CARDIOVASCULAR RESPONSES OF INDIVIDUALS WITH TYPE A BEHAVIOR PATTERN AND PARENTAL CORONARY HEART DISEASE*

DAVID B. NEWLIN† and ROBERT W. LEVENSON‡

Abstract—Cardiovascular and other responses to stress of subjects considered to be at risk for coronary heart disease (CHD) were studied in order to evaluate two hypothesized pathways in the link between CHD risk factors and the disease process. Forty-seven male subjects with and without a parental history of CHD and with either the Type A or Type B behavior pattern were exposed to two psychosocial stressors (reaction time and Stroop Color-Word test) and one physical stressor (isometric hand grip). Subjects with a parental history of CHD showed larger finger pulse amplitude responses to the two psychosocial stressors, and Type A subjects had larger diastolic blood pressure responses to all three stressors. These results indicated that subjects at greater risk for CHD had more substantial peripheral vascular responses to the stressors compared to low risk subjects; there were no differences in sympathetic cardiac responses related to contractility. The results are discussed in terms of potential mediating mechanisms in the development of CHD.

THE ASSOCIATION between the Type A coronary-prone behavior pattern and coronary heart disease (CHD) has been extensively studied over the past 20 years. Recent studies using a prospective methodology indicate heightened risk for CHD among Type A individuals, whether measured by the interview method [1] or the Jenkins Activity Survey (JAS) [2].

Several hypothesized pathways from the Type A behavior pattern to the manifestation of CHD have been proposed and studied. However, pathways related to heightened cardiovascular reactivity among Type A individuals have been the most extensively investigated. Conceptually, findings of increased Type A reactivity have been consistently interpreted in terms of heightened sympathetic activation among Type A individuals exposed to stressors relevant to the coronary-prone behavior pattern [3, 4]. These findings have included results showing greater physiological responses among Type A individuals in SBP [3, 4, 5, 6, 7, 8, 9, 10], DBP [5, 10, 11], heart rate [5, 6, 8], finger pulse amplitude (FPA) [12], and pulse transmission time (PTT) [15]. The interpretation that Type A individuals show heightened sympathetic reactivity in response to laboratory stressors is consistent with the view that coronary-prone individuals have chronically elevated cardiac responses to life demands [14], which, in turn, increase the demand for oxygen by the heart.

While this theoretical view has been widely promoted, there is a second possible mediational pathway involving heightened cardiovascular reactivity that has not been considered, perhaps because the theoretical development relevant to this alternative pathway has been both recent and controversial. It has been proposed that the immediate cause of CHD may be vasospastic attacks of the coronary

arteries. Oliva [15] and Hellstrom [16] have critically reviewed this research. Coronary artery vasospasm, in which constriction of the coronary arteries is mediated by alpha-adrenergic mechanisms, has been documented during angiographic examinations of anginal patients who subsequently had infarctions, as well as in myocardial infarction patients immediately following the attacks [15]. This theory, although also involving cardiovascular reactivity, emphasizes acute limitations in blood supply to the heart due to vasoconstrictive activity of the coronary arteries rather than chronic increases in demand.

The primary purpose of this research was to more fully examine the heightened cardiovascular reactivity of Type A individuals in response to stress in order to evaluate these two cardiovascular pathways. Although noninvasive measurement of coronary vasospasm is not yet possible, measures of vasoconstrictive activity in the periphery are available. These include FPA, which measures local vasoconstriction in the arteries and arterioles of the finger, and DBP, which is determined primarily by total peripheral resistance of the arterial system, and hence, vasoconstrictive activity in the entire body. It should be noted, however, that the relationship between peripheral vasoconstriction and vasospasm of the coronary arteries is unknown, and represents an avenue for further research. FPA and DBP are determined in large part by alpha-adrenergic mechanisms. A number of primarily beta-adrenergic measures are available, including SBP, PTT, which has been proposed as an indirect measure of blood pressure [17], or myocardial contractility [18], and pre-ejection period (PEP), which has been advocated as a relatively pure measure of myocardial contractility and sympathetic (beta-adrenergic) cardiac activation [19].

