artery disease. Annu Rev Med 29:543-562
4. Neuberger O, Allerbeck M (1978) Messung und Analyse von Arbeitszufriedenheit. Huber, Bern
5. Sales SM (1969) Organizational role as a risk factor in coronary heart disease. Adm Sci Q 14:325-336
6. Schäfer H, Blohmke M (1978) Herzkrank durch psychosozialen Stress. Hüthig, Heidelberg
7. Williams RB, Friedman M, Glass D, Herd JA, Schneiderman N (1978) Mechanisms linking behavioral and pathophysiological process. In: Demkroski TM, Weiss SM, Shields JL, Haynes SG, Feinleib M (eds) Coronary-prone behavior. Springer, Berlin, Heidelberg, New York, pp 120-128

Different Patterns of Stress in Patients with Internal Diseases*

G. Pepping

Introduction

This study was designed to find differential patterns of stressors characterizing different chronic diseases. As this workshop deals with coronary heart disease, only data directly relevant to this disease will be presented.

This investigation looks at chronic stressors associated with work and takes psychological, sociological, and clinical variables into account. These groups of variables have up to now been analysed separately in most studies (for an overview cf. 2, 3).

Variables

Our results are based on two questionnaires and a set of clinical findings.

The Sociological Questionnaire

This instrument provided information on the physical and social characteristics of the subjects' occupational environment. Examples of physical stressors are noise, heat, and prolonged standing. One sum score of these variables was used in regression models (see below).
Six social stressors were included in the regression equations; examples are high responsibility, high concentration demands, and conflict with colleagues.

* This study was carried out with Dr. L.v. Ferber. It was financed by a grant from the Deutsche Forschungsgemeinschaft

The Psychological Questionnaire

The items in this questionnaire concerned behavioral dispositions and attitudes toward work, leisure time, social relations, one's own body, and illness. They have been classified by factor and item analysis into the following three dimensions: competitive achievement striving (five variables); time urgency (four variables); and sensitivity to bodily processes (three variables). The first two dimensions are similar in content to concepts employed in describing type A behavior (cf. 1).

Clinical Data

These included information pertaining to the traditional risk factors of coronary heart disease and alcohol consumption (cigarette smoking, blood pressure, cholesterol level, blood lipids, body weight, and glucose level).

Sample

Four hundred male patients, 45 - 55-years-old, were examined while undergoing treatment in rehabilitation clinics. The sample consisted of four subsamples of 100 subjects each and was characterized by the following four diagnoses: myocardial infarction, functional heart disorders, apoplexy, and degenerative spinal column disease. (The group of apoplectic patients is not considered here.) Patients with spinal column disease or those with functional heart disorders served as control groups.

The use of such groups as controls is subject to criticism; however, because these subjects lived under the same conditions as the coronary patients, i.e., both received treatment in rehabilitation clinics, they enabled us to discover the specifics about coronary heart disease.

Data Analysis

The variance in the diagnosis was explained by means of multiple regression models; the different variable groups (sociological,

psychological, and clinical) were employed as predictors in the regression analysis.

Results

The contributions of the sociological variables to the diagnosis variance are presented in Table 1. The regression coefficients are low, but two of them proved to be significant.

Table 1. Multiple R between occupational stressors and diagnosis (myocardial infarction as opposed to spinal column disease; (n, 102)

	Multiple R	Explained Variance (%)	P
Physical stressors	0.13	2	n.s.
Social stressors	0.24	6	<0.05
All stressors	0.41	16	<0.01

Table 2 shows results of the same procedure with another control group. We had expected the patients with functional heart disorders to differ sharply from coronary patients, which would have resulted in high regression coefficients, but this did not occur.

While the multiple correlation coefficients between psychological dimensions and diagnosis are somewhat higher (Table 3), the group of traditional risk factors accounts for the biggest part of variance (Table 4).

When, in addition to these traditional predictors, the psychological and/or sociological variables are entered into larger

Table 2. Multiple R between occupational stressors and diagnosis (myocardial infarction as opposed to functional heart disorders; n, 82)

	Multiple R	Explained Variance (%)	P
Physical stressors	0.02	0	n.s.
Social stressors	0.14	2	n.s.
All stressors	0.25	6	n.s.

