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Approaches to altering the Type A behavior pattern

ABSTRACT: The Type A behavior pattern, a risk factor for coronary heart disease, is described in terms of the expanded cognitive social learning model that takes into account the interdependent influences of behavioral, environmental, cognitive, and physiologic components. Six studies aimed at altering the behavior pattern are discussed in light of this conceptual framework. Among the issues involved in designing and conducting treatment programs are the targets of behavior (e.g., impatience or hostility) for change; the type of client populations to treat (e.g., teenagers, post-coronary patients); and treatment techniques (e.g., relaxation procedures, cognitive restructuring, mass media programs).

For at least three decades, cardiovascular and cerebrovascular diseases have been the most significant cause of death and disability in the United States and other western cultures. About 600,000 Americans die every year from coronary heart disease (CHD) with about 35% of these deaths (over 200,000) occurring prematurely in persons under 65 years of age.[1]

Medical research has made us realize that the vascular diseases and other chronic disorders, especially those involving the heart, are complex multidimensional problems not caused directly by any single factor, such as excessive dietary intake of cholesterol or cigarette smoking. Some investigators are beginning to realize the magnitude of the problem, specifically, the intimate relationship of heart disease and contemporary lifestyle, especially daily habit patterns, and a few are starting to understand the imperative for a broad inclusive framework in conceptualizing the many factors contributing to CHD.[2] Even when elaborate multivariate statistical analyses are performed, less than one half of the variance associated with CHD is accounted for by risk factors such as hypertension, cigarette smoking, body weight, family history, physical activity, and serum cholesterol level.

The association of CHD with combinations of cigarette smoking, hypertension, elevated serum cholesterol, excessive dietary fats, and other factors leads to the question of what causes these behaviors and patterns. Obviously, other factors play a role in the development of CHD, because (1) not all persons with a high rate of risk factors develop CHD, and (2) many persons suffering myocardial infarction, stroke, or angina are known to have relatively low levels, if any, of these risk factors.

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The Type A behavior pattern

Building on the psychosocial or "personality" perspective of earlier investigators,[3,4] Friedman and Rosenman, in the late 1950s, started to observe the behavior and characteristics of their patients with coronary conditions. They identified a constellation of observable behaviors, which became known as the Type A behavior pattern.[5]

With remarkable consistency they noted a ubiquitous hurriedness, impatience, sense of "time urgency," a pervasive pattern of competitive striving, and frequently and readily aroused angry and hostile feelings and behavior. The key theme they noted was *struggle*—incessant efforts to overcome real or imagined obstacles imposed by time, events, and especially other people. They defined the Type A behavior pattern as:

> an action-emotion complex that is exhibited by those individuals who are engaged in a *chronic and incessant* struggle to achieve more and more in less and less time (thus giving rise to a sense of time urgency or "hurry sickness") and who also usually (but not always) exhibit a free-floating but well-rationalized hostility.[6]

In a series of laboratory and field studies, results began to confirm a relationship between the observable pattern of behaviors and a number of physiologic indicators associated with CHD.[7] Two major prospective studies, the Western Collaborative Group Study and the Framingham Study, have since convincingly shown that persons manifesting the Type A pattern are at significantly greater risk for all forms of cardiovascular disease.[8,9] Other investigators have provided collaborative evidence in a variety of ways that shows a significant relationship exists between the Type A pattern and physiologic factors, especially elevated levels of serum cholesterol, epinephrine, norepinephrine, and triglycerides; increased blood coagulation time; and postprandial sludging of erythrocytes.[10] A meeting convened by the National Heart, Lung, and Blood Institute of medical and behavioral scientists recently concluded that evidence of a substantial predictive relationship between the Type A behavior pattern and CHD was clearcut.[11]

Cognitive social learning model

When we turn to the few published studies of treating Type A behavior, almost all suffer from serious methodologic and conceptual limitations. The absence of an adequate conceptual framework to encompass major components and themes of the Type A pattern has been a major obstacle in developing effective treatment studies. How one conceptualizes the Type A pattern strongly influences how one attempts to alter it. For example, a narrow conceptualization of Type A behavior as the chronic stimulation of physiologic variables has led to the development of intervention strategies aimed primarily at reducing physiologic arousal. For example, while various forms of relaxation training may help reduce physiologic arousal, they may have little impact on other features of the behavior pattern, such as beliefs fostering hostile behavior or social behavior encouraging excessive commitments. What is needed is a conceptual framework broad enough to capture the complexity of the Type A pattern, yet flexible enough to be readily altered by research data.

