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CARDIOVASCULAR AROUSAL IN INDIVIDUALS WITH AUTISM SPECTRUM DISORDER: AN IDIOGRAPHIC ANALYSIS

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CARDIOVASCULAR AROUSAL IN INDIVIDUALS WITH AUTISM

SPECTRUM DISORDER: AN IDIOGRAPHIC ANALYSIS

BY

DANIELLA MARIA AUBE

A THESIS SUBMITTED IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE OF MASTERS OF ARTS

IN

PSYCHOLOGY

UNIVERSITY OF RHODE ISLAND

MASTER OF ARTS THESIS

OF

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ABSTRACT

 Stress in individuals with autism spectrum disorder (ASD) is poorly understood, yet can be detrimental to the functioning of these individuals. Stress-related problems are more common in ASD than the typical population, and individuals with ASD often have poorer coping skills. It is crucial to understand stress responses in these individuals, to help them better learn, cope, and prevent problem behavior associated with stressful events and heightened arousal. However, traditional measures of stress (e.g. self-reports) are often unreliable in this population, due to communication deficits in ASD. Studying physiological responses is an alternative, potentially more accurate, way to study stress in ASD.

This idiographic study systematically examines heart rate (HR) responses to six stressors in 39 individuals with ASD. Patterns of response for each individual are discussed. Examples of four hypothesized physiological subtype responders were identified. These subtypes include: hyperarousal (characterized by high baseline HRs, with low variation in response to different stressors), hyporesponsive (characterized by low/normal baseline HR, with low variation in response to different stressors), reactive responsivity (characterized by HR that increases significantly throughout the assessment and fail to return to baseline level), and normal responsivity (characterized by normal baseline HR that varies during stressor phases, but returns to baseline level during subsequent baseline phases). Clinical and general implications of these findings are discussed, as well as directions for future research.

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Chapter 1. Overview of Stress, Anxiety, and ASD

 According to the American Psychiatric Association (*DSM-IV-TR*, 2000), autism spectrum disorder (ASD) encompasses a group of disorders, including autistic disorder, Asperger's syndrome, Pervasive Developmental Disorder not otherwise specified (PDD-NOS), Childhood Disintegrative Disorder, and Rett Syndrome. ASD is behaviorally defined and characterized by a broad constellation of symptoms (Eigsti & Shapiro, 2003), including: qualitative impairments in social interaction (i.e. eye contact, facial expression, body posture, emotional reciprocity, and gestures), communication (i.e. lack of or delays in spoken communication), and restrictive, repetitive, and stereotyped patterns of behavior (i.e. preoccupation with an interest that is abnormal in intensity or focus, routines, or stereotyped, and repetitive motor mannerisms), and these impairments are evident before or at 36 months (American Psychiatric Association, 2000). ASD affects an estimated 1/110 individuals (Department of Health & Human Services, 2009), and is the fastest growing developmental disability (California Health & Human Services, 2003). Because of its heterogeneous nature, and purely behavioral definition, ASD is likely to have multiple possible etiologies that are not fully understood (Eigsti & Shapiro, 2003). Studying ASD not only sheds a light on the disorder itself, but can also improve understanding of normal functioning and development (Cicchetti & Rogosch, 1996). *Comorbidity in ASD*

Individuals with ASD are more likely than the general population to have a range of comorbid diagnoses. Seventy-five percent of individuals with ASD also have mental retardation (MR), while 25% have intellectual abilities that range from low average to above average (Eigsti & Shapiro, 2003). These individuals are at higher risk for seizure

disorders (20-30% lifetime prevalence), which is even more likely for those with MR (Rapin, 1996). Individuals with Fragile X are also at greater risk (3-25% incidence) for ASD (Baileyet al. 1993). In addition, multiple studies have shown higher rates of stressrelated problems in ASD than the general population, including: anxiety (Bellini, 2004; Gillot, Furniss, & Walter, 2001; Gillot & Standen, 2007; Kim, Szatmari, Bryson, Streiner & Wilson, 2000; Muris, Steerneman, Merckelbach, Holdrinet, & Meesters, 1998), depression (Kim et al., 2000), and fears and phobias (Evans, Canavera, Kleinpeter, Maccubbin, & Taga, 2005; Knapp, Barrett, Groden & Groden, 1992; Matson & Love, 1990). Gillot & Standen (2007) note that compared to typical adults, adults with ASD have more difficulty coping with change, anticipation, sensory stimuli, and unpleasant events. Wood & Gadow (2010) suggest that stress may moderate ASD symptom severity (e.g. social skills deficits, and repetitive behaviors). Since individuals with ASD are likely to experience stress-related problems, it is crucial to understand how individuals with ASD experience stress. Due to the heterogeneity of ASD, and the likelihood of comorbid diagnoses, it is also necessary to acknowledge that stress experience varies by individual.

Stress and ASD

 According to Selye (1974), stress is the physiological reaction of the body to either positive or negative events, or stressors. Stressors are events that place a demand on an organism and require an organism to make an adjustment to maintain homeostasis (Lazarus & Folkman, 1984). Groden, Cautela, Prince, & Berryman (1994) propose that individuals with ASD are at greater risk for experiencing high stress levels, and respond to stressors differently than the typically developing population. This may be due to

social and communication deficits, as well as difficulty adapting to new situations. As many as 50% of individuals with ASD fail to develop spoken language (Bryson, Clark, & Smith, 1988) making it very difficult to communicate feelings of anxiety. Stress and anxiety can affect the cognitive, behavioral, and physiological responses of people with ASD (King, Hamilton & Ollendick, 1994). Therefore, it is crucial to understand how stress affects these individuals, in order to improve their quality of life, create targeted interventions and prevention programs, and better understand the nature of ASD. *Assessment of Stress in ASD*

Self-Reports. While self-reports are a commonly used tool to assess stress in typical populations, many individuals with ASD have communication deficits that make self-report measures unreliable (Hill, Berthoz, & Frith, 2004). Two alternatives for measurement are parental and caretaker ratings, and physiological measures.

The Stress Survey Schedule. The Stress Survey Schedule (SSS) developed by Groden & colleagues (2001) and validated by Goodwin & colleagues (2007), is completed by parents or caretakers, and measures stressors highly relevant to individuals with ASD. From this measure, eight domains of commonly experienced stressors for individuals with ASD were identified. These domains include: *Changes and Threats, Anticipation/Uncertainty, Unpleasant Events, Pleasant Events, Sensory/Personal Contact, Food-Related Activity, Social/Environmental Interactions, and Ritual-Related Stress*. Although parent/caretaker reports may be more accurate than self-report with this population, scores are based on overt behavior observations, which may not always adequately reflect an individual's true stress or arousal level.

Physiological Measures. Another alternative to stress measurement is physiological measurement. It has been suggested that passive, physiological measurement is especially appropriate to use with this population due to heterogeneity in ASD in regard to chronological age, developmental level, and linguistic and sensorimotor skills and capabilities, and potential behavioral/physiological dysynchrony (Berntson, Ronca, Tuber, Boysen, & Leland, 1985; Tuber, Ronca, Berntson, Boysen, & Leland, 1985). Autonomic nervous system (ANS) arousal is a good physiological indicator of one's stress level at rest and in the presence of different stimuli. If the system becomes aroused, changes in the cardiovascular system, immune system, endocrine glands, and brain regions involved in memory and emotion occur (Sapolsky, 1998). Cardiovascular activity (including HR) is a commonly measured ANS stress indicator (Andreassi, 2000). HR quickens to more intense stimulation and slows to less intense stimulation, which is presumed to be a defensive response to perceived danger (Lacey & Lacey, 1958). Kootz & Cohen (1981) suggested that a heightened ANS activity is indicated by high HR. Lower HR indicates focused attention, and blockade of external stimuli, also called an orienting response (Cohen, & Johnson, 1977). Romanczyk & Matthews (1998) proposed physiological state could be an antecedent to problem behavior often seen in ASD, and Freeman, Horner, & Reichle (1999) demonstrated HR changes before, during and after episodes of self-injury, aggression, and other problem behaviors in individuals with developmental disabilities.