These two types of cardiovascular measures were included in the present research in order to evaluate mediational pathways involving cardiovascular reactivity—one pathway involving heightened sympathetic (beta-adrenergic) influences on the heart and the other concerning increased sympathetic (alpha-adrenergic) influences on the vascular system. Other noncardiovascular measures were assessed to determine whether cardiovascular differences were specific to coronary risk, or were part of a larger pattern of differences that included electrodermal activity and general somatic activity (ACT). These pathways and variables are schematized in Table I. The subjects were Type A and B individuals classified by a student version of the JAS [20]. This self-report questionnaire has been validated in a college student population in terms of expected behavioral differences between behavior types [14].

TABLE I.—Hypothesized Mediational Pathways and Associated Response System

Mediational pathway (mechanism)	Relevant physiological measure
Sympathetic Cardiac Responses (primarily beta-adrenergic)	Systolic Blood Pressure (SBP) Pre-ejection Period (PEP) Pulse Transmission Time (PTT)
Vasoconstrictive Activity (primarily alpha-adrenergic)	Diastolic Blood Pressure (DBP) Finger Pulse Amplitude (FPA)
Generalized Physiological Activity (mixed physiological influences)	Heart Rate (HR) Skin Conductance Level (SCL) Skin Conductance Responses (SCR) General Motor Activity (ACT)

However, although the JAS has documented predictive validity for CHD [2], the ability of the student version to predict CHD has not been evaluated.

In addition to the coronary-prone behavior pattern, we selected individuals with and without a parental history of CHD. This was found in the Western Collaborative Group Study to be a significant risk factor for CHD [21]. This additional risk factor was studied in order to determine whether mediating physiological pathways were common to two different risk factors, or whether a final common pathway for CHD risk could be identified. In addition, we were interested in the interaction of these two risk factors, a phenomenon that has proved important for other chronic diseases. College-age subjects were chosen for this study because they are temporally removed from the clinical manifestation of disease. Thus, mediational pathways may be assessed without contamination by secondary effects of CHD or early manifestations of the disease (e.g. atherosclerosis) so that correlates of risk can be attributed to causal rather than secondary factors.

While a number of studies have examined cardiovascular responses of Type A individuals, none have included a full complement of measures that would allow assessment of cardiac vs vascular influences, while at the same time including another risk factor for CHD (i.e. parental CHD). Thus, the present research places cardiovascular responsivity of Type A individuals within a broader context of patterns of mediational pathways from risk factors to clinical CHD.

METHOD

Subjects

Forty-seven male subjects, ages 18–25, participated in the experiment. They were recruited by two newspaper advertisements, the first offering $2.50 per hour "for males, ages 18–30 to serve as subjects in a Psychology experiment studying the effects of personality type on stress responses." The second advertisement was identical except for the additional request for subjects whose "biological mother and/or father has had coronary heart disease or a heart attack." Subjects who expressed interest were contacted by phone and told the nature of the tasks, as well as the possibility of receiving "a moderately painful electric shock." All subjects were asked if they had a personal history of CHD or essential hypertension, and those who had were excluded.

Subjects were assigned to groups according to their scores on the student JAS and their responses on a parental medical history questionnaire. In our sample, the median of the JAS was between 6 and 7. Therefore, subjects with JAS scores of 8 and above were designated Type A, and those scoring 5 and below were designated Type B. Within these groupings, subjects who reported* that their biological father and/or mother had a history of CHD were given a Parental History (PH) designation, and those reporting no such history were designated as No Parental History (NPH). This yielded four groups: PH-A (N = 14), PH-B (N = 9), NPH-A (N = 11), and NPH-B (N = 13).

