ABSTRACT

Title of dissertation: THE SOCIAL ENGAGEMENT SYSTEM: FUNCTIONAL DIFFERENCES IN INDIVIDUALS WITH AUTISM

John William Denver, Doctor of Philosophy, 2004

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The Polyvagal Theory links the evolution of the autonomic nervous system to affective experience, emotional expression, facial gestures, vocalization and social engagement behavior. Therefore, the theory provides a plausible explanation for the bio-behavioral indices of several psychiatric disorders. The vagus as a "system" provides a rich organizing principle to investigate several of the behavioral, psychological, and physiological features associated with compromised social behavior in several psychiatric disorders. The Polyvagal Theory describes this integrated system as the Social Engagement System. Observations of the behaviors and physiological responses of autistic individuals suggest that they have great difficulties in recruiting the neural circuit that regulates the social engagement system. This model predicts that a deficit in the system would produce atypical social behaviors such as social withdrawal; improper communication (i.e., poor intonation and prosody); difficulty listening (inability to extract human voice from background noise); poor eye contact; inappropriate facial expressivity (i.e., flat affect); and atypical visceral functioning (i.e., low cardiac vagal tone). These indices are directly

related to the atypical behaviors associated with autism, and several other psychiatric disorders. In the current study, measures related to the functioning of these components were obtained to test the hypothesis that autistic individuals have a compromised social engagement system. Forty subjects participated in the study (20 autistic, 31 males, ages 9-24). Data were collected to assess autonomic functioning (i.e., cardiac vagal tone), the ability to extract human voice from a compromised environment, an estimate of right ear advantage, and looking behavior (i.e., eye contact). Analyses showed that autistic individuals scored poorer on all measures assessing social engagement system functioning. Compared to controls, the autistic group had lower mean cardiac vagal tone and shorter heart periods, performed poorer on extracting human voice from a compromised environment, on a dichotic listening task, and on a measure of right ear advantage. They also spent significantly less time fixating on the eyes and more time fixating off of the face when viewing a movie of a person telling them a story. Results support the hypothesized relation between a compromised social engagement system and the atypical features associated with autism.

THE SOCIAL ENGAGEMENT SYSTEM: FUNCTIONAL DIFFERENCES IN INDIVIDUALS WITH AUTISM

By

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DEDICATION

This paper is dedicated to my wife Elaine, my children, and my entire family. Without their strong support, unending patience, personal sacrifice, and love, this journey would have never have had a beginning or an ending. It is also specially dedicated to the memory of my father, who did not have the chance to see the end of this journey, but whom I know is watching…and is proud.

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A meaningful graduate career begins with successful individuals who take the time to provide challenges and opportunities while at the same time demanding commitment, enthusiasm, and productivity from their students. I have had the good fortune throughout my studies to meet, engage with, and to learn from several such individuals. At the forefront is my advisor and mentor Dr. Stephen Porges who has shaped my thinking, has shown me the excitement and value of good research, and has allowed me to grow as a researcher and as a person, and has always treated me as a colleague.

I also need to acknowledge all of the members of my committee. Each has influenced my thinking about what it means to be a good researcher and what it takes to be committed to my field. Either directly or indirectly, each of the committee members has enhanced my experience as a student, as a researcher, and as a colleague-in-training.

Finally, a special acknowledgement goes to and Keri Heilman who helped design and create the video stimuli for the study, and George Nijme who was kind enough to play the part of the storyteller for the video stimuli in this study.

TABLE OF CONTENTS

LIST OF TABLES

Table 11: Summary of analyses of eye-tracker variables for each group. As hypothesized, significant differences are seen for the Off and Eye regions of interest. The only significant difference for the Mouth region was for fixation count percent. SFD = sum of fixation duration; FDP = fixation duration percent; SFC = sum of fixation counts; FCP = fixation count percent; MFD = mean fixation duration.

LIST OF FIGURES

CHAPTER I

INTRODUCTION

This study investigates the hypothesized relation between the core behavioral deficits of social communication in autism and underlying neurobiological processes. Fundamental to this hypothesized relation are several key points. First, diagnostic features for several psychiatric disorders (i.e. autism, behavior disorders, attention deficit disorders, language and reading disorders, internalizing disorders) include compromised social interactions, deficits in verbal and nonverbal communication, and atypical patterns of behaviors. Second, these features emerge as a "cluster" of functional deficits in many disorders. Third, it is plausible that underlying this cluster of behaviors is a common neurophysiological system. Fourth, from a systems perspective, predictable relations between system component functioning and the cluster of atypical behaviors can be proposed. Finally, these proposed relations can be objectively tested to inform theory, research, and practice.

Many DSM-IV (APA, 1994) classifications include a number of similar criteria as defining characteristics of a disorder. For instance, difficulties in social settings or atypical social behaviors are criteria for anxiety disorders (i.e., agoraphobia, social phobia), mood disorders (i.e., depression), and several of the disorders diagnosed in infancy, childhood, or adolescence (i.e., learning disorders, communication disorders, pervasive developmental disorders, attention deficit/disruptive behavior disorders). Within these disorders, a "cluster" of common diagnostic features can be extracted that includes deficits in functions related to social engagement such as initiating or maintaining social interactions, appropriate eye

contact, appropriate facial expression and appropriate social communication. The fact that several of disorders share common behavioral features supports the hypothesis that there is a common underlying system involved in the normal functional regulation of these behaviors and that the specific disorders may lie along a continuum of severity of deficit within that system. A system such as this would have to be able to account for the neurobiological, as well as the behavioral aspects associated with the relevant clinical features in a cohesive way. For example, the system would have to be able to link aspects of the atypical social behaviors of social phobia (i.e., social withdrawal) with the physical components (i.e., increased heart rate) of the disorder. At the same time, understanding a cluster of diagnostic features as evidence of a "system" deficit would allow for a better understanding of the dynamics of the disorder and would aid in the design of effective treatment strategies. The current study was designed to assess and to expand on information that supports the involvement of a specific neurobiologically based system in this core of atypical behaviors, and posits that these behaviors may be the adaptive "emergent properties" of a deficit in the functioning of this system.

The Social Engagement System (Porges, 2001, 2003) defines a neurobiologically based system that describes the intricate functioning of several neural pathways related to social engagement behaviors. This system is involved in the coordination of autonomic activity with social behavior through the use of hierarchically organized adaptive strategies for coping with the environment. Derived from an understanding of the evolution of the mammalian nervous system, the social engagement system integrates the phylogenetic development of links between various

anatomical and neural components and describes the way in which these links support adaptive strategies for dealing with the environment. The system is comprised of circuits involved in the regulation of visceral state including heart rate, and control muscles of the face and head (i.e., middle ear muscles, striated facial muscles). The social engagement system model describes the integration of visceromotor and somatomotor functioning of specific components of the autonomic nervous system (ANS) that are involved in social engagement (see Figure 1).

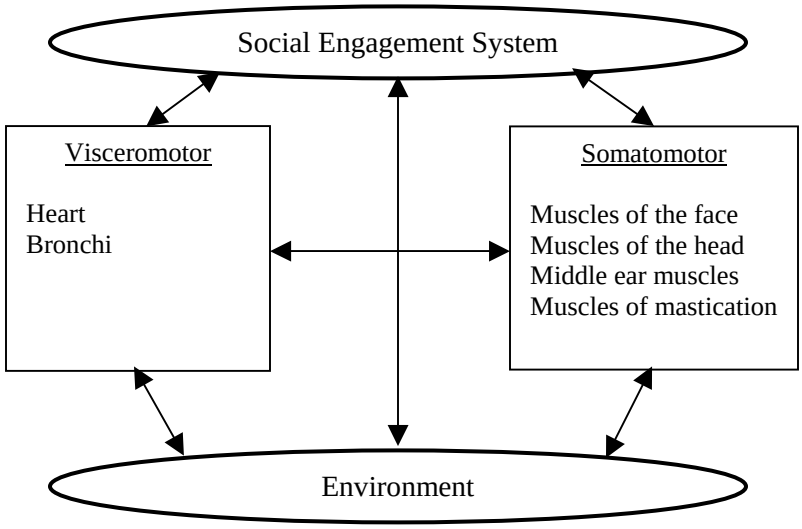

Figure 1: Representation of the Social Engagement System showing the integration of components relevant to social engagement (adapted from Porges, 1995).