We have found that an expanded cognitive social learning model provides a useful framework for designing, implementing, and evaluating treatment. This framework recognizes the interdependent and reciprocal influences of behavioral, environmental, cognitive, and physiologic factors.[12,13] The model denies that any one of these factors is sole influence and instead offers a perspective much like the biopsychosocial framework proposed by Engel.[14]

One advantage of the cognitive social learning model is that it provides ways of studying relationships among components of the framework, thus facilitating the development of treatment strategies. For instance, hostility is a Type A characteristic that has been shown to be related to CHD.[15] According to a cognitive social learning model, an adequate conceptualization of the role of hostility in the Type A pattern must consider (1) *cognitive factors* (e.g., the belief that one must beat the other person before getting beaten); (2) *behavioral factors* (e.g., angry verbal outbursts); (3) *physiologic factors* (e.g., increases in heart rate during an angry outburst); and (4) *environmental factors* (e.g., media depicting hostility as a "normal" reaction to stress).

Of the above four factors, cognitive processes such as basic beliefs, attributions, and expectancies have received the least amount of attention from researchers. We suspect, however, that it is the person's perceptions of himself or herself, work, family, community, and God that create his or her reality, the foundation on which the other three factors are based. In advancing cognition factors as the core, we seek to direct attention to an area

that well deserves rigorous inquiry.

But is an expanded cognitive social learning model the best or the only valid way of thinking about the Type A behavior pattern? Probably not. However, at this point it does appear quite useful, especially in planning for treatment. Undoubtedly, the model will be altered on the basis of empirical findings and clinical experiences in the way that the early theoretical and treatment orthodoxy within behavior therapy has been expanded dramatically in the past decade.[13,16] Using our expanded cognitive social learning model as a guide, let us now examine the few major efforts to modify the Type A pattern.

Selected treatment studies

Suinn[17] conducted some of the earliest published attempts at altering the Type A pattern. The brief treatment program (five hours) used for postmyocardial infarction patients consisted of: (1) training in progressive muscle relaxation; (2) identifying varying levels of muscle tension that accompany stress; (3) practicing relaxation as a coping response to stressful imagery; and (4) using imagery to practice behavior incompatible with the Type A pattern. Compared with a control group, the patients reported more lifestyle changes and greater improvements in their subjective and physiologic reactions to stress. Unfortunately, these results are difficult to interpret since Type A behavior was not objectively assessed, potential nonspecific treatment effects were not controlled, and a statistical analysis of the findings was not reported.

More recently Suinn and Bloom[18] tested a slightly modified version of the earlier program with a sample of healthy Type A persons. Unlike the earlier study, subjects were not asked to rehearse behavior incompatible with the Type A pattern. The investigators used the Jenkins Activity Survey, a self-report measure of Type A behavior that consists of "job involvement," "speed impatience," and "hard driving" scales. After the treatment program, subjects in the treatment group reported lower scores on the "hard driving" scale, compared with those of control subjects. Contrary to earlier findings, no significant differences were found on physiologic measures.

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Jenni and Wollersheim[19] compared Suinn's program with a cognitive treatment approach consisting of the reevaluation and reappraisal of tension-related thoughts concerning time-pressure, hostility, and competitiveness. The cognitive treatment was significantly more effective in reducing scores, on the Bortner Rating Scale, for subjects classified as "high" Type A persons than for other subjects. No differences in blood pressure or serum cholesterol were found between the experimental and control subjects. A major problem in this study was the use of a paper and pencil measure (the Bortner Scale) which has not been validated; unlike the Structured Interview (the method used in the Western Collaborative Group Study), the Jenkins Activity Survey, and the Framingham Type A Scale, the Bortner Scale has not been demonstrated to predict coronary heart disease.