The ANS

The ANS is comprised of two separate systems: the sympathetic nervous system (SNS) and the parasympathetic nervous system (PNS). SNS responses in the presence of

stressors include increased HR and respiration, pupil dilation, increased perspiration, inhibition of salivation and digestion, increased respiration, blood pressure increase, inhibition of reproductive organs, and adrenaline discharging into the system. Once the perceived threat has passed, the PNS constricts the pupils, stimulates salivation, decreases HR, slows respiration, stimulates digestive activity, and stimulates reproductive organs enabling a return to a homeostatic state (Sapolsky, 2002). Gellhorn (1957) suggested that trying to maintain balance between the SNS and PNS activates either system, but also excitation in one system may result in activation of the complementary one. He suggests that PNS activity could directly relate to the specific intensity, frequency, and duration of the preceding SNS stimulation. This is called the "principle of reciprocity" and involves maintaining neurochemical homeostasis between dynamic branches of the ANS.

ANS Dysfunction in ASD

Rubin (1962) suggests that individuals with ASD may have deficiencies in regulation between the two ANS branches. Compared to children with ASD, typicallydeveloping children were found to have significantly greater capacity for SNS activity, greater reactivity to their environment (specifically to changes in stimulation), and greater capacity to inhibit this reactivity and return to a state of homeostasis. Hirstein, Iverson, and Ramachandran (2001) suggested that the ANS in individuals with ASD cannot regulate itself appropriately, and requires additional behaviors for regulation (i.e. selfinjurious and stereotyped behavior). Porges (1976) suggests that studying this autonomic imbalance using physiological measures in ASD, early in a child with ASD's development, may facilitate positive and successful intervention.

ANS Research in ASD

Some studies have examined ANS responses of people with ASD to one particular stimulus assumed to be stressful. These stimuli include: auditory stimuli (Palkovitz & Wiesenfeld, 1980; Stevens & Gruelier, 1984; Tuber et al., 1985; van England, 1984; Zahn, Rumsey & Van Kammen, 1987); visual stimuli (Althaus, Mulder, Mulder, Aarnoudse & Minderaa, 1999; Kootz & Cohen, 1981; Hirstein et al., 2001; James & Barry, 1980; Sigman, Dissanayake, Corona & Espinosa, 2003; Tuber et al., 1985); somatosensory stimuli (Berntson et al., 1985; Tuber et al., 1985); social tasks (Jansen, Gispen-de Wied, van der Gaag & van Engeland, 2003; Jansen, Gispen-de Wied, Wiegant, Westenberg, Lahuis, & van Engeland, 2006; Kootz & Cohen, 1981; Sigman, et al., 2003); experimenter distress (Corona, Dissanayake, Arbelle, Wellington & Sigman, 1998); mental tasks (Toichi & Kamio, 2003); attentional tasks (Cohen & Johnson, 1977); and environmental load (Graveling & Brooke, 1978). Previous research suggests a variety of physiological stress patterns exist in ASD. For instance, some studies find general hyperarousal in the presence of stressors in ASD compared to the typical population (Cohen & Johnson, 1977; Goodwin, Groden, Velicer, Lipsitt, Baron, Hofmann & Groden, 2006; James & Barry, 1980; Kootz & Cohen, 1981; Stevens & Gruzelier, 1984; Zahn et al., 1987), others find hyporesponsivity (Graveling & Brooke, 1978; Palkovitz & Wiesenfeld, 1980), some find both (Hirstein et al., 2001), others still find no differences (Sigman et al., 2003; van England, 1984). Some also found slower habituation (or ability to differentiate between novel and previously presented stimuli, as evidenced by decreased physiological reactivity across multiple stimulus presentations) in individuals with ASD (Cohen & Johnson, 1977; James & Barry, 1980; Stevens &

Gruzelier, 1984). James and Barry (1980) noted that this finding is suggestive of an immature physiological system in ASD; namely, physiological responses of individuals with ASD were more similar to very young children without ASD than individuals of the same chronological age without ASD. Baranek (2002) noted sensory processing abilities in ASD appear uneven and fluctuating, and one may see hyper- and hypo-responses in the same child. These behavioral response patterns are reflective of poor arousal modulation in the central nervous system.

Responsivity to Stressors in ASD

Difficulty in modulation, or hyper-/hyporesponsivity to stimuli can lead to a range of problems in ASD. Hyperresponsivity (responding inappropriately with high arousal levels to innocuous stimuli) or hyperarousal (being in a chronically high state of heightened arousal) may lead to behavior problems such as self-injury and aggression, which interferes with learning and attention, and may require pharmacological intervention (King, 2000). Hyporesponsivity may make an individual appear to be lethargic or unfocused, and could also interfere with learning. Understanding how people with ASD experience stress is integral to improving their quality of life. If stress responses are better understood in this population, it may be possible to help these individuals better deal with stress, so that they are able to better focus their attention, learn, and reduce problem behaviors and the likelihood of developing other diagnoses (such as anxiety, and mood disorders).

Physiological Subtypes in ASD

Findings from a few physiological studies suggest subgroups exist in ASD. Cohen and Johnson (1977) identified three subgroups. One small subgroup had normal

HRs for their age with little change throughout the assessment. A second had rapid HRs which decreased during rest phases. However, the majority of their sample had tachycardias (high HRs between 110-150 bpm) that seemed unrelated to environmental demands. Hirstein et al., (2001) identified two subgroups. One had high electrodermal activity that could be reduced by sensory activity, and a larger range of skin conductance responses when compared to other groups (suggestive of hyperarousal). The other subgroup had no to few skin conductance responses produced only by extreme activities (i.e. self-injurious behavior, etc.) (suggestive of hyporesponsivity). Others have found greater within group differences among individuals with ASD than between group differences in ASD compared to a control group (Berntson et al., 1985; Kootz & Cohen, 1981). These findings suggest that it may be more appropriate to analyze physiological data in ASD idiographically, rather than nomothetically. Baranek (2002) suggested that identifying specific individual physiological patterns that differentiate responder types would be very useful when planning interventions in ASD.

Limitations of Prior Research

While informative, previous physiological studies in ASD consist of small samples and vary widely in their use of physiological measures and experimental stimuli, making it difficult to generalize findings. Experimental stimuli also usually consisted of one or few potential stressors. Older instruments used to measure physiology often required that the participant must restrict movement (Kootz & Cohen, 1981; Stevens & Gruzelier, 1984), which would most likely prove quite difficult for the majority of participants with ASD, resulting in error. Many studies were also published before publication of the DSM-III, potentially resulting in non-ASD individuals being included

in their samples. Most studies use group-level analyses, which can wash out effects (i.e. high and low responders will be averaged together, and look "normal"). The current study attempts to overcome these limitations by analyzing data from a larger sample of individuals with ASD ($N=39$) assessed on one physiological measure (HR) while exposed to a standardized variety of potential stressors. Use of time series analysis (TSA) at the idiographic (individual) level can provide detailed data on individual stressresponse patterns.

An Idiographic Analysis of HR in ASD

No studies to the author's knowledge look at multiple stimuli with many replications across many individuals, but one study examines cardiovascular responses to a variety of potential stressors identified by the SSS, in a small sample of individuals with ASD and a typically-developing age-/sex-matched control group (Goodwin et al., 2006). They found that individuals with ASD have higher baseline HR and less HR variability to different stressors than the control group. Also, individuals in the control group had more significant responses to different stressors than the individuals with ASD. This indicates that some people with ASD may be in a constant state of cardiac over-arousal, and may experience high levels of stress on a more continuous basis than those in the typical population. Another explanation could be that the individuals with ASD in this study were a subset of individuals who exhibit hyperarousal, and that other response patterns exist in ASD.

This Investigation

The present study, a secondary data analysis, replicates and extends the Groden et al. (2005) and the Goodwin et al. (2006) studies by examining clinical HR assessment

data from 39 students enrolled at the Groden Center Day School, Providence, RI; a program serving the academic and behavioral needs of children with developmental disabilities. These assessments contributed to a functional behavior assessment by identifying stressors that may serve as antecedents to problem behavior for each of the 39 participants. These assessments provide 39 replications, more than the typical 5 or 6 replications typically recommended in single-case design research (Barlow & Hersen, 1982).

