# Idiodynamic Profiles of Cardiovascular Activity: A P-technique Approach

BRUCE H. FRIEDMAN, PH.D.<sup>1</sup> AIMEE K. SANTUCCI, M.S.<sup>2</sup>, PH.D.\*

<sup>1</sup>Department of Psychology, Virginia Polytechnic Institute and State University <sup>2</sup>Department of Psychology, Virginia Polytechnic Institute and State University

A study was conducted to expand the conventional view of cardiovascular (CV) reactivity by using the idiodynamic paradigm for investigation of individuals. Patterns of autonomic CV regulation were assessed in six subjects across diverse laboratory tasks on three separate occasions. Individual CV profiles were derived from these data with P-technique factor analysis, and then group aggregated with chain P-technique. The composite pattern suggested a three-component solution consisting of cardiac rate, cardiac contractility, and peripheral resistance factors. Individual profiles were compared to the composite pattern; these profiles differed in the number of components derived, percentage variance explained by these components, and relative dominance of specific CV components. A hypothesis that emerged is that the subjects differed in the complexity of CV control. It appears that the idiodynamic framework, combined with novel research designs and statistical methods, may help expand the view of CV reactivity beyond the traditional unitary view as response magnitude.

# Introduction

THE IDIODYNAMIC PERSPECTIVE of psychology views the individual as the foundation of behavioral science. This conceptualization entails a distinctive methodology that has been applied to diverse areas such as personality assessment, psychodiagnosis, and psychohistory (see Rosenzweig, 1984, 1986 for overviews). However, idiodynamic investigations of physiology have been limited (e.g., Rosenzweig, 1973). Yet, idiodynamics transcends topical boundaries and holds broad research potential (Rosenzweig, 1986). The present paper explores the potential of idiodynamics for the study of cardiovascular (CV) activity as a means to capture the dynamic quality of CV responding in individuals. A primary goal of this venture is to spark new depictions of CV *reactivity*, with concomitant hypotheses relevant to health. To date, reactivity has been defined typically as a CV response change from resting baseline to a discrete stimulus (Manuck, 1994; Sherwood & Turner, 1992), and has been studied with "large-N" research designs. This approach has been central in testing the "reactivity hypothesis", which links large CV responses to stress with disease risk, and has generated voluminous research, much of it supportive (Blascovitch & Katkin, 1991; Krantz & Manuck, 1984; Manuck, 1994; Rozanski,

Integrative Physiological & Behavioral Science, October–December 2003, Vol. 38, No. 4, 295–315.

Address for Correspondence: Bruce H. Friedman, Department of Psychology, Virginia Polytechnic Institute and State University, Blacksburg VA 24061–0436. *Email: bhfriedm@vt.edu*, Phone: (540)231–9611, *Fax:* (540)231–3652

<sup>\*</sup>Aimee Santucci is now affiliated with the Department of Psychology, University of Pittsburgh.

Blumenthal, & Kaplan, 1999). However, conspicuous negative results suggest that this model cannot explain fully the link between CV responding and disease (Carroll, Smith, Sheffield, Shipley, & Marmot, 1995; Foflick et al., 1990; Gerin, Rosofsky, Pieper, & Pickering, 1994; Julius et al., 1991; Pickering & Gerin, 1990; van Doornen, Snieder, & Boomsa, 1998). Accordingly, calls for new approaches to reactivity have been made (Light, 2001; Linden, Gerin, & Davidson, 2003; Thayer, 1998).

A drawback of the prevailing reactivity model is its insensitivity to CV temporal dynamics (Jennings, van der Molen, & Somsen, 1998; Thayer, 1998; Thayer & Friedman, 2000). Intensive investigations of individuals over time and situations afford increased sensitivity to temporal patterns (Denenberg, 1982; Endicott, 1993; Thoman, 1986), and so may be valuable in this regard. Idiodynamics views the individual as a series of dynamic temporal events, and offers a platform to launch such studies of CV reactivity. This proposal is explored here, first from a theoretical perspective, followed by an empirical study intended as an exemplar of this approach.

# Idiodynamics, Individual Research, and Systems Theory

The distinction between *nomothetic* and *idiographic* research has a storied past. The former typically refers to "large-N" designs, and the latter to studies of individuals (Allport, 1937, 1962). Although nomothetic designs have long dominated the behavioral and medical sciences, individual research offers its own distinct values (e.g., see Valsiner, 1986 for review). The idiodynamic approach to individual research is summarized below, followed by consideration of the general advantages of individual research for a systems view of responding.

# Idiodynamics: The Basics

Idiodynamics is founded on the study of individual dynamics (Rosenzweig, 1958). Normative data in idiodynamics derive from the unique population of events in an individual life, defined as the *idioverse* (Rosenzweig, 1984, 1986). This approach contrasts the common sense of "individual differences", in which individual data are meaningful only in reference to group norms (cf. Anastasi, 1958). Idiodynamic norms reflect recurrent event patterns (*markers*) in the idioverse, which is situated in biologic and sociocultural milieus (Figure 1). These influences fuse with personal history in the present, and all these factors must be considered in explaining and predicting individual behavior.

This notion is formalized in the idodynamic schema of three norms: nomothetic, *demo-graphic*, and idiodynamic (Rosenzweig, 1984, 1986). Interpretive mode, not population size, determines these norms, which go beyond equating nomothetic with "group", and idiographic with "individual" (Rosenzweig & Fisher, 1997). In the idiodynamic view, nomothetic norms are universal, and demographic norms apply to comparisons based on group variables such as gender, race, or age. Idiodynamic norms describe lawful individual patterns, and with rare exception, are absent from psychophysiological research (cf., Allen, Boquet, & Shelley, 1991; Fahrenberg, 1986). Yet nomothetic and demographic norms are uniquely exhibited in individuals, and so the scientific study of individuality is vital to apply these norms to individuals (Rosenzweig, 1950, 1986).

Idiodynamics perceives responses primarily in reference to each other (*response dominance*; R-R), rather than as reactions to discrete stimuli (S-R; Rosenzweig, 1951). The R-R view of stimulus and response is as a mutually defined complex in a global temporal



Fig. 1. Parameters of the idioverse: A future paradigm for dynamic psychology (from Rosenzweig, 1986; printed with permission from author).

structure (*configuration dominance*). The R-R concept underpins the idiodynamic view of the individual as a unique, unbroken series of events that are defined as "dynamic behavioral reconstructions" reflecting the interplay of internal and external factors, or stimulus and response (Rosenzweig, 1986, p. 242). This model is distinct from that of trait psychology (Rosenzweig & Fisher, 1997), in which uniqueness is expressed in internalized traits, conceptually similar to common traits used to compare individuals (Allport, 1961, 1966). The environment can moderate the expression of, but never define, such traits themselves.

# Individual Research and Systems Theory

Although many general scientific principles have derived from a small number of intensively studied individuals (Garmezy, 1982), there is a pervasive bias against individual data, which are held as irrelevant to general laws (Danziger, 1990; cf., Eysenck, 1954; Miranda & Borkovec, 1999). This view overlooks the benefits of individual studies, which stem from their multi-occasion formats that yield a more representative sample of the diverse organismic and environmental determinants of behavior than do single-occasion group designs (Brown & Moskowitz, 1999). Individual research is thus well suited to the systems perspective, in which self-regulating organismic systems are governed by a multiplicity of interactive internal and external rhythms and processes (Nesselroade & Ford, 1987). Group averaging of data within a single task or occasion can mask these underlying differences in processes (Larsen & Cutler, 1996; Mandell & Selz, 1992; Ohman & Magnusson, 1987; Thayer, 1998). Wide-ranging inter-individual variations in physiological regulation have been found within a common stimulus situation (Sargent & Weinman, 1966), and conversely, individuals can show consistent physiological response patterns across diverse conditions (Malmo & Shagass, 1949). Both of these instances of psychophysiological individuality can hold significant scientific insights, but neither is detectable with single-occasion group designs (Fahrenberg, 1986).

Individual data are best seen as complementary to group and universal norms. For example, replication of individual patterns across persons can lead to generally applicable principles (Jones & Nesselroade, 1990; Zevon & Tellegen, 1982; Valsiner, 1986). Alternatively, demographic and nomothetic norms can identify parameters for individual comparisons (Denenberg, 1982; Nesselroade & Ford, 1987). The mutual dependence of individual and group data is pivotal in linking clinical case studies with epidemiological research (Ohman & Magnusson, 1987; Rose, 1992). Moreover, systems theory emphasizes that similar processes can apply at multiple planes of analysis (Mandell, Stewart, & Russo, 1981; Miller, 1978; Thayer & Friedman, 1997). In sum, multiple norms—individual, group, and universal—are essential to a comprehensive understanding of human behavior.

