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The purpose of the present study was to empirically identify individuals who differed in their patterns of components derived from the structured interview (SI), and to evaluate whether individuals characterized by the different patterns varied in terms of their risk for coronary heart disease (CHD). The present study represents a reanalysis of data from the Western Collaborative Group Study in which components of Type A were individually related to risk for CHD. Subgroups of individuals who differed in the patterns of their component scores were identified by means of cluster analytic techniques and were found to vary in their risk of CHD. As expected, a pattern of characteristics in which hostility was salient was found to be predictive of CHD. Moreover, another pattern of characteristics that appears to reflect pressured, controlling, socially dominant behavior in which hostility was not salient also was found to be predictive of CHD. Further, two patterns of characteristics were identified that were unrelated to CHD risk. Finally, two patterns of characteristics were identified that were related to reduced risk of CHD. Overall, these results suggest that future research should investigate variables in addition to hostility in regard to risk for and protection from CHD.

Key words: cluster; analysis; dominance; competitiveness; CHD risk.

## INTRODUCTION

The Type A behavior pattern, the focus of considerable research and controversy, has been described as vague both conceptually (1) and operationally (2). Classifying individuals as Type A by the Structured Interview (SI) does not require that they exhibit all or the same components of Type A behaviors. Individuals are classified as Type A if they exhibit a "preponderance" of an array of characteristics (2, 3). Consequently, individuals who are categorized as Type A are heterogeneous in regard to their characteristics. Thus, individuals who are classified as exhibiting the Type A behavior pattern probably exhibit different behavior patterns. An underlying assumption to the SI assessment procedure is that the various patterns of Type A characteristics as well as the individual characteristics that comprise the patterns are equivalent in regard to predicting coronary heart disease (CHD), the ultimate criterion for the existence and measurement of this concept.

Relatively little research has been conducted to evaluate the relation between different patterns of Type A components and CHD. One study in which this was attempted was that by Matthews et al. (4) in which scores for SI-derived character-

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istics from the Western Collaborative Group Study (WCGS) were factor analyzed. Five factors, labeled competitive drive, past achievements, impatience, nonjob achievement, and speed, were identified, each of which represented a different pattern of characteristics. The patterns of characteristics reflected in both the competitive drive and impatience factors tended to be associated with occurrence of CHD. Further, subsequent analyses of individual characteristics revealed that hostility and certain voice stylistics were more predictive of CHD than other components of the Type A pattern.

Stimulated in part by the findings of this investigation, several studies in the past few years have been conducted with the aim of further exploring which SI-derived components of Type A are predictive of CHD. In a study by Chesney et al. (5) of SI-derived Type A components and incidence of CHD in the WCGS, univariate analyses revealed that hostility, immediateness of responses, verbal competitiveness, speech rate, and Type A content of responses were each significantly, positively associated with incidence. However, in a multivariate analysis in which the inter-relatedness of these components was controlled, only hostility was found to be significantly, positively related to CHD risk, while self-aggrandizement was found to be significantly negatively related to CHD risk (6). The predictive significance of hostility received further support from a 22-year follow-up of the WCGS by Ragland and Brand (7), which revealed that while subsequent CHD mortality in patients who suffered an initial coronary event was lower in Type A than in Type B subjects, a further analysis of these follow-up data using components rather than global Type A confirmed that hostility assessed at intake into the WCGS was significantly related to subsequent coronary mortality (8).

Dembroski et al. (9), employing a somewhat different scoring procedure from Chesney et al. (5), conducted a study of individual SI-derived components that predicted incidence of CHD in the Multiple Risk Factor Intervention Trial. Employing one-tailed tests of statistical significance, only hostility was found to be significantly, positively associated with incidence of CHD in univariate analyses and in multivariate analyses in which traditional risk factors were controlled.

Powell and Thoresen (10), who employed a scoring procedure that differed from both Chesney et al. (5) and Dembroski et al. (9), conducted a study of individual characteristics that were associated with recurrence of cardiac events in participants in the Recurrent Coronary Prevention Project. Each of 15 characteristics was found to be significantly, positively related to recurrent cardiac events in univariate analyses. In a multivariate analysis in which the interrelatedness of the characteristics was controlled, four of the 15 remained significant: intensity (i.e., strength of positive or negative responses), self-involvement, periorbital pigmentation, and anger arousal while driving.