This study will:

- 1) Explore variation in individual HR responses to stressors specifically by number, type, and combination of significant responses.
- 2) Examine individual patterns of responses reflective of four predicted subtypes: hyperarousal- high baseline HR and low variation in response across stressors; hyporesponsive- low/normal baseline HR and low variation in response across stressors; reactive responsive- HR increases throughout the assessment, and fails to return to baseline level; and normal responsivenormal baseline HR with some variation in HR during stressor phases, but HR returns to baseline level during subsequent baseline phases.

Chapter 2. Method

Participants

Participants included 39 former and current clients (males=33, females=6) from the Groden Center Day School. Written consent from guardians of each participant was obtained to collect these data. Participants ranged in age from 3 years 2 months to 19 years 11 months (*m*= 11 years 10 months, median= 12 years 7 months) (See Figure 1 for frequency of participants in each age range). Only participants who had a primary or secondary diagnosis of ASD made by a licensed psychologist familiar with the DSM-IV were included in this study. Thirty-six participants (92%) had a primary diagnosis of ASD, and 3 (8%) had a secondary diagnosis of ASD (See Table 1). All participants with available blood pressure data were normotensive (<90 mmHg diastolic blood pressure) (See Table 2 for more participant characteristics). Overall, 16 participants (41%) were verbal, 17 (44%) were non-verbal, and 6 (15%) had limited verbal ability, determined by a speech-language pathologist. Level of functioning was measured by the Vineland Adaptive Behavior Scales-Expanded Interview Form (VABS) or the Vineland Adaptive Behavior Scales-Expanded Interview Form-Second Edition (VABSII), depending on which scale was the most recent when the participant was assessed. These scales assess adaptive behavior of individuals with disabilities on the behavior domains of: Communication, Daily Living Skills (DLS), and Socialization in preparation for educational programming (for more on these scales, see Sparrow, & Cicchetti, 1989). Thirty participants were assessed with the VABS, six were assessed with the VABSII, and three had missing data (alternate measures are reported when available). VABSII scores are expressed as range scores, so the mean was taken for each individual's domain

scores in order to be comparable with the VABS. Mean level of functioning for Communication was 2 years 8 months, for DLS it was 3 years 5 months, and for Socialization it was 2 years 2 months. Twenty participants were on at least one medication at the time of the assessment (not all of which affected ANS arousal), 10 were on none, and data were not available for nine. No participants had low baseline HRs, and 6 had baselines that were high for their age (Participants 4, 7, 11, 25, 33, and 36), and three of these participants only had slightly above average heart rates (Participants 25, 33, and 36). Of these six participants, only Participants 7 and 11 were on medications that could have raised their heart rate. Ways to compensate for this will be discussed further. *Multicultural Representation*

Participants include individuals with ASD, as the goal is to see individual patterns of cardiovascular response to a variety of potentially stressful stimuli. According to the American Psychological Association (2000), males are four to five times more likely than females to have ASD. This accounts for more males $(n=33)$ being included in this study than females ($n=6$). Seventy-nine percent of participants were Caucasian ($n=31$), and 21% participants were racial/ethnic minorities (African American (n=3), Latino (n=4), and Asian American (n=1)) (See Figure 2).

Setting

Assessments took place in a sound-attenuated laboratory room with plain white walls, low incandescent lighting, a neutral-colored carpet, and a one-way mirror (to allow discrete viewing from an adjacent observation room). The glass was covered by a blind, so that participants were not distracted by their reflections.

Instruments

Cardiovascular responses were recorded using the *Lifeshirt* (Vivometrics, Inc.). This non-invasive vest telemetrically recorded HR, respiration, electrocardiograph (ECG) data, and motor movement. Data were continuously stored (i.e., beat-to-beat) on a portable battery-powered electronic recorder worn on the body. Motor movement and posture changes were recorded by a dual-axis accelerometer inside of the *Lifeshirt* positioned on the anterior surface of the ribcage. Movement data were collected to control for HR changes due to increased physical demands. See Wilhelm, Roth & Sackner (2003) and Heilman & Porges (2007) for a more complete description of this system, including reliability and validity data. Groden et al. (2005) found that individuals with ASD could tolerate the *Lifeshirt* system well. Data were collected and transferred onto a personal computer, were exported into Excel, and were later analyzed in SAS. *Materials*

A familiar staff was present during the assessments and was given a sheet listing the phases. A vacuum, remote control car, edible, two small dish towels, and stationary bike were used during the phases of the assessments. Researchers recorded start and end times of each phase using a data sheet and stopwatch in the adjoining room to the laboratory.

All assessments were videotaped using a discrete camera mounted in the upper corner of the lab room. A cushioned chair was provided for participants. Across the room was another chair for the familiar staff. There was a rectangular table pushed against the wall during most of the assessment (except for "*Difficult Task*," which required the table to be moved between the participant and staff).

Original Data Collection Procedure

 The current study is a secondary data analysis of HR data that were collected as part of a clinical assessment that is a regular part of the intake assessment at the Groden Center. The intake assessment is a two month period where students become acclimated to the center. HR analysis became a regular part of this assessment to integrate HR data into a functional analysis of behavior; to provide a physiological and behavioral baseline for evaluating program interventions over time; and to increase understanding of individual differences in individuals with autism.

 Most participants had been assessed during their first two months at the Center (n=24, 62%). In some cases, assessments were delayed by unavailability of equipment. The majority of assessments were done within the participants' first year at the Center (n=33, 85%). Only two participants (5%) had their assessment between one year to one year and six months at the Center, two (5%) had it between two to four years at the Center, and 2 (5%) had it after 10 years at the Center (See Table 2).

Before the assessments, all participants went through a familiarization period. This served to increase comfort level related to the lab room and the *Lifeshirt* before participants had their assessments, and also to control for the novelty of these factors accounting for HR changes. Accompanied by a familiar staff, all participants were introduced to the lab room as well as the *Lifeshirt*, at least one time before their assessment. Number of visits varied depending on the needs of the participant. Once the participant had at least one exposure, and appeared to grow comfortable with the room and *Lifeshirt*, they underwent the full HR assessment.

HR Assessment Description

For the HR assessment, the client entered the now familiar lab room with a familiar staff. A researcher put on the *Lifeshirt*, and connected three adhesive electrodes to the participant (on the left and right upper part of the chest, and on the left side of the torso, below the ribcage). The client was then seated in a chair across the room from their staff. Participants were instructed to sit quietly, at which point an initial baseline phase began. This phase served as a comparison to subsequent phases (stressors and rests) during statistical analyses.

The assessment consisted of 14 phases and followed an

A1BA2CA3DA4EA5FA6GA7H design, where A represented a baseline phase, B through G represented stressor phases, and H was an additional phase described below (See Figure 3 for a detailed description of each phase). A fixed order was used across all assessments to maximize comparability of exposures. Six stressor phases were adapted from five domains of the SSS to be examined empirically. These domains included: *Sensory/Personal Contact*, *Anticipation/Uncertainty*, *Pleasant Event*, *Changes/Threats*, and *Unpleasant Event*. *Sensory/Personal Contact* was represented by the *Loud Noise* phase of the assessment. *Anticipation/Uncertainty* was represented by the *Remote Control Robot*, and *Unstructured Time* phases. *Pleasant Event* was represented by the *Eating a Preferred Food* phase. *Changes/Threats* was represented by the *Difficult Task* phase. *Unpleasant Event* was represented by the *Change in Staff* phase. Two additional phases were included: *Physical Exertion* and *Transition*. *Physical Exertion* was included to show participants' HRs could increase. *Transition* was an artifact of the study design,

and consisted of a pool of all time in-between stressor and baseline phases. This HR assessment was well tolerated in individuals with ASD (Groden et al, 2005).