Table 3. Multiple R between psychological dimensions and diagnosis (myocardial infarction as opposed to spinal column disease; n, 102)

	Multiple R	Explained Variance (%)	P
Competitive achievement striving	0.36	13	0.05
Sensitivity to bodily processes	0.38	15	0.01
Time urgency	0.23	5	n.s.
Competitive achievement striving + Sensitivity to bodily processes + Time urgency	0.54	29	n.s.

Table 4. Multiple R between traditional risk factors and diagnosis (myocardial infarction as opposed to spinal column disease; n, 102)

	Multiple R	Explained Variance (%)	P
Traditional risk factors	0.73	53	<0.01

regression models, they add only a small and insignificant amount to the explained variance (Table 5, 6; Figure 1).

Discussion

In view of the above results, some readers might be inclined to argue against the significance of psychosocial factors in coronary heart disease. But it should be kept in mind that the sociological

Table 5. Increase in multiple R by adding psychological variables to traditional risk factors (n, 102)

	Multiple R_{full}	Additional explained variance (%)	P
Traditional risk factors + competitive achievement striving	0.77	6	n.s.
Traditional risk factors + sensitivity to bodily processes	0.75	3	n.s.
Traditional risk factors + Time urgency	0.75	3	n.s.
Traditional risk factors + Competitive achievement striving + sensitivity to bodily processes + time urgency	0.80	7	n.s.

Table 6. Increase in multiple R by adding psychological variables and occupational stressors to traditional risk factors

	Multiple R	Additional explained variance (%)	P
Traditional risk factors + psychological variables + occupational stressors	0.81	12	n.s.

and psychological variables did correlate significantly with the diagnosis. Their negligible role in the larger regression models can be attributed to the order in which the variables were entered into the equations. Thus, overlapping portions of explained variance are attributed solely to the traditional risk factors.

The results presented here suggest that the psychosocial factors do not help to increase substantially the number of predictable cases of myocardial infarction. Instead, we may infer from the data that traditional and psychosocial risk factors overlap to some extent in their pathogenic effect. Further research must be done to disentangle their interplay.

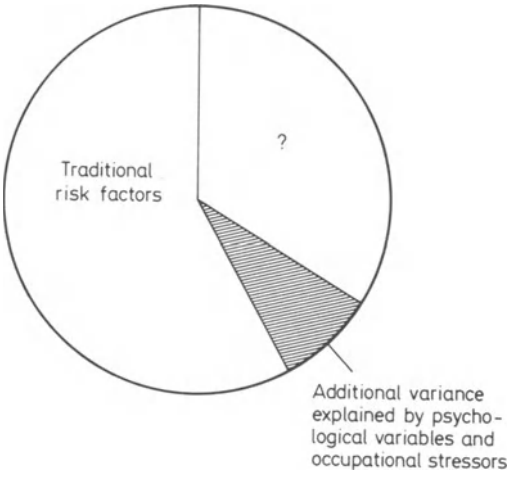

Fig. 1. Increase in explained variance by adding psychological variables and occupational stressors to traditional risk factors

References

1. Dembroski TM, Weiss SM, Shields JL, Haynes SG, Feinleib M (eds) (1978) Coronary-prone behavior. Springer, New York, Berlin, Heidelberg

2. Jenkins CD (1971) Psychological and social precursors of coronary disease. N Engl J Med 284:244-255, 307-317

3. Jenkins CD (1979) Recent evidence supporting psychologic and social risk factors for coronary disease. N Engl J Med 294: 987-994, 1033-1036

Myocardial Infarction and Psychosocial Risks: Concluding Remarks

J. Siegrist

Introductory Remarks

Official statistics of causes of death do not accurately reflect mortality from coronary heart disease (CHD). Underestimates as well as overestimates have been reported (46). There can be no doubt, however, that in the last few decades mortality from CHD has increased considerably in all industrial countries [e.g., by 33% to 53% in individual countries from 1956 to 1967 (45)], except Japan and the United States where a marked decrease during the last 20 years has been reported (13). There are wide variations in terms of age- and sex-specific morbidity and mortality from CHD in general as well as from its most dangerous complications, acute myocardial infarction (AMI) and sudden cardiac death. The rates for men are higher than for women (14), but the differences have diminished in recent years. Various factors, such as smoking, increased occupational activity of women, and oral contraceptives have been cited as possible explanations.