Overall, the findings tend to suggest that hostility is the most salient and reliable single predictor of CHD assessed via the SI.[1] However, the findings as to whether there are other components than

[1] Hostility has also been assessed via a self-report scale developed by Cook and Medley from the MMPI. See Williams (11) and Smith (12) for reviews of research on the association between CHD and hostility assessed via the Cook and Medley Hostility (Ho) scale.

hostility that predict CHD vary depending on the method that is used for scoring components and the method of analysis. Both the univariate and multivariate results from the study by Powell and Thoresen (10) and the univariate analyses from the Hecker et al. (6) study suggest that there are characteristics other than hostility that may also predict CHD.

The approach of investigating the relation between risk of CHD and individual components or characteristics has ignored the possibility that certain combinations or patterns of components may predict risk of CHD. The study by Matthews et al. (4) represented an attempt to do this, though it was not very successful perhaps because it employed factor analysis. Factor analysis of variables is a technique for identifying classes of variables, each of which possesses a common pattern of characteristics. Further, it assumes that all individuals belong to the same group, i.e., there are no subgroups of people who differ qualitatively (13).

If there are qualitative differences between groups of people in regard to the characteristics that relate to CHD, they will not be identified via a factor analysis of variables, as was performed in the Matthews et al. (4) study. In this instance, a more appropriate analytic technique is a cluster analysis of people. Cluster analysis of people is a technique that assumes there are qualitative differences between subgroups of people and attempts to identify subgroups on the basis of the variables examined (14). The notion that there are qualitative differences between groups of people in regard to the characteristics that relate to CHD was contained in the original formulation concerning Type A (15) and is contained in more recent formulations concerning behavioral patterns that may be associated with CHD risk (16).

Therefore, it may be fruitful to attempt to identify different patterns of SI-derived Type A characteristics via cluster analytic techniques and relate them to CHD. This was the purpose of the present study, namely, to empirically identify individuals who differ in their patterns of components and then to evaluate whether such individuals vary in terms of their risk for CHD. Considering the findings concerning hostility mentioned above, it was expected that a pattern of characteristics in which hostility was salient would be identified and would be predictive of CHD. Additionally, considering the possibility that there are other components that may be related to risk, it was expected that one or more patterns of characteristics in which hostility was not salient would be identified that would be predictive of CHD. Moreover, since some components have not been found in univariate analyses to be related to risk, it was expected that one or more patterns of characteristics would be identified that would be unrelated to risk. Finally, considering that the Type B behavior pattern, which ostensibly is protective for CHD, is heterogeneous and negative relations between individual component scores and risk for CHD have been reported (6), it was expected that one or more patterns of characteristics would be identified that would be related to reduced risk of CHD.

The present study represents a reanalysis of data from the WCGS in which components of Type A were individually related to risk for CHD (5, 6). Specifically, subgroups of individuals who differed in the patterns of their component scores were identified by means of cluster analytic techniques (17–19). An evaluation was then made as to whether these groups differed in their risk for CHD.

## METHOD

### Subjects

Individuals who participated in the WCGS provided the subjects for the current investigation. The original cohort for the WCGS consisted of 3154 males between 39 and 59 years of age. They were free of CHD at intake into the study and were followed prospectively for an average of 8.5 years. The participants were employed by 10 companies in the state of California and were involved primarily in white-collar occupations. The SI was administered to the entire sample at intake for assessment of Type A behavior. Conventional risk factors for coronary heart disease, evidence of clinical CHD, and other data were assessed at intake and at annual intervals throughout the follow-up period. By the end of the 8.5-year follow-up period, 257 participants had developed clinical manifestations of CHD (i.e., coronary death, electrocardiographically confirmed nonfatal myocardial infarction, or angina pectoris).

The present study focuses on these 257 participants who developed CHD. Of these men, recorded interviews for seven subjects were either inaudible or lost, reducing the sample of CHD cases to 250. Each of the 250 cases was matched with two controls selected from the 2,897 participants who did not develop CHD during the follow-up period, The 250 cases and 500 controls were matched on the basis of company of employment, date of entry into the study, and as closely as possible on age. In those isolated instances where a match of the same age was impossible, a control was chosen who was one year younger or older than the case. The combined mean age at intake for CHD cases and controls was 48.5

### Component Scoring

The component scoring procedure is described in detail elsewhere (5). The procedure involves the division of the SI into 20 segments. The responses made by the subject during each segment were scored in terms of 12 carefully operationalized components, which included facets of the Type A behavior pattern that have previously been described (20) and other variables considered to be related to risk for CHD. The 12 components are acceleration, competitiveness, despondency, exactingness, hard voice, hostility, immediateness, loudness of voice, self-aggrandizement, speaking rate, syllabic emphasis, and Type A content. Brief definitions for these components are presented in Table 1.