Roskies and associates[20] compared the relative effectiveness of behavioral group therapy and psychodynamic group therapy in altering the Type A behavior pattern in 25 healthy men in one of the very few well designed studies of this type to date. Behavioral group therapy consisted of training in general relaxation skills and daily self-monitoring of tension levels. In psychodynamic group therapy, analytic interpretation was focused on increasing the subjects' awareness of their current Type A behavior; the pattern was presented to the subjects as a reenactment of an outdated family script. After 14 weeks of treatment, no differences between the two groups were found. However, significant improvements over 14 weeks in serum cholesterol, systolic blood pressure, self-reported health, family and employment satisfaction, and sense of time pressure were found for both treatments. No differences were found on measures of state and trait anxiety, diastolic blood pressure, or serum triglycerides. Data from seven men, who were later treated in a special behavior therapy group but excluded from the original data analysis because they had CHD, were later analyzed and compared with the original two groups.[21] Post-treatment results after six months resembled earlier findings, with some exceptions. Unlike the initial results, diastolic blood pressure levels for these seven subjects were significantly reduced, and their perceptions of work responsibility were lowered. Further, after six months significant differences in serum cholesterol levels were found between the three treatment groups (psycho-

dynamic therapy, 240 mg; behavior therapy, 220 mg; special behavior therapy, 187 mg). At the six-month follow-up observation, treatment effects were maintained less successfully in the psychodynamic group than in the behavioral group. The delayed effects of treatment suggest that outcome may depend on the combined influence of the type of treatment, time, and the person's clinical status. The duration of the benefits of treatment seems a far more meaningful index than the results obtained immediately after treatment.

The only other noteworthy treatment study reported to date assessed the relative effectiveness of behavior therapy, supportive group therapy, and "minimal treatment" for healthy male executives.[22] Those receiving "minimal treatment" completed assessment measures, met for one therapy session, and were urged to reduce Type A behavior. Behavior therapy consisted of stress management training coupled with self-control techniques designed to reduce the frequency and intensity of individually specified Type A behaviors. Supportive group therapy was intended to help subjects become aware of their Type A behaviors, identify Type A behavior in their daily living, and change this behavior. However, specific behavioral techniques were not offered. Both treatment approaches had more favorable effects on several aspects of the Type A pattern than did "minimal treatment." Significant reductions in stress, according to self-ratings and physiologic indices, were found, although relief was less marked in the group receiving supportive therapy.

Commendably, this study employed several psychometric, physiologic, and self-report measures, thus providing a comprehensive evaluation of treatment effects. The group receiving "minimal treat-

Table—Studies on Treatment of Type A Behavior

	Treatment targets				Measures† used to assess treatment			
Study	Cognitive	Behavioral	Physiologic	Environmental	Cognitive	Behavioral	Physiologic	Environmental
Suinn et al[17] N = 20* Treatment: 2 wk (5 hr)		X	X		S		B	
Suinn & Bloom[18] N = 14 Treatment: 3 wk (6 hr)			X		S,P	P	E,B	
Jenni & Wollersheim[19] N = 42 Treatment: 6 wk (9 hr)	X		X		P	S,P	P,E,B	
Roskies et al[20,21] N = 33 Treatment: 20 wk (14 hr)	X		X		S,P	S,P,E	S,P,E,B	
Levenkron[22] N = 38 Treatment: 8 wk (12 hr)		X	X	X	S,P	S,P	S,P,E,B	S
Recurrent Coronary Prevention Program[23,24] N = 1,035* Treatment: 5 yr (est 120 hr)	X	X	X	X	S,P	S,P,E	S,E,B	S,P

*Subjects were postcoronary patients.
†Measures: S = Self-report; P = Standardized psychosometric instrument; E = External observers; B = Biochemical assay.

ment" allowed evaluation of the possible effects on subjects of volunteering for treatment and completing various assessment measures. The identification of individualized Type A behavior was a most interesting aspect of the study. The value of personalized treatment goals well deserves further study.

A growing body of research suggests that the Type A behavior pattern is multidimensional with cognitive, behavioral, physiologic, and environmental components interacting in a complex system. The intervention studies just described paid little or no attention to the behavioral, cognitive, and environmental components as targets for intervention. As the Table illustrates, in these studies intervention focused primarily on the physiologic aspect of the Type A pattern (i.e., reducing the physiologic arousal caused by stressful situations). While not denying the importance of teaching Type A persons to cope with stress, more attention might be given to helping them modify the conditions that set the stage for behavior characteristic of the Type A pattern.