Each phase was two minutes long, with the exception of the initial baseline, which was five minutes long. The first three minutes of the initial baseline were not included in the analyses to allow participants time at the beginning of the assessment to grow acclimated with the environment. Once these data were discarded, all phases were equal in length allowing for TSA to be performed. While the length of phases was standard for 21 participants, 18 participants had shortened phases presented in the same order (after confirming that HR responsivity was not statistically significantly different between two minutes and one minute). For these participants, the initial baseline was two minutes, while each subsequent baseline and stressor phase was one minute. This shortened assessment was given to very young participants, or when a familiar staff requested this assessment due to special behavioral concerns for the participant. One participant (29) required *Unstructured Time* phase to occur later in the assessment than typical for safety reasons.

Analyses

Data analyses consisted of 39 separate univariate interrupted time series analyses (Crosbie, 1993; Glass, Willson, & Gottman, 1975; Velicer & Colby, 1997; Velicer & Fava, 2003) performed on each participant for the dependent variable HR. Time series analysis can model change over time and requires a large number of observations at equally spaced intervals. In TSA, sample size is the number of observations over time rather than the number of subjects. Each full-length HR assessment generated over 3,000 data points per participant. However, since all data were taken from a single participant,

there is serial dependency in the data. Group level analyses assume that data are independent. Therefore, for the present study, traditional group-level analyses are inappropriate unless the data are transformed to be independent. TSA addresses the issues of dependency in the data by determining the degree of autocorrelation that transforms the data to be independent. After this transformation dependency is removed and standard general linear model procedures can be employed.

Time series is a regression-based technique that uses an autoregressive integrated moving average (ARIMA) models of the order (p, d, q) to model the serial dependence of the data. The *p* represents the autoregressive term that shows the degree to which the data are dependent on previous observations. The *d* term represents the number of times a series has to be differenced in order to make it stationary. The *q* represents the moving average term that describes the persistence of a previous shock to the system (Box & Jenkins, 1970).

Velicer & Harrop (1983) caution that the correct ARIMA model underlying a time series is difficult to determine. Therefore, this study employs the General Transformation Approach (Velicer & McDonald, 1984). This approach uses an ARIMA (5, 0, 0) model for all TSA and has been shown to adequately approximate most commonly encountered time series analyses in the behavioral sciences (Velicer & McDonald, 1984). Missing data were handled using the maximum likelihood procedure, which has been identified to best approximate missing data, when compared to other procedures, with up to 40% of data missing (Velicer & Colby, 2005).

For the present study, PROC ARIMA was used in SAS. The dependent variable was HR. Shape, level, and variability were examined. T-tests were done on all data to

test for significant differences between stressor phases and the initial baseline phase. Results for significant phases are reported as a change in level (stressor phase mean HRbaseline mean HR=change in level).

 Power Calculation. Based on Goodwin et al. (2006) pilot studies, power calculations were the same for each test of intervention (stressor) effects. There are approximately 480 observations in the original baseline phase (A1) and approximately 192 observations in the intervention phases (B, C, D, E, F, G and H). If HR level is assumed to be 85 at baseline with a standard deviation of 3.5 and a change in HR of 5 for the intervention, power is .99, since the effect size was found to be large (eta-squared= .338).

Chapter 3. Results

Individual Results

Participant 1 had a mean baseline HR of 91.75 (*sd*=17.55). He had HR responses significantly higher than baseline for all stressor phases (*Loud Noise* (change in level= 43.41, *t*(2400)= 5.90, *p*<.05); robot (change in level= 18.90, *t*(2400)= 3.52, *p*<.05); *Unstructured Time* (change in level= 5.49, *t*(2400)= 2.75, *p*<.05); *Eating a Preferred Food* (change in level= 15.12, *t*(2400)= 3.80, *p*<.05); *Difficult Task* (change in level= 9.46, *t*(2400)= 3.42, *p*<.05); and *Change in Staff* (change in level= 10.41, *t*(2400)= 3.77, *p*<.05)) and also for *Physical Exertion* (change in level= 21.88, *t*(2400)= 3.94, *p*<.05), and *Transition* (change in level= 15.36, *t*(2400)= 3.73, *p*<.05) phases. Autocorrelations were 0.92 for lag 1, 0.86 for lag 2, 0.83 for lag 3, 0.80 for lag 4 and 0.74 for lag 5 (See Figures 4-42 for all participants' HR graphs, and Table 3 for HR for all participants).

Participant 2 had a mean baseline HR of 65.83 (*sd*= 5.36). He had HR responses significantly higher than baseline for two stressor phases (*Eating a Preferred Food* (change in level= 8.93, *t*(2347)= 2.20, *p*<.05) and *Difficult Task* (change in level= 11.20, *t*(2347)= 2.16, *p*<.05)) and also for *Physical Exertion* (change in level= 43.89, *t*(2347)= 7.49, *p*<.05) and *Transition* (change in level= 10.56, *t*(2347)= 2.94, *p*<.05) phases. Autocorrelations were 0.98 for lag 1, 0.96 for lag 2, 0.95 for lag 3, .92 for lag 4 and .90 for lag 5.

Participant 3 had a mean baseline HR of 105.10 (*sd*=9.10). He had no HR responses significantly different than baseline, although his HR did elevate slightly (though not statistically significant) during the *Physical Exertion* phase. Autocorrelations were 0.85 for lag 1, 0.76 for lag 2, 0.68 for lag 3, 0.63 for lag 4 and 0.58 for lag 5.

Participant 4 had a mean baseline HR of 115.35 (*sd*= 6.65). He had no HR responses significantly different than baseline, although his HR did elevate slightly (though not statistically significant) during the *Physical Exertion* phase. Autocorrelations were 0.95 for lag 1, 0.91 for lag 2, 0.89 for lag 3, 0.86 for lag 4 and 0.83 for lag 5.

Participant 5 had a mean baseline HR of 98.30 (*sd*= 13.70). He had a HR response significantly different than baseline for one stressor phase (*Unstructured Time* (change in level= 4.14 , $t(3564)=2.17$, $p<.05$)) and *Physical Exertion* (change in level= 25.07, *t*(3564)= 2.04, *p*<.05). Autocorrelations were 0.94 for lag 1, 0.91 for lag 2, 0.90 for lag 3, 0.89 for lag 4 and 0.86 for lag 5.

Participant 6 had a mean baseline HR of 99.00 (*sd*= 6.30). He had a HR response significantly different than baseline for all stressor phases (*Loud Noise* (change in level=, *t*(3707)= t, *p*<.05 *m*= 102.80, *sd*= 7.0); *Remote Control Robot* (change in level= 2.70, *t*(3707)= 2.68, *p*<.05); *Unstructured Time* (change in level= 10.00, *t*(3707)= 2.12, *p*<.05); *Eating a Preferred Food* (change in level= 6.20, *t*(3707)= 3.54, *p*<.05); *Difficult Task* (change in level= 8.80, *t*(3707)= 3.66, *p*<.05); and *Change in Staff* (change in level= 4.90, *t*(3707)= 2.86, *p*<.05)) and *Physical Exertion* (change in level=, *t*(3707)= t, *p*<.05 *m*= 133.50, *sd*= 27.70) and *Transition* (change in level=, *t*(3707)= t, *p*<.05 *m*= 123.40, *sd*= 22.40). Autocorrelations were 0.97 for lag 1, 0.94 for lag 2, 0.91 for lag 3, 0.90 for lag 4 and 0.88 for lag 5.

Participant 7 had a mean baseline HR of 121.09 (*sd*= 4.55). He had a HR response significantly different than baseline for *Physical Exertion* only (change in level= 10.24, *t*(1699)= -2.16, *p*<.05). Autocorrelations were 0.97 for lag 1, 0.93 for lag 2, 0.89 for lag 3, 0.85 for lag 4 and 0.80 for lag 5.

Participant 8 had a mean baseline HR of 98.66 (*sd*= 4.01). He had a HR response significantly different than baseline for two stressor phases (*Remote Control Robot* (change in level= 1.38, *t*(2612)= 2.04, *p*<.05); and *Difficult Task* (change in level= -2.85, *t*(2612)= -2.41, *p*<.05)) and *Physical Exertion* (change in level= 7.63, *t*(2612)= 4.93, *p*<.05). Autocorrelations were 0.86 for lag 1, 0.72 for lag 2, 0.65 for lag 3, 0.60 for lag 4 and 0.57 for lag 5.