Two raters scored the 750 interviews. Neither had knowledge of the CHD status of subjects. Both raters scored 36 interviews in order to assess inter-rater reliability. All other interviews were scored by only one rater. The inter-rater reliability for scoring the components, as indexed by the intraclass correlation coefficient, ranged from 0.38 for self-aggrandizement to 0.93 for immediateness (6).

### Cluster Analysis

Aldenderfer and Blashfield (14) and Tyron and Bailey (18) recommend that prior to conducting a cluster analysis of individuals, the number of dimensions, in particular redundant dimensions, should be reduced in order to increase the likelihood of achieving a satisfactory solution for the cluster analysis. Consequently, a variable cluster analysis, employing the Pearson product-moment correlation coefficient as the proximity measure, was performed on the 12 components using an average linkage procedure. The standardized scores for the variables identified in the variable cluster analysis were then employed in a cluster analysis of subjects. First, Ward's (21) hierarchical clustering procedure was employed. This procedure uses the squared Euclidian distance to determine the similarity between subjects' profiles on the variables. At each stage of fusion, clusters are formed so as to yield the least increase in the error sum of squares, defined as the sum of the distances from each individual's profile to the centroid of its parent cluster. Thus, those individuals whose profiles have similar elevations and patterns will be grouped together. The procedure results in minimum-variance clusters in which there is a relatively high level of within-cluster homogeneity on the variables employed and maximal discrimination among the defined clusters. The optimal number of clusters was determined empirically when there was a marked discontinuity in the fusion coefficient value associated with merger and reduction to a smaller number of clusters.

Following Wishart's suggestion (19), a K-means relocation cluster analysis was conducted to evaluate the stability of the clusters and to iteratively relocate subjects into the cluster whose centroid was closest. The K-means clustering is terminated when each subject has been placed in the cluster whose centroid is closest to the subject.

TABLE 1. Brief Definitions of Components[a]

| Component | Definition |
|---|---|
| Acceleration | Temporary increases in speaking rate, usually at the end of sentences. |
| Competitiveness | Competitive behavior exhibited during the interview (e.g., interrupting the interviewer, asking irrelevant questions, or requesting unnecessary clarification). |
| Despondency | Depressed viewpoint or mood evidenced in content or tone of responses (e.g., statements of loss, pessimism, sadness, and withdrawal as a coping style). |
| Exactingness | Excessive and unnecessary attention to detail, both in interpreting questions and providing answers (e.g., volunteering numerical information, requesting additional information before responding to questions). |
| Hard voice | Speech that reflects excessive muscular tension in the laryngeal structures. |
| Hostility | Reports of anger or irritation involving others or unpleasant situations, and hostility expressed toward the interviewer (e.g., complaints about others, depreciation of interviewer). |
| Immediateness | Quickness with which the subject responds to the key questions. The inverse of response latency. |
| Loudness of voice | Loudness of voice during responses. |
| Self-aggrandizement | Claims of superiority relative to others (e.g., conceit, pompous statements, boasting). |
| Speaking rate | Speed of speaking in passages that are free of thought pauses and emotional interruptions. |
| Syllabic emphasis | Sudden increases in loudness that emphasize particular syllables, i.e., explosive speech. |
| Type A content | Content of the subject's responses indicative of Type A behavior (e.g., report of heavy job responsibility). |

[a] Adapted from Chesney et al. (5).

## Risk Analysis

The relations between cluster membership and CHD incidence were examined using the multiple logistic model. Effects coding was used to render the information of cluster membership, one effect variable corresponding to each cluster. Effects coding is a particular type of dummy variable coding where each group (i.e., cluster) is compared with all the other groups combined, rather than with a single reference group as is usually done in regression analysis with dummy variables (see Cohen and Cohen (22) for a description of the basic procedure). Since all of the clusters were so represented, the logistic model without an intercept term was employed. Each resulting logistic regression coefficient represents the difference in risk between that cluster and the others combined and, furthermore, is adjusted for the other cluster differences. A composite score was also calculated as the linear combination of all the cluster effects which best predicts CHD incidence.

The risk analyses were conducted in three steps: a) unadjusted, b) adjusted for three traditional risk factors (serum cholesterol, diastolic blood pressure (DBP), and cigarettes smoked per day), and c) adjusted for the three traditional risk factors and for the hostility component of the scoring procedure. To facilitate comparisons among the predictor variables, results from all logistic analyses are presented in terms of relative risk standardized to a variance of 0.25 in the predictor variables. This equates all predictors to the norm of a 50–50 dichotomy and is equivalent to comparing subjects with scores 1 SD above the mean with those 1 SD below the mean. Since the sample of cases and controls was matched, the stratified conditional-likelihood approach (23) was employed to estimate logistic parameters as implemented in the SAS procedure MCSTRAT.