Considering that in the Federal Republic of Germany, for example, more than 100.000 persons per year die of CHD and about 120.000 contract the disease, the significance of this disease complex for public health is quite evident. It is reasonable to assume, of course, that preventive efforts based on a knowledge of risk factors are helping to reduce the incidence of the disease [most markedly so in the United States and occasionally in Finland (13)], and that the well-known improvements in the field of diagnostics, coronary care units, and therapy have also had a beneficial effect.

Nevertheless, present-day medicine, with its theoretical knowledge and guidelines to be followed by the patients, is less successful in the area of CHD than in various other areas. The principal reasons are likely to be (1) the limited applicability of the so-called risk factor concept and (2) the insufficient attention paid to the conditions responsible for the development of these risk factors.

Point 1: Various prospective epidemiologic studies have shown that pronounced coronary risk factors can account for only 50%-60% of the total variance within the period of observation (13, 26). For high density lipoprotein (HDL)-cholesterol, an extensive secondary analysis recently showed that its prognostic value for exclusion of CHD is low and its specificity slight; the authors therefore warn against the indiscriminate use of HDL values as a routine diagnostic test (35). Better predictive value of HDL_2/HDL_3 quotient, as indicated by recent findings (34), has still to be validated in larger groups.

The proportion of risk carriers without manifest CHD is substantial, as is that of victims of infarction lacking any distinct risk factors. Of late there have also been increased findings of juvenile infarction in the absence of any marked coronary atherosclerosis (3). On the other hand, autopsies have revealed advanced coronary sclerosis with no infarction (6).

It is further evident that individuals having extremely high values for one risk factor very frequently have additional risk factors at the same time and that these are numerically small groups; the statistical data concerning relative risks are even less valid.

These are some of the arguments that favor an epidemiologic extension of pathogenetic model concepts. In this connection the influence of higher nervous activity on the cardiovascular system is of special significance.

Point 2: Higher nervous activity has also been considered as a condition for the development of essential hypertension (2,19). Other risk factors such as cigarette smoking, faulty diet, and

lack of exercise point to behavioral and attitudinal patterns which are learned and reinforced in definable psychosocial contexts. Effective prevention, therefore, cannot be confined to the investigation and treatment of somatic risk factors alone. Theoretical as well as practical considerations suggest the need for research into psychosocial stress constellations in premorbid phases.

Possible Mechanisms Linking Higher Nervous Activity to CHD

Animal studies (20) and psychophysiologic research (10) today increasingly support the stress concepts of Cannon and Selye and their differentiation by Lazarus and Mason: The neural-autonomic and hormonal components of strong CNS excitations (sympatheticotonic reactivity) are influenced through the hypothalamus, viz., via the sympathetic nervous system, on one hand, and the pituitary-ACTH-adrenal cortex axis, on the other. The sympathetic nervous system can have a direct effect on organ innervation (intensified by the action of glucocorticoids), and it can also release epinephrine, which reaches the myocardium by way of the blood stream via the adrenal medulla. The physiologic functions of the catecholamines include cardiovascular control, metabolic regulation, and regulation of endocrine secretion (7). The adrenal cortex also releases corticosteroids into the blood.

Two types of effects may be expected to result from such massive emotional excitements:

1) Effects resulting from continuous, long-term repetitions of homeostatic regulation which, acting as a "bias" (52), gradually become pathogenetic (e.g., continuous increase in blood pressure, heart rate, oxygen demand, renin secretion, and plasma fatty acids).

2) Precipitating states of excitement which acutely trigger the morbid event after prior damage (e.g., derangements of myocardial metabolism, coronary spasms, increased sinus rate and extrasystoles, and increased electrical instability of the myocardium).