Participant 9 had a mean baseline HR of 117.25 (*sd*= 7.12). He had a HR response significantly different than baseline for two stressor phases (*Remote Control Robot* (change in level= -4.98, *t*(1546)= -2.61, *p*<.05); and *Change in Staff* (change in level= 6.46, *t*(1546)= 1.97, *p*<.05)) and *Physical Exertion* (change in level= 10.06, *t*(1546)= 2.68, *p*<.05). Autocorrelations were 0.85 for lag 1, 0.70 for lag 2, 0.56 for lag 3, 0.48 for lag 4 and 0.43 for lag 5.

Participant 10 had a mean baseline HR of 102.50 (*sd*= 6.10). He had a HR response significantly different than baseline for three stressor phases (*Loud Noise* (change in level= 12.58, *t*(1981)= 7.61, *p*<.05); *Remote Control Robot* (change in level= - 0.14, *t*(1981)= 2.17, *p*<.05); *Eating a Preferred Food* (change in level= 0.76, *t*(1981)= 3.03, *p*<.05)) and *Transition* (change in level= 2.51, *t*(1981)= 2.34, *p*<.05). Autocorrelations were 0.94 for lag 1, 0.90 for lag 2, 0.86 for lag 3, 0.83 for lag 4 and 0.80 for lag 5.

Participant 11 had a mean baseline HR of 105.04 (*sd*= 4.96). She had a HR response significantly different than baseline for 7 stressor phases (*Loud Noise* (change in level= 2.14, *t*(3785)= 2.02, *p*<.05); *Unstructured Time* (change in level= 2.02, *t*(3785)= 3.16, *p*<.05); *Eating a Preferred Food* (change in level= 8.30, *t*(3785)= 4.11, *p*<.05);

Difficult Task (change in level= 5.86, *t*(3785)= 4.01, *p*<.05); and *Change in Staff* (change in level= -1.86, *t*(3785)= 2.08, *p*<.05)) and *Physical Exertion* (change in level= 24.25, *t*(3785)= 9.43, *p*<.05) and *Transition* (change in level= 3.20, *t*(3785)= 4.25, *p*<.05). Autocorrelations were 0.94 for lag 1, 0.88 for lag 2, 0.83 for lag 3, 0.82 for lag 4 and 0.79 for lag 5.

Participant 12 had a mean baseline HR of 84.42 (*sd*= 6.45). He had a HR response significantly different than baseline for three stressor phases (*Unstructured Time* (change in level= 2.80, *t*(2355)= -2.17, *p*<.05); *Difficult Task* (change in level= 5.34, *t*(2355)= -3.08, *p*<.05) and *Change in Staff* (change in level= -4.66, *t*(2355)= -2.56, *p*<.05)) and *Transition* (change in level= 15.53, *t*(2355)= -2.52, *p*<.05). Autocorrelations were 0.94 for lag 1, 0.91 for lag 2, 0.88 for lag 3, 0.85 for lag 4 and 0.83 for lag 5.

Participant 13 had a mean baseline HR of 83.04 (*sd*= 6.63). He had a HR response significantly different than baseline for two stressor phases (*Eating a Preferred Food* (change in level= 13.47, *t*(2932)= 7.13, *p*<.05) and *Difficult Task* (change in level= 4.55, *t*(2932)= 3.21, *p*<.05) and *Physical Exertion* (change in level= 23.16, *t*(2932)= 10.86, *p*<.05) and *Transition* (change in level= 8.84, *t*(2932)= 5.57, *p*<.05). Autocorrelations were 0.94 for lag 1, 0.85 for lag 2, 0.78 for lag 3, 0.71 for lag 4 and 0.65 for lag 5.

Participant 14 had a mean baseline HR of 96.44 (*sd*= 4.40). He had a HR response significantly different than baseline for no stressor phases nor *Physical Exertion* and *Transition*, but data for *Unstructured Time* and *Difficult Task* phases were not available for this participant. Autocorrelations were 0.89 for lag 1, 0.83 for lag 2, 0.81 for lag 3, 0.81 for lag 4 and 0.77 for lag 5.

Participant 15 had a mean baseline HR of 97.90 (*sd*= 4.80). He had a HR response significantly different than baseline for three stressor phases (*Remote Control Robot* (change in level= 5.67, *t*(1763)= 1.98, *p*<.05); *Unstructured Time* (change in level= 15.58, *t*(1763)= 5.68, *p*<.05); *Eating a Preferred Food* (change in level= 14.08, *t*(1763)= 5.57, *p*<.05)) and *Transition* (change in level= 19.00, *t*(1763)= 4.43, *p*<.05). Data were missing for *Difficult Task*, *Change in Staff* and *Physical Exertion*. Autocorrelations were 0.90 for lag 1, 0.87 for lag 2, 0.82 for lag 3, 0.79 for lag 4 and 0.74 for lag 5.

Participant 16 had a mean baseline HR of 86.73 (*sd*= 6.34). He had a HR response significantly different than baseline for five stressor phases (*Loud Noise* (change in level= 6.37, *t*(3127)= 2.77, *p*<.05); *Remote Control Robot* (change in level= 10.97, *t*(3127)= 4.33, *p*<.05); *Unstructured Time* (change in level= 16.75, *t*(3127)= 5.16, *p*<.05); *Eating a Preferred Food* (change in level= 8.03, *t*(3127)= 4.71, *p*<.05); and *Difficult Task* (change in level= 4.86 , $t(3127)$ = 3.63 , $p<05$)) and *Physical Exertion* (change in level= 21.19, *t*(3127)= 6.78, *p*<.05) and *Transition* (change in level= 9.67, *t*(3127)= 5.58, *p*<.05). Autocorrelations were 0.93 for lag 1, 0.90 for lag 2, 0.86 for lag 3, 0.82 for lag 4 and 0.79 for lag 5.

Participant 17 had a mean baseline HR of 123.37 (*sd*= 5.48). She had a HR response significantly different than baseline for one stressor phase (*Loud Noise* (change in level= -3.84, *t*(1911)= -2.74, *p*<.05)) and *Physical Exertion* (change in level= 16.60, *t*(1911)= 4.88, *p*<.05) and *Transition* (change in level= -3.53, *t*(1911)= -2.19, *p*<.05). Autocorrelations were 0.88 for lag 1, 0.77 for lag 2, 0.70 for lag 3, 0.65 for lag 4 and 0.61 for lag 5.

Participant 18 had a mean baseline HR of 80.93 (*sd*= 6.45). He had a HR response significantly different than baseline for 4 stressor phases (*Unstructured Time* (change in level= 8.40 , $t(2463)=2.24$, $p<05$); *Eating a Preferred Food* (change in level= 12.43, *t*(2463)= 5.82, *p*<.05); *Difficult Task* (change in level= 11.72, *t*(2463)= 4.93, *p*<.05); and *Change in Staff* (change in level= 11.26, *t*(2463)= 4.81, *p*<.05)) and *Physical Exertion* (change in level= 30.72, $t(2463)$ = 1309, $p<0.05$) and *Transition* (change in level= 8.40, *t*(2463)= 4.45, *p*<.05). Autocorrelations were 0.95 for lag 1, 0.92 for lag 2, 0.88 for lag 3, 0.84 for lag 4 and 0.80 for lag 5.

Participant 19 had a mean baseline HR of 113.91 (*sd*= 5.69). He had a HR response significantly different than baseline for one stressor phase (*Remote Control Robot* (change in level = -3.41, $t(1829)$ = -2.67, $p<.05$)) and *Transition* (change in level = -1.90, *t*(1829)= -2.07, *p*<.05). Autocorrelations were 0.90 for lag 1, 0.83 for lag 2, 0.77 for lag 3, 0.72 for lag 4 and 0.67 for lag 5.