Apparatus

Physiological measures were recorded on a Grass Model 7 Polygraph and analyzed on-line using a PDP 11/10 minicomputer. The following measures were obtained: (a) cardiac interbeat interval (IBI)—the electrocardiogram (EKG) was detected using Beckman miniature electrodes placed on opposite sides of the chest, with a ground electrode clipped to the earlobe. IBI was computed as the interval between successive R-waves; (b) pulse transmission time to the finger (PTT)—the interval from the Q-wave of the EKG to the initial upstroke of the finger pulse (located using a digital slope-detection algorithm) was measured using a Grass PTT1-6 photoplethysmograph attached to the middle finger of the left hand to detect the finger pulse wave; (c) pre-ejection period (PEP)—using a Hewlett-Packard No. 780—16 photoplethysmograph attached to the pinna of the right ear to detect the ear pulse, a locally constructed

---

*It was not possible to confirm parental histories by either contacting the parents or the parent's physicians. Thus the validity of our designations are based on the accuracy of the subjects' reports. Despite the certainty of these subjects concerning their parents having or not having the criterion problems, the potential for erroneous reporting must be recognized.

circuit to differentiate the ear pulse signal, and a Narco Biosystems No. 705—0016 heartsounds micro-phone to detect the phonocardiogram. PEP was determined using a procedure described in detail by Newlin and Levenson [19]*; (d) finger pulse amplitude (FPA)—the height of the finger pulse wave from trough (foot of upstroke) to peak (maximum point in cardiac cycle) was determined; (e) skin conductance level (SCL)—a constant voltage device was used to pass a small current through large Beckman electrodes attached to the second phalanges of the index and middle fingers of the right hand using KC1-Unibase electrode paste. This yielded a calibrated D.C. measure of SCL in μMho's; (f) skin conductance responses (SCR)—the output of the SCL channel was rerouted to an A.C. coupled polygraph channel. SCR were defined as changes in SCL greater than 0.5 cm after setting the sensitivity of the polygraph preamplifier so that the average SCR amplitude during the initial baseline period was 1 cm; and (g) general motor activity (ACT)—an electromagnetic sensor attached to the subject's chair generated a signal with bodily movement that was integrated using a Grass 7P10 Integrator. This signal was converted into arbitrary A/D units that were comparable between subjects. In addition, SBP and DBP were obtained manually using an electronic sphygmomanometer that had been modified to allow inflation and determination of pressures in a room adjacent to the subject room. The cuff was always inflated 30 mm Hg above the previous SBP value in order to track increases in SBP. The Korotkoff sounds were initially monitored on an oscilloscope to insure that the light and buzzer display on the electronic sphygmomanometer was properly triggered. SBP and DBP were recorded once per minute.

A four digit LED display controlled by the computer was placed on a table in front of the subject and was used to signal the subject during the experimental procedure. A Stoelting hand dynamometer was used for the isometric handgrip task.

Procedure

Subjects were first asked to sign informed consent forms and then to complete the JAS and a question-naire concerned with parental medical history. Maximum handgrip was determined by having the subject squeeze the dynamometer as hard as possible three times using the left hand. After electrodes and transducers were attached, subjects were given oral instructions concerning the experimental procedures and told to refer to a poster summarizing the instructions that was mounted on the opposing wall. A 10 min adaptation period ensued during which no data were collected, then each subject was exposed to three stressors.

Reaction time (RT). Following a five min baseline, subjects saw the number "9999", signalling the RT task, on the display device. They had been instructed previously to watch for a change to the number "1" at which time they were to press a response key attached to the arm of the chair. Subjects were told that they could avoid receiving shock by pressing the key quickly after the "1" appeared. To equalize the stressor across subjects, the task was divided into 5 RT trials of 1 min duration. Following the appearance of the "1" in the third trial, (and only at that time), they received a brief shock to the left wrist regardless of their performance. The number "9999" appeared on the display throughout the RT task except when the "1" appeared.