# CV reactivity: An Idiodynamic Approach

Idiodynamics offers a systematic framework for individual studies of CV regulation. The potential of this approach described below, vis-à-vis the S-R model of CV reactivity.

#### The S-R Approach to Reactivity

In the S-R view, CV reactivity is framed as a discrete response to a specific external event; i.e., CV variables are group aggregated and compared within rest and stress epochs (Kamarck, 1992; Manuck, 1994). This model conforms to classical notions of experimental control, and promotes values such as task homogeneity, internal consistency, and testretest reliability to attain conventional standards of psychometric reliability (Kamarck, 1992; Manuck, Kasprowicz, Monroe, Larkin, & Kaplan, 1989). The use of homogeneous tasks is predicated on sympathetic hyperresponsivity being the key process in linking reactivity with CV disease, a commonly held view (Rozanski, Blumenthal, & Kaplan, 1999). As such, this strategy does increase the reliability of reactivity indices, but at the cost of sensitivity to other autonomic (ANS) mechanisms.

An alternative is to identify the range of stable regulatory processes prior to hypothesis testing about mean group differences (Mandell & Selz, 1992; Stallone & Stunkard, 1991). CV sampling across diverse, rather than similar, tasks may enhance detection of such processes. Resultant cross-situational profiles may be lawful for an individual, though not strictly "stable" (Magnusson & Endler, 1977; Nesselroade & Ford, 1987; Nesselroade, Pruchno, & Jacobs, 1986). These profiles reflect regulatory processes that can be more

#### IDIODYNAMIC PROFILES

variable than the outcome measures they control (Mandell & Selz, 1992; Stallone & Stunkard, 1991). For example, measures of sweat gland activity, which serves thermoregulation, are more variable than body temperature itself (Sargent & Weinman, 1966). In the systems view, stability is sustained by variability in component function across shifting milieus (Nesselroade & Forde, 1987; Weiss, 1969). This constancy is built on system flexibility, resilience, and coherence, and may not conform to classical reliability standards (Goldberger, 1991; Kaplan et al., 1991; Lipsitz & Goldberger, 1992; Peng et al., 1994).

# An Idiodynamic View of CV Activity

An idiodynamic implementation of reactivity research designs begins with the idiodynamic three-norm schema. The S-R reactivity hypothesis does not apply nomothetically because a universal association between reactivity and CV disease across individuals and CV variables has not been found (cf. Carroll et al., 1995; Ewart & Kolodner, 1992; Foflick et al., 1990; Gerin et al., 1994; Julius et al., 1991; Pickering & Gerin, 1990; Sheffield & Carroll, 1994; van Doornen et al., 1998). This finding is not surprising in view of the myriad pathways to CV disease. Demographic norms have most typically characterized reactivity research, which frequently involves comparisons based on individual difference variables (see Turner, Sherwood, & Light, 1992 for a review). Some consistent findings have emerged at this level, but others have been mixed. Idiodynamic reactivity research, as conceived here, involves the intensive study of CV response patterns in individuals over time and a range of conditions, with the aim of generate new reactivity models and filling a void of individual CV data.

The idiodynamic R-R "configuration dominance" model stands in contrast to the S-R approach to CV reactivity. Consistent with traditional experimental design, the latter excludes response variability not directly attributed to specific external stimuli (manipulated variables) (Manuck, Kasprowicz, Muldoon, 1990). Though straightforward, this paradigm cannot account for complex temporal relationships between external events and CV measures (Levenson, 1988) or intrinsic CV rhythms that are independent of external stimuli (Goldberger, 1991). Hence, the framing of reactivity as a stable response to a precise external event enhances experimental control and reliability, but is constrained in modeling the dynamic interactive nature of CV activity. A related concern is the assumption of a stationary CV baseline that is requisite to the recurrent activation version of the S-R reactivity model (Edwards & Hill, 1967; Jennings et al., 1998). Consistent with a homeostatic view of reactivity as deviation from steady state, this model is predicated on linking chronic large changes in CV levels from baseline (rest) to stress with disease (Manuck et al., 1989). However, the notion of a static biological equilibrium is belied by findings that healthy cardiac dynamics are often marked by high variability, low predictability, and nonequilibrium in "baseline" states (Friedman & Thayer, 1998a; Friedman & Thayer, 1998b; Goldberger, 1991; Kaplan et al., 1991; Lipsitz & Goldberger, 1992; Peng et al., 1994).

In contrast, the R-R model perceives responses in global temporal configurations, and so holds all sources of variability as potentially relevant. The aim is to extract functional relationships among ongoing processes across the response patterns (O'Connor, 1990). One way to implement this view experimentally is by post-hoc removal of boundaries between diverse experimental conditions, followed by response aggregation across these epochs. This design models reactivity as a continuous process that traverses externally imposed epochs, as opposed to the more segmental baseline-stressor S-R view (Figure 2).



FIG. 2. Contrast between S-R (A) and R-R (B) models of reactivity.

The goal of this approach is detect recurrent response patterns from this simulated CV idioverse, and emulates the Rosenzweig *Picture-Frustration study* (P-F), an idiodynamic instrument to assess individual response patterns to frustration (Rosenzweig, 1956, 1978). The P-F evokes distinct response styles across a range of diverse frustrating situations. Similarly, a spectrum of non—heterogeneous CV challenges could be used in reactivity studies. Furthermore, the P-F blends nomothetic, demographic, and idiodynamic norms in analyzing responses. This schema is applicable to CV responding, in which individual cross-situational response profiles could be compared to group norms, and understood in terms of general psychophysiological principles.

In sum, several tenets of idiodynamics show relevance for individual studies of CV responding. Next, a specific design and analysis strategy is outlined to implement this approach.

# Idiodynamic Profiles: A Methodology for the Study of Reactivity

Numerous means are potentially compatible with idiodynamics and also applicable to the study of CV reactivity. One such methodology and accompanying analytic strategy is described below, followed by details of the method used in the present study.

#### IDIODYNAMIC PROFILES

# Data Aggregation within Individuals and P-technique Factor Analysis

A key element of many individual research designs entails data aggregation within individuals over time and diverse situations, which has corresponding *within-personal correlational* and *pooled cross-sectional* time series statistics (Dielman, 1983; Jaccard & Wan, 1993; Michela, 1990). Individual data acquired in this manner can be group aggregated if desired by within-subject standardization, which avoids confounding between and within-subject variance. Examples of this strategy can be found studies in which individual CV control patterns were extracted from a range of heterogeneous ANS tasks, and then group aggregated (Friedman et al., 1993; Friedman & Thayer, 1998a).

*P-technique* factor analysis is one statistical tool that has been used in tandem with such designs (Cattell, 1988a). P-technique extracts factors from multiple response variables sampled over repeated occasions in a single individual (Jones & Nesselroade, 1990; Luborsky & Mintz, 1972). The obtained factor patterns reflect underlying individual processes rather than static traits, and so fit the idiodynamic focus on events. P-technique can be expanded with *chain* P-technique, in which individual data are aggregated into a single factor pattern (Jones & Nesselroade, 1990). Scores are standardized within individuals before aggregation, and individual patterns are then compared to the group via between-factor correlations. Thus, this approach potentially engages idiodynamic, demographic, and nomothetic modes.

# Overview of the Present Study

The present study was designed to apply the idiodynamic perspective to CV reactivity. A small group of individuals engaged in a large set of diverse CV tasks over multiple experimental sessions. These tasks were chosen to generate a broad range of autonomic patterns (Freeman, 1990; Friedman, Thayer, & Tyrell, 1996). A representative set of ANS indices was used to capture the scope of CV function. These measures were aggregated within-subjects and across tasks and occasions, and then subjected to P-technique factor analysis to extract individual CV factor patterns. Finally, chain-P technique was applied to allow inter-individual comparisons of these patterns.