The visceromotor component of this system is the Xth cranial nerve (vagus), which is responsible for the regulation of the heart (i.e., visceral homeostasis). The somatomotor component consists of special visceral efferent (SVE) pathways traveling through cranial nerves V, VII, IX, X and XI that are responsible for the regulation of the striated muscles of the face and head. The system consists of

pathways that regulate the middle ear muscles (i.e., extraction of human voice from background sounds, listening), eye-lid opening (i.e., looking behavior), facial muscles (i.e., facial expressivity), laryngeal and pharyngeal muscles (i.e., vocalization and intonation), head turning muscles (i.e., social gesture, orienting), and muscles of mastication (i.e., ingestion). According to this model, the bi-directional interactions of the visceromotor and somatomotor components serve to regulate social experience both internally and externally based on an evaluation of the environment (i.e., safe, dangerous, life-threatening). A well functioning social engagement system supports our ability to approach and interact with others, which includes maintaining appropriate eye contact, listening and communicating, and making appropriate social gestures and facial expressions. Alternatively, a deficit in this system would predict atypical features in some or all of these components of social engagement.

The atypical behaviors predicted by a deficit in the social engagement system are contained in the diagnostic features of several psychiatric disorders. For instance the DSM-IV communication disorders all list difficulties in the reception of language (i.e., accurately extracting human voice), and/or the expression of language (i.e., vocalization, prosody) that interfere with social communication. Similarly, attention deficit disorders and conduct disorders list difficulties in listening, social skills and regulation of behavior. Relevant to the current study, the diagnostic criteria of the pervasive developmental disorders (PDD) in general, and autism in particular, specifically relate to features predicted by deficits in components of the social engagement system. More specifically, the diagnostic criteria of these disorders include impairments in initiating and maintaining social interactions, facial expression,

eye contact, social gesture, vocalization and intonation, and atypical behavior regulation. Based on the relation between the diagnostic criteria of certain psychiatric disorders and the predictions of features related to a deficit in the social engagement system, it is possible to propose that certain disorders (and atypical social behaviors) may lie on a continuum of deficits in social engagement system functioning (i.e., from shy to PDD). While research has been conducted that provides evidence to support a deficit in specific components described by the model, data integrating the deficits across components is lacking. The current study was designed to test the predicted features of a deficit in social engagement system functioning by assessing measures of the somatomotor and visceromotor components of the system in autistic individuals. Given the tight overlap in the diagnostic features of autism and predicted indices of a compromised social engagement system, it is possible that autism represents an extreme example of the emergent properties of a compromise in this system. To support this hypothesis, functional differences between clinical populations and normal controls on the indices predicted by the model would need to be quantified. In the current study, a number of these predictable indices are tested between individuals with autism and normal controls.

The Social Engagement System model, derived from the Polyvagal Theory, provides the basis for understanding features common to a number of psychiatric disorders. From the approach that these features emerge as a cluster of behaviors and are associated with a social engagement system, evidence for a deficit in an underlying neurobiological system can be quantified based on specific predictable relations among the components of the system. Although research on several psychiatric

disorders provides support for the concept of an underlying system deficit, very little has been done in the way of understanding the diagnostic features related to autism from an integrated neurobiological system perspective. The hypotheses of the current study are derived directly from the social engagement system model, and the emergent properties that are predicated by a deficit in this system. An understanding of the behavioral and physiological outcomes of a system deficit facilitates a better understanding of both the diagnostic features of certain psychiatric disorders but also intervention and treatment strategies that may effectively return the system to a more optimal level of functioning.

CHAPTER II

POLYVAGAL THEORY AND THE SOCIAL ENGAGEMENT SYSTEM

Overview of the Polyvagal Theory

The Polyvagal Theory (Porges, 1995) was developed to relate the evolutionary shift in the neural regulation of the autonomic nervous system to the range and regulation of emotion expressed and experienced by humans. The theory proposes that our physiological state limits the range of available behaviors and psychological experiences. The Polyvagal Theory emphasizes the integration via evolution of the facial muscles (i.e., facial expression, looking, listening) and the neural regulation of visceral organs (i.e., heart) to regulate behavioral states. The Polyvagal Theory proposes that the evolution of the autonomic nervous system provides an organizing principle from which we can interpret the adaptive significance of emotional processes. The Polyvagal Theory links the evolution of the autonomic nervous system to affective experience, emotional expression, facial gestures, vocalization and social engagement behavior. Therefore, the theory provides a plausible explanation for the bio-behavioral indices of a number of psychiatric disorders.

Porges (1995, 1997, 1998, 2001) proposed the Polyvagal Theory as a means of describing the evolutionary shifts in neuroanatomical structures that have evolved through the mammalian phylogeny, as an organizing principle for understanding the expression of social and emotional behavior in mammals and especially humans. According to the Polyvagal Theory, the mammalian nervous system has evolved for

the physiological support of emotional and social behavior and communication. Specifically, the theory focuses on the phylogenetic shifts in the neural regulation of the viscera (i.e., heart) by the 10th cranial nerve (the vagus) and how these shifts have evolved to facilitate specific psychological processes (Porges, 1995). The theory emphasizes the neurophysiological and neuroanatomical distinctions between two branches of the vagus nerve that evolved sequentially. "Polyvagal" is the term used to emphasize the functional and neuroanatomical differences between the two branches of the vagus and their source nuclei located in the brainstem. The dorsal motor nucleus is the source of the primitive unmyelinated vagal efferents while the nucleus ambiguus gives rise to the more modern myelinated vagal efferents.

The vagus is a primary component of the autonomic nervous system that originates in brainstem structures and branches off to regulate muscles of the face and head (i.e., facial muscles, middle ear muscles, muscles of mastication, and the larynx and pharynx muscles) and visceral organs (i.e., heart). The theory proposes that each branch of the vagal system is associated with a different adaptive behavioral strategy. Specifically, the Polyvagal Theory describes the phylogenetic shifts that gave rise to three hierarchically organized behavioral strategies that emerged during the evolution of the mammalian nervous system. The phylogenetic stages reflect the emergence of three distinct autonomic subsystems, 1) the dorsal vagal complex (DVC), the sympathetic nervous system (SNS), and 3) the ventral vagal complex (VVC). The most primitive system, the DVC, is linked to immobilization strategies (i.e., freezing, death feigning) and provides neural regulation via the dorsal motor nucleus of the vagus (DMNX). The SNS is related to mobilization strategies (i.e., fight-flight

behaviors). The VVC is behaviorally linked to social engagement (i.e., visceral regulation, vocalization, facial expressivity, listening) and is associated with the mammalian myelinated vagus, which provides neural regulation via pathways originating in the nucleus ambiguus (NA). These three circuits can be conceptualized as providing the phylogenetically organized adaptive responses to environmental states of safety (i.e., VVC), danger (i.e., SNS), or life threat (i.e., DVC).

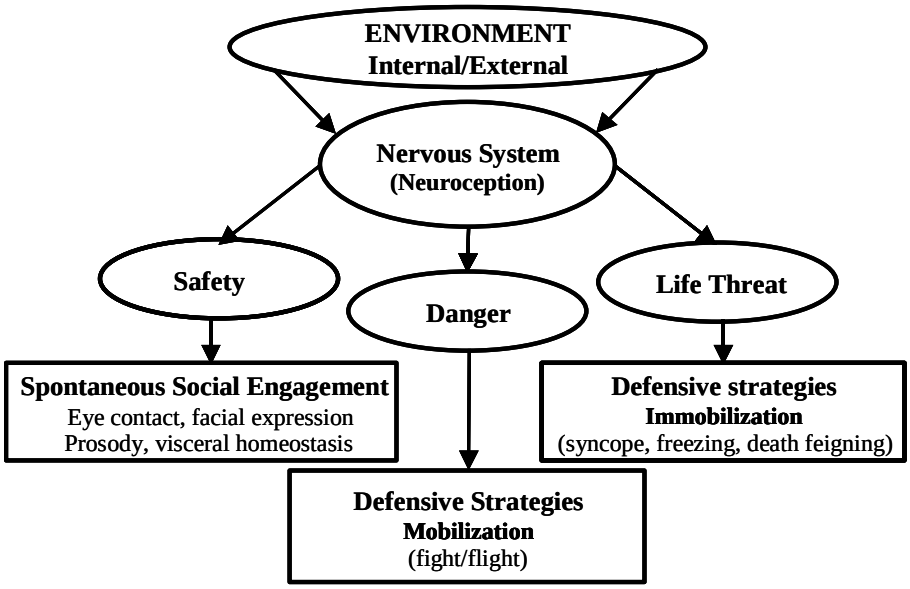

Figure 2: Functional representation of the components of the Polyvagal Theory (adapted from Porges, 2003).