Stroop Color-Word Test. The Stroop Color-Word Interference Test consisted of a set of names of colors printed in ink colors different from the written color name (e.g., "blue" printed in red ink). After a five min baseline, the number "5555" appeared on the display device. Subjects had been instructed to call out the *ink colors* "as quickly as possible" whenever the "5555" display was on. The signal was on for the first 30 s of each 1 min trial. The board on which the Stroop stimuli were printed was placed face down on a rack on the arm of the subject's chair before and after the Stroop task, and between the 30 s Stroop trials. Subjects were told to rest during the 30 s between each Stroop trial. Prior to the third trial, the subject was criticized regardless of performance by telling him, "Most people do it about twice that fast. Try to do it faster the next time."

Isometric handgrip. Following a five minute baseline, the number "1111" appeared on the display device. Subjects had been instructed to remove the finger plethysmograph, pick up the dynamometer, and squeeze it.

Half of the subjects in each condition received the Stroop stressor first followed by the RT stressor, and the other half received the RT first followed by the Stroop. All subjects received the isometric handgrip stressor last.

Subjects then received a Psychosomatic Symptomatology Questionnaire (PSQ) [22] in oral form. The PSQ is a short true-false test with somatic items from the Minnesota Multiphasic Personality Inventory and other sources. This test was administered to determine whether risk subjects had greater somatic complaints, an effect that would have implications for the generality of stress responding in these groups.

Following completion of the session, subjects were paid ($2.50 per hour) and fully debriefed.

---

*In this system, PEP is determined by measuring the interval from S^2 on the phonocardiogram to the nadir of the differentiated ear pulse signal. This quantity (which represents the true transit time of the pulse from the heart to the ear) is subtracted from the interval between the Q wave and the upstroke of the differentiated ear pulse signal to derive PEP.

RESULTS

Physiological data obtained during the experiment were averaged into 1 min periods. The dependent variables (SBP, PEP, PTT, DBP, FPA, IBI, SCL, SCR, and ACT) were submitted to two sets of analyses of variance (ANOVAs). In the first set, data obtained during the RT and Stroop stressors were analyzed in a $2 \times 2 \times 2 \times 2 \times 5$ repeated measures ANOVA in which Parental History and Behavior Type (A vs B) were between-subject factors, and Stressor (RT vs Stroop), Baseline vs stress, and Minute (five 1 min periods) were within-subject factors. The data obtained during the handgrip stressor were analyzed in a similar ANOVA with Baseline vs stress as the only repeated measure, using the 1 min period before the handgrip period as the baseline. The ANOVA's used the unweighted means solution to handle unequal N.

Separate multivariate analyses of variance (MANOVA) were performed on the combined RT and Stroop data and the Handgrip data with Parental History and Behavior Type as between-subject factors. Due to limitations in the available MANOVA program, it was necessary to collapse across the within-subject factors (Stressor, Baseline vs stress, and Minute). To accomplish this, changes from baseline were computed for the RT and Stroop stressors and then averaged. The dependent measures that were specifically predicted to differentiate between groups based on previous research, SBP, DBP, and FPA, were entered into the MANOVA.

Additional analyses were performed and will be described below.

Stressor effects

The RT, Stroop, and Handgrip stressors produced significant responses in all measured variables. The means and F values for the responses to the stressors are presented in Table 2. The responses to the RT and Stroop stressors were combined since our primary concern in this analysis was to verify the overall efficacy of the stressors; in subsequent analyses involving the CHD risk factors, the responses to the three stressors were handled separately.

Order effects

Although the ordering of the RT and Stroop stressors were counter-balanced within the risk conditions, we were interested in determining whether there were, in

TABLE II.—RESPONSES TO STRESSORS

| Measure | RT and Stroop | | | Handgrip | | |
	Baseline	Stress	F(1/43)	Baseline	Stress	F(1/41)
SBP (mm Hg)	120.7	125.5	48.0‡	118.5	130.0	77.4‡
PEP (ms)	83.1	77.1	72.0‡	89.7	83.6	19.2‡
PTT (ms)	242.0	237.0	25.5‡	*	*	
DBP (mm Hg)	73.0	74.5	15.7†	73.5	84.4	160.8‡
FPA (units)	24.6	18.2	28.8‡	*	*	
IBI (ms)	848.4	826.3	9.6†	867.0	778.5	78.9‡
SCL (μmho)	7.8	9.4	70.6‡	8.5	10.6	61.5‡
SCL (no.)	2.7	5.3	83.3‡	3.1	7.8	34.9‡
ACT (units)	7.1	82.0	8.4†	76.5	131.3	28.9‡