Participant 20 had a mean baseline HR of 95.00 (*sd*= 7.15). He had a HR response significantly different than baseline for one stressor phase (*Eating a Preferred Food* (change in level= 6.30, *t*(3092)= 2.19, *p*<.05)) and *Physical Exertion* (change in level= 18.12, *t*(3092)= 2.11, *p*<.05). Autocorrelations were 0.92 for lag 1, 0.85 for lag 2, 0.81 for lag 3, 0.78 for lag 4 and 0.77 for lag 5.

Participant 21 had a mean baseline HR of 86.86 (*sd*= 5.57). He had a HR response significantly different than baseline for two stressor phases (*Loud Noise* (change in level= 0.72, *t*(2983)= -2.80, *p*<.05) and *Difficult Task* (change in level= 8.34, *t*(2983)= 1.08, *p*<.05)) and *Physical Exertion* (change in level= 11.46, *t*(2983)= 3.40, *p*<.05).

Autocorrelations were 0.95 for lag 1, 0.85 for lag 2, 0.74 for lag 3, 0.62 for lag 4 and 0.52 for lag 5.

Participant 22 had a mean baseline HR of 90.30 (*sd*= 7.45). He had a HR response significantly different than baseline for no stressor phases and data were missing for *Physical Exertion*. Autocorrelations were 0.77 for lag 1, 0.69 for lag 2, 0.62 for lag 3, 0.66 for lag 4 and 0.57 for lag 5.

Participant 23 had a mean baseline HR of 97.47 (*sd*= 12.86). He had a HR response significantly different than baseline for two stressor phases (*Loud Noise* (change in level= 12.47 , $t(3264)$ = 3.12 , $p<0.05$) and *Remote Control Robot* (change in level= 15.31, *t*(3264)= -2.93, *p*<.05)) and *Physical Exertion* (change in level= 21.10, *t*(3264)= 4.54, *p*<.05). Autocorrelations were 0.83 for lag 1, 0.77 for lag 2, 0.72 for lag 3, 0.69 for lag 4 and 0.64 for lag 5.

Participant 24 had a mean baseline HR of 83.98 (*sd*= 7.35). He had a HR response significantly different than baseline for one stressor phase (*Remote Control Robot* (change in level= 7.68, $t(3283) = 2.44$, $p<.05$)). Autocorrelations were 0.83 for lag 1, 0.68 for lag 2, 0.62 for lag 3, 0.60 for lag 4 and 0.55 for lag 5.

Participant 25 had a mean baseline HR of 101.85 (*sd*= 9.01). He had a HR response significantly different than baseline for two stressor phases (*Eating a Preferred Food* (change in level= 11.68, *t*(2100)= 3.29, *p*<.05) and *Change in Staff* (change in level= 15.65, *t*(2100)= 4.95, *p*<.05)) and *Transition* (change in level= 9.28, *t*(2100)= 3.46, *p*<.05). Data were not available for *Unstructured Time*, *Difficult Task* or *Physical Exertion* phases. Autocorrelations were 0.92 for lag 1, 0.90 for lag 2, 0.88 for lag 3, 0.86 for lag 4 and 0.84 for lag 5.
Participant 26 had a mean baseline HR of 96.30 (*sd*= 5.18). He had a HR response significantly different than baseline for four stressor phases (*Loud Noise* (change in level= 10.72 , $t(3163)=1.98$, $p<.05$); *Remote Control Robot* (change in level= 4.47, *t*(3163)= 2.39, *p*<.05); *Eating a Preferred Food* (change in level= 7.42, *t*(3163)= 1.98, *p*<.05) and *Difficult Task* (change in level= 9.17, *t*(3163)= 2.48, *p*<.05)) and *Physical Exertion* (change in level= 28.23 , $t(3163)= 6.41$, $p<.05$) and *Transition* (change in level= 18.26, *t*(3163)= 2.58, *p*<.05). Autocorrelations were 0.96 for lag 1, 0.92 for lag 2, 0.89 for lag 3, 0.86 for lag 4 and 0.83 for lag 5.

Participant 27 had a mean baseline HR of 100.07 (*sd*= 7.07). He had a HR response significantly different than baseline for three stressor phases (*Loud Noise* (change in level= -7.83, *t*(1736)= -2.90, *p*<.05); *Remote Control Robot* (change in level= -3.04, *t*(1736)= -2.01, *p*<.05); and *Change in Staff* (change in level= 8.34, *t*(1736)= 2.58, *p*<.05)). Autocorrelations were 0.80 for lag 1, 0.68 for lag 2, 0.57 for lag 3, 0.54 for lag 4 and 0.47 for lag 5.

Participant 28 had a mean baseline HR of 106.59 (*sd*= 9.89). He had a HR response significantly different than baseline for one stressor phase (*Unstructured Time* (change in level= 25.00, *t*(1853)= 2.32, *p*<.05)) and *Transition* (change in level= 7.64, *t*(1853)= t, *p*<.05). Autocorrelations were 0.88 for lag 1, 0.76 for lag 2, 0.66 for lag 3, 0.57 for lag 4 and 0.51 for lag 5.

Participant 29 had an unusual order for his assessment. *Unstructured Time* was skipped in its normal position in the assessment order, for safety reasons, but then placed at the end of the assessment, before *Physical Exertion*. He had a mean baseline HR of 88.08 (*sd*= 4.86). He had a HR response significantly different than baseline for two

stressor phases (*Loud Noise* (change in level= 12.09, *t*(1888)= 2.89, *p*<.05); and *Remote Control Robot* (change in level= 26.56, *t*(1888)= 4.95, *p*<.05)) and *Physical Exertion* (change in level= 18.26, *t*(1888)= 3.59, *p*<.05). Autocorrelations were 0.95 for lag 1, 0.90 for lag 2, 0.87 for lag 3, 0.86 for lag 4 and 0.84 for lag 5.

Participant 30 had a mean baseline HR of 79.64 (*sd*= 7.76). He had a HR response significantly different than baseline for three stressor phases (*Eating a Preferred Food* (change in level= 14.36, *t*(2705)= 2.75, *p*<.05); *Difficult Task* (change in level= 10.61, *t*(2705)= 2.87, *p*<.05); and *Change in Staff* (change in level= 7.19, *t*(2705)= 2.68, *p*<.05)) and *Physical Exertion* (change in level= 36.39, *t*(2705)= 7.06, *p*<.05) and *Transition* (change in level= 14.14, $t(2705)$ = 4.05, $p<0.05$). Autocorrelations were 0.96 for lag 1, 0.88 for lag 2, 0.81 for lag 3, 0.76 for lag 4 and 0.73 for lag 5.

Participant 31 had a mean baseline HR of 110.08 (*sd*= 7.57). She had a HR response significantly different than baseline for 5 stressor phases (*Loud Noise* (change in level= 6.44, *t*(819)= 2.05, *p*<.05); *Remote Control Robot* (change in level= 9.49, *t*(819)= 3.10, *p*<.05); *Unstructured Time* (change in level= 8.08, *t*(819)= 2.15, *p*<.05); *Eating a Preferred Food* (change in level= -8.74, *t*(819)= -3.32, *p*<.05); and *Change in Staff* (change in level= 6.84 , $t(819)= 2.70$, $p<.05$. Autocorrelations were 0.60 for lag 1, 0.48 for lag 2, 0.42 for lag 3, 0.39 for lag 4 and 0.31 for lag 5.

Participant 32 had a mean baseline HR of 81.70 (*sd*= 3.90). He had a HR response significantly different than baseline for four stressor phases (*Remote Control Robot* (change in level= 3.80, *t*(2339)= 3.56, *p*<.05); *Unstructured Time* (change in level= 6.00, *t*(2339)= 6.07, *p*<.05); *Eating a Preferred Food* (change in level= 6.50, *t*(2339)= 6.09, *p*<.05); and *Difficult Task* (change in level= 9.10, *t*(2339)= 8.33, *p*<.05)) and *Physical Exertion* (change in level= 9.30, *t*(2339)= 8.01, *p*<.05) and *Transition* (change in level= 3.74, *t*(2339)= 4.74, *p*<.05). Autocorrelations were 0.89 for lag 1, 0.80 for lag 2, 0.71 for lag 3, 0.63 for lag 4 and 0.55 for lag 5.