# Method

# Subjects

Seven female university students (mean age = 21.7 years) were recruited for the study, which included three approximately 2–3 hour experimental sessions spaced one week apart. Data from one subject were not included due to problems related to physiological recording and experimental protocols. All subjects were nonsmokers who reported no health problems, and had abstained from caffeine and alcohol for at least 12 hours prior to each lab session. Body mass index average was 20 (range: 17-24). Subjects were paid per session, with a bonus incentive for completing all three sessions and for performance on the video game task. Each session lasted approximately two hours and began around the same time (~ 4:00 p.m.).

# Apparatus

The electrocardiogram (ECG) and impedance cardiogram (ICG) were recorded with Ag-AgCl electrodes in conjunction with the Ambulatory Monitoring System v4.4 (AMS; Vrije Universiteit, Amsterdam, the Netherlands). The validity of this device is comparable to that of a non-ambulatory device in deriving ECG and ICG (Willemsen, De Geus, Claver, Van Doornen, & Carroll, 1996). The amplified ECG was passed through a bandpass filter of 17 Hz. The R-spike was recognized as with a level detector with automatic level adjustment in the analog ECG, and the resolution of the IBI time series was 1 msec. Electrode placement for the ECG and ICG was on the front and back torso, in accordance with the configuration guidelines described in the user manual for the AMS. BP was monitored with an IBS SD–700A automated BP monitor (Industrial & Biomedical Sensors Corp., Waltham, MA). A video car race game (Test Drive 4, Accolade, Inc., 1997) was used as the stimulus in the video game task, and a hand dynamometer (Model 78010, Lafayette Instrument Company, Lafayette, IN) was used in the handgrip task.

Dependent measures. Measures derived from the ECG were HR, which reflects mixed vagal and sympathetic  $\beta$ -adrenergic influences, and high frequency variability (HF), a HR variability index strongly correlated with cardiac vagal activity (Saul, 1990). The SAS Spectra procedure (SAS 10.0, Carey, NC) was used to determine the HF values, defined as spectral power in the .15-.40 Hz bandwidth (Malliani, Pagani, Lombardi, Cerutti, 1991). A finite Fourier transform was used to generate a periodogram, and then smoothed with a Bartlett window to get spectral estimates. The time series was detrended with linear detrending; the residuals of that line resulted in the detrended time series. Contractility indices based on systolic time intervals were derived from the ICG and served as measures of cardiac sympathetic b-adrenergic influences, including pre-ejection period (PEP), left ventricular ejection time (LVET), and Heather index of myocontractility (HI) (Sherwood et al., 1990). DZ/dt is sampled at 250 Hz during a 512 ms period around each R-wave to yield the impedance cardiogram. The R-wave DZ/dt data blocks are averaged over a default period of 60-s. The B-point is calculated as the time between the Q-wave onset and the DZ/dt upstroke in msec. Mean arterial pressure (MAP) indicates the average hemodynamic driving force through the cardiac cycle, and is affected by vascular sympathetic aadrenergic activity as well as by cardiac vagal and b-adrenergic input (Fox, 1999). Total Peripheral Resistance (TPR), which reflects relatively pure vascular sympathetic a-adrenergic influences, was derived from BP and cardiac output (CO), the latter being assessed with impedance cardiography (Sherwood et al., 1990).

# Procedure

For the first session, subjects arrived at the lab, signed informed consent, and completed a health screening form. The Institutional Review Board of Virginia Tech approved all experimental procedures. At the beginning of the second and third sessions, subjects noted any changes to this information. The ECG and ICG were recorded with six thoracic electrodes that were applied in accord with AMS configuration guidelines (AMS User Manual, 1998, v1.2. Vrije Universiteit, Amsterdam, the Netherlands). The blood pressure cuff was then applied to the upper arm of the subjects' non-dominant hand. The investigator gave all instructions to the subject while two research assistants operated recording equipment and assisted in task administration. Subjects engaged in the following 10 laboratory tasks per session, with task duration of three minutes<sup>1</sup> (see Table 1):

		J	
TASK (ABBREVIATION)	SESSION 1	SESSION 2	SESSION 3
Quiet rest (QR)	1. QR	1. QR	1. QR
Video game (VG)	2. VG	2. VG	2. VG
Facial cooling (FC)	3. FC	3. FC	3. FC
Video game/facial cooling (VG/FC)	4. HC	4. HG	4. OS
Orthostatic stress (OS)	5. SR	5. PB	5. SR
Hand cold pressor (HC)	6. OS	6. OS	6. HC
Supine rest (SR)	7. PB	7. SR	7. PB
Hand grip (HG)	8. HG	8. HC	8. HG
Paced breathing (PB)	9. VG/FC	9. VG/FC	9. VG/FC
Recovery (RC)	10. RC	10. RC	10. RC

 TABLE 1

 Task Abbreviations and Order of Tasks by Session

(1) Quiet rest (QR): The subject sat quietly and relaxed in a comfortable lounge chair with eyes closed. This task is characterized generally by cardiac vagal activity.

(2) *Video game (VG)*: The subject played a challenging race car video game with a cash bonus incentive based on overall performance in comparison with other subjects in the study. This task represents "active coping" that elicits predominantly sympathetic  $\beta$ -adrenergic activity and vagal withdrawal (Obrist, 1981).

(3) Facial cooling (FC): The subject, who sat quietly with eyes closed, wore a chilled gel facemask that covers the forehead and cheeks. Mask temperature was maintained between  $1-2^{\circ}$  C (mean temperature across subjects and sessions was  $1.2^{\circ}$  C), and the subject was told to remove the mask if at anytime it became uncomfortable (this never occurred). This procedure elicits a reflexive parasympathetic cardiac and sympathetic–a-adrenergic vascular response (Friedman et al., 1996).

(4) Hand cold pressor (HC): The subject placed the non-dominant hand up to the wrist in a bucket of iced water maintained between  $3-5^{\circ}$  C (mean temperature =  $3.9^{\circ}$  C). The BP cuff was placed on the opposite arm. The hand was removed from the water after 1.5 minutes, and then re-emerged after 10 seconds to allow a total immersion time of three minutes with minimal discomfort. This task is characterized primarily by sympathetic  $\alpha$ adrenergic activation (Saab et al., 1993).

(5) Supine rest (SR): The subject reclined fully in an armchair with eyes closed, eliciting parasympathetic activation and sympathetic withdrawal (Fox, 1999).

(6) Orthostatic stress (OR): The subject stood from a seated position and remained standing during the task, evoking sympathetic CV activation and vagal withdrawal (Pagani et al., 1995).

(7) *Paced breathing (PB)*: The subject breathed at a relatively slow pace set by a metronome, while sitting quietly with eyes closed (8 breaths per minute). The metronome rate was adjusted such that the respiration cycle was evenly divided at three beats for inspiration and three beats for expiration. This procedure elicits a cardiac vagal response (Malliani, Pagani, Lombardi, & Cerutti, 1999).

(8) *Hand grip (HG)*: The seated subject squeezed a hand dynamometer at one-third maximal grip strength, which was determined before the task began. This task evokes sympathetic CV activation and parasympathetic withdrawal (Pagani et al., 1995).

(9) Combined video game/facial cooling (VG/FC): The subject played the video game while wearing the chilled gel facemask (average temperature =  $3.6^{\circ}$  C), evoking sympathetic and vagal co-activation (Friedman et al., 1996).

(10) *Recovery (RC)*: The subject sat quietly in the lounge chair with eyes closed, in an identical manner to the QR task.

Task order was partially counterbalanced within-subjects and across sessions; certain tasks appeared in a fixed position across subjects and sessions for specific experimental reasons. First, each session began with QR, which served as a "baseline" period, and ended with RC. Tasks were then ordered to alternate sympathetic and parasympathetic dominance. By design, one task was chosen to evoke sympathovagal co-activation; this task (VG/FC) always came in position order 9. In order to allow for maximum temporal separation of VG/FC and its component tasks, while at the same time keeping sympatheticparasympathetic alternation, VG always appeared in the 2<sup>nd</sup> position order, followed by FC (OR, always  $1^{st}$ , is primarily vagal, hence the  $2^{nd}$  task must be sympathetic). The order of the remaining tasks (3-8) was counterbalanced across sessions. Table 1 shows the task order for the three sessions that resulted from these considerations. The purpose of selecting this diverse array of tasks was to simulate the range of autonomic variability that a CV system may encounter in everyday life, rather than to focus on CV responses in any one particular task. As such, counterbalancing did not have the same significance that it does in a typical repeated-measures study. On the contrary, as long as the sequences were consistent across individuals, the resultant CV profiles should reflect comparable idiodynamic patterns among the individuals.