*Not measured during Handgrip.
†$p < 0.01$.
‡$p < 0.001$.

fact, order effects. This analysis was performed by adding Order (RT first vs Stroop first) as a between-subject factor. As might be expected, when a stressor came first, it tended to produce a larger response than when it came second. This was true for PEP, PTT, IBI, SCL, and ACT as reflected in significant Order × Stressor × Baseline vs stress interactions; $(F(1/39) = 11.0, p < 0.01)$, PTT $(F(1/39) = 11.9, p < 0.01)$, IBI $(F(1/39) = 7.5, p < 0.01)$, SCL $(F(1/39) = 9.3, p < 0.01)$, ACT $(F(1/39) = 10.1, p < 0.01)$. The more critical order effects involving interactions with the Parental History and Behavior Type factors were not significant, indicating that task order had no effect on the differential responding to stress of the CHD risk groups.

Homogeneity of variance

Assumptions of homogeneity of variance were tested using Hartley's F_{max} test [23], and the results are presented in Table 3. Assumptions of homogeneity were supported in all cases except for the Behavior Type interaction for FPA (for which there was no mean effect). The Parental History × Behavior Type interaction with the BL vs stress factor was also tested because mean effects were found for FPA. Assumptions of homogeneity of variance were violated $(F_{max} (8,12) = 17.3, p < 0.01)$ for this effect.

TABLE III.—TESTS OF HOMOGENEITY OF VARIANCE FOR INTERACTIONS OF RISK FACTORS WITH BL VS STRESS FACTOR

Variable	RT and Stroop		Handgrip	
	Parental History $F(4,12)$	Behavior Type $F(4,12)$	Parental History $F(4,12)$	Behavior Type $F(4, 12)$
IBI	1.07	1.28	1.54	1.89
SBP	1.90	1.83	2.70	3.17
DBP	1.73	1.72	2.77	2.58
PEP	1.13	1.31	1.44	1.34
FPTT	1.45	1.47	†	†
FPA	3.74	7.65*	†	†
SCL	2.88	1.75	2.33	1.81
SCR	2.60	2.47	2.10	1.90
ACT	1.38	1.48	2.65	2.34

*$p < 0.05$.
†Not measured during Handgrip.

Risk for CHD

Group assignment. The mean JAS score was 10.8 for the Type A group and 4.1 for the Type B group. Interestingly, the mean JAS score for the PH group (8.3) did not significantly differ from the mean JAS score for the NPH group (6.9; $F(1/45 = 1.7)$. The Type A group had faster reaction times averaged across the 5 reaction time stressor trials compared to the Type B groups ($\overline{X} = 379$ vs $\overline{X} = 425$ msec; $F(1/144) = 6.3, p < 0.05$). The PH and NPH groups did not differ in reaction times. Analysis of the makeup of the four experimental groups revealed no significant differences in average age (Type A: $\overline{X} = 21.1$, Type B: $\overline{X} = 21.6$, $F(1/43) 1$; (PH: $\overline{X} = 20.9$, NPH: $\overline{X} = 21.9, F(1/43) = 2.6, p < 0.05$). There were no significant baseline differences among the 47 subjects assigned to the four experimental groups in any of the measured physiological variables.

Parental history of CHD

PH subjects reported significantly ($F(1/45) = 5.6$, $p < 0.05$) more somatic complaints on the Psychosomatic Symptomatology Questionnaire than NPH subjects (PH: $\overline{X} = 4.2$, NPH: $\overline{X} = 2.4$).