This increased complexity and the development of opposing neural systems (i.e., SNS and ANS) allows us to assess and react to others (i.e., friend, foe) and the environment (i.e., safe, dangerous, life threatening) in adaptive ways (i.e., social interactions or survival behaviors). For instance, when the environment is perceived as safe (i.e., interacting with a friend, a familiar setting), the system is responsible for

the tonic regulation of visceromotor components (i.e., slowing the heart, promotion of calmness) as well as somatomotor components involved in social engagement (i.e., looking, listening, communicating). This state is associated with physiological and behavioral strategies that promote appropriate social engagement behaviors. Alternatively, vagal control of the heart can be withdrawn in order to react to stimuli in the environment (i.e., approach of a stranger) without the need to immediately recruit the metabolically demanding sympathetic-adrenal system. The myelinated vagus has an inhibitory effect at the sino-atrial node of the heart, which serves to reduce heart rate below the intrinsic rate produced by the cardiac pacemaker cells. In essence, the myelinated vagus acts like a brake on the heart. The removal of the "vagal brake" (Porges et. al., 1996) allows for an increase in heart rate and metabolism for transitory periods. The reapplication of the vagal brake promotes a return to visceral homeostasis. These transitory changes in state are adaptive to mammals in that the demands on the nervous system are minimized unless the perception of safety is removed (i.e., the stranger becomes threatening). A perception of danger would bring about the inhibition of the vagal system and the recruitment of the SNS (i.e., mobilization, fight/flight reactions), which is metabolically costly but adaptive under certain circumstances. Under life threatening conditions, inhibition of both the VVC and SNS and recruitment of the primitive vagal system (DVC) would serve to immobilize the organism (i.e., freezing, death feigning) in order to promote survival. In mammals, and especially humans, the recruitment of this primitive system can prove lethal (i.e., via shutdown of necessary functions).

According to the Polyvagal Theory, the increased neural complexity associated with the phylogenetic development of the autonomic subsystems allows for the enrichment of an organism's affective and behavioral repertoire (Porges, 1997) and provides us with the neurobiologically based organizing principle for understanding behaviors as "emergent properties" of autonomic nervous system functioning (Porges, 1998).

The Social Engagement System

The Polyvagal Theory can provide a model for understanding the deficits in spontaneous social behaviors prominent in autism and several other psychiatric disorders. Collectively, the integration of the visceromotor and somatomotor components described by the Polyvagal Theory and their related structures have been labeled the Social Engagement System (Porges, 2001). The social engagement system model was proposed as a construct to understand an integrated neural feedback system that is responsible for the regulation of social and emotional functioning.

During embryological development, components of several of the cranial nerves (V, VII, IX, X, XI) develop together (Parent, 1996) and form the basis for a social engagement system (see also Porges, 1998; 2001). This system is comprised of a control component in the cortex (i.e., upper motor neurons) that regulates the brainstem nuclei (i.e., lower motor neurons) to control specific functions related to social engagement. As illustrated in Figure 3, these functions include the regulation of visceral organs (i.e., heart, bronchi), facial muscles (i.e., eyelid opening, facial expression), middle ear muscles (i.e., the extraction of human voice from background

noise), laryngeal/pharyngeal muscles (i.e., vocalization and intonation), head turning muscles (i.e., orienting and social gesture), and muscles of mastication (i.e., ingestion).

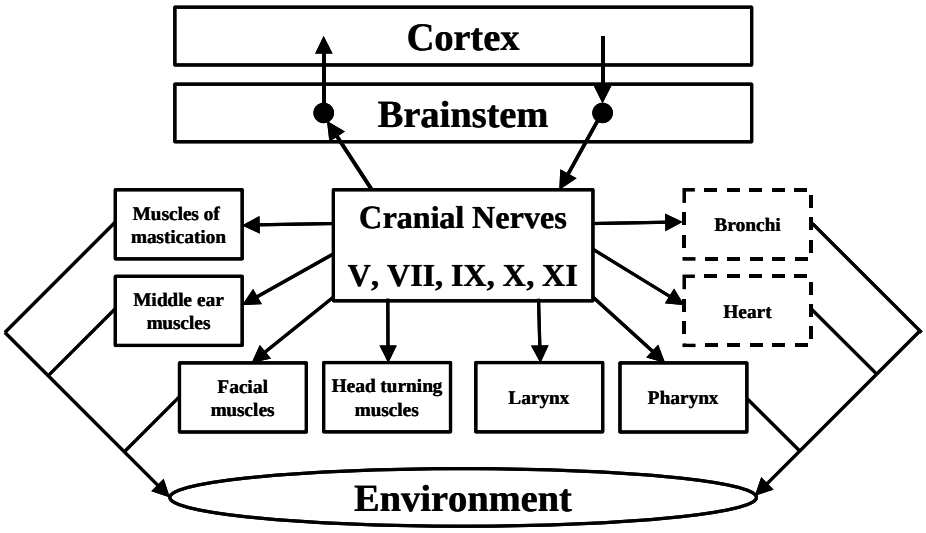

Figure 3: The social engagement system consists of a somatomotor component (i.e., special visceral efferent pathways that regulate the muscles of the head and face) and a visceromotor component (i.e., the myelinated vagus that regulates the heart and bronchi). Solid blocks indicate the somatomotor component. Dashed blocks indicate the visceromotor component. (adapted from Porges, 2001).

Functionally, these muscles are involved in limiting social stimuli and determining physiological availability for engagement with the environment (Porges, 2002). The model proposes that a key component of social engagement is the availability of the system to initiate and maintain these engagements. In order for us to initiate and reciprocate engagement with our environment, the system must be functioning properly. This allows for proper social orienting and appropriate communication, listening, eye contact and facial expression and reduces arousal to promote calmness and relaxation. Relevant to the current study are the components

involved in regulation of the viscera (i.e., the heart), the facial muscles (i.e., facial expression, eye gaze) and the middle ear muscles (i.e., listening).

Component Functioning

Paralleling the phylogenetic shift in the neural mechanisms involved in regulating cardiac output were shifts in behavioral strategies in response to stimuli in the environment. Changes from non-vagal to vagal, and finally to a multi-vagal system has lead to an enriched behavioral and affective repertoire in mammals. In order to regulate the heart, several efferent structures have evolved. These structures represent the sympathetic-adrenal system, which includes chromaffin tissue and the spinal sympathetics, and a vagal system that is a major component of the parasympathetic nervous system (Porges, 1995). The vagal system contains branches that originate in medullary nuclei that include the dorsal motor nucleus of the vagus and the nucleus ambiguus. A list of the regulatory structures that influence the heart is seen in Table 2 (Porges, 1995, Santer, 1994). A number of key points concerning phylogenetic changes in cardiac control can be extracted. First, there is a phylogenetic shift from chromaffin (endocrine) regulatory systems to unmyelinated and finally myelinated neural control. Second, there is a development of opposing systems for excitatory and inhibitory innervation. Third, the increase in cortical development provides for greater control over brainstem structures through direct and indirect neural pathways for cranial nerves V, VII, IX, X, and XI for regulation of the face, head, and viscera. Finally, the brainstem structures responsible for regulation of the muscles of the face and head are intimately linked to the regulation of autonomic state (Porges, 2001, 2003).

Phylogenetic Stage	Autonomic nervous system component	Behavioral Function	Lower motor neurons
III	Myelinated Vagus	Social engagement, communication, self-soothing and calming, visceral homeostasis, inhibition of sympathetic nervous system	Nucleus Ambiguus
II	Symathetic-Adrenal	Mobilization (fight/flight)	Spinal column
I	Unmyelinated Vagus	Immobilization (syncope, freezing, death feigning)	Dorsal motor nucleus

Table 1: The three phylogenetic stages of the neural control of the heart proposed by the Polyvagal Theory (adapted from Porges, 1995).

Third, the increase in cortical development provides for greater control over brainstem structures through direct and indirect neural pathways for cranial nerves V, VII, IX, X, and XI for regulation of the face, head, and viscera. Finally, the brainstem structures responsible for regulation of the muscles of the face and head are intimately linked to the regulation of autonomic state (Porges, 2001, 2003).