# RESULTS

## Cluster Analysis

The variable cluster analysis performed on standardized scores for the 12 compo-

TABLE 2. Clusters and Means for Components

| | Clusters | | | | | |
|---|---|---|---|---|---|---|
| | 1 | 2 | 3 | 4 | 5 | 6 |
| *N* | 142 | 224 | 54 | 140 | 147 | 43 |
| Competitiveness | 0.24 | −0.54 | 0.30 | 1.11 | −0.47 | −0.34 |
| Immediateness | −0.66 | −0.14 | 0.33 | 0.95 | −0.15 | −0.10 |
| Hostility | 0.19 | −0.32 | 2.51 | −0.09 | −0.45 | −0.29 |
| Type A content | 0.17 | −0.17 | 0.45 | 0.42 | −0.33 | −0.47 |
| Despondency | −0.09 | −0.36 | 0.03 | −0.21 | 0.03 | 2.80 |
| Speaking rate | 0.32 | 0.50 | 0.44 | 0.42 | −1.48 | −0.55 |
| Voice composite | 0.92 | −0.40 | 0.97 | 0.07 | −0.39 | −1.07 |
| Loudness | 0.83 | −0.35 | 0.81 | 0.10 | −0.40 | −0.88 |
| Emphasis | 0.89 | −0.23 | 0.71 | 0.07 | −0.58 | −0.90 |
| Acceleration | 0.47 | −0.46 | 0.67 | 0.09 | 0.04 | −0.46 |
| Hard voice | 0.50 | −0.12 | 0.61 | −0.05 | −0.20 | −0.86 |
| Self-aggrandizing/exacting composite | 1.12 | −0.66 | 0.59 | 0.14 | −0.35 | −0.22 |
| Exactingness | 0.92 | −0.63 | 0.47 | 0.16 | −0.19 | −0.20 |
| Self-aggrandizement | 1.01 | −0.52 | 0.55 | 0.08 | −0.42 | −0.17 |
| % CHD cases | 32 | 32 | 52 | 43 | 26 | 19 |
| % Type A | 70 | 46 | 82 | 76 | 36 | 28 |
| DBP (mm Hg) | 84 | 82 | 86 | 83 | 82 | 84 |
| Cholesterol (mg/dl) | 224 | 240 | 243 | 239 | 232 | 228 |
| Cigarettes/Day | 10 | 13 | 18 | 14 | 12 | 13 |
| Age | 49 | 48 | 49 | 49 | 49 | 47 |

nents resulted in a parsimonious yet meaningful solution involving eight variable clusters. Each of six variable clusters was identified by a single component (namely, competitiveness, despondency, hostility, immediateness, speaking rate, and Type A content), while one cluster contained four voice-related components (namely, loudness of voice, syllabic emphasis, acceleration, and hard voice) and another contained self-aggrandizement and exactingness. Standardized scores for the four components that comprised the voice-related variable and the two components that comprised the self-aggrandizing/exacting variable were summed and then restandardized in order to make their means and standard deviations comparable with the standardized scores for the other components.

Employing the standardized scores for the eight variables identified in the variable cluster analysis, application of Ward's cluster analysis resulted in six clusters of individuals. The K-means relocation procedure terminated in a small number of iterations indicating that stable cluster solutions had been found.

Table 2 presents for each cluster the mean standardized scores for the six separate components employed in the cluster analysis. Table 2 also presents for each cluster the mean standardized scores for the voice-related variable and the self-aggrandizing/exacting variable as well as the mean standardized scores for the components comprising each of these variables.

Cluster 1 is notable for its elevations on both the voice-related and self-aggrandizing/exacting variables. Cluster 2 is distinguished by its below average values on all variables except speaking rate. Cluster 3 is marked by its substantial elevation on hostility and elevation on the voice-related variable. Cluster 4 is notable for its elevations on competitiveness and immediateness. Cluster 5 is marked by its below average values on most variables and very low value on speaking rate. Cluster 6 is distinguished by its marked elevation on despondency and below average values on most variables, particularly, the voice-related variable. A brief description for each cluster is presented in Table 3.