Following completion of the last task, recording equipment was detached, and subjects were asked for any comments or questions from that day's session. Payment was made at the end of each session, and at the last session, subjects received bonuses for completing the study and for task performance incentives.

# **Results**

The overall data were first examined to determine the appropriateness of principal components analysis (PCA). The Kaiser-Meyer-Olkin measure of sampling adequacy was .65, and Bartlett's Test of Sphericity was 338.674, p < .001, indicating sufficient inter-item correlations for PCA. Further support for PCA was provided by the high communalities (range: 65'—.80), indicating that a substantial portion of variance in each variable was accounted for by the factors.

To generate the individual pattern matrices, CV variables were standardized within subjects and aggregated across experimental conditions and sessions, avoiding the confounding of within-person with between-condition and between-session variance (Dielman, 1983; Jaccard & Wan, 1993; Michela, 1990). This procedure resulted in means for eight physiological variables for each of the 10 conditions per subject. A promax rotation (kappa = 4) was chosen since oblique rotations are often more theoretically and empirically realistic than orthogonal rotations (Hair, Anderson, Tatham, & Black, 1998). The resultant loadings in the pattern matrices indicate the importance of that variable to the factor with the other variables partialed out (Hetzel, 1996).

Using the eigenvalues > 1 criterion, three subjects yielded a two-component solution (Table 2), and three subjects yielded a three-component solution (Table 3). Two-component solutions explained an average of 70.6% of total variance (range: 65.8-78.6%), and three-factor solutions explained an average of 82.8% total variance (range: 79.2-86.3%). Sequential increments in variance explained by these components differed among individuals. In two-component subjects, the first component accounted for a range of 46.7-60.0% variance (mean = 51.5%), and the second component explained between 18.6-

# IDIODYNAMIC PROFILES

SUBJECT 1						
	2 Componer Comp	t Solution 3 Component Solution onent Component		Maguaganaa		
	1	2	PR	Ι	С	tasks and sessions
HF Power (ms <sup>2</sup> ) HI (ohm/s*s) HR (bpm) LVET (ms) MAP (mmHg) PEP (ms) TPR (PRU) Eigenvalues: % of Variance Total variance explained: Correlation with Grand:	$\begin{array}{c} -0.03\\ 0.86\\ 0.49\\ 0.02\\ 0.95\\ -0.12\\ 0.94\\ 3.27\\ 46.66\\ 67.43\%\end{array}$	-0.66 -0.03 0.44 -0.73 -0.12 0.88 0.02 1.45 20.77	-0.09 <b>0.87</b> 0.43 0.12 <b>0.92</b> -0.11 <b>0.93</b> 0.92 13.16 80.59 0.913	-0.92 0.12 0.03 0.02 -0.17 0.76 0.06 3.27 46.66 0.464	0.18 -0.14 <b>0.57</b> - <b>0.98</b> 0.08 0.29 -0.01 1.45 20.77 0.498	5779.75 -8.52 86.17 281.49 96.81 117.91 0.0092
		SU	JBJECT 3			
	2 Componer Comp	nt Solution onent	3 Com	3 Component Solution Component		Maans across
	1	2	PR	Ι	С	tasks and sessions
HF Power (ms <sup>2</sup> ) HI (ohm/s*s) HR (bpm) LVET (ms) MAP (mmHg) PEP (ms) TPR (PRU) Eigenvalues: % of Variance Total variance explained: Correlation with Grand:	$\begin{array}{c} -0.86\\ -0.73\\ 0.76\\ -0.87\\ 0.37\\ 0.60\\ 0.4263\\ 3.31\\ 47.22\\ 65.79\%\end{array}$	$\begin{array}{c} 0.01\\ 0.03\\ -0.18\\ -0.03\\ 0.74\\ 0.34\\ -0.7802\\ 1.3\\ 18.57\end{array}$	0.11 -0.12 -0.04 -0.09 <b>0.75</b> 0.18 <b>-0.80</b> 0.79 11.24 77.03 0.027	-0.75 -0.86 -0.21 -0.16 0.20 0.87 0.18 3.31 47.22 0.595	-0.20 -0.06 <b>1.06</b> -0.83 0.28 -0.19 0.23 1.3 18.57 0.819	15977.45 -5.17 77.26 272.97 84.63 132.8 0.0075
		SU	JBJECT 5			
	2 Component Solution Component		3 Com	ponent Sc Componen	olution t	Means across
HF Power (ms <sup>2</sup> ) HI (ohm/s*s) HR (bpm) LVET (ms) MAP (mmHg) PEP (ms) TPR (PRU)	$-0.18 \\ -0.85 \\ -0.25 \\ -0.19 \\ 0.87 \\ 0.70 \\ 1.04$	-0.76 0.07 1.03 -0.71 -0.02 0.29 -0.18	-0.48 0.10 0.05 0.38 <b>0.72</b> 0.23 0.25	0.03 -0.91 -0.20 -0.55 0.49 0.67 0.99	-0.04 -0.04 1.02 -0.54 0.03 0.23 -0.26	14623.74 -9.48 83.95 271.69 86.74 121.55 0.0068
Eigenvalues: % of Variance Total variance explained: Correlation with Grand:	4.2 59.94 78.60%	1.31 18.68	0.67 9.59 88.21 0.492	4.19 59.94 0.607	1.31 18.68 0.951	

 TABLE 2

 Pattern Matrices for Two-component Subjects

PR = peripheral resistance, I = inotropic, C = chronotropic

#### FRIEDMAN AND SANTUCCI

		SUBJE	CT 2	*
	3 Co	mponent Solu	ition	
	Component			
	PR	Ι	С	Means across tasks and sessions
HF Power (ms <sup>2</sup> )	-0.16	-0.07	0.72	120966.55
HI (ohm/s*s)	0.51	-0.69	0.18	-5.86
HR (bpm)	0.75	-0.03	-0.39	60.14
LVET (ms)	0.09	0.01	0.89	312.86
MAP (mmHg)	0.94	-0.22	0.14	91.45
PFP (ms)	_0.31	0.92	-0.12	125.48
TPR (PRI)	0.89	0.07	-0.04	0.0084
Figenvalues:	2.81	1.31	1 42	0.0004
C of Variance	40.12	1.51	20.24	
Total variance	40.12	10.70	20.34	
Completion with Complete	/9.24	0.953	0.963	
Correlation with Grand:	0.889	0.852	0.862	
		SUBJE	CT 4	
	3 Co	mponent Solu	tion	
		Component		
	PR	Ι	С	Means across tasks and sessions
HF Power (ms <sup>2</sup> )	0.12	-0.07	-0.84	46807.64
HI (ohm/s*s)	0.23	-0.83	0.07	-10.2
HR (bpm)	-0.13	0.20	0.85	69.52
LVET (ms)	0.10	-0.82	-0.25	287.8
MAP (mmHg)	0.41	-0.24	0.75	86.27
PFP (ms)	0.03	1.03	-0.15	112.12
TPR (PRI)	0.05	0.14	_0.08	0.0127
Figenvalues.	1.00	3.69	1.23	0.0127
% of Variance	13.81	52.65	17.50	
	13.01	52.05	17.59	
Correlation with Grand	84.05 0.744	0.965	0.918	
	0.711	0.905	0.910	
		SUBJE	CT 6	
	3 Co	mponent Solu	tion	
	Component			
	PR	Ι	С	Means across tasks and sessions
HF Power (ms <sup>2</sup> )	-0.13	-0.17	-0.74	3244.82
HI (ohm/s*s)	-0.09	-0.63	0.55	-5.73
HR (bpm)	-0.13	-0.33	1.08	97.7
LVET (ms)	0.10	0.06	-0.97	230.96
MAP (mmHg)	0.98	-0.20	-0.13	88.51
PEP (ms)	-0.21	1.00	-0.31	133.63
TPR (PRU)	0.38	0.48	0.32	0.0123
Eigenvalues:	1.01	1.52	3 52	
% of Variance	14 42	21.68	50.23	
Total variance explained	86 33	21.00	50.25	
Correlation with Grand	0.845	0.741	0.867	
	0.045	0.741	0.007	

 TABLE 3

 Pattern Matrices for Three-component Subjects

PR = peripheral resistance, I = inotropic, C = chronotropic

Composite Pattern Matrix					
	COMPONENT				
	PR	Ι	С	Means Across Tasks and Sessions	
HF Power (ms <sup>2</sup> )	0.12	0.21	0.88	34566.66	
HI (ohm/s*s)	0.43	-0.59	0.03	-7.49	
HR (bpm)	0.25	0.11	-0.66	79.12	
LVET (ms)	0.09	-0.54	0.52	276.29	
MAP (mmHg)	0.92	-0.29	-0.06	88.99	
PEP (ms)	-0.17	0.99	0.14	123.91	
TPR (PRU)	0.79	0.16	0.13	0.0095	
Eigenvalues:	2.883	1.087	1.031		
% of Variance	41.18	15.53	14.73		
Total variance explained:	71.45%				

TADLE 4

PR = peripheral resistance, I = inotropic, C = chronotropic

20.8% variance (mean = 19.3%). Among three-component subjects, variance explained ranged from 40.1-52.7% by the first component (mean = 47.7%), 17.6-21.7% by the second component (mean = 19.9%), and 13.81-18.8% by the third component (mean = 15.7%).