This concept implies that CNS excitations can lead to CHD in various ways through an increase in sympathetic tone as well as by way of neurohormonal reactions: (a) by long-term exacerbation of atherosclerosis in the coronaries, by a rise in blood pressure, and changes in blood composition (21); (b) by damage to the myocardial metabolism (37); (c) by induction of coronary spasms (31); and (d) by induction of arrhythmias (28).

Many specific research questions regarding individual mechanisms, their pathophysiologic significance, and temporal dynamics, as well as the diagnostic differentiation of varying neurohormonal reactions remain to be settled. For example, correlations between catecholamine measures and psychosocial stress indicators are usually low. This is illustrated, among others, by several contributions in this volume, especially the papers of Theorell, Glass, and Haider et al. Is this partly due to methodological difficulties (2) and to reduced comparability of baseline values? [For a critical analysis of the stress-immune system see Rogers et al. (41)]. Is it an argument against the study of single hormonal reactions, as Mason repeatedly maintains (32)? Several authors plead for a broader analytic framework in the stress field. Brown et al. (4) recently suggested that neurohormonal activation may not occur in every stress situation, but only to the extent that higher nervous reactions are no longer able to "cope" with stressful information input. And Hamilton and Warburton (18) point to the relations between information input and electrocortical activity, which is interrelated with neurohormonal reactions. These suggestions are highly important for future research, although the problem of adequate understanding of how stress experiences are "transduced" into neural and neurohormonal impulses (52) remains unsolved. They indicate that effective research in this area requires new forms of interdisciplinary cooperation between the biochemical, physiologic, and psychosociological sciences.

Psychosocial Risk Constellations: Present Knowledge and Open Questions

If there is sufficient evidence of an interrelationship between strong and/or prolonged CNS excitations and the development of CHD, the question arises as to what conditions provoke such excitations. Are they external situations, and if so, can they be specified? Or are stress reactions mainly due to the individuals themselves, to specific personality or behavioral traits? What kind of interaction between person and situation may be assumed?

The Present State of Knowledge

In the area of sociologically determinable stress factors, research has only recently established clearly defined features that are correlated with increased rates of CHD. A number of studies have emphasized the significance of social change (24): The process of urbanization, of geographic mobility within and between generations, and incongruity of status favor an increased incidence of CHD, in part irrespective of standard risk factors (48). Lack of social support and loss of ties to primary groups, often associated with chronic familial problems (33), frequently increase the incidence and prevalence of CHD as well (30).

The extent to which stressful experiences are attributed to one's family and private sphere may be influenced, as has been shown by Orth-Gomer, by the sociocultural context. In addition, chronic familial problems and lack of social support seem to be related to a larger number of life changes and therefore to a greater vulnerability in the premorbid phase.

The role of subacute life changes before onset of AMI or sudden cardiac death has been the object of numerous retrospective, and at least one prospective, study (see, e.g., 8, 38-40, 49-51). A highly significant rise in cardiovascular mortality could be demonstrated among approximately 4500 widowers during the first few months after the wife's death (36). Nearly one-half of 177 cases of sudden death studied by Engel (12) experienced significant interpersonal losses prior to onset of illness (see also 29).

Starting from results of Rahe et al. (38), Rissanen et al. (40) have shown that subacute and chronic stresses were greatest in those cases of sudden cardiac death in which manifest CHD had been absent. In the Marburg study referred to in this volume, male patients with premature first AMI experienced significantly more (and more severe) events, as well as higher subjective stress levels (mainly due to higher frequency and severity) than did matched controls. Similar results could be found in a study using a different methodological approach (8). Total number of life changes, as measured by the Holmes-Rahe technique (22), was not significantly related to the incidence of CHD cases in a prospective Swedish study (50), but work-related life changes showed a significant effect, which could even be increased by introducing variables such as "concrete work" and "psychosocial work load" in a stepwise multiple regression (see the paper of Theorell in this volume).

Life events which threaten an individual's sense of control have been discussed by Glass (16), who pointed to the possible consequences of depression and hyporesponsiveness in coronary-prone individuals. This issue has also been elaborated on by Appels (1). However, it should be emphasized that, "There is no evidence, to date, which bears on the interactive effects of depression and type A on clinical CHD."