Participant 33 had a mean baseline HR of 103.01 (*sd*= 7.43). He had a HR response significantly different than baseline for one stressor phases (*Eating a Preferred Food* (change in level= 6.46, *t*(1282)= 2.68, *p*<.05)). There were no data for *Physical Exertion*. Autocorrelations were 0.77 for lag 1, 0.58 for lag 2, 0.41 for lag 3, 0.35 for lag 4 and 0.31 for lag 5.

Participant 34 had a mean baseline HR of 87.09 (*sd*= 6.70). She had a HR response significantly different than baseline for no stressor phases but did have a significantly different response for *Physical Exertion* (change in level= 23.31, *t*(1756)= 5.10, *p*<.05). Autocorrelations were 0.87 for lag 1, 0.77 for lag 2, 0.68 for lag 3, 0.60 for lag 4 and 0.52 for lag 5.

Participant 35 had a mean baseline HR of 91.45 (*sd*= 3.59). He had a HR response significantly different than baseline for no stressor phases but did have a significantly different response for *Physical Exertion* (change in level= 12.15, *t*(2709)= 6.19, *p*<.05). Autocorrelations were 0.84 for lag 1, 0.79 for lag 2, 0.77 for lag 3, 0.74 for lag 4 and 0.67 for lag 5.

Participant 36 had a mean baseline HR of 102.94 (*sd*= 9.18). She had a HR response significantly different than baseline for one stressor phase (*Unstructured Time* (change in level= -7.91 , $t(1830) = -2.12$, $p<0.05$)) and *Physical Exertion* (change in level= 19.50, *t*(1830)= 4.54, *p*<.05). Autocorrelations were 0.92 for lag 1, 0.82 for lag 2, 0.77 for lag 3, 0.74 for lag 4 and 0.70 for lag 5.

Participant 37 had a mean baseline HR of 90.12 (*sd*= 3.29). He had a HR

response significantly different than baseline for four stressor phases (*Unstructured Time* (change in level= 9.46 , $t(1540) = 2.65$, $p<.05$); *Eating a Preferred Food* (change in level= 13.15, *t*(1540)= 3.26, *p*<.05); *Difficult Task* (change in level= 10.07, *t*(1540)= 2.67, *p*<.05); and *Change in Staff* (change in level= 12.08, *t*(1540)= 3.58, *p*<.05)) and *Physical Exertion* (change in level= 23.61, $t(1540) = 6.03$, $p<0.05$) and *Transition* (change in level= 9.36, *t*(1540)= 3.45, *p*<.05). Autocorrelations were 0.90 for lag 1, 0.82 for lag 2, 0.80 for lag 3, 0.79 for lag 4 and 0.74 for lag 5.

Participant 38 had a mean baseline HR of 71.50 (*sd*= 6.60). He had a HR response significantly different than baseline for no stressor phases but did have a significantly different response for *Physical Exertion* (change in level= 39.27, *t*(4534)= 11.17, *p*<.05). There were no data for *Change in Staff* phase. Autocorrelations were 0.93 for lag 1, 0.88 for lag 2, 0.85 for lag 3, 0.84 for lag 4 and 0.83 for lag 5.

Participant 39 had a mean baseline HR of 115.91 (*sd*= 9.03). She had a HR response significantly different than baseline for 5 stressor phases (*Remote Control Robot* (change in level= 4.27 , $t(2286) = 2.41$, $p<0.05$); *Unstructured Time* (change in level= 11.69, *t*(2286)= 4.94, *p*<.05); *Eating a Preferred Food* (change in level= 14.69, *t*(2286)= 4.65, *p*<.05); *Difficult Task* (change in level= 5.79, *t*(2286)= 3.39, *p*<.05); and *Change in Staff* (change in level= 6.48, *t*(2286)= 3.76, *p*<.05)) and *Physical Exertion* (change in level= 17.24, *t*(2286)= 5.12, *p*<.05) and *Transition* (change in level= 7.72, *t*(2286)= 2.27, *p*<.05). Autocorrelations were 0.93 for lag 1, 0.88 for lag 2, 0.83 for lag 3, 0.79 for lag 4 and 0.75 for lag 5.

Subtypes. Four examples of each of the four hypothesized subtype responder were identified using visual inspection of level of the baseline compared to stressor phases, scatter, and shape of participants' graphs (taking into account phase significance and if HR returns to baseline during rest phases). There were at least four participants fitting the criteria for hyperarousal, hyporesponsive, reactive responsive, and normal responsive (See Figures 43-58 for graphs exemplars).

Chapter 4. Discussion

 This study went beyond the current body of related literature by idiographically examining 39 replications of cardiovascular responsivity in individuals with ASD to a variety of systematically selected stressors. This is the only study, to the author's knowledge, that has idiographically examined physiological responses in a large group of individuals with ASD, and identified potential subtype responders (warranting future confirmatory analyses). Much of the prior research in this area examined a small number of participants using nomothetic methods to compare individuals with ASD to control groups. Group level analyses washes out high and low responders. Idiographic analyses allow examination of each responder. This allows for tailored interventions for the needs of individuals, before, during, or after exposure to a stressor to help the individual cope, learn, and reduce problem behaviors.

Based on mixed research findings, prior research suggestive of subtype physiological responders in ASD (Cohen, & Johnson, 1977; Hirstein et al, 2001), and findings of larger intraindividual than interindividual variation in ASD when compared to a control group when using group-level statistics (Berntson, et al., 1985; Kootz & Cohen, 1981), idiographic analyses to identify individual patterns of physiological response to stimuli was warranted. As expected, individual HR patterns varied. Typically very high autocorrelations were found for participants (around .95 for the first lag). This result would likely be the expected pattern for HR data taken from very short intervals.

There were four hypothesized responder types in this study. Examples of each were identified. These subtypes include: hyperarousal (i.e. have a high baseline HR, and low variation in response across stressors), hyporesponsive (i.e. have a low/normal

baseline HR, and low variation in response across stressors), reactive responsive (i.e. HR increases throughout the assessment, and fails to return to baseline level), and normal responsive (i.e. normal baseline HR that varies during stressor phases, but returns to baseline during subsequent baseline phases). These findings warrant future investigation, discussed below.

Individuals who fit the hyperarousal subtype could include teaching relaxation techniques, and to prompt using these techniques multiple times in a day. Individuals who fit the hyporesponsive subtype may need to do physical or sensory activities to get their arousal up so that they are better able to focus throughout the day. Individuals who fit the reactive responsive subtype would also probably need to learn relaxation techniques that are used throughout the day, especially before and after events that are known stressors. Individuals who fit the normal responsive subtype could also benefit from relaxation strategies that are used specifically prior to exposure to known stressors.

One limitation of the current study was that it was a secondary data analysis. Demographic and medical information (i.e. medications) were difficult to gather (as many participants had left the Center, and some data had not been collected close to the time of the assessment), which makes it difficult to compare individual results with respect to these data. Follow-up studies will further examine individual results with respect to different participant characteristics outlined in Table 2. Medications are one factor that can affect HR. Although many participants were on medications, the majority of participants (n=33) had HRs that were in the normal range for their age at baseline. Only six had high baseline HRs (participants 4, 7, 11, 25, 33, and 36), and none had low baseline HRs. All participants with high baseline HRs were over 10 years old. Follow-

up investigation could attempt to partial out HR changes due to medication, or could include only participants not on medications (however, this will substantially decrease participant pool, and may not be representative of individuals with ASD).

It is possible that HR changes may have been due to being in a laboratory setting, rather than the different stressor phases, per se. However, this may have been controlled by the familiarization period with the lab and *Lifeshirt*, and also by being accompanied by a familiar staff at all times. It is possible that baseline HR here was not a true indicator of one's resting HR. The first three minutes in the lab may not have been enough time for all individuals to habituate to their environment, or HR may have been artificially high due to being observed in an artificial setting. Attempts were made to control for this with the familiarization period, and the three minutes at the beginning of baseline that are discarded. Also, sitting quietly in a comfortable chair with a noninvasive vest and a familiar staff were other attempts to control for this. Only six individuals had high baseline HRs, so it doesn't appear that this was a potential problem for most. This study does not correlate overt behavioral responses with physiological responses, although individuals with high HRs during the assessment often showed little to no overt behavioral signs of distress. It would be informative to systematically investigate if there is synchrony or dysynchrony between behavior and physiology in ASD, and a follow-up study could be done examining the correlation between behavior and physiological measures. .