Next, the standardized data were aggregated across subjects to generate the chain Ptechnique group composite pattern matrix. Examination of the composite component correlation matrix showed correlations that were > .32 (range: .35-.42), suggesting a 10% or greater overlap in variance among components, further justifying oblique rotation (Tabachnick & Fidell, 2001). This composite matrix was subjected to PCA, followed by a promax rotation (kappa = 4). The eigenvalues > 1 criterion yielded a three-component solution for the composite pattern matrix, accounting for 71.45% of the variance<sup>2</sup> (Table 4). Inspection of this matrix suggests a peripheral resistance (blood pressure) component, a flow-resistance (cardiac contractility) component, and a chronotropic (cardiac rate) component, replicating previous models of CV activation components (Stemmler, Grossman, Schmid, & Foerster, 1991; Stemmler, 1993).

Individual pattern matrices were then compared to the composite by calculating Pearson correlations between loadings for comparable components (Cliff, 1996; Guadagnoli & Velcier, 1991). In order to compare two-component subjects to the group, a three-component solution was forced on their data. Correlations between conceptually similar components for the individual and group matrices are displayed in Tables 2 and 3. To facilitate comparisons with the composite matrix, individual subject components are displayed to match the order of the variance explained by components in the composite matrix (peripheral resistance, inotropic, and chronotropic).

A standard of r > .75 has been suggested to infer similarity between individual and composite components in chain-P technique (Cliff, 1966). Across all subjects, the range of r's between the individual and group components were .02 -.91 for peripheral resistance (mean = .65), .46-.96 for inotropic (mean = .70), and .49-.95 for chronotropic (mean = .65), .46-.96.82). Three-component patterns more closely matched the composite; mean r's for two- vs. three-component subjects were, respectively, .48 vs. .82 for peripheral resistance, .56 vs. .85 for contractility, and .75 vs. .88 for chronotropic.

Finally, for descriptive purposes, mean physiological measures across tasks and sessions

	2 Compone	ent Subjects	3 Component Subjects		
	Mean	SE	Mean SE		
HF Power (ms <sup>2</sup> )	12126.98	1427.175	57006.33	8629.967	
HI (ohm/s*s)	-7.72	0.287	-7.26	0.255	
HR (bpm)	82.46	1.047	75.79	1.951	
LVET (ms)	275.38	1.719	277.21	4.144	
MAP (mmHg)	89.31	0.997	88.68	1.080	
PEP (ms)	124.08	1.312	123.74	1.339	
TPR (PRU)	0.008	0.001	0.011	0.002	

TABLE 5
Means and Standard Errors Across Tasks and Sessions for Two
vs. Three Component Subjects

are shown for individual subjects in Tables 2 and 3, and group means for two—and threecomponent subjects are shown in Table 5.

# Discussion

The primary aims of this study are exploratory, and these data are best cast in an "inductive-deductive upward spiral" model, in which systematic observations made in exploratory studies lead to testable hypotheses, which in turn spawn more inductive research (Cattell, 1988b). The design and analysis were selected to extract CV response patterns from the detailed study of a small group of subjects across multiple conditions and sessions. The results are examined here for potential hypotheses about CV reactivity, and the "three-norm" schema is applied to assess the degree of individuality or generality of the subjects' CV response patterns.

In this spirit, consideration begins with inspection of the individual pattern matrices. A distinction emerged between subjects who naturally yielded two-component versus threecomponent solution. As one would expect, variables that reflect diverse aspects of CV function tended to load on a single general component in the reduced solution. A clear example of this trend can be found in subject #5, whose loading pattern suggests a combined peripheral resistance/contractility factor and a rate factor. It is conceivable that such patterns imply less complex, more rigidly coupled CV dynamics and ANS regulation. Indeed, the mixing of contractility and flow variables implies a blurred distinction between sympathetic a—and b-adrenergic processes. In contrast, three-component subjects tended to show distinct rate, contractility, and resistance factors, which suggests increased sensitivity in CV responding. This pattern may indicate flexibility and resiliency, traits that marks healthy response systems in which outputs reflect multiple physiological inputs (Friedman & Thayer, 1998a,b; Friedman et al., 1996; Goldberger, 1991; Kaplan et al., 1991; Lipsitz & Goldberger, 1992; Mandell & Schlesinger, 1990).

This interpretation is amplified by the variance explained by components in two—vs. three-component subjects. The first general component in two-component subjects explained nearly half or more total variance (range for 1<sup>st</sup> component: 47–60%; mean = 50%). In contrast, explained variance was somewhat more evenly distributed in three-component subjects (range for 1<sup>st</sup> component: 40–50%; mean = 45%). The presence of a global factor reinforces the view of autonomic regulation as less versatile in two-component subjects.

One way to assess this CV "complexity" notion is see if the number of components predicts either mean resting or reactivity levels in standard CV tasks. The small number of subjects in the present study precludes deductive contrasts, but such tests would be feasible in a larger scale research program focused on individual reactivity. Still, if the gross limitations of such contrasts in the present study are acknowledged from the onset, a few suggestive group comparisons can be observed (Table 5). With data aggregated across all tasks and sessions, the two-component subjects had lower HF power and greater HR than three-component subjects. These differences imply reduced cardiac vagal control in the former, a quality associated with CV stress (Pagani et al., 1995), reduced CV complexity (Friedman et al., 1996; Friedman & Thayer, 1998a), and CV pathology (Malliani et al., 1991).

Hence, the number of components mapped directionally onto standard measures of cardiac rate and variability, suggesting a hypothesis for further testing: A reduced factor solution, obtained over diverse situations, repeated occasions, and a range of CV variables, is associated with low cardiac vagal control. Studies might further explore if these idiodynamic CV profiles predict "S-R" determined reactivity, as well as psychosocial variables thought related to CV health such as hostility and social support. Furthermore, it may be that in-depth CV profiles obtained in this manner—i.e., sampled broadly across situations, occasions, and variables—might better predict CV health in individuals than single-measure S-R reactivity indices.

Another way in which the matrices might prove informative is to examine which specific component explained the most variance for a particular subject. This component might reflect which aspect of CV regulation, and accompanying autonomic mechanisms, might predominate in that individual. For example, if more variance was explained by the chronotropic factor, cardiac rate control (vagal and  $\beta$ -adrenergic) dominates, and if peripheral resistance accounted for more variance, dominance,  $\alpha$ -adrenergic mechanisms prevail. The relationship between these components could be compared the "cardiac" vs. "vascular" reactor distinction made in the reactivity literature (Kamarck, Jennings, Pogue-Geile, & Manuck, 1994) by assessing how S-R reactivity in cardiac and vascular variables relates to variance explained in individual factor structures.

The next step in evaluating these CV profiles involves comparing individuals with the composite pattern. This stage bridges the idiodynamic, demographic, and nomothetic levels. The composite matrix (Table 4) showed three distinct components: one marked by high loadings for MAP and TPR; one with high loadings for PEP, LVET, and HI; and one with high loadings for HF, HR, and LVET. This matrix might be viewed as demographic, based on subject similarities (i.e., all were college-aged females). Yet, this group structure replicates established nomothetic data that show, respectively, an  $\alpha$ -adrenergic peripheral resistance, a  $\beta$ -adrenergic contractility, and a vagal-sympathetic chronotropic component in CV regulation (Stemmler et al., 1991; Stemmler, 1993).