Morphological changes occurred in the vagus (Morris & Nilsson, 1994) and in mammals the vagus consists of two branches. The first branch is unmyelinated and originates in the DMNX in the brain stem. This branch provides the primary neural regulation of subdiaphragmatic organs such as the digestive tract. At the level of the heart, the DMNX fibers do not play a major role in the normal dynamics of cardiac functioning (Parent, 1996). The second branch of the vagus consists of myelinated fibers that arise from the NA. During embryological development in mammals, cells migrate from DMNX to the NA (Schwaber, 1986) where they form the cell bodies for the myelinated vagal visceromotor pathways that provide potent inhibitory regulation

(i.e., the vagal brake) to the sinoatrial node (SA) of the heart. In general, this specialization has led to greater control over the heart via the mammalian vagal system. This specialization allows for up- and down-regulation of cardiac responses without a specific need for innervation via sympathetic-adrenal; systems that has a high metabolic cost. As mammals, we can rapidly increase and decrease metabolic output via vagal withdrawal, while at the same time maintain the ability to reverse this response in order to quickly return to a homeostatic state. A prolonged challenge will still invoke sympathetic activation, but the reciprocal inhibitory effect of vagal re-innervation, which inhibits the sympathetic response, allows us to self-sooth and calm down. Table 2 provides information regarding the basic anatomy of the many components of the vagal system and their general functions. These components are referred to as the branchial motor, visceral motor, general sensory, and special sensory components, and each plays a distinct role in vagal innervation dynamics.

Component	Function	Cells of origin or termination	Related Components
Branchial motor	Supplies voluntary muscles of pharynx and larynx	NA	CN IX
Visceral motor	Innervation to the sinoatrial node of the heart	NA	CN X
	Innervation to parasympathetic ganglia in the head and abdomen	DMNX	
Visceral sensory	Receives virtually all sensory information from the parasympathetic system	Terminates in NTS	CNs V, IX, X

Table 2: Components of the vagal system and their function. Related connections include integration of input/output with other cranial nerves. NA, nucleus ambiguus; DMNX, dorsal motor nucleus of the vagus; NTS, nucleus of the solitary tract.

The vagal system has many functions that can be grouped into three main categories, each corresponding to one of the medullary nuclei. The NA is a motor nucleus that sends axons to innervate striated muscles throughout the neck and thorax. This includes the palate and pharynx, and to regulate the parasympathetic innervation of the heart. The DMNX is a parasympathetic nucleus, which innervates ganglia in the head and abdomen, as well as providing projections to the heart. The NTS is the sensory nucleus of the vagus that receives all of the visceral sensory information including sensory inputs from blood pressure receptors, blood-oxygen receptors, sensations from the pharynx/larynx and trachea, and stretch receptors in the gut. It should be noted that the majority of information related to the vagal system is afferent and that roughly 80% of vagal fibers are afferent. Thus, the NTS and the solitary tract function as the afferent limb of the vagal system. The various components of the vagal system serve many autonomic functions and play an integral role in autonomic regulation. Primarily, the system can be thought of as a tripartite system that includes a primitive component (i.e., vagal fibers originating in the dorsal motor nucleus), a modern component (i.e., vagal fibers originating in the nucleus ambiguus), and an afferent component (i.e., fibers traversing and terminating in the solitary tract). Table 3 summarizes the response of autonomic innervation including both motor nuclei of the system and the sympathetic nervous system's input and shows the excitatory and inhibitory responses that result from these inputs. From this we can see that the vagal system is integrated with numerous functions of social engagement. The Social Engagement System model was proposed as a means of directly relating the neurobiology of the nervous system, to the physiological and behavioral components

of social engagement. It is also possible to extract information pertaining to outcomes associated with both normal and deficient functioning within the system. Under normal conditions, the social engagement system facilitates and modulates our communicative, behavioral, and emotional interactions with others, which in turn provides others with the information and feedback necessary to facilitate reciprocal social engagement. For instance, when we are engaged with others, either directly or indirectly, we expect certain features to be present.

	VVC	SNS	DVC
Heart rate	+/-	+	-
Bronchi	+/-	+	-
Gastrointestinal		-	+
Vasoconstriction		+	
Sweat		+	
Adrenal		+	
Tears	+/-		
Vocalization	+/-		
Facial muscles	+/-		
Eyelids	+/-		
Middle ear	+/-		

Table 3: Functional effects of innervation by components of the autonomic nervous system. SNS= sympathetic fibers; VVC = ventral vagal complex including motor fibers originating in the NA and the source nuclei of V and VII; DVC = dorsal vagal complex including the motor fibers originating in the dorsal motor nucleus of the vagus and the sensory fibers terminating in the nucleus of the solitary tract (adapted from Porges, 1997).

We expect that the person we are engaged with will look at us and make appropriate eye contact, show appropriate facial expressions and affect, gestures, and have prosody in their voice. When we speak we know that others are listening because of their eye contact, facial expressions and gestures. When confronted with individuals

who do not conform to these expectations, we may interpret their behaviors as stemming from an illness as in the case of a sick child, as a lack of interest in what we are communicating, or a disinterest in us personally, which is often accompanied by feelings of visceral discomfort and anxiousness.

A well functioning social engagement system provides us with the neural control necessary to engage others in our environment, as well as to interpret our own internal states in meaningful ways. The somatomotor components responsible for controlling the striated muscles of the head and face allow us to orient our heads toward the person we are engaging. These same neural pathways are responsible for providing some of the muscle control to our faces, allowing for appropriate facial expressions. They allow us to raise our eyelids to look at an individual we are engaged with, and these same circuits are linked to the middle ear muscles that when flexed are responsible for allowing the transmission of the human voice frequencies to pass to the auditory pathways for the perception of speech. Therefore, when we open our eyes during a conversation, our middle ear muscles are also helping us to extract the human voice frequencies and dampen the low frequency noise from within our environments. For listening, the middle ear muscles have evolved in a way that specifically allows for the extraction of the human voice frequencies from background sounds (i.e., a "listening system").

A "listening system"

The pathways from five cranial nerves control the muscles of the face and head. Collectively, these pathways are labeled as special visceral efferent. The special visceral efferent pathways (i.e., the vagal system) regulate the muscles of

mastication (e.g., ingestion via CN V), muscles of the middle ear (e.g., listening to human voice via CN VII, V), muscles of the face (e.g., emotional expression via CN VII), muscles of larynx and pharynx (e.g., prosody and intonation via CN X, IX), and muscles controlling head tilt and turning (e.g., gesture via CN XI). In fact, the neural pathway that raises the eyelids also tenses the stapedius muscle in the middle ear, which facilitates hearing human voice. Thus, the neural mechanisms for making eye contact are shared with those that extract human voice from background sounds. As a cluster, the difficulties in gaze, extraction of human voice, facial expression, head gesture and prosody are common features of individuals with autism.

Sound pressure waves from our environment reach the eardrum and cause it to vibrate. The vibrations are transduced from the eardrum to the inner ear by the small bones in the middle ear (i.e., ossicles). When innervated, the stapedius muscle (innervated via a branch of the facial nerve) and the tensor tympani (innervated via a branch of the trigeminal nerve), stiffen the ossicles, which functionally dampen the amplitude of the low frequency sounds reaching the inner ear. The impact of these muscles on the perceived acoustic environment is to markedly attenuate low frequency sounds and to facilitate the extraction of high frequency sounds associated with human voice. For example, our acoustic environment is often dominated by loud low frequency sounds that have the functional effect of masking the soft high frequency sounds associated with human voice. In humans, the ossicular chain is regulated primarily by the stapedius muscle and tensing the stapedius prevents this masking effect (Borg & Counter, 1989). Individuals who can voluntarily contract middle ear muscles exhibit an attenuation of approximately 30 db at frequencies below 500 Hz,

while there is no or minimal attenuation at frequencies above 1000 Hz (see Kryter, 1985; see also Burns, Harrison, Bulen, & Keefe, 1993). In a study by Djupesland (1965, see also Djupesland 1976) it was shown that manually raising the eyelids also contracts the middle ear muscles, which was assumed to be related to an orienting reflex. Salomon & Starr (1963) also showed that the middle ear muscle reflex is elicited during certain movements of the head and neck as well as during vocalization. Thus, specific neural components described by the social engagement system may be conceptualized as a *listening system.*

The evolution of the mammalian middle ear enabled low amplitude relatively high frequency airborne sounds (i.e., sounds in the frequency of human voice) to be heard, even when the acoustic environment was dominated by low frequency sounds. This ability to hear low amplitude high frequency airborne sounds in an acoustic environment dominated by loud low frequency sounds, could only be accomplished when the middle ear muscles are tensed to create a rigidity along the ossicular chain. The tensing of these muscles prevents the low frequency sounds from being transduced through the middle ear bones from the eardrum to the cochlea, and the subsequent masking of high the frequency sounds associated with human voice.