HR is a robust measure of arousal, however, HR alone does not reveal which system (SNS or PNS) is controlling HR responses. HR variability (HRV) is a measure that allows one to infer which system is working (or may be deficient). HRV data were

collected during all sessions, and a follow-up study will be done examining HRV responses in these 39 individuals.

With ideographic analyses, it is important to see if findings generalize across time, stimuli, and settings. Follow-up studies to examine this can involve testing a subset of this sample again at a six month follow-up session to assess generalization across time. Different stimuli representing the same construct could be used at a follow-up session with a subset of this sample. Finally, assessments could be done in a classroom setting, or other real-world setting to see if results generalize across setting with a subset of this sample.

Since ASD is solely defined by behavioral characteristics, it is useful to be able to break down this group into subtypes based on other characteristics, to better understand various phenotypes and to tailor interventions and prevention programs. A follow-up study will be conducted exploring different endophenotypes that may exist in ASD, using the four hypothesized responder types as a guide. These patterns may offer a better understanding of how stress operates in individuals with ASD, and may have direct clinical applications. Dynamic cluster analysis will be performed on the current data in a future study to see exactly what subtypes of responders exist. Cluster analysis categorizes inter-individual heterogeneity in intra-individual change as indices of different sub-populations that are characterized by different trajectories (Dumenci $\&$ Windle, 2001). This method allows researchers to identify patterns of change when group membership is not known *a priori*. Three reasons identified by Hoeppner, Goodwin, Velicer, Mooney, & Hatsukami (2008) as to why this method is so useful is that it "(1) parsimoniously represent(s) individual differences in intra-individual stability

and change, (2) evaluate(s) taxonomic developmental theories of change, and (3) facilitate(s) the development of models for early intervention and prevention programs by determining predictors and outcomes specific to a certain growth pattern." (p. 625).

Typically, cluster analysis is used for data collected from many people at a single time point. This future study will use dynamic cluster analysis, since it is based on a single variable measured on multiple occasions over time (Norman, Velicer, Fava, & Prochaska, 1998; Norman, Velicer, Fava, & Prochaska, 2000; Prochaska, Velicer, Guadagnoli, Rossi & DiClemente, 1991). Identifying factors that may be predictive of one's typology could help groups of individuals engage in treatments to prevent problems, and/or create tailored interventions to suit the needs of different subgroups. If an individual with ASD can be identified as a certain type of stress responder early, it may help these individuals receive better services sooner. In addition, understanding these differences can give insight to underlying biology, cognitive style, sensory sensitivity, and genetic, neural and physiological underpinnings in ASD. Different groups may represent different endophenotypes. These endophenotypes may help determine genetic, neurological, cognitive and behavioral differences in ASD. If HR can reliably distinguish subtypes, this would be very useful to guide etiologic, developmental, and intervention research.

The current study idiographically analyzed HR responses (measured telemetrically with a non-invasive vest) in 39 individuals with ASD to a variety of stressors. Individual patterns were identified, stressors were ranked, and examples of subtypes were found. No other study has looked at data from this many individuals with ASD idiographically using physiological measures. These findings could directly benefit

each individual, and could help identify subtypes of responder. This could allow for the creation of better interventions, but also a better understanding of ASD in general.

Table 1

Participant Diagnoses

Table 2

Participant Characteristics-Part 1

Table 2 (continued)

Participant Characteristics-Part 2

Table 3

HR for All Phases for All Participants

 Figure 1. Number of participants in each age range

Figure 2. Participant race

Detailed Description of the HR Assessment

A1) Initial baseline (5 minutes)- Participant sits quietly with staff.

B) *Loud Noise* (2 minutes)- A vacuum is turned on outside of the lab room door.

A2) Return to baseline (2 minutes)- Participant sits quietly with staff.

C) *Remote Control Robot* (2 minutes)- A hidden remote control car, controlled by the researcher in the adjoining room, drives around the room..

A3) Return to baseline (2 minutes)- Participant sits quietly with staff.

D) *Unstructured Time* (2 minutes)- The familiar staff leaves the room. The participant is instructed to sit quietly until the staff comes back in.

A4) Return to baseline (2 minutes)- Participant sits quietly with staff.

E) *Eating a Preferred Food* (2 minutes)- Participant is given a preferred food.

A5) Return to baseline (2 minutes)- Participant sits quietly with staff.

F) *Difficult Task* (2 minutes)- The participant is instructed to imitate their staff

who folds a towel quickly and are told to try again when they are unsuccessful.

A6) Return to baseline (2 minutes)- Participant sits quietly with staff.

G) *Change in Staff* (2 minutes)- The familiar staff leaves, and an unfamiliar staff sits quietly across from the participant.

A7) Return to baseline (2 minutes)- Participant sits quietly with staff.

H) *Physical Exertion* (2 minutes)- Participant rides a stationary bike or does

jumping jacks. This phase is included to make sure that the client is physically able to increase their HR.

Figure 3. Detailed description of HR assessment

Figure 4. HR Graph for Participant 1

 Figure 5. HR Graph for Participant 2

Participant 3

Figure 6. HR Graph for Participant 3

Figure 7. HR Graph for Participant 4

Figure 8. HR Graph for Participant 5

Figure 9. HR Graph for Participant 6

Figure 10. HR Graph for Participant 7

Figure 11. HR Graph for Participant 8

Figure 12. HR Graph for Participant 9

 Figure 13. HR Graph for Participant 10

Figure 14. HR Graph for Participant 11

Figure 15. HR Graph for Participant 12

 Figure 16. HR Graph for Participant 13

Figure 17. HR Graph for Participant 14

Figure 18. HR Graph for Participant 15

Figure 19. HR Graph for Participant 16

 Figure 20. HR Graph for Participant 17

Figure 21. HR Graph for Participant 18

 Figure 22. HR Graph for Participant 19

Figure 23. HR Graph for Participant 20

Figure 24. HR Graph for Participant 21

Figure 25. HR Graph for Participant 22

Figure 26. HR Graph for Participant 23

Figure 27. HR Graph for Participant 24

Figure 28. HR Graph for Participant 25

Figure 29. HR Graph for Participant 26

 Figure 30. HR Graph for Participant 27

Figure 31. HR Graph for Participant 28

 Figure 32. HR Graph for Participant 29

Figure 33. HR Graph for Participant 30

Figure 34. HR Graph for Participant 31

Figure 35. HR Graph for Participant 32

Figure 36. HR Graph for Participant 33

 Figure 37. HR Graph for Participant 34

Figure 38. HR Graph for Participant 35

Figure 39. HR Graph for Participant 36

Figure 40. HR Graph for Participant 37

Figure 41. HR Graph for Participant 38

Figure 42. HR Graph for Participant 39

Figure 43. Hyperarousal Exemplar Example 1

Figure 44. Hyperarousal Exemplar Example 2

Figure 45. Hyperarousal Exemplar Example 3

Figure 46. Hyperarousal Exemplar Example 4

Figure 47. Hyporesponsive Exemplar Example 1

Figure 48. Hyporesponsive Exemplar Example 2

Figure 49. Hyporesponsive Exemplar Example 3

Figure 50. Hyporesponsive Exemplar Example 4

Figure 51. Reactive Responsivity Exemplar Example 1

Figure 52. Reactive Responsivity Exemplar Example 2

Figure 53. Reactive Responsivity Exemplar Example 3

Figure 54. Reactive Responsivity Exemplar Example 4

Figure 55. Normal Responsivity Exemplar Example 1

Figure 56. Normal Responsivity Exemplar Example 2

Figure 57. Normal Responsivity Exemplar Example 3

Figure 58. Normal Responsivity Exemplar Example 4

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