Objective work load as well as subjectively perceived chronic work stressors have been evaluated in a large number of studies (for recent summaries see, e.g., 9). The work of Theorell already mentioned and summarized in this issue, is of special importance. The same holds true for the prospective study of Belgian employees of a private bank and a semigovernmental banking institution undertaken by Kornitzer et al. (27). This study disclosed an incidence of CHD (after 5 and 10 years) twice as high among the employees of the private bank, even though the standard risk factors initially showed approximately the same distribution. Additional surveys referring to a job stress scale revealed significant relationships. A more recent study by Kornitzer et al. involving more than 2000 industrial workers is presented in

this volume and brings to light the highly significant relationship between the presence of angina pectoris symptoms and job stress values.

Several studies point to the fact that subjective job stress and job dissatisfaction are related to occupational characteristics (23,43,44) and to the size of the company; interesting results concerning this latter point are given in the paper of van Dijkhuizen. What are the stressful dimensions of the work experience? This question is still controversial. Frankenhaeuser et al. suggested that certain combinations of quantitative or qualitative "underload" and "overload" may serve as a common denominator of stress experience (15). Theorell concentrates on "high work demands" and "job decision latitude", while others refer to variables such as time urgency, degree of social control, responsibility, inconsistency of work demands, level of concentration, and number of distractions and interruptions (9). Von Ferber tries to introduce an approach to occupational stress by theory of action. Conceptual as well as operational problems - of special importance are questions of validity - will persist in research on this topic during the coming years, and we feel that special emphasis should be placed on examining the interactions between work stressors and conditions in the recreational private sphere, i.e., to accumulations of stressful experiences in everyday patterns of living.

The coronary-prone behavior pattern (type A) described by Rosenman and Friedman (42), which encompasses such psychological characteristics as ambition, latent hostility, zealous devotion to work, a desire to dominate, and resistance to relaxation, has meanwhile been confirmed as an independent coronary risk factor in several prospective studies. Experimental studies on different mental and physical reaction patterns of so-called type A individuals, in contrast with individuals having the opposite characteristics (type B), have been carried out in laboratory situations on a number of occasions (for an overview see, for examplex 10). The papers of Glass, Dembroski et al., Haider et al., and Langosch in this volume represent trends of current research in the field.

Although relatively well-established data are available in the area of type A research and although the evidence for a correlation with pathophysiologic mechanisms is comparatively strong in this area, various questions concerning, e.g., the causes of the pattern, its relation to culture and class, and its consistency over time can as yet be answered only in part. The question of the social and environmental influences that promote development of the type A pattern leads to a broadening of the research perspective in that features of risk situations and predispositions are considered in terms of their systematic interrelationship. Much work in this field remains to be done: "Serious considerations should be given to the notion that type A behavior is an outgrowth of a person-situation interaction. It follows from this view that efforts need to be directed toward a delineation of the classes of environmental stimuli that elicit the primary facets of the behavior pattern." Attention should also be given to conceptualizing type A behavior adequately. Is it possible to connect the three approaches delineated by Glass: the " competition-hostility" model, the model of "self-involvement," and the model of "ambitions of control" (17)? What is the motivational basis and how do cognitive processes operate in these models? Can notions such as "unrealistic demand appraisal" and "lack of self-distance" be integrated into these models?

One promising step in the direction of greater ecological validity of experimental research on type A, stress, and autonomic reactivity is contained in a proposal developed by Dembroski et al., who try to investigate "the ways in which personality characteristics, situational factors in the work environment, and individual patterns of autonomic nervous system activity are related to stress, illness, and job productivity."

Several of the empirical studies in this issue add further evidence to the person-situation interaction, or, as we might say, psychosocial risk constellations (van Dijkhuizen, Kornitzer et al., Orth-Gomer, Pepping, Siegrist et al., and Theorell). But theoretical models integrating both aspects are still poorly developed (18).

It goes without saying that this short summary of present knowledge in the field of psychosocial risks and myocardial infarction is severely limited and that even important issues addressed by the contributions in this volume could not be adequately evaluated. But summing up some major trends of current work gives us the opportunity to develop critical comments and suggestions for further research.