However, individuals showed a wide range of variability from the composite structure. In order to make specific comparisons between individuals and the composite, it was necessary to "force" a three-component solution on two-component subjects. In some cases, this process made comparisons problematic, since inconsistencies in variable loadings led to difficulties in labeling the components. Still, this concern reinforces the good fit between "natural" three-component subjects and the composite pattern, as shown by high inter-component correlations (mean r = .84; range: .74 –.89). All correlations in these subjects nearly met or exceeded the previously cited figure of r > .75 to denote similarity between composite and individual components in chain-P technique (Cliff, 1966). Hence,

three-component subjects can be said to more closely resemble the nomothetic structure of CV regulation (Stemmler et al., 1991; Stemmler, 1993). This finding is consistent with the previous depiction of three components indicating healthy complexity in CV regulation.

In contrast, two-component subjects showed much lower correlations with the group structure (mean r = .66; range; .03 - .95). This deviation from the nomothetic norm further reinforces the alleged rigidity in CV regulation inferred from the natural two-component structure in this group. Thus, chain P-technique proved helpful in expanding upon information suggested by inspection of the individual (idiodynamic) matrices.

Beyond the drawbacks inherent in any study of a small number of subjects, certain limitations of these findings should be acknowledged. The present study was meant to serve as a prototype for future idiodynamic research on CV reactivity, and undoubtedly requires replication and extension with more individuals, diverse conditions, and occasions. The experimental parameters of six subjects, three occasions, and ten conditions were chosen to assess the feasibility of multi-occasion/multi-condition formats for future research. That is, it was uncertain how subject compliance would fare when one was required to return to the lab at three weekly intervals for extensive autonomic testing. One effect of this concern was to restrict the individual observation-to-variable ratios to less than what is optimal for stability in factor analysis (Hair et al., 1998). Our experience in the present study suggests that the number of conditions and sessions could be increased, and such research is planned for the future. This work will help assess the robustness of the present findings, allow for further investigation of the hypotheses generated herein, and expand the base upon which to build an idiodynamic foundation for CV research.

Another methodological point concerns the usage of LVET as an inotropic index. LVET tends to be correlated with HR, which is evident in this study in the significant loadings of LVET on the chronotropic component. Although LVET does show the correct directional relationship with HR when both indices load on this component, it also tends to load in the opposite direction of PEP when both load on the inotropic component. It should be noted that in the individual matrices, LVET generally loaded on the chrontropic rather than inotropic component, and the association of PEP and HI with the inotropic component was in the predicted direction. It may be that potential multicollinearity of LVET and HR precludes using both in component analyses of CV activity.

Another issue concerns relevance to the reactivity literature, which is focused on the relationship between psychological challenge and CV response. The present tasks were chosen on autonomic, rather than psychological, criteria to generate a broad range of ANS activity from which to sample. These tasks do in fact elicit affective responses (Santucci & Friedman, 2001), but were not selected on this basis. A future study could involve a similar design as the present, however using a broad range of affective or cognitive, rather than ANS, tasks.

The methodology of this study offers but a glimpse into the potential of an idiodynamic perspective on CV research; there are multitudes of other available research designs and statistical methods. For example, many forms of individual research (e.g., Garmezy, 1982; Kratochwill & Levin, 1993), temporally sensitive designs (Brown & Moskowitz, 1998), nonlinear dynamics methodologies (eg, Schmidt & Morfill, 1995; Thayer & Friedman, 1997), and other formats with multivariate systems perspectives (Thayer & Friedman, 2000) are consonant with idiodynamics. The present use of P-technique is merely a starting point to realize in-depth studies of CV regulation in individuals.

In sum, the present study suggests that the idiodynamic framework may have fruitful empirical applications to the study of CV activity in individuals. The synthesis of idiodynamics and psychophysiology aims for a systematic, multidimensional depiction of CV reactivity, the study of which has been dominated by traditional group designs ensconced in a one-dimensional view of reactivity as response magnitude. There is a much ground to cover in this quest, and it is hoped that the present research will encourage development in this direction.

## Acknowledgements

This research was supported by a grant from the College of Arts and Sciences at Virginia Tech. Portions of these data were presented at the annual meetings of the Societies for Psychophysiological Research (October, 1999) and Behavioral Medicine (April, 2000). The authors would like to thank Dr. Saul Rosenzweig for his insights and support, Israel Christie for technical and statistical assistance, and Erin Curtis, Lyn Balan, Elena Kyrgos, and Ben Pumphrey for their roles in data collection.

#### Notes

1. Subjects completed a brief 12-item emotion adjective questionnaire after each task to assess their affective responses, the results of which will be reported elsewhere.

2. In an oblique rotation, although both the pattern and structure matrices should be consulted, the obtained pattern matrix is generally preferred for interpretation. Similar components were found in both the pattern and structure matrices; however, the structure matrix did not have simple structure. HR, HI, and LVET all loaded highly (>.50, + or-) on two different components. Since the pattern matrix captures only the independent relationship between each variable and factor, it is a common finding that the pattern matrix is more likely to have simple structure, as was found with our data. Overall, both matrices generally support the finding of blood pressure, contractility, and rate components.

#### References

- Allen, M.T., Boquet, A.J., Shelley, K.S.(1991). Cluster analyses of cardiovascular responsivity to three laboratory stressors. *Psychosomatic Medicine*, 53, 272–288.
- Allport, G.W. (1937). Personality: A Psychological Interpretation. New York: Holt.
- Allport, G.W. (1961). Pattern and growth in personality. New York: Holt, Rinehart, & Winston.
- Allport G.W. (1962). The general and the unique in psychological science. *Journal of Personality*, 30, 405–22. Allport, G.W. (1966). Traits revisited. *American Psychologist*, 21, 1–10.
- Anastasi, A. (1958). *Differential psychology: individual and group differences in behavior* (3<sup>rd</sup> ed.). New York: Macmillan.
- Blascovitch, J., & Katkin, E.S., (Eds.) (1991). Cardiovascular Reactivity to Psychological Stress and Disease. Washington, DC: American Psychological Association.
- Brown, K.W., & Moskowitz, D.S. (1998). It's a function of time: a review of the process approach to behavioral medicine. *Annals of Behavioral Medicine*, 1998, 109–117.
- Carroll, D., Smith, G.D., Sheffield, D., Shipley, M.J., & Marmot, M.G. (1995). Pressor reactions to psychological stress and prediction of future blood pressure: data from the Whitehall II study. *British Medical Journal*, 310, 771–76.
- Cattell, R.B. (1988a). The data box: its ordering of total resources in terms of possible relational systems. In J.R. Nesselroade & R.B. Cattell (Eds.), *Handbook of multivariate experimental psychology* (2<sup>nd</sup> ed) (pp. 69–130). Chicago: Rand McNally.
- Cattell, R.B. (1988b). Psychological theory and scientific method. In J.R. Nesselroade & R.B. Cattell (Eds.), *Handbook of multivariate experimental psychology* (2<sup>nd</sup> ed) (pp. 3–20). Chicago: Rand McNally.

Cliff, N. (1966). Orthogonal rotation to congruence. Psychometrika, 31, 33-41.

- Danziger, K. (1990). Constructing the subject: historical origins of psychological research. Cambridge: Cambridge University Press.
- Denenberg, V.H. (1982). Comparative psychology and single-subject research. In D.W. Fiske (Series Ed.), A.E. Kazdin & A.H. Tuma (Vol. Eds.), New directions for methodology of social and behavioral sciences: single-case research designs (Vol. 13) (pp. 9–31). San Francisco: Jossey-Bass.