Also presented in Table 2 for each cluster are the Ns for the clusters, mean values for the traditional risk factors, mean age, percentage of individuals classified as Type A, and percentage of CHD cases. Significant differences between clusters were found for percentage of individuals classified as Type A ($\chi^2$ (5) = 92.85, $p < 0.0001$), percent of CHD cases ($\chi^2$ (5) = 22.39, $p < 0.0005$), cigarette use ($F$ (5, 744) = 2.87, $p < 0.02$), and cholesterol ($F$ (5, 744) = 2.35, $p < 0.04$). Differences between clusters for DBP and age were not significant ($F$s (5, 744) = 2.09 and 1.86, respectively, both NS). Because risk analyses were performed (see below) in which relative risk for CHD was calculated adjusting for cholesterol, cigarette consumption, and DBP, the differences between clusters in the aforementioned variables were not explicated further.

**TABLE 3. Cluster Descriptions**

Cluster 1
Persons who have a characteristic speech pattern that is somewhat loud and explosive, quickens within phrases, and indicates muscular tension. In addition, response content is self-centered and exacting about details.

Cluster 2
Persons who show some continuous quickness in speech that does not speed up or slow down. Responses are not self-centered or exacting about details and do not indicate competitiveness or hostility.

Cluster 3
Persons who are markedly hostile and who speak somewhat loudly, with explosiveness, quickening pace, and muscular tension. Response content suggests individuals who are somewhat self-centered and exacting about details.

Cluster 4
Persons who are verbally competitive, alert, and quick to respond.

Cluster 5
Persons who are soft-spoken, and who show a speech pattern that is slow and lacks emphasis or a self-centered focus. Their speech does not indicate competitiveness or hostility.

Cluster 6
Persons who are dejected, quiet, or soft-spoken and whose speech lacks emphasis.

## Risk Analysis

Unadjusted and adjusted relative risks (RR) for CHD associated with each of the six clusters and with the cluster composite are presented in Table 4.

The results of the unadjusted analyses reveal that the cluster composite was significantly associated with risk for CHD. Moreover, individual clusters 3 and 4 were significantly, positively related to CHD incidence while clusters 5 and 6 were significantly, negatively related to CHD incidence.

After adjustment for the three traditional risk factors, it was found that the cluster composite remained significantly associated with risk for CHD. Further, cluster 4 was significantly, positively related to CHD incidence, cluster 3 approached ($p = 0.061$) being significantly, positively related to CHD incidence, cluster 6 was significantly, negatively related to CHD incidence, and cluster 5 approached ($p = 0.068$) being significantly, negatively related to CHD incidence.

TABLE 4. Logistic Analysis of Component Clusters and CHD Risk

| Cluster | Standardized Logistic Coefficient | Standardized Relative Risk | | |
|---|---|---|---|---|
| | | Value | 95% Confidence Interval | *p* Value |
| Unadjusted | | | | |
| 1 | −0.0807 | 0.92 | 0.67–1.28 | 0.626 |
| 2 | −0.1104 | 0.90 | 0.65–1.24 | 0.508 |
| 3 | 0.4587 | 1.58 | 1.17–2.14 | 0.003 |
| 4 | 0.4211 | 1.52 | 1.12–2.07 | 0.007 |
| 5 | −0.3511 | 0.70 | 0.51–0.97 | 0.033 |
| 6 | −0.3877 | 0.68 | 0.47–0.98 | 0.038 |
| Composite | 0.7150 | 2.04 | 1.50–2.79 | <0.001 |
| Adjusted for traditional risk factors[a] | | | | |
| 1 | 0.0757 | 1.08 | 0.75–1.54 | 0.679 |
| 2 | −0.0810 | 0.92 | 0.64–1.32 | 0.658 |
| 3 | 0.3268 | 1.39 | 0.99–1.95 | 0.061 |
| 4 | 0.3682 | 1.45 | 1.03–2.03 | 0.033 |
| 5 | −0.3237 | 0.72 | 0.51–1.02 | 0.068 |
| 6 | −0.4954 | 0.61 | 0.40–0.93 | 0.021 |
| Composite | 0.6206 | 1.86 | 1.33–2.61 | <0.001 |

[a] Total cholesterol, diastolic blood pressure, and cigarettes per day.

Thus, both unadjusted and traditional-risk-factor-adjusted analyses indicated that two of the clusters, viz., clusters 3 and 4, are predictive of CHD and two of the clusters, viz., 5 and 6, are protective of CHD, though after adjustment for the risk factors, the results for one predictive cluster (cluster 3) and one protective cluster (cluster 5) did not quite reach the 0.05 level of significance.