Studies have demonstrated that the neural regulation of middle ear muscles, a necessary mechanism to extract the soft sounds of human voice from the loud sounds of low frequency background noise, is defective in individuals with language delays, learning disabilities and autistic spectrum disorders (Smith et al., 1988; Thomas et al., 1985). Other studies have shown similar findings with introverted and socially withdrawn children (Bar-Haim, 2002; Bar-Haim & Marshall, 2001). Disorders that

degrade the neural function of the facial nerve (i.e., Bell's Palsy), not only influence the stapedius reflex (Ardic et al., 1997; Qui, Yen, Stucker, & Hoasjoe, 1997), but also affect the patient's ability to discriminate speech (Wormald et al., 1995).

The stapedius reflex arc (Figure 5) consists of auditory afferent pathways (i.e., outer, middle and inner ear structures and the auditory nerve), the brainstem (i.e., the auditory nucleus, superior olivary complex, and facial nucleus), the facial nerve, and the stapedius muscle (Jerger & Jerger, 1977).

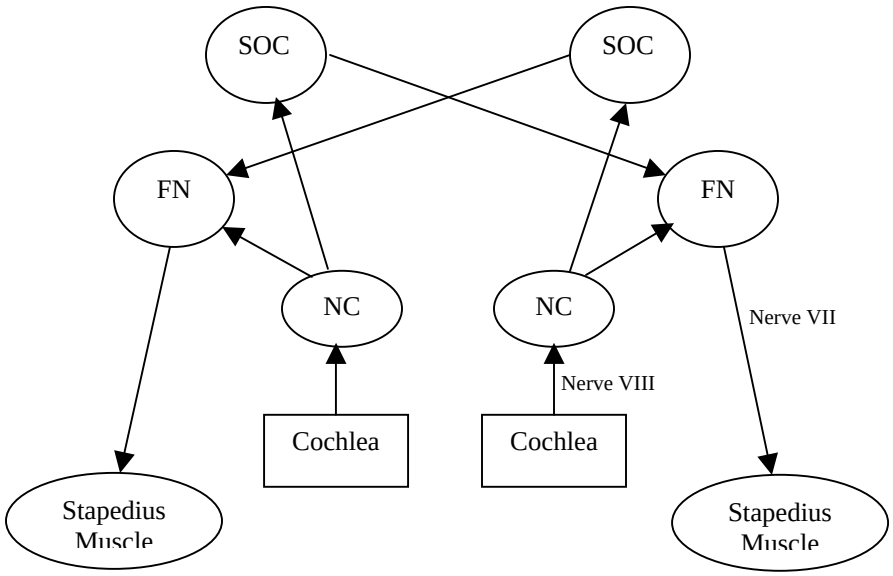

Figure 4: The reflex arc of the middle ear acoustic reflex: NC, cochlear nucleus; SOC, superior olivary complex; FN, facial motor nucleus; nerve VIII, auditory nerve; nerve VII, facial nerve motor neuron. The facial nerve also innervates facial muscles responsible for control of looking behavior and facial expression.

Bi-directional communication between the components of this reflex arc provides for the proper functioning of the arc. Thus, it is plausible to assume that bi-directional communication also exists between these components and components of the social

engagement system via common neural structures (i.e., facial nerve, brain stem nuclei proximity).

Further evidence of the neural connections comprising a "listening system" is found in research investigating the relation between the stapedius reflex and olivocochlear reflexes. Olivocochlear reflexes are elicited by excitation of the outer hair cells (OHC) in the inner ear and are dependent on stapedius reflex dynamics (Burns, Harrison, Bulen, & Keefe, 1993). The outer hair cells are responsible for "fine-tuning" the frequencies reaching the basilar membrane. It is widely accepted that OHC motility in response to stimulation is the key element of a mechanical feedback loop on the basilar membrane (Buki, Wit, & Avan, 2000). When properly functioning, this feedback loop serves to amplify the input to sensory cells by as much as 40dB in a frequency selective manner (Dallos & Evans, 1995; Liberman & Dodds, 1984). Relative to the concept of a listening system, middle ear muscle functioning dynamically regulates the olivocochlear reflex. When the middle ear muscles are functioning properly, the extraction of human voice frequencies is enhanced and the OHCs serve to amplify this range of frequencies. However, at the threshold when a middle ear muscle reflex is elicited, the olivocochlear reflex is dampened and the amplification of these frequencies is lowered. To reiterate, the dynamic functioning of the middle ear is accomplished via muscles innervated by the facial nerve (CN VII), which is directly related to the fine-tuning of the frequencies reaching the basilar membrane (and therefore the perception of sound) as well as facial expression and eyelid opening (i.e., looking behavior). It is still unclear what the physiological meaning of the olivocochlear reflex is. It has been proposed that it may serve to

regulate the dynamic range of the cochlea and the cochlea's ability to extract low-level sounds within background noise (Kawase & Liberman, 1993; Liberman & Guinan Jr., 1998). A final important note is that the olivocochlear efferents also originate in the brainstem at the superior olivary complex. This complex is located directly adjacent to origin of the vagal system and has direct ties to vagal system pathways (i.e., facial nerve). It is plausible to assume that communication between the nuclei (direct and possibly indirect via vagal special sensory pathway connections to vestibulocochlear and optic nerves [see Table 2]) may integrate the regulation of the stapedius muscle and the olivocochlear reflex into the dynamic feedback loops of the social engagement system to enhance the looking-listening connection. Thus, the observed difficulties that many autistic individuals have in extracting human voice from background sounds may be dependent on the same neural system that regulates facial expression, looking behaviors and consequently, visceral homeostasis.

The Polyvagal Theory describes the integration of the evolutionary development of the mammalian nervous system and several components of the nervous system into a system (i.e., the Social Engagement System) that facilitates social engagement behaviors. Phylogenetic shifts in regulatory components of the mammalian nervous system have evolved to facilitate the interaction between neurobiological components and environmental influences that allows for social communication as a behavioral strategy for maneuvering in our environment. The model describes the anatomical and neurological changes that have taken place over time, and proposes that the integration of specific neural feedback loops facilitates specific adaptive strategies for engagements under various environmental settings. In

a safe environment, a properly functioning system enhances the physiological regulation of the viscera, listening and looking behaviors, communicative behavior, and social gesture behaviors. In unsafe environments, phylogenetically older systems are recruited and defensive mobilization strategies (i.e., fight/flight) are used to protect the organism. In life-threatening environments, the primitive system is recruited and defensive immobilization strategies (i.e., syncope, death feigning) are used to promote survival. Several behavioral and physiological predictions that can be extracted from an understanding of the functional properties of this system are manifested in psychiatric disorders, where evidence of defensive behavioral strategies (i.e., withdrawal, mobilization, immobilization, shut down) often mirror diagnostic criteria (psychological, physiological, and physical symptoms).

The vagus as a "system" provides a powerful organizing principle to investigate the behavioral, psychological, and physiological features associated with several psychiatric diagnoses. Observations of the behaviors and physiological response of autistic individuals suggests that they have great difficulties in recruiting the regulatory neural circuits of the Social Engagement System. Rather, it appears that autism is associated with autonomic states that support the adaptive defensive strategies of mobilization (i.e., fight-flight behaviors) or immobilization (i.e., shut-down). Behaviorally, the withdrawal of the neural regulation of the Social Engagement System would be expressed as limited use and regulation of the muscles of the face and head. Functionally, this retraction would limit facial expressions and head gestures, and would result in difficulties extracting human voice from background sounds, and a lack of prosody. It may also compromise the regulation of

visceral organs such as the heart. Although there is a limited scientific literature evaluating the role of the vagus in autism, the current study was designed to assess the plausibility of system-specific predictions, a key component of which is assessment of vagal functioning at the level of the heart.

Since vagal efferent pathways to the heart are cardioinhibitory, changes in cardiac vagal tone can influence the metrics used to monitor heart rate and heart rate variability (Porges, 2003). In general, greater cardiac vagal tone produces slower heart rate and regulates the transitory changes in heart rate in response to stimulation. The myelinated vagal efferents that synapse on the sino-atrial node have a respiratory rhythm. This rhythmic increase and decrease in cardioinhibitory activity through the vagus produces a heart rate rhythm known as respiratory sinus arrhythmia (RSA). The greater the cardioinhibitory influence through the vagus, the greater the rhythmic increases and decreases in the heart rate pattern. Therefore, the amplitude of RSA provides a sensitive index of the functional impact that the myelinated vagus has on the heart. The rapid changes in heart rate in response to specific stimuli are primarily under vagal control. The dynamic increases or decreases in cardioinhibitory activity through the myelinated vagus provide the characteristic heart rate pattern changes of an immediate deceleration, followed by either a continued deceleration or an acceleration. The literature suggests that autism is associated with reliable differences in the amplitude of respiratory sinus arrhythmia. For instance, an early publication by Hutt et al. (1975) reported that normal children suppressed respiratory sinus arrhythmia more than autistic children. Similarly, Althaus et al. (1999) found that PDD-NOS children did not suppress respiratory sinus arrhythmia. Studies have also

reported that autistic children have dampened heart rate responses to a variety of stimulation. Zahn et al. (1987) reported unusually small heart rate decelerations to auditory stimulation. Palkovitz and Wiesenfeld (1980) reported dampened heart rate responses to socially relevant speech, nonsense phrases, and a 500 Hz tone. Corona et al. (1998) reported that the heart rate of children with autism did not change across conditions.