Further, five of these six studies failed to evaluate all four target areas—cognitive, behavioral, physiologic, environmental. The environmental component in particular was largely ignored. Another issue involves the method of assessment. Only the most recent studies have assessed treatment by using multiple measures within a given target area. We feel the complexity of the Type A behavior pattern demands a variety of assessment strategies, i.e., self-report questionnaires, standardized psychometric instruments, external observation, and biochemical assays.

Recurrent coronary prevention program

In contrast to the treatment studies already discussed, the Recurrent Coronary Prevention Program (RCPP), a five-year trial currently underway, is seeking to demonstrate reduced morbidity and mortality by altering the Type A pattern in more than 1,000 post-infarction men and women in the San Francisco Bay Area.[23,24] The major treatment program, based on an expanded cognitive social learning model, is offered in small groups of eight to ten participating persons.

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Treatment in the cognitive area includes self-instructional training, evaluation of basic beliefs, active listening, and mental relaxation. In the behavioral part of the program, participants learn to alter certain speech patterns (e.g., interrupting), psychomotor actions (e.g., emphatic gesturing), and other physical activities (e.g., hurried walking). In the environmental area, specific features of the work and home settings are scrutinized. For example, spouses of participants are encouraged to change social behavior that may elicit unnecessary stress. The basics of social problem-solving are presented to help participants alter stressful environmental factors, such as a place of work where a supervisor engenders excessive stress. In the physiologic aspect of treatment, participants are informed about biochemical processes in stress reactions, such as the role of norepinephrine in the cardiovascular system in response to events perceived as threatening or challenging. They learn that anger, irritation, aggravation, and impatience are associated with increased production of the catecholamines and of serum cholesterol. The comparison treatment in the study is provided in cardiologist-led small groups focused on diet, medication, and physiology.

Based on various measures (e.g., self-report, biochemical assay) of treatment of the targets discussed, results to date are encouraging. The recurrence rates of infarctions for the behavior change treatment and the cardiologist-led group treatment are below the rates for control subjects, post-infarction patients receiving routine medical care, as well as for nonsmoking subjects receiving placebos in the National Coronary Drug Project (a cohort highly similar to that in the current RCPP).[25] However, not all treated subjects are showing clearcut changes in stress-related behavior, although some have dramatically reduced their commitments and modified their sense of time urgency and hurriedness. A few have altered their angry behavior and their related feelings of hostility. Most, however, are still working on understanding their hostile feelings and are trying to change them. Efforts in this direction seem crucial; angiographic studies of pre-coronary subjects suggest that chronic hostility may be the major pathogenic facet of the Type A pattern.[15]

Discussion

Among the issues involved in designing and conducting treatment

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programs to alter the Type A behavior pattern are the targets of behavior change, the kinds of persons who make appropriate clients, and choices of treatment methods.

■ *Treatment targets.* Many possible features of the Type A pattern might be the targets for change: readily observable behavior, such as speech interruptions or emphatic gesturing; psychomotor activities, such as rapid eating or walking; composite behavior including hostility or a sense of urgency; specific biochemical markers, including excessive physiologic reactivity; and basic beliefs or fears, especially those concerned with oneself, career, and family. A major issue is whether to attempt to change selected specific behaviors or the global lifestyle. Another issue concerns the magnitude of change. Should treatment focus on reducing the severity of the Type A pattern or on changing the pattern substantially enough to approximate the behavioral characteristics of a Type B pattern?

At present we simply do not know which features of the behavior pattern are pathogenic. Not all characteristics of the Type A behavior pattern necessarily represent a predilection to coronary events or even to disease, in general. However, in a study of matched pairs of men in the Western Collaborative Group Study, "competitive drive" and "impatience" differentiated those who developed CHD from those who did not.[26] Angiographic studies and preliminary findings from the RCPP coupled with clinical experience suggest that excessive anger and hostility may be the crucial pathogenic aspects of Type A behavior. However, until empirical findings further substantiate this view, it seems prudent to help persons alter the Type A pattern in a more general way.