One interpretation of a decline in *p* levels for predictive and protective clusters following adjustment for traditional risk factors is that the traditional risk factors mediated, in part, the association between the cluster and CHD. Inspection of Table 2, in which it may be seen that relative to the other clusters, levels of the traditional risk factors were highest for individuals in cluster 3 and low for individuals in cluster 5, suggest that some of the CHD risk attributable to individuals in cluster 3 is due to greater consumption of cigarettes and elevations in cholesterol and DBP and some of the decreased CHD risk attributable to individuals in cluster 5 is due to relatively lower consumption of cigarettes and lower levels of cholesterol and DBP.

Considering the importance hostility has been shown to have in association with risk for CHD, question could be raised whether certain clusters were merely a manifestation of hostility and thus any relation to CHD incidence is due to its underlying association with hostility. To evaluate this question, the hostility component score was added to the previous model to control for hostility in addition to the traditional risk factors. These results are presented in Table 5. Even after adjustment for hostility as well as the risk factors, it was found that the cluster composite was significantly associated with risk for CHD. This indicates that hostility is not alone in being signifi-

**TABLE 5. Logistic Analysis of Component Clusters Controlling for Risk Factors and Hostility**

| Cluster | Standardized Logistic Coefficient | Standardized Relative Risk | | |
|---|---|---|---|---|
| | | Value | 95% Confidence Interval | *p* Value |
| Adjusted for hostility and traditional risk factors[a] | | | | |
| 1 | −0.1259 | 0.88 | 0.63–1.23 | 0.459 |
| 2 | 0.0145 | 1.01 | 0.72–1.43 | 0.933 |
| 3 | 0.0528 | 1.05 | 0.69–1.61 | 0.806 |
| 4 | 0.4297 | 1.54 | 1.13–2.10 | 0.007 |
| 5 | −0.1917 | 0.83 | 0.59–1.16 | 0.263 |
| 6 | −0.3474 | 0.71 | 0.49–1.02 | 0.066 |
| Composite | 0.5348 | 1.71 | 1.19–2.45 | 0.004 |

[a] Total cholesterol, diastolic blood pressure, and cigarettes per day.

cantly associated with CHD risk. After adjustment for hostility in addition to the risk factors, the relation between CHD incidence and cluster 4 not only remained significant but increased slightly in RR, indicating that cluster 4 is not merely a proxy for hostility and does not derive its predictiveness because of an underlying association with hostility. After adjustment for hostility in addition to the risk factors, as one would expect, the relation between CHD incidence and cluster 3 in which hostility was most salient dropped to a level that did not even approach significance. Moreover, the RR for cluster 5 decreased to a level that did not approach significance while the RR for cluster 6 dropped to a level that did approach significance ($p = 0.066$).

Theoretical implications aside, some readers may be interested in how findings bearing on the configuration of characteristics associated with cluster 4 may in a practical sense be used in future research. To address this issue, the following analyses were conducted. Risk analyses using a logistic regression model were employed to examine the relationship between CHD incidence and the linear, unit-weighted combination of immediateness and competitiveness (the two components with the highest scores in the profile for cluster 4) after controlling for the traditional risk factors. In addition, risk analyses were employed to examine the relationship between CHD incidence and the linear, unit-weighted combination of immediateness, competitiveness, and speaking rate (the three behavioral ratings with the highest scores in the profile for cluster 4) after controlling for the traditional risk factors. The standardized RR for the combination of immediateness and competitiveness after adjustment for the traditional risk factors was 1.63, $p = 0.011$, and the standardized RR for the combination of immediateness, competitiveness, and speaking rate after adjustment for the traditional risk factors was 1.75, $p = 0.002$. Further, these analyses were repeated controlling for hostility in addition to the traditional risk factors. The standardized RR for the combination of immediateness and competitiveness after controlling for hostility and the traditional risk factors was 1.47, $p = 0.047$; and the standardized RR for the combination of immediateness, competitiveness, and speaking rate after controlling for hostility and the traditional risk factors was 1.56, $p = 0.017$.

Besides their potential value in regard to future research, these ancillary analyses further indicate that a set of components independent of hostility as well as traditional risk factors, is predictive of CHD risk.

In addition to considering the clusters that were either predictive or protective of CHD, note should be taken that two of the clusters (namely, clusters 1 and 2) were neither positively nor negatively related to CHD incidence in either the unadjusted or adjusted analyses.