There were significant differences between the parental history groups in FPA responses to the stressors. Subjects with parental history of CHD had larger FPA decreases to both the RT and Stroop tasks. Although the MANOVA main effect for Parental History did not reach statistical significance ($F(3/41) = 2.4$, $p < 0.08$, see Table 4), ANOVA was performed to determine the significance of the FPA changes because this effect was specifically hypothesized. The univariate Parental History × Baseline vs stress interaction was significant ($F(1/43) = 5.5$, $p < 0.05$). This effect was consistent across the two psychosocial stressors; the interaction with the Stressor factor was not significant ($F(1/39) < 1$).

TABLE IV.—MANOVA SUMMARY OF DIFFERENCE SCORE RESULTS

Effect	RT and Stroop*		Handgrip†	
	$F(3/41)$	$p<$	$F(2/42)$	$p<$
Parental History	2.4	0.08	<1	
Behavior Type	3.4	0.05	4.9	0.05
Parental History × Behavior Type	2.1		<1	

*SBP, DBP, and FPA entered in MANOVA.
†SBP and DBP entered in MANOVA (FPA not measured).

Since RT was found to be a significant determinant of the magnitude of physiological responses to stress, a covariance analysis was performed in order to determine whether the FPA difference was significant after removal of the variance associated with the average RT. The FPA effect was significant ($F(1/43) = 4.2$, $p < 0.05$) with average RT as a covariate.

Type A behavior pattern

Type A subjects also reported significantly ($F(1/45) = 4.8$, $p < 0.05$) more somatic complaints on the Psychosomatic Symptomatology Questionnaire then Type B subjects (Type A: $\overline{X} = 7.0$, Type B: $\overline{X} = 4.9$).

Subjects manifesting the Type A behavior pattern evidenced differences in DBP and SBP responses to the stressors compared to Type B subjects. The DBP differences were reflected in greater DBP responses for the Type A subjects during all three stressors. The interaction of Behavior Type × Baseline vs stress was significant for the RT and Stroop stressors ($F(1,43) = 5.0$, $p < 0.05$) and for the handgrip stressor ($F(1/35) = 5.5$, $p < 0.05$). This was supported by a significant effect of Behavior Type in the MANOVA ($F(3/41) = 3.4$, $p < 0.05$). DBP increased +2.5 mm Hg for Type A subjects compared to increases of +0.7 for the Type B subjects during the RT and Stroop stressors; during the Handgrip, DBP increased +12.9mm hg for Type A's and +8.9 mm Hg for Type B's.

Covariance analysis was also performed on DBP using average RT as a covariate.

The DBP responses of Type A subjects were still significantly ($F(1/43) = 5.2$, $p < 0.05$) larger than Type B subjects after removal of the variance associated with RT.

The SBP difference was reflected in a significant interaction of Behavior Type × Baseline vs stress for the Handgrip stressor ($F(1/36) = 5.1$, $p < 0.05$). Type B subjects had larger (+14.4 mm Hg) SBP responses than Type A subjects (+8.5 mm. Hg) to this stressor.

Combined risk. We found little evidence of interaction of Parental History with Type A risk. The interaction of the risk factors was not significant ($F(1/43) = 2.2$) for the Symptom Questionnaire. The only significant interaction of Parental History × Behavior Type × Baseline vs stress was for FPA in the RT and Stroop ANOVA ($F(1/43) = 5.1$, $p < 0.05$). However, it was noted above that the assumption of homogeneity of variance was violated for this interaction effect. Therefore, this effect is not interpretable. The Parental History × Behavior Type interaction in the MANOVA was nonsignificant ($F(3/41) = 2.1$).

DISCUSSION

The major findings of this study were the larger DBP responses of Type A subjects to three different stressors (RT, Stroop, and Handgrip), and the larger FPA responses of subjects with parental histories of CHD to both the RT and Stroop stressors.* In both cases, these effects represented heightened reactivity of the peripheral vasculature for subjects considered to be at greater risk for CHD. Further, response differences between subjects at varying risk were limited to these and other specialized cardiovascular measures; they did not extend to the more general measures of arousal such as HR, electrodermal response, or general somatic activity. There was no evidence of an interactive effect of these two risk factors.