- Dielman, T.E. (1983). Pooled cross-sectional and time series data: a survey of current statistical methodology. American Statistician, 37, 111–22.
- Edwards, A.E., & Hill, R.A. (1967). The effect of data characteristics on theoretical conclusions concerning the physiology of emotions. *Psychosomatic Medicine*, 24, 303–310.
- Endicott, J. (1993). The return of the case report to psychosomatic medicine. Psychosomatic Medicine, 55, 11.
- Ewart, C.K., & Kolodner, K.B. (1992). Diminished pulse pressure response to psychological stress: early precursor of essential hypertension? *Psychosomatic Medicine*, *54*, 436–446.
- Eysenck, H.J. (1954). The science of personality: Nomothetic. Psychological Review, 61, 339–342.
- Fahrenberg, J. (1986). Psychophysiological individuality: a pattern analytic approach to personality research and psychosomatic medicine. Advances in Behavioral Research and Therapy, 8, 43–100.
- Foflick, M.J., Ahom, D.K., Gorkin, L., Nisura, R.S., Herd, J.A., Ewart, C., Schron, E.B., Kornfield, D.S., & Capone, R.J. (1990). Relation of psychosocial and stress reactivity variables to ventricular arrhythmias in Cardiac Arrhythmia Pilot Study (CAPS). *American Journal of Cardiology*, 86, 53–67.
- Fox, S.I. (1999). Human Physiology (6th ed). Dubuque, IA: WC Brown Publishers.
- Freeman, R. (1990). Autonomic nervous system. In J.H. Stein (Ed.), *Internal medicine* (3<sup>rd</sup> ed) (pp. 1894–1903). Boston: Little Brown & Co.
- Friedman, B.H., & Thayer, J.F. (1998a). Anxiety and autonomic flexibility: a cardiovascular approach. Biological Psychology, 49, 313–23.
- Friedman, B.H., & Thayer, J.F. (1998b). Autonomic balance revisited: panic anxiety and heart rate variability. Journal of Psychosomatic Research, 44,133–151.
- Friedman, B.H., Thayer, J.F., Borkovec, T.D., Tyrrell, R.A., Johnsen, B.H., & Colombo, R. (1993). Autonomic characteristics of nonclinical panic and blood phobia. *Biological Psychiatry*, 34, 298–310.
- Friedman, B.H., Thayer, J.F., & Tyrrell, R.A. (1996). Spectral characteristics of heart period variability during cold face stress and shock avoidance in normal subjects. *Clinical Autonomic Research*, 6, 47–152.
- Garmezy, N. (1982). The case for the single case in research. In D.W. Fiske (Series Ed,), A.E. Kazdin & A.H. Tuma (Vol. Eds.), New directions for methodology of social and behavioral sciences: single-case research designs (Vol. 13) (pp. 5–17). San Francisco: Jossey-Bass.
- Gerin, W., Rosofsky, M., Pieper, C., & Pickering, T.G. (1994). A test of generalizability of cardiovascular reactivity using a controlled ambulatory procedure. *Psychosomatic Medicine*, 56, 360–68.
- Goldberger, A.L. (1991). Is the normal heartbeat chaotic or homeostatic?-News in Physiological Science, 6, 87-91.
- Guadagnoli, E., & Velicer, W. (1991). A comparison of pattern matching indices. *Multivariate Behavioral Research*, 26, 323–343.
- Hair, J.F., Anderson, R.E., Tatham, R.L., & Black, W.C. (1998). *Multivariate data analysis* (5<sup>th</sup> ed). Upper Saddle River, NJ: Prentice Hall.
- Hetzel, R.D. (1996). A primer on factor analysis with comments on patterns of practice and reporting. In B. Thompson (Ed.), Advances in social science methodology (Vol. 4) (pp. 175–206). Greenwich, CT: JAI Press 1996.
- Jaccard, J., & Wan, C.K. (1993). Statistical analysis of temporal data with many observations: issues for behavioral medicine. *Annals of Behavioral Medicine*, 15, 41–50.
- Jennings, J.R., van der Molen, M.W., & Somsen, J.M. (1998). Changes in heart beat timing: reactivity, resetting, or perturbation? *Biological Psychology*, 47, 227–42.
- Jones, C.J., & Nesselroade, J.R. (1990). Multivariate, replicated, single-subject, repeated measures designs and P-technique factor analysis: a review of intraindividual change studies. *Experimental Aging Research*, 16, 171–183
- Julius, S., Jones, K., Schork, N., Johnsen, E., Krause, L., Nazzaro, P., & Zemva, A. (1991). Independence of pressure reactivity from pressure levels in Tecumseh, Michigan. '*Hypertension*, 17 (Supp. 3), H12-H21.
- Kamarck, T.W. (1992). Recent developments in the study of cardiovascular reactivity: contributions from psychometric theory and social psychology. *Psychophysiology*, *29*, 491–503.
- Kamarck, T.W., Jennings, J.R., Pogue-Geile, M., & Manuck, S.B. (1994). A multidimensional measurement model for cardiovascular reactivity: stability and cross-validation in two adult samples. *Health Psychology*, 13, 471–78.
- Kaplan, D., Furman, M.I., Pincus, S.M., Ryan, S.M., Lipsitz, L.A., Goldberger, A.L. (1991). Aging and the complexity of cardiovascular dynamics. *Biophysiological Journal*, 59, 945–49.
- Krantz, D.S., & Manuck, S.B. (1984). Acute psychophysiologic reactivity and risk of cardiovascular disease: a review and methodologic critique. *Psychological Bulletin*, 96, 435–64.
- Kratochwill, T.R., & Levin, J.R., (Eds.) (1992). Single-case research design and analysis: new directions for psychology and education. Hillsdale NJ: Lawrence Erlbaum Associates.

- Larsen, R.J., & Cutler, S.E. (1996). The complexity of individual emotional lives: a within-subject analysis of affect structure. *Journal of Social and Clinical Psychology*, 15, 206–230.
- Levenson, R.W. (1988). Emotion and the autonomic nervous system: a prospectus for research on autonomic specificity. In H.L. Wagner (Ed.), *Social psychophysiology and emotion: theory and clinical applications* (pp. 17–42). New York: John Wiley & Sons.
- Light, K.C. (2001). Hypertension and the reactivity hypothesis: the next generation. *Psychosomatic Medicine*, 63, 744–746.
- Linden, W., Gerin, W., & Davidson, K. (2003). Cardiovascular reactivity: Status quo and a research agenda for the new millennium. *Psychosomatic Medicine*, 65, 5–8.
- Lipsitz, L.A, & Goldberger, A.L. (1992). Loss of complexity and aging: potential applications of fractals and chaos theory to senescence. *Journal of the American Medical Association*, 267, 806–809.
- Luborsky, L., & Mintz, J. (1972). The contribution of P-technique to personality, psychotherapy, and psychosomatic research. In R.M. Dreger (Ed.), *Multivariate personality research: contributions to the understanding of personality in honor of Raymond B. Cattell* (pp. 387–410). Baton Rouge, LA: Claitor's Publishing Division.
- Magnusson, D., & Endler, N.S. (Eds.) (1977). Personality at the crossroads: current issues in interactional psychology. Hillsdale, NJ: Erlbaum.
- Malliani, A., Pagani, M., Lombardi, F., & Cerutti, S. (1991). Cardiovascular neural regulation explored in the frequency domain. *Circulation*, 84, 482–492.
- Malmo, R.B., & Shagass, C. (1949). Physiologic study of symptom neurosis in psychiatric patients under stress. *Psychosomatic Medicine*, 11, 25–29.
- Mandell, A.J., & Selz, K.A. (1992). Dynamical systems in psychiatry: now what? *Biological Psychiatry*, 31, 299–301.
- Mandell, A.J., & Schlesinger, M.F. (1990). Lost choices: parallelism and topological entropy decrements in neurobiological aging. In S. Krasner (Ed.), *The ubiquity of chaos* (pp. 35–46). Washington, DC: American Association for the Advancement of Science.
- Mandell, A.J., Stewart, K.D., & Russo, P.V. (1981). The Sunday syndrome: from kinetics to altered consciousness. *Federal Proceedings*, 40, 2693–2698.
- Manuck, S. (1994). Cardiovascular reactivity in cardiovascular disease: "Once more unto the breach." International Journal of Behavioral Medicine, 1, 4–31.
- Manuck, S.B., Kasprowicz, A.L., Monroe, S.M., Larkin, K.T., & Kaplan, J.R. (1989). Psychophysiologic reactivity as a dimension of individual differences. In N. Schneiderman, S.M. Weiss, & P.G. Kaufmann, (Eds.), *Handbook of research methods in cardiovascular behavioral medicine* (pp. 365–382). New York: Plenum Press.
- Manuck, S.B., Kasprowicz, A.L., & Muldoon, M.F. (1990). Behaviorally-evoked cardiovascular reactivity and hypertension: conceptual issues and potential associations. *Annals of Behavioral Medicine*, 12, 17–29.
- Michela, J.L. (1990). Within-person correlational design and analysis. In C. Hendrick & M.S. Clark (Eds.), *Research methods in personality and social psychology* (pp. 279–311). Newbury Park, CA: Sage Publications.
- Miller, J.G. (1978). Living systems. New York: McGraw-Hill.
- Miranda, J., & Borkovec, T.D. (1999). Reaffirming science in psychotherapy research. *Journal of Clinical Psychology*, 55, 191–200.
- Nesselroade, J.R., & Ford, D.H. (1987). Methodological considerations in modeling living systems. In M.E. Ford & D.H. Ford (Eds.), *Humans as self-constructing living systems: putting the framework to work* (pp. 47–79). Hillsdale, NJ: Lawrence Erlbaum Associates.
- Nesselroade, J.R., Pruchno, R., & Jacobs, A. (1986). Reliability and stability in the measurement of psychological states: an illustration with measures of anxiety. *Psychologische Beitrage*, 28, 255–264.
- Obrist, P.A. (1981). Cardiovascular psychophysiology. New York: Plenum Press.
- O'Connor, K. (1990). Towards a process paradigm in psychophysiology. *International Journal of Psychophysiology*, 9, 209–223.
- Ohman, A., & Magnusson, D. (1987). An interactional paradigm for research on pychopathology. In D. Magnusson & A. Ohman (Eds.), *Psychopathology: an interactional perspective* (pp. 3–21). New York: Academic Press.
- Pagani, M., Lucini, D., Rimoldi, O., Furlan. R., Piazza. S., & Biancardi, L. (1995). Effects of physical and mental exercise on heart rate variability. In M. Malik & A.J. Camm (Eds.), *Heart–rate variability* (pp. 245– 266). Armonk, NY: Futura Publishing Co.
- Peng, C.K., Buldyrev, S.V., Hausdorff, J.M., Havlin, S., Mietus, J.E., Simons, M., Stanley, H.E., &