The mean ages of individuals in the various clusters were highly similar (see Table 3) and not significantly different from one another. Therefore, the results for the protective clusters cannot be attributed to their containing older, perhaps more biologically resilient individuals, and the results for the clusters predictive of CHD risk cannot be attributed to their containing younger, perhaps more vulnerable individuals.

## DISCUSSION

Several interesting findings emerged from the present study. The results suggest that there may be more than one pattern of Type A characteristics that is positively related to CHD incidence, more than one pattern that may be unrelated to CHD incidence, and more than one pattern that may be negatively related to CHD incidence.

As expected, a pattern of characteristics in which hostility was salient (viz., cluster 3) was found in the unadjusted analyses to be significantly, positively related to CHD incidence. Additionally, a pattern of behavior that involved quick responses and interrupting the interviewer, but not hostility (viz., cluster 4), was found in both unadjusted and adjusted analyses to be positively related to CHD incidence. It is interesting to note that SI-derived measures of quick responses and interrupting the interviewer but not hostility have been found to be positively associated with the desire for control (24) and that simultaneous speech and interruptions have been found to be associated with dominance (25–27). Thus, the pattern of components associated with cluster 4 suggest that these individuals are characterized by being pressured, controlling, and socially dominant. It is noteworthy that effortful attempts to exert dominance or social control have been found to be associated with enhanced cardiovascular reactivity (28, 29), dominance has been reported to be associated with accelerated coronary atherosclerosis in male monkeys threatened by loss of control (30), and interruptive simultaneous speech has been found to be associated with severity of coronary occlusion in humans (31).

The characterization of individuals in cluster 4 as pressured, controlling, and socially dominant is speculative and additional information is needed on their psychological attributes and behaviors. Moreover, consideration should be given to the possibility that a pattern of behavior that involves quick responses and interrupting the interviewer as well as controlling, socially dominant behaviors may reflect covert hostility which may contrast with overt expressions of hostility as embodied in the individual hostility component that was assessed. The hostility component only correlated $r = 0.16$, $p < 0.01$, and $r = 0.00$, NS, respectively, with competitiveness and immediateness, the two most salient components of cluster 4, which indicates that the hostility component is assessing a rather different construct from competitiveness and immediateness. All told, it may be that both a pressured, controlling, socially dominant pattern of behaviors and a relatively separate pattern of behaviors characterized by overt expression of hostility are associated with risk of premature CHD. This possibility is intriguing and warrants further study to increase our understanding of and ability to identify people at risk for CHD.

It is interesting to place these findings in historical context. The pressured, controlling, socially dominant pattern fits better than does a pattern characterized by hostility the often quoted description by Sir William Osler (32) of the typical patient with angina as "not the delicate neurotic person . . . but the robust, the vigorous in mind and body, and the keen and ambitious man, the indicator of whose engines is always set at full speed ahead." The latter description contains no direct indication of hostility. It seems that hostility as a salient characteristic may be seen more directly in Dunbar's (33) description of coronary patients as manifesting a calm facade concealing underlying aggression and resentment, or Menninger and Menninger's (34) characterization of heart patients as exhibiting repressed hostility and compulsive traits.

After adjustment for the traditional risk factors, the RR for the pattern in which hostility was salient was 1.39, which approached significance ($p = 0.061$) but did not achieve the traditional 0.05 level. Hecker et al. (6) report that the RR for the hostility component individually after adjustment for the risk factors was 1.84, $p < 0.001$. What accounts for the discrepancy in RR and its significance for the cluster in which hostility was salient and that for the hostility component considered separately? Differences in how RR is calculated in the two situations and the average values of hostility in the different cluster profiles provide the answer. When the hostility component is considered separately, the RR represents the ratio of the CHD incidence of individuals high on hostility to that of individuals low on hostility. When the cluster in which hostility was salient is considered, the RR represents the ratio of CHD incidence for individuals in that cluster to the incidence for individuals in all other five clusters, one of which (cluster 1) was slightly higher than average on hostility, another of which was essentially average on hostility (cluster 4), and three of which were below average in hostility (clusters 2, 5 and 6).

The profiles of components for the clusters that were protective of premature CHD are also interesting. The profile for cluster 5 that was found to be significantly, negatively related to CHD in the unadjusted analysis was notable for its below average values on most characteristics and very low value on speaking rate. Considering that rapid, loud speech frequently is associated with hostility-related emotions, e.g., anger (35), perhaps these individuals are relatively placid and unreactive in terms of hostility-related emotions and behavior and thereby do not experience the exaggerated pituitary adrenocortical and/or sympathetic-adrenomedullary arousal that has been hypothesized to link hostility with coronary atherosclerosis and CHD (36).