Some Criticisms and Suggestions

Much of the contemporary work in this area can still be characterized by one or more of the following weaknesses:

Imprecise definition of the criterion variable;
Insufficient theoretical basis for the predictor variables and their interrelationships;
Unsatisfactory measurement of psychosocial stress and coping mechanisms and incomplete statistical procedures; and
Lack of data pertaing to process of mechanisms linking stress and CHD resulting from longitudinal studies.

Various quoted studies define the criterion variable by means of administrative data, the reliability and validity of which are often, statistically and epidemiologically, questionable. The criterion variables are often referred to as CHD without specifying subclasses or precise disease entities.

Even with AMI, there is no differentiation between first infarction and later cardiac events, although internal mechanisms of the cardiovascular system presumably play an important part in additional infarctions. In an exact description of the dependent variable, localization, type of infarction, and degree of extension or of coronary stenoses should be documented. Only a few psychosocial studies in the field use these specifications and integrate data obtained by refined diagnostic procedures (such as coronary angiography).

The insufficient theoretical basis for predictor variables has been discussed briefly with regard to current models of concep-

tualizing type A behavior and with regard to work stressors. The same holds true for life events. For a long time the number of life changes occurring a certain time before onset of illness was considered to be important. More recently, attempts have been made to differentiate between subjective experience of stress, psychological and social coping potentials, and contextual circumstances. The dimensions of stress experience considered by different authors have not as yet been given a consistent theoretical basis, although there is some consensus on the role of predictability, controllability, active coping, and social support in dealing with stressful life events (5,11,25). The shortcomings mentioned here are closely connected with a still unsatisfactory research situation in current stress theory (18,47). Integrating medical, psychological, and sociological variables in a dynamic model which is able to explain the relationships between intensity of stress experience, length of exposure, and pathophysiologic processes remains a task for future research.

The questionnaires and interview schedules used for collecting sociological and psychological data in CHD studies have often been inappropriate. Short questions covering a wide range of different experiences were used, and global questions on "work dissatisfaction," "marital discord," etc., were seen as replacements for time-consuming procedures of intensive data collection. Obviously, little attention has been given to the reliability and validity of instruments. Besides type A research, the situation may be characterized by a lack of standardized measurements. Until now few studies have relied on observational data. Most of the work was done by written questionnaires whose methodological fallacies and shortcomings are well known. Bias due to situational and sociocultural factors is rarely discussed.

Again and again, statistical procedures used for data handling have been too simplistic. Multivariate analysis has only recently been integrated into the scientific community. Statistical analyses of tests have been conventional and norm-oriented rather than criterion-oriented, using probabilistic test models.

In summary, the methodological difficulties and problems in the field may be comparable to the theoretical ones mentioned above.

Finally, our critical argument was that there is a lack of data on mechanisms linking stress and CHD resulting from longitudinal studies. Several prospective studies show correlations between psychosocial stress indicators and rates of new CHD cases. But mechanisms postulated by theory are not studied. "The crucial test of relating blood or urinary catecholamine levels prospectively to the occurrence of CHD has not yet been effectuated to our knowledge," as cardiologist Kornitzer writes. We suggest that further basic studies in the field should start by defining psychosocial high-risk groups, which would be followed for several years, controlling for standard risk factors. Continuous measurements of stress experiences as well as neurohormonal reactions should be carried out as far as possible. Perhaps one could even integrate stress-reducing interventions such as counseling, relaxation, or psychotherapy in defined subgroups of the study sample.

Studies of this kind, which are carefully designed and which integrate current knowledge of psychosocial risk constellations on a theoretical and methodological level, might lead to a better understanding of the mechanisms linking social, behavioral, and pathophysiologic processes.

Practical Considerations

A deeper knowledge of risk factors and pathophysiologic processes in the field of CHD provokes new forms of prevention and intervention. One structural prerequisite is extensive documentation in the medical case history. This assumes in the long run that medical personnel will have the knowledge to understand, collect, and evaluate psychosocial information relating to risk groups and to patients and that medical personnel will be trained in handling such information to the advantage of their clients. New forms of counseling and psychotherapy may influence the organizational context of medical work as well as styles of professional cooperation and teamwork.