Goldberger, A.L. (1994). Non-equilibrium dynamics as an indispensable characteristic of a healthy biological system. *Integrative Physiological and Behavioral Science*, *29*, 283–293.

- Pickering, T.G., & Gerin, W. Cardiovascular reactivity in the laboratory and the role of behavioral factors in hypertension: a critical review. *Annals of Behavioral Medicine*, *12*, 3–16.
- Rose, G. (1992). Strategies of prevention: the individual and the population. In M. Marmot & P. Elliot (Eds.), Coronary heart disease epidemiology (pp. 311–324). New York: Oxford University Press.
- Rosenzweig, S. (1950). Norms and the individual in the psychologist's perspective. In M.L. Reymert (Ed.), Feelings and emotions: the Mooseheart symposium (1<sup>st</sup> ed.)(pp. 327–335). New York: McGraw Hill.
- Rosenzweig, S. (1951). Idiodynamics in personality theory with special reference to projective methods. *Psychological Review*, 58, 213–223.
- Rosenzweig, S. (1956). Projective methods and psychometric criteria. *Australian Journal of Psychology*, 8, 152–155.
- Rosenzweig, S. (1958). The place of the individual and of idiodynamics in psychology: a dialogue. *Journal of Individual Psychology*, 14, 3–21.
- Rosenzweig, S. (1984). Idiodynamics. In R.J. Corsini (Ed.), Encyclopedia of psychology (2<sup>nd</sup> ed.) (pp. 205–208). New York: Wiley & Sons.
- Rosenzweig, S. (1986). Idiodynamics vis-a-vis psychology. American Psychologist, 41, 241-245.
- Rosenzweig, S. (1973). Human sexual autonomy as an evolutionary attainment, anticipating proceptive sex choice and idiodynamic bisexuality. In J. Zubin & J. Money (Eds.), *Contemporary sexual behavior: Critical issues in the 1970s*. Baltimore, MD: The Johns Hopkins University Press.
- Rosenzweig, S. (1978). Aggressive behavior and the Rosenzweig Picture-Frustration Study. New York: Praeger.
- Rosenzweig, S., & Fisher, S.L. (1997). "Idiographic" vis-a-vis "Idiodynamic" in the historical perspective of personality: remembering Gordon Allport, 1897–1997. *Journal of Historical Behavioral Science*, 33, 405–419.
- Rozanski, A., Blumenthal, J.A., & Kaplan, J. (1999). Impact of psychological factors on the pathogenesis of cardiovascular disease and implications for therapy. *Circulation*, 99, 2192–2217.
- Saab, P.G., Llabre, M.M., Hurwitz, B.E., Schneiderman, N., Wohlgemuth, W., Durel, L., Massie, C., & Nagel, J. (1993). The cold pressor test: vascular and myocardial responses patterns and their stability. *Psychophysiology*, 30, 366–373.
- Santucci A.K., & Friedman, B.H. (2001). Cardiovascular and affective responses to laboratory tasks [abstract]. Psychophysiology, 38 (Suppl. 1), S84.
- Sargent, F., & Weinman, K.P. (1966). Physiological individuality. Annals New York Academy of Sciences, 134, 696–719.
- Saul, J.P. (1990). Beat-to-beat variations of heart rate reflect modulation of cardiac autonomic outflow. News in Physiological Science, 5, 32–37.
- Schmidt, G., Morfill, G.E. (1995). Nonlinear methods for heart rate variability assessment. In M. Malik & A.J. Camm (Eds.), Heart rate variability (pp. 87–98). Armonk, NY: Futura Publishing Co.
- Sheffield, D., & Carroll, D. (1994). Social support and cardiovascular reactions to active laboratory stressors. *Psychology and Health*, 9, 305–316.
- Sherwood, A., Allen, M.T., Fahrenberg, J., Kelsey, R.M., Lovallo, W.R., & van Doornen, L.J.P. (1990). Methodological guidelines for impedance cardiography. *Psychophysiology*, 27, 1–23.
- Sherwood, A., & Turner, J.R. (1992). A conceptual and methodological overview of cardiovascular reactivity research. In J.R. Turner, A. Sherwood, & K. Light (Eds.), *Individual differences in cardiovascular re*sponses to stress (pp. 3–32). New York: Plenum Press.
- Stallone, D.D., & Stunkard, A.J. (1991). The regulation of body weight: evidence and clinical implications. Annals of Behavioral Medicine, 13, 220–230.
- Stemmler, G. (1993). Receptor antagonists as a tool for structural measurement in psychophysiology. *Neuropsychobiology*, 28, 47–53.
- Stemmler, G., Grossman, P., Schmid, H., & Foerster, F. (1991). A model of cardiovascular activation components. *Psychophysiology*, 28, 367–82.
- Tabachnick, B.G., & Fidell, L.S. (2001). Using multivariate statistics (4th ed). Boston: Allyn & Bacon.
- Thayer, J.F. (1998). New approaches to reactivity. Biological Psychology, 47, 225–226.
- Thayer, J.F., & Friedman, B.H. (1997). The heart of anxiety: nonlinear dynamics. In A. Vingerhoets A, F. van Bussel, & J. Boelhouwer (Eds.), *The (non) expression of emotion in* health *and disease* (pp. 39–49). Tilburg, The Netherlands: Tilburg University Press.
- Thayer, J.F., & Friedman, B.H. (2000). The design and analysis of experiments in engineering psychophysiol-

ogy. In R.W. Backs & W. Boucsein (Eds.), *Engineering psychophysiology: issues and applications* (pp. 59–78). Mahwah, NJ: Lawrence Erlbaum & Associates, Inc.

- Thoman, E. (1986). The time domain in individual subject research. In J. Valsiner (Ed.), The individual subject and scientific psychology (pp. 181–200). New York: Plenum Press.
- Turner, J.R., Sherwood, A., & Light, K. (1992). Individual differences in cardiovascular responses to stress. New York: Plenum Press.

Valsiner, J. (Ed.) (1986). The individual subject and scientific psychology. New York: Plenum Press.

- van Doornen, L.J.P., Snieder, H., & Boomsa, D.I. (1998). Serum lipids and cardiovascular reactivity to stress. *Biological Psychology*, 30, S10.
- Weiss, P. (1969). The living system: determinism stratified. In A. Koestler & J.R. Symthies (Eds.), *Beyond reductionism* (pp. 3–55). Boston: Beacon Press.
- Willemsen, G.H.M., De Geus E.J.C., Claver, C.H.A.M., Van Doornen, L.J.P., & Carroll, D. (1996). Ambulatory monitoring of the impedance cardiogram. *Psychophysiology*, 33, 184–93
- Zevon, M.A., & Tellegen, A. (1982). The structure of mood change: an idiographic/nomothetic analysis. Journal of Personality and Social Psychology, 43, 111–22.