Subjects at greater risk for CHD responded specifically in terms of peripheral vasoconstrictive activity. This lends support to the alternative formulation of a cardiovascular pathway derived from (but not necessarily confirming) theories of coronary vasospasm. No support was found for the hypothesis of greater sympathetic cardiac activation among Type A and parental history subjects, despite the inclusion of three measures (PEP, PTT, SBP) intended to assess myocardial contractility and beta-adrenergic cardiac influences.

Our findings of greater vascular responsivity of subjects considered to be at risk for CHD are consistent with previous research. It should be recalled that Keys *et al.* [24] found that DBP increase to the cold pressor was the best single predictor of subsequent CHD in the 20 year Minnesota study. The effect of greater vasomotor responsivity of subjects with parental histories of CHD is a new finding, but has direct parallels to research on vasomotor responses of actual CHD patients. Cromwell *et al.* [25] examined FPA responses of small samples of myocardial infarction patients and medical controls to an insoluble cognitive task, and found greater vasomotor reactivity in the cardiac patients. Klorman *et al.* [26] replicated these results, finding greater FPA decreases both in acute myocardial infarction

---

*It should be noted that the Parental History finding was not supported by the multivariate analysis. However, we feel the finding is still valid as it was consistently supported by univariate statistics utilized on an *a priori* basis.

patients and in individuals with fully healed infarctions. This suggested that heightened vasomotor reactivity might be a stable trait of individuals with a history of CHD. Our results, showing a similar effect for subjects with a parental history of CHD, may indicate that this characteristic is also present in individuals at familial risk for CHD. It is possible that the heightened vasomotor reactivity found by Cromwell *et al.* [25], Klorman *et al.* [26], and in the present research, reflects similar mechanisms to those involved in coronary artery spasms. Although this link has not been established, it represents an important avenue for further research, particularly the question concerning the possible association of peripheral vasoconstrictive activity with coronary vasospasm.

We had expected that subjects at higher risk for CHD would show evidence of heightened beta-sympathetic cardiac response, and included measures of PEP and PTT in addition to SBP to more fully evaluate this response system. In fact, our results comparing SBP responses of Type A and B subjects ran opposite to prediction, with Type B subjects showing larger SBP responses during the handrip stressor. It is conceivable that the relationship between Type A behavior pattern and SBP reactivity emerges only under certain kinds of environmental manipulations. Further research is needed in which the environmental situations eliciting DBP vs SBP differences between behavior types are formally studied.

We see the results as supporting the hypothesis that Type A and parental CHD risk factors reflect slightly different pathways in the development of CHD. However, some evidence of common features were that both risk groups responded in terms of heightened peripheral vasoconstriction and both Type A and parental CHD groups endorsed more somatic complaints on the Psychosomatic Symptomatology Questionnaire.

We have presented evidence that the ultimate convergence of these risk pathways may be increased peripheral vascular responding. If this finding is supported by future research, it may provide a useful conceptual link between two well documented indicators of risk for CHD (Type A behavior pattern and parental CHD) and new findings concerning vasomotor factors in the disorder.