The social engagement system model predicts that a deficit in the system would produce atypical social engagement behaviors such as a social withdrawal; improper communication (i.e., poor intonation and prosody); difficulty listening (inability to extract human voice from background noise); poor eye contact; inappropriate facial expressivity (i.e., flat affect); and atypical visceral functioning (i.e., low cardiac vagal tone). As discussed, these are precisely the indices associated with autism, as well as several other psychiatric disorders. In the current study, data related to a number of these indices were obtained to test the hypothesis that the engagement behaviors of autistic individuals may be the emergent properties of an adaptive strategy elicited by deficits in the functioning of the social engagement system.

CHAPTER III

AUTISM

> "We must, then assume that these children have come into the world with innate inability to form the usual, biologically provided affective contact with people…(Kanner, 1943)"

Definition

The DSM-IV (APA, 1994) criteria describe autism as a pervasive developmental disorder that is defined by impairments in social and communication functions, and by atypical behavior patterns. Areas in which social deficiencies occur include poor eye contact, a reduction in the ability to interpret emotion states, failure to develop peer relations and deficiencies in social-emotional reciprocity (APA, 1994). The central communication problems include either a delay or lack of expressive language, as well as a marked impairment in nonverbal communicative behavior (facial expression, gesturing). Reduced prosody and intonation, as well as impairments in the use of figurative language are also associated with autism (Ornitz, 1988). In cases where individuals do develop language, they often have deficits in initiating and sustaining conversations (APA, 1994). The following sections describe research on the core diagnostic components of autism as they relate to the social engagement system model.

While many autistic individuals exhibit normal intelligence, there is a clear dysfunction in the way in which they interact with the people in their surroundings.

Recently, there has been a shift in autism research to gain an understanding of the etiology of the disorder and researchers have focused on specific biological, neurological, and genetic indicators of the disorder. While there is still along way to go, studies in the area of autism have provided useful information for guiding continuing research, and technological advances have provided the tools for advancing methodologies used in this area. Currently, researchers are studying brain functioning (i.e., EEG, fMRI), auditory functioning (i.e., auditory brainstem responses; ABR), genetic markers, and eye gaze (i.e., eye tracking systems) to name a few. Studies in these areas are uncovering evidence supporting the concept that autism, and disorders with similar features (i.e., anxiety, depression, language and communication disorders), is best described as a "spectrum" disorder. Although the concept of autism as a spectrum disorder is not novel, research is beginning to make connections between the core deficits of autism and specific biological systems. Advances in technology have provided less invasive and more powerful tools that have enhanced the ability to begin making these connections and gain a better understanding of the etiological and functional issues associated with the disorder.

The two primary indices of autism are deficits in social interactions and social communication. The following sections describe research in both of these areas as they relate to a specific biologically based systems model for understanding autism-related behaviors as well as similar behaviors associated with other clinical diagnoses

Social Interaction

The social engagement system model defines a specific system of neural feedback loops responsible for the regulation of our ability to be socially engaging.

More specifically, components within the model are intimately linked to eye gaze, facial affect, prosody and intonation, and in general our physiological "availability" for initiating and maintaining social engagements. This section describes research related to the somatomotor components of the social engagement system and how these components are related to the core deficits of autism.

Faces represent the most important stimuli available in any social interaction (Posamentier & Abdi, 2003). When we look at faces, two important sources of information become available to us, identity and emotion. Normal individuals have a looking preference for the eyes and mouths of human faces (Yarbus, 1967; Walker-Smith, Gale, & Findlay, 1977; Mertens, Siegmund, & Gruesser, 1993). It has been argued that the eyes and mouth portray the largest amount of information conveyed from the face. For instance, information about the mental state of a person and affective information can be perceived by looking at another person's face. From this information we have the ability to decide whether or not to engage with a person (familiar, unfamiliar) and even how to engage (based on facial expression and emotional cues). For most of us, this decision is made with relative ease and speed, suggesting a specialized system for engagement. In contrast, individuals who cannot (or do not) decipher such facial information may have great difficulty in actively engaging with, or maintaining interactions with others. The social engagement system proposes just such a specialized system.

The social engagement system model proposes that a well functioning system enhances our ability to interact with others both verbally and nonverbally. A key component of nonverbal communication relies on facial information exchanged

between individuals. This includes appropriate eye contact and facial expression, as well as facial tone and social gestures (i.e., head nodding and orientating). Components of the social engagement system provide the efferent and afferent neural connections that help us regulate gaze behavior, orienting, and the facial muscles associated with these behaviors.

The literature on the looking behaviors of autistic children has provided important information regarding differences in gaze behaviors between children with autistic spectrum disorders and normal children. Since Kanner's (1943) original description of autism, one of the most frequently reported clinical observations of autistic children is that they display abnormal looking behaviors towards other individuals (Baron-Cohen, 1988, 1995; Rutter, 1978; Volkmar & Mayes, 1990). Several studies have shown that autistic children have a lower frequency of looking at others as compared to normal children (Hutt & Ounsted, 1966; Klin, Jones, Schultz, Volkmar, & Cohen, 2002; Pedersen, Livoir-Petersen, & Schelde, 1989; Volkmar & Hayes, 1990). For instance, Klin et. al. (2002) studied visual fixations in a group of 15 autistic males and a matched control group. Using eye-tracking technology, fixation parameters for specific areas of interest (i.e., eyes, mouth, body, object) were assessed and compared between groups. Fixation patterns were also correlated with measures of social competence. Klin et. al. reported that individuals with autism show atypical fixation patterns to all of the areas of interest. More specifically, the autistic individuals fixated twice as much on the mouth, body, and object regions, and half as much on the eye regions, than the control group when looking at naturalistic social situations. The best indicator of social competence was the fixation patterns towards

the face in general, and more specifically towards the eyes. This means that individuals who spend more time fixating on the face perform better on measures of social competence, lending further support to the proposal that the face is a key component of social interaction. In contrast to the Klin et. al. study (2002) which utilized a video clip of a movie in which actors engaged with other actors but not with the subject, the current study utilized a video clip of an individual talking directly to, and maintaining eye contact with the subject.

Other studies have reported that autistic individuals look at another individual as much as normal controls, but that the *way* autistic individuals look is different. For instance, Buitelaar, van Engeland, de Kogel, de Vries, and van Hooff (1991) have shown that autistic individuals will look at the other's face as long as their attention is drawn to it. Even so, the specific regions that are focused on are different than those of normal control individuals. These results suggest that autistic individuals perform similar looking behaviors as controls if they are not actively engaged in the situation. Hobson and Lee (1998) showed similar findings in a study designed to test engagement-behavior differences between autistic and age and verbal IQ-matched non-autistic, mentally retarded individuals. In this study, individuals engaged with an unfamiliar adult through a number of stages (i.e., greeting, conversation, farewell). They found that those with autism were less likely to offer spontaneous verbal and nonverbal gestures for both the greeting and farewell stages, and were less likely to establish eye contact even when they were offered a greeting. There were also fewer autistic subjects who smiled, or who waved goodbye.

A recent study by van der Geest, Kemner, Camfferman, Verbaten, and van Engeland (2002a), used eye tracking technology to test whether autistic children show a *general* looking problem reflected in different overall fixation parameters, and whether autistic children have a *specific* looking problem related to social stimuli. The results of the study, which used a human figure and neutral objects within the same drawing, showed that in general, autistic individuals show similar numbers of fixations as normal individuals on the human figure within the drawing. As far as a *general* looking problem, the study showed that autistic children spent the same amount of time inspecting the pictures and looking at the human figure, suggesting that the looking behaviors are not necessarily a general looking problem or a social stimuli problem.

Another study on the gaze behavior of children with PDD by van der Geest, Kemner, Verbaten, and van Engeland (2002b) used pictures of human faces in different orientations as stimuli and reached similar conclusions. The children with PDD showed the same fixation patterns as controls for upright faces regardless of emotional expression and while normal children looked less at upside-down faces, the fixation times of the PDD children did not differ according to face orientation. The authors concluded that the abnormal looking behaviors were not due to the presence of a social stimulus per se, but instead may be related more to other factors such as requirements for social interaction.