Prevention and intervention in the field of stress and CHD may not be limited to professional help and medicine. We must further explore the potential of self-help and the benefits of good emotional relationships and social support in the private sphere. Finally, it is thought that an individualistic perspective is too narrow. Occupational and community medicine as well as intervention programs on a community or organizational level are more adequate in coping with several kinds of risk situations. This type of social, and health and political crisis prevention and intervention does, however, require various legal changes and new developments in professional cooperation.

Before such far-reaching suggestions can be seriously approached research into the problems mentioned must be given absolute priority. We have stated the difficulties that are to be expected here, but the benefits which may result from these lines of sociomedical research have also become evident.

References

1. Appels A (1979) Myocardial infarction and depression. Act Nerv Sup 21:65-66
2. Birkenhäger WH, Falke HE (1978) Circulating catecholamines and blood pressure. Bunge, Utrecht
3. Blacket RB (1973) Coronary heart disease in young men: a study of seventy patients with a critical review of etiological factors. Aust NZ J Med 3:39
4. Brown GM, Seggie J, Ettigi P (1979) Stress, hormon responses and cancer. In: Taché J, Selye H, Day St B (eds) Cancer, stress and death. Plenum New York, London
5. Brown GW, Harris TO (1978) Social origins of depression. Tavistock, London
6. Brunner D (1973) Studies in preventive cardiology. The Cardiovascular Research Unit, Jaffa, Tel Aviv
7. Christensen NJ (1979) The role of catecholamines in diseases. Acta Med Scand 624:9-18
8. Connolly J (1976) Life events before myocardial infarction. J Human Stress 2:3-17
9. Cooper C, Payne R (eds) (1978) Stress at word. Wiley, New York
10. Dembroski TM, Weiss SM, Shields JL, Haynes SH, Feinleib M (eds) (1978) Coronary prone behavior. Springer, Berlin, Heidelberg, New York

11. Dohrenwend BP, Dohrenwend BS (eds) (1974) Stressful life events. Wiley, New York

12. Engel GL (1971) Sudden and rapid death during psychological stress. Ann Intern Med 74:771-782

13. Epstein FH (1977) Highlights of epidemiological research in western countries. In: Schettler G, Goto Y, Hata Y, Klose G (eds) Arteriosclerosis IV. Springer, Berlin, Heidelberg, New York, pp 471-478

14. Feinleib M (1979) Epidemiology of coronary heart disease. In: Zülch KJ, Kaufmann W, Hossmann KA, Hossmann V (eds) Brain and heart infarct II. Springer, Berlin, Heidelberg, New York, pp 4-19

15. Frankenhaeuser M, Gordell B (1976) Underload and overload in working life. J Human Stress 2:35-46

16. Glass DC (1976) Stress, behavior pattern and coronary disease. Am Sci 65:177-187

17. Glass DC (1977) Behavior patterns, stress and coronary disease. Erlbaum, Hillsdale

18. Hamilton V, Warburton DM (1979) Human stress and gocnition. Wiley, Chichester

19. Hebeisen H, Halhuber MJ (1977) Epidemiologie des Hochdrucks. In: Blohmke M, Ferber Chr v, Kisker KP, Schaefer H (eds) Handbuch der Sozialmedizin II. Enke Stuttgart, pp 67-82

20. Henry JP, Stephens P (1977) Stress, health and the social environment. Springer, Berlin,Heidelberg, New York

21. Herd JA (1978) Physiological correlates of coronary prone behavior. In: Dembrosko TM, Weiss SM, Shields JL, Haynes SG, Feinleib M (eds) Coronary prone behavior. Springer, Berlin, Heidelberg, New York, pp 129-136

22. Holmes TH, Rahe RH (1967) The social readjustment rating scale. J Psychosom Res 11:213-218

23. House JS (1972) The relationship of intrinsic and extrinsic work motivations to occupational stress and coronary heart disease risk. PhD dissertation, University of Michigan

24. Jenkins CD (1977) Epidemiological studies of the psychosomatic aspects of coronary heart disease. Adv Psychosom Med 9:1-19