REFERENCES

1. ROSENMAN RH, BRAND RJ, JENKINS CD, FRIEDMAN M, STRAUS R, WURM M. Coronary heart disease in the Western Collaborative Group Study: Final follow-up of 8.5 years. *J Am Med Ass* 1975; **233:** 872.
2. JENKINS CD, ROSENMAN RH, ZYZANSKI SJ. Prediction of clincial coronary heart disease by a test for coronary-prone behavior pattern. *N Eng J Med* 1974; **290:** 1271.
3. DEMBROSKI TM, MACDOUGALL JM, SHIELDS JL, PETTITO J, LUSHENE R. Components of the Type A coronary-prone behavior pattern and cardiovascular responses to psychomotor performance challenge. *J Behav Med* 1978; **1:** 159.
4. MANUCK SB, GARLAND FN. Coronary-prone behavior pattern, task incentive, and cardiovascular response. *Psychophysiology* 1979; **16:** 136.
5. DEMBROSKI TM, MACDOUGALL JM, SHIELDS JL. Physiologic reactions to social challenge in persons evidencing the Type A coronary-prone behavior pattern. *J Hum Stress* 1977; **3:** 2.
6. DEMBROSKI TM, MACDOUGAL JM, HERD JA, SHIELDS JL. Effect of level of challenge on pressor and heart rate responses in Type A and B subjects. *J Appl Soc Psychol* 1979; **9:** 209.
7. MANUCK SB, CRAFT S, GOLD KJ. Coronary-prone behavior pattern and cardiovascular response. *Psychophysiology* 1978; **15:** 403
8. PITTNER MS, HOUSTON BK. Response to stress, cognitive coping strategies and the Type A behavior pattern. *J Persp Soc Psychol* 1980; **39:** 147.

9. Lovallo WR, Pishkin V. A psychophysiological comparison of Type A and B men exposed to failure and uncontrollable noise. *Psychophysiology* 1980; **17:** 29.

10. Sherwitz L, Berton BS, Leventhal H. Type A behavior, self-involvement, and cardiovascular response. *Psychos Med* 1978; **40:** 593.

11. Schell SM, Lusche DJ. Psychophysiological response patterns of coronary prone and non-coronary prone males. *Psychophysiology* 1981; **18:** 139.

12. Van Egeren LF. Social interactions, communications, and the coronary-prone behavior pattern: A psychophysiological study. *Psychos Med* 1979; **41:** 2.

13. Goldband S. Stimulus specificity of physiological response to stress and the Type A coronary-prone behavior pattern. *J Pers Soc Psychol* 1980; **39:** 670.

14. Glass DC. *Behavior patterns, stress, and coronary disease.* New Jersey: Erlbaum, Hillsdale, 1977.

15. Oliva PB. Role of coronary artery spasm in ischemic heart disease. *Adv Card* 1978; **22:** 164.

16. Hellstrom HR. Coronary artery vasospasm: The likely immediate cause of acute myocardial infarction. *Br Heart J* 1979; **41:** 426.

17. Steptoe A, Smulyan H, Gribbin B. Pulse wave velocity and blood pressure change: Calibration and applications. *Psychophysiology* 1976; **13:** 488.

18. Newlin DB. Relationships of pulse transmission times to pre-ejection period and blood pressure. *Psychophysiology* 1981; **18:** 316.

19. Newlin DB, Levenson RW. Pre-ejection period: Measuring beta-adrenergic influences upon the heart. *Psychophysiology* 1979; **16:** 546.

20. Krantz DS, Glass DC, Snyder ML. Helplessness, stress level, and the coronary prone behavior pattern. *J Exp S Psy* 1974; **10:** 284.

21. Sholtz RI, Rosenman RH, Brand RJ. The relationship of reported parental history to the incidence of coronary heart disease in the Western Collaborative Group Study. *Am J Epidem* 1975; **102:** 350.

22. Reyher J, Basch JA. Degree of repression and frequency of psychosomatic symtoms. *Perc Mot Skills* 1970; **30:** 559.

23. Winer BJ. *Statistical principles in experimental design.* New York: McGraw-Hill, 1971.

24. Keys A, Taylor HL, Brozek J, Anderson JT, Simonson E. Mortality and coronary heart disease among men studied for 23 years. *Arch In Med* 1971; **128:** 201.

25. Cromwell, RL, Butterfield EC, Brayfield FM, Curry JJ. *Acute myocardial infarction: Reaction and recovery.* St. Louis: Mosby, 1977.

26. Klorman RS, Klorman R, Cromwell RL, Shah PH. Plethysmographic responses to experimental stress in acute and recovered myocardial infarction patients. *Psychophysiology* 1978; **15:** 271.