■ *Client selection.* Three levels of intervention with the Type A pattern have been suggested by Roskies: (1) *primary prevention* seeking to prevent the development of the behavior pattern among young adolescents as well as adults entering high-risk life situations (e.g., women re-entering the work force); (2) *secondary prevention* for persons who are obviously Type A and therefore are at increased risk for CHD, and (3) *tertiary prevention* for patients who have had an infarct or who suffer from angina.[27] At the level of secondary prevention, specific groups at particularly high risk could be identified. For example, the Framingham data suggest that employed Type A women with three or more children are at substantial risk for CHD.[28] Such women as well as other Type A persons who have a number of other risk factors, such as hypertension and smoking, may be appropriate candidates for intervention. It is clear that it is not wise to wait until a heavy smoker has had a myocardial infarction or has developed emphysema before suggesting ways to stop smoking. Similarly, reserving treatment for only post-myocardial infarction patients indeed might well be offering too little too late to too few.

Perhaps a combination of community-based media programs and face-to-face group sessions would prove effective.

Primary prevention with the Type A pattern is an intriguing

proposition. Should we develop educational programs to stem at least the extreme versions of the Type A pattern rather than wait for chronic disease to develop? Some evidence shows that the behavior pattern is clearly present in young children and early adolescents,[29,30] and a substantial number of studies have demonstrated various features of the pattern in college students.[31] Currently at Stanford we are initiating a series of studies to assess the behavior pattern in children and adolescents with an eye to planning primary prevention (and intervention) studies at a later time.

■*Treatment methods.* Intervention has ranged from an exclusive focus on altering physiologic responses to stressful situations, using progressive muscle relaxation (sometimes coupled with teaching awareness of behavior via self-monitoring), to an inclusive focus on altering all facets of the pattern via a group/administered program using multiple techniques. Which treatment modalities are most effective? That will depend on the targets selected for treatment, and there are not yet sufficient data to provide a definitive answer. Our experience suggests that treatment should be directed at physiologic, environmental, behavioral, and cognitive aspects of the Type A pattern. A multi-component treatment approach, such as the one used in the RCPP, introduces clients to a variety of skills and strategies (e.g., physical and mental relaxation skills, communication skills, modeling, daily drills, and cognitive restructuring techniques such as self-instructional training). Self-managed behavioral changes, whereby the participants themselves learn how to apply skills to a variety of situations, are highlighted. In keeping with the expanded cognitive social learning model discussed earlier, treatment addresses the environmental component by trying to engage the spouse, other family members, and the client's co-workers in the treatment program. A major effort is made to provide social support through the treatment group and to secure the support of significant persons in the client's life.

To date all intervention has been face-to-face in format. Our experience suggests that this is crucial for adults with well-established Type A patterns, especially post-coronary patients. The Stanford Heart Disease Prevention Program has been relying primarily on a media-based format (e.g., radio and TV announcements, newspaper articles, brochures) to reduce heart disease risk factors in all residents of a community.[32] It remains to be seen if substantial reductions in the Type A pattern can be effected with such a mass-media strategy. Certainly such innovative approaches deserve thoughtful consideration. Perhaps a combination of community-based media programs and face-to-face group sessions would prove effective.

Another intriguing possibility is offering treatment in the work environment. We suspect that intervention programs built into the working day would not only be well received by participants but would have a significant influence on Type A behavior. Work- and school-based programs appear especially promising ways of implementing preventive efforts. □