The profile for cluster 6 that was found to be significantly, negatively related to CHD in both unadjusted and adjusted analyses was notable not only for its below average values on most of the variables but its particularly low value on the voice style variable and particularly high value on despondency. It should be noted that the total pattern of characteristics for cluster 6 is important to consider because in univariate analyses of these data (6), despondency alone was found to be negatively, but not significantly associated with CHD ($p = 0.13$). What was said above in regard to cluster 5 may also apply to cluster 6. Perhaps these individuals are relatively placid and unreactive in terms of hostility-related emotions and behavior. In addition, due to their dejection, they also may tend to withdraw from and avoid stressful situations and engaging in potentially stressful behaviors, such as pressured, controlling, socially dominant acts. Such avoidant behavior could further reduce their experiencing exaggerated pituitary adrenocortical and sympathetic-adrenomedullary arousal.

Reference to Table 1 indicates that the hostility component was defined in terms of both hostility-related emotion and behavior. It may be recalled that in the adjusted analyses in which hostility was controlled along with the traditional risk factors, cluster 5 was no longer found to be significantly, negatively related to CHD incidence and cluster 6 only approached being significantly, negatively related to CHD incidence. These findings support the interpretation that cluster 5 derived much of its protectiveness by virtue of individuals in this cluster being unreactive emotionally and/or behaviorally to provocation. Additionally, these findings are congruent with the interpretation that cluster 6 derived much of its protectiveness because individuals in this cluster not only are unreactive to provocation but also do not engage in the pressured, controlling, socially dominant behaviors that characterize the at-risk individuals in cluster 4.

While research on individual differences that confer protection from disease has been pursued less vigorously than research on factors that confer risk, it is no less important. The results obtained in the present study concerning patterns of characteristics which are associated with decreased CHD incidence are intriguing and warrant further investigation in regard to protection from coronary disease.

No association with CHD risk was found for individuals in cluster 1 who were characterized by an elevation on the self-aggrandizing/exacting variable and, to a lesser extent, the voice style variable. This result is generally congruent with the finding that self-referencing was not associated with CHD risk in this sample (37). Interestingly, reference to Table 2 reveals that 70% of the individuals in cluster 1 had been classified as Type A, while 54% of the individuals in cluster 2, which also was unrelated to CHD risk, had been classified as Type B. An implication of these findings for other epidemiological studies is that the extent to which studies of global Type A obtain statistically significant relations with cardiac end points probably depends on the proportion of individuals who exhibit the different patterns identified in the present study. For instance, the greater the proportion of subjects there are in a study who exhibit patterns of characteristics similar to those for clusters 1 and 2, the less likely it is that global Type A will be found to be associated with CHD.

The present study, then, succeeded in its purpose. Individuals were empirically identified who differed in their patterns of SI-derived components and their risk of CHD. As expected, a pattern of characteristics in which hostility was salient was found in unadjusted analyses to be predictive of CHD. Moreover, another pattern of characteristics which appears to reflect pressured, controlling, socially dominant behavior in which hostility was not salient also was found to be predictive of CHD. Further, two patterns of characteristics were identified that were unrelated to CHD risk. The latter finding makes intuitive sense, as it is likely that there are various behavioral characteristics and patterns of characteristics that are unrelated to CHD risk. Finally, two patterns of characteristics were identified that were related to reduced risk of CHD. Overall, these results suggest that future research should investigate variables in addition to hostility in regard to risk for and protection from CHD.

The present study was exploratory; therefore the results should be interpreted cautiously. The cluster profiles and their associations with CHD risk are clearly in need of replication with other samples of men as well as women who vary in ethnicity. It should be noted, though, that in a previous study of 101 college women which involved cluster analysis of only six SI-components (38), four clusters were found (which were labeled quick, vigorous; loud, emphatic, hostile; verbally competitive; and easy going) that are quite similar to clusters 1, 3, 4, and 5 obtained in the current study. (CHD status was not investigated in the previous study, however.) Such a finding lends credibility to the cluster structures and their interpretation in the present study as well as suggests that similar profiles of components may be found across genders. Caution should also be exercised concerning the implications of the present study for the overall health outlook of men characterized by patterns that were found to be unrelated to CHD risk or related to reduced risk of CHD. It should not be inferred that these patterns are favorable signs for health in general, because their relation to risk for diseases other than CHD, e.g., cancer, is unknown. Another interesting question for future research, then, is to examine the relations between the various patterns of characteristics identified in the present study and a variety of disease endpoints.

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