It should be noted that in the first study reported above, the cartoon-like human figure drawings within the stimuli picture (similar to what a young child might draw) were considered the social stimuli. This type of stimuli may be inherently different

than what might be seen during an interaction with another person, or even watching a video of a person telling a story. Similarly, the latter study used static pictures of human faces in various orientations as social stimuli. Again, the gaze behaviors expected from this type of stimuli may be quite different than those elicited from a true social interaction. These studies provide information regarding the gaze behaviors of autism individuals when interaction is removed from the social stimuli, and therefore provide support for the current studies proposal that the actual interaction is the key to understanding deficits in social behavior of individuals with autism. To date there have been no reported studies that have attempted to simulate a more realistic situation of a social interaction between an individual with autism and another person in order to quantify the gaze behaviors using objective (i.e., eye trackers vs. coding of video) measurements for quantifying these behaviors. Advance has allowed more interactive studies of gaze behaviors accessible to researchers, providing a greater understanding of the dynamics of these interactions for autism and several other psychiatric disorders.

The current study focuses on the gaze behaviors of individuals with autism during social interactions, and the specific locations (regions of interest) that individuals are fixating upon (i.e., eyes, mouth, or off target) when the stimuli are more socially engaging. From a social engagement system model differential looking behaviors should be quantifiable in individuals with autism over specific regions of interest (ROIs), especially when the stimuli used represents more closely a direct social interaction.

Social Communication

The DSM-IV lists a number of qualitative abnormalities in communication associated with autism. These include a delay or a total lack of development of spoken language, a failure to initiate or sustain a conversation and a lack of reciprocity and communicative spontaneity. Taken alone, these criteria could describe a number of psychiatric diagnoses. However, when coupled with the atypical social behaviors described above, a diagnosis of autism primarily follows. The lack of intonation and prosody exhibited by autistic individuals is incorporated into the diagnostic criteria (APA, 1994), and several recent reviews have outlined the qualitative impairments in social communication in individuals with autism (see for instance Dissanayake & Sigman, 2001; Mundy, 1995).

Communication differences may also include: echolalia (repeating what has been heard; inflexible thinking; obscure speech; repetitive questions and persistent monologue about favorite interests without regard for the listener's response. Most of these language issues are a part of *pragmatic* language which includes facial expressions, gestures, body language, and the unspoken nuances of language that give meaning to what we communicate. The social engagement system provides a basis for understanding these difficulties. For example, if the social engagement system were not functioning properly, an individual would have difficulty extracting human voice from the environment (via middle ear muscle and facial nerve functioning). It is a well-established fact that impairments in audition lead to speech impairments, which may account for a number of the speech deficits of autism. Adding to this plausible relation is the fact that a deficit in the social engagement system is also related to

neural pathways for the regulation of intonation and prosody (i.e., via vagal and glossopharyngeal fibers). The fact that most of the issues are a part of pragmatic (socially relevant) language problems further supports the role of the social engagement system. The social engagement specifically predicts deficits in the pragmatic components of language (i.e., facial expression, gesture, body language etc.) as an outcome of a compromise in the system.

Summary

The Social Engagement System model was proposed to integrate the neurobiological feedback loops that provide the basis for social engagement behaviors. Based on the Polyvagal Theory's description of the phylogenetic shifts in autonomic nervous system regulation and dynamics, the Social Engagement System was conceptualized to describe the integration of visceromotor and somatomotor components of the vagal system that enrich or restrict our social behavior repertoire. The model predicts that a deficit in the system would produce atypical social engagement behaviors such as a social withdrawal; improper communication (i.e., poor intonation and a lack of prosody); difficulty listening (inability to extract human voice from background noise); poor eye contact; inappropriate facial expressivity (i.e., flat affect); and atypical visceral functioning (i.e., low cardiac vagal tone). These components are precisely the indices associated with autism. Thus, it is plausible to hypothesize that autism represents the extreme end of the emergent properties of functional deficits in the system. Specific hypotheses can be extracted from the social engagement system model, many of which directly relate to clinical features of several psychiatric disorders. Currently, the literature on deficits as integrated components of

a system is lacking. However, research on specific components within the social engagement system does support the model of an underlying neurobiological system deficit. The current research was designed to test several social engagement system components from the position that relations predicted by this system model would provide further support for specific atypical behaviors (i.e., those associated with autism) as representing a compromised social engagement system. Thus, research, assessment and intervention strategies designed on this system model may provide a better understanding of psychiatric disorders such as autism and other disorders with similar features.

Hypotheses

Based on predictions derived from the Polyvagal Theory and the Social Engagement System model, the following hypotheses were formulated for the current study. As compared to a non-autistic control group, individuals with autism will have:

1). Lower levels of mean cardiac vagal tone
2). Poorer SCAN FW scores
3). Poorer SCAN CW scores
4). Lower indications of REA
5). Fewer EYE fixations
6). More OFF fixations

CHAPTER IV

JUSTIFICATION OF THE MEASURES

Subjects

All of the autistic individuals were able to complete the tasks presented in the study. A general characterization of this group would be that they ranged from mid- to high functioning. While it is common for researchers to match autistic subjects on age and mental ability and/or verbal ability, the variables included in the current study required no cognitive demands that would justify the use of mental age or verbal ability as a controlling factor. The SCAN test, which represents the only cognitive-type task in the study, was designed as a simple repetition task to avoid the possibility of influencing results based on cognitive ability. This test also provides normative data allowing for standardization of scores and enables direct comparisons across age groups. Individual subtest scores were converted to their standardized values using standardization tables provided with the test administration manual. Statistically the use of a control variable (i.e., mental age) makes the assumption that there is a linear relation between the control variable and the dependent variables and therefore, this possible relation was assessed for both the control (using age) and autistic group (using age, mental age, and age controlling for mental age) on the research variables (presented below). The use of a matching variable other than chronological age for physiological measures may be inherently confounding. Furthermore, several studies using age and verbal ability or IQ have reported that no relation was found between matching variables and research variables (i.e., Langdell, 1978; Valentine, 1988;

Volkmar, Sparrow, Rende, and Cohen, 1989; Tantam, Monaghan, Nicholson, and Sterling, 1989, Klin et. al., 1999).

Audiogram

A standard audiogram procedure was used prior to administration of the SCAN tasks and the eye-tracking task for two main purposes. First, the audiogram was used as a screening tool to assess the level of cooperation that the autistic individuals would be able to provide during subsequent tasks. While a standard audiogram is not an invasive procedure to most of us, the concept of was constitutes an invasive procedure is often very different in individuals with autism. For instance, many autistic individuals are sensitive to sound, light, and contact by both people and objects (i.e., headphones). A primary focus of the current research was to remain non-invasive and to consider the difficulties individuals may have with the testing procedures. Thus, an a priori decision of the research was to exclude those who were unable to accomplish the screening tasks. Second, the audiogram test was presented to rule out hearing impairments that might possibly affect the SCAN measures, and confound the gaze analyses (i.e., more fixations on mouth due to difficulty hearing the story). All of the individuals who agreed to participate completed the hearing assessment, and none had difficulties that required withdrawal from the study.

SCAN

The SCAN tests (Keith, 1994, 2000) are diagnostic tools for auditory processing disorders. Subtests for both the adult and child versions (A and C respectively) were selected to measure specific features of auditory functioning. The "Filtered Words" (FW) subtest was designed to assess an individual's ability to perceive speech that is compromised by a poor acoustic environment. The FW subtest represents the functional auditory abilities in everyday listening situations. These situations include when speech is heard in the presence of background noise. This subtest was chosen because one prediction from the social engagement system model states that a deficit in the system would lead to difficulties in the extraction of human voice from background noise. The "Competing Words" (CW) subtest is a dichotic listening task. This test assesses the development of the auditory system, auditory maturation, and hemispheric lateralization. Dichotic listening performance, and auditory lateralization measures are often used for assessing the difficulties seen in children with language and learning disorders (Scanlon & Vellutino, 1996). The diagnostic criteria for autism also include difficulties in language and speech. Because the primary pathway for the perception of human speech is from right ear to left hemisphere, most (but not all) individuals show a right ear advantage for processing human speech. The differential score between performances on the right vs. left ear of the SCAN CW subtest provides an indication of the right ear advantage (Keith, 1994, 2000). Extracting from the social engagement system model, autistic individuals should show deficits in tasks designed to assess dichotic listening ability and the right ear advantage. This conceptualization comes from the fact that the dynamic functioning of the middle ear muscles is related to the ability to extract human voice from background sounds.