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REFERENCES

1. Havlik RJ, Feinleib M: Proceedings of the Conference on the Decline in Coronary Heart Disease Mortality. Bethesda, Md, National Heart, Lung, and Blood Institute, Oct 24-25, 1978.
2. Farquhar JW: *The American Way of Life Need Not Be Hazardous to Your Health.* Stanford, Calif, The Portable Stanford, 1978.
3. Dunbar HF: *Psychosomatic Diagnosis.* New York, Paul B. Hoeber, 1943.
4. Osler W: *Lectures on Angina Pectoris and Allied States.* New York, Appleton, 1892.
5. Friedman M, Rosenman, RH: *Type A Behavior and Your Heart.* Greenwich, Conn, Fawcett, 1974.
6. Friedman M: Type A behavior pattern: Some of its pathophysiological components. *Bull NY Acad Med* 53:593-604, 1977.
7. Friedman M: *Pathogenesis of Coronary Artery Disease.* New York, McGraw-Hill, 1969.
8. Rosenman RH, Brand RJ, Jenkins CD, et al: Coronary heart disease in the Western Collaborative Group Study. Final follow-up experience of 8 1/2 years. *JAMA* 233:872-877, 1975.
9. Haynes SG, Feinleib M, Kannel WB: The relationship of psychosocial factors to coronary heart disease in the Framingham Study pt III, Eight-year incidence of coronary heart disease. *Am J Epidemiol* 111: 37-58, 1980.
10. Dembroski TM, Weiss SM, Shields, JL, et al: *Coronary-Prone Behavior.* New York, Springer-Verlag, 1978.
11. *Coronary-prone behavior and coronary heart disease: A critical review.* Report of the Coronary-Prone Behavior Review Panel, Bethesda, Md, National Heart, Lung, and Blood Institute, National Institutes of Health, December, 1978.
12. Bandura A: *Social Learning Theory.* Englewood Cliffs, NJ, Prentice-Hall, 1977.
13. Thoresen CE, Coates TJ (eds): *The Behavior Therapist.* Monterey, Calif, Brooks/Cole, 1980.
14. Engel GL: The need for a new medical model: A challenge for biomedicine. *Science* 196:129-196, 1977.
15. Williams RB, Haney TL, Lee KL, et al: Type A behavior, hostility, and coronary atherosclerosis. *Psychosom Med* 42:539-549, 1980.

(continued)

16. Krumboltz JD, Thoresen CE (eds): *Counseling Methods*. New York, Holt, Rinehart & Winston, 1976.
17. Suinn RM: The cardiac stress management program for Type A patients. *Cardiac Rehabilitation* 5:13-15, 1975.
18. Suinn RM, Bloom LJ: Anxiety management training for Pattern A behavior. *J Behavioral Medicine* 1:25-35, 1978.
19. Jenni MA, Wollersheim JP: Cognitive therapy, stress management training, and the Type A behavior pattern. *Cognitive Therapy and Research* 3:61-73, 1979.
20. Roskies E, Spevack M, Surkis A, et al: Changing the coronary-prone (Type A) behavior pattern in a nonclinical population. *J Behav Med* 1:201-216, 1978.
21. Roskies E, Kearney H, Spevack M, et al: Generalizability and durability of treatment effects in an intervention program for coronary-prone (Type A) managers. *J Behav Med* 2:195-207, 1979.
22. Levenkron JC: Modifying the Type A coronary-prone behavior pattern: A comparison of three intervention approaches. St. Louis, Washington University, 1979, Unpublished doctoral dissertation.
23. Friedman M: Type A behavior: A progress report. *The Sciences* 20:10-11, 28, February 1980.
24. Friedman M, Thoresen CE, Gill JJ: Type A behavior: Its possible role, detection, and lateralization in patients with ischemic heart disease, in Hurst JW (ed): *Heart Update V*. New York: McGraw-Hill, 1981, pp 88-99.
25. The Coronary Drug Project: Clofibrate and niacin in coronary heart disease. *JAMA* 231:360-381, 1975.
26. Matthews KA, Glass DC, Rosenman RH, et al: 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, 1977.
27. Roskies E: Considerations in developing a treatment program for the coronary-prone (Type A) behavior pattern, in Davidson P (ed): *Behavioral Medicine: Changing Health Life Styles*. New York, Brunner/Mazel, 1979, pp 299-333.
28. Haynes SG, Feinleib M: Women, work, and coronary heart disease: Prospective findings from the Framingham Heart Study. *Am J Public Health* 70:133-141, 1980.
29. Matthews KA, Angulo J: Measurement of the Type A behavior pattern in children: Assessment of children's competitiveness, impatience-anger, and aggression. *Child Dev* 51:466-475, 1980.
30. Siegel JM, Leitch CJ: Type A behavior in adolescence: The Tacoma Study. Read before the 20th Conference in Cardiovascular Disease Epidemiology, San Diego, Calif, March 1980.
31. Dembroski TM, MacDougall JM, Herd JA, et al: The Type A coronary-prone behavior-pattern: A review. National Heart, Lung, and Blood Institute, 1980.
32. Maccoby N, Farquhar JW, Wood PW, et al: Reducing the risk of cardiovascular disease: Effects of a community-based campaign on knowledge and behavior. *Community Health* 3:100-114, 1977.