25. Kasl S (1977) Epidemiologic studies in psychosomatic medicine. Adv Psychosom Med 9:160-223

26. Keys A, Aravanis C, Blackburn H, Van Buchem FSP, Buzina R, Djordjevic BS, Fidanza F, Karvonen MJ, Menotti A, Puddu V, Taylor HL (1972) Probability of middle-aged men developing CHD in five years. Circulation 45:815-828

27. Kornitzer MD, Dramaix M, Gheyssens H (1979) Incidence of ischemic heart disease in two Belgian cohorts followed during 10 years. Eur J Cardiol 9/6:455-472

28. Lown B, Verrier RL, Rabinowitz St.H (1977) Neural and psychological mechanisms and the problem of sudden cardiac death. Am J Cardiol 39:890-902

29. Lynch J (1979) Das gebrochene Herz. Rowohlt, Hamburg

30. Marmot MG (1975) Acculturation and coronary heart disease in Japanese-Americans. PhD dissertation, University of California

31. Maseri A, Pesola A, Marzilli M, Severi S, Parodi O, L'Abbate A, Ballestra AM (1977) Coronary vasospasm in angina pectoris. The Lancet I, 8014: 713-719

32. Mason JW (1971) A reevaluation of the concept of nonspecifity in stress theory. J Psychiatr Res 8:323-333

33. Medalie JH, Kahn MA, Neufeld HN (1973) Five year myocardial infarction incidence. J Chronic Dis 26:329

34. Miller NE (1980) Communication. Medical Tribune May 8, 1980

35. Noe DA, Rock RC (1979) Comment. N Engl J Med 14:798-799

36. Parkes CM, Benjamin B, Fitzgerald RG (1969) Broken heart: a statistical study of increased mortality among widowers. B Med J 1:740-743

37. Raab W (1970) Preventive myocardiology. Thomas, Springfield

38. Rahe RH, Romo M, Bennett L, Siltanen P (1974) Recent life changes, myocardial infarction and abrupt coronary death. Arch Intern Med 133:221-228

39. Rahe RH, Arajänvi H, Arajänvi S, Punsar S, Kanvonen MJ (1976) Recent life changes and CHD in East vs West-Finland. J Psychosom Res 20:431-437

40. Rissanen V, Romo M (1978) Premonitory symptoms and stress factors proceeding sudden death from ischeamic heart disease. Acta Med Scand 204:389-396

41. Rogers MP, Dubey D, Reich P (1979) The influence of the psyche and the brain on immunity and disease susceptability: a critical review. J Psychosom Med 41:147-164

42. Rosenman RH, Friedman M (to be published) Beiträge zur Typus A Forschung. In: Dembroski TM, Halhuber MJ (eds) Das koronargefährdende Verhaltensmuster. Springer, Berlin, Heidelberg, New York

43. Sales SM (1969) Organizational role as a risk factor in coronary disease. Adm Sci 14:325-336

44. Sales SM, House J (1971) Job dissatisfaction as a possible risk factor in coronary heart disease. J Chronic Dis 23: 861-873

45. Schaefer H, Blohmke M (1977) Herzkrank durch psychosozialen Stress. Hüthig, Heidelberg

46. Schettler G, Nüssel E (1974) Neuere Resultate aus der epidemiologischen Forschung in Heidelberg. Dtsch Med Wochenschr 99:2001

47. Selye H (1975) Confusion and controversy in the stress field. J Human Stress 1:37-44

48. Syme SL, Bouhani NO, Brechley RW (1965) Cultural mobility and the occurence of coronary heart disease. J. Health Hum Behav 6:78-89

49. Theorell T (1974) Life events before and after the onset of a premature myocardial infarction. In: Dohrenwend BP, Dohrenwend BS (eds) Stressful life events. Wiley, New York, pp 101-117

50. Theorell T (1976) Selected illnesses and somatic factors in relation to two psychosocial stress indices. J Psychosom Res 20:7-20

51. Theorell T, Lind E, Floderus B (1975) The relationship of disturbing life changes and emotions to the early development of myocardial infarction and other serious illnesses. Int J Epidemiol 4:281-293

52. Weiner H (1977) Psychobiology and human disease. Elsevier, New York, Amsterdam

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