Thus, if there were a deficit in this system, it is plausible that hemispheric lateralization may also be affected. The argument would follow that if regulation of the middle ear muscles were compromised, the ability to extract human voice would be compromised, which in turn may have an affect on the development of lateralization for human voice.

Tympanometry measurements, where the stapedius reflex threshold differences between the left and right ears, are often used to confirm the ear advantage. However, tympanometry tests are somewhat invasive, and often aversive due to the level of stimulus that must be presented in order to elicit the reflex (i.e., 70-80 dB above threshold). While the use of tympanometry was considered for the current study, pilot testing with a few individuals with autism proved that the measure would be difficult to consistently obtain from many of the individuals. Therefore, in the interest of maintaining a non-invasive, non-aversive protocol, it was decided that tympanometry would not be used to confirm the results of the SCAN test for ear advantage.

The SCAN tests provide standardization algorithms that allow for comparisons between age groups. Because it was assumed that a wide range of participants would be involved, the SCAN test provided a way to accurately test individuals of all ages and make comparisons between the ages using standardized scores. Importantly, the SCAN test was also chosen because of the low task demands of this particular test. The SCAN is an imitative test that requires only that subjects repeat the stimulus words. It requires no higher level cognitive functioning, which might influence the scores of special populations such as individuals with autism. This type of test also

avoids the cross modality and cognitive aspects of having to point at a picture in response to a word.

Finally, the SCAN test was chosen because of the validity and reliability metrics reported. For all ages, Cronbach's alpha for measures of internal consistency and test-retest reliability, were above .60 for each of the subtests used. Tables 5 and 6 provide psychometric data for the FW and CW subtests for SCAN-A and SCAN-C respectively.

Internal Consistency			**Test-Retest Reliability**	
Test	Mean (SD)	Cronbach's alpha (SEM)	Means Test/retest	SD Test/retest
FW	35.2 (3.0)	.65 (1.7)	34.7 / 34.9	2.1 / 2.5
CW	53.5 (4.0)	.69 (2.2)	55.0 / 56.1	3.1 / 2.6

Table 4: Psychometric data for the SCAN-A subtests. FW = Filtered words; CW = Competing Words. The SCAN-A test is for individuals 12yrs. and older (adapted from Keith, 1994).

The variation in scores on each of the SCAN-A subtests in the test-retest was too limited, therefore alpha coefficients for individual tests were not reported (Keith, 1994). The test-retest alpha coefficient for total test score for the SCAN-A was .69.

Internal Consistency			Test-Retest Reliability		
Age	Test	Alpha	Means Test/retest	SD Test/retest	Alpha (corrected)
5	FW CW	.83 .87			
6	FW CW	.77 .89	**FW** 10.17/11.63	2.88/2.95	.66
7	FW CW	.77 .87	**CW** 10.82/11.99	2.67/3.06	.82
8	FW CW	.76 .86			
9	FW CW	.70 .85	**FW** 9.71/10.79	3.12/2.68	.75
10-11	FW CW	.64 .79	**CW** 9.65/11.43	3.00/3.01	.78

Table 5: Psychometric data for the SCAN-C subtests. FW = Filtered words; CW = Competing Words. The SCAN-C test is for individuals 5-11yrs. old. The test-retest metrics were computed on combined groups (i.e., 5-7yrs. & 8-11yrs.) and Cronbach's alpha was corrected for age (adapted from Keith, 2000).

Eye tracking

One of the predominant features of autism is the lack of eye contact. As discussed previously, the majority of research on individuals with autism makes note of the lack of eye contact. Recent research on the gaze behaviors of individuals with autism also shows differing patterns of looking behavior (Klin et. al., 2002; van der Geest et. al. 2002a, 2002b). However, the extent to which these studies used a social stimulus that was directly relevant to the individual is questionable (i.e., a movie of other people's social interactions, photos, and stick-figure drawings). The current study was designed to test the gaze behavior of autistic individuals using a stimulus that was more directly targeted to the subject. The videos used in this study were of a person telling a story while looking directly at the individual watching. Because of the importance of information extracted from the eyes and mouth regions, specific regions

of interest (ROI) were the variables of interest. Three specific ROIs were defined a priori that included the eye region, the mouth region, and an off region, which included any fixations on (or slightly beyond) the presentation monitor, but not in the other ROIs (i.e., EYE or MOUTH). Using this design, differences in the gaze behaviors of autistic individuals can be tested when the stimulus more closely resembled a direct social interaction.

Vagal regulation of the heart

The study of cardiac vagal tone (i.e. the amplitude of RSA) is well established in the literature as a means of assessing vagal control of the heart. The amplitude of RSA (i.e., vagal tone) provides a sensitive index of the functional impact that the myelinated vagus has on the heart. As mentioned, the literature suggests that autism is associated with reliable differences in the amplitude of respiratory sinus arrhythmia. Quantification of cardiac vagal tone was accomplished using collection, editing, and analysis methods designed and standardized by Porges (1985). These standardized methods have proven to be reliable measures of the functional impact of the vagus nerve on the sino-atrial node of the heart, and are being used by over 100 labs worldwide (Porges, personal communication). A recent study by Denver & Porges (in press) showed that this method is related to accurate quantification methods in both the time and frequency domains to near unity (see also Porges and Byrne, 1992).

CHAPTER V

METHODS

Participants

Inclusion Criteria: Only subjects who passed the audiometry test and had verbal capabilities high enough to complete the SCAN task (i.e., were able to repeat words verbatim and without pronunciation errors) were considered for the study. Also, participants who were taking medication that may affect cardiac vagal tone (i.e., antihistamines, anxiolytics, anti-psychotic medications) were excluded from the study.

A total of forty participants (31 male) between the ages of 9-24 (mean = 16.2, SD 3.6) participated in the study. The majority of the autistic group was recruited through the Easter Seals Day School in Chicago. School officials identified an initial pool of 35 candidates who were diagnosed as autistic and currently on no medications. Of these 35 potential subjects, 21 met the inclusion criteria. Of these 21 individuals, 17 provided consent and participated in the study. The remaining three autistic individuals contacted the lab to inquire about participating in the study after learning of the research through advertisements, flyers, or presentations. The diagnosis for these individuals was confirmed using the ADI-R (Lord, Rutter, & LeCouteur, 1994). The characteristics of the groups are presented in Table 6.

	Group	
	Autistic	Control
	Mean (SD)	Mean (SD)
N	20	20
Males	18	13
CA	15.9 (2.9)	16.5 (4.2)
MA	10.1 (2.7)	
Ethnicity		
AA	11	4
Hisp	3	3
Cauc	6	13

Table 6: Characteristics for autistic and control groups. CA = chronological age; MA = mental age; AA= African American; Hisp. = Hispanic; Cauc. = Caucasian.

None of the control group reported a psychiatric diagnosis and each of these individuals (or parents) reported appropriate grade-level school performance. Also, no measure of mental age was taken for the control group. Therefore, the chronological age of the control group was also used as the mental age index. Mental age for the autistic group was provided from school records.

Apparatus

Hearing Assessment

An MA 41 portable audiometer (Maico Diagnostics, Eden Prairie, MN.) was used to perform standard pure-tone audiometric tests. Specific tones were presented via headphones four times each to each ear 500, 1000, 2000, and 4000Hz in a random order.

Auditory Processing Assessment

The SCAN-A (over age 12) and SCAN-C (6-12 yr-olds) tests for auditory processing disorders (Keith, 1994, 2000) were used to assess the ability to extract human voice from background noise, performance on a dichotic listening task, and to obtain an index of right ear advantage (REA). The specific subtests used were 1) Filtered Words and 2) Competing Words. Presentation of the SCAN tests was performed on a stand-alone CD player (Marantz model CC4000, Marantz America, Itasca, IL.). A digital sound level meter (Radioshack SLM 33-2055, Ft. Worth, TX.) was used to calibrate the decibel (dB) level of the SCAN test to 60dB-C using the first track of the SCAN CD (a 13 second calibration pure tone). This level was selected because it was shown to be a comfortable level for this room during pilot testing. Beyerhauser model 801 professional circum-aural, noise-limiting headphones were used to present the SCAN subtests.

Eye-Gaze

To collect and quantify the eye-gaze data, an ASL model 504 Eye-Tracking System was used (ASL, Bedford, MA.). This system is composed of an illuminated optical pan/tilt/zoom camera, a magnetic head tracker unit, a control PC, and a scene presentation PC. To optimize the accuracy of the pupil coordinates obtained by the optical camera, the ASL 504 is equipped with a Flock of Birds magnetic head tracking unit (Ascension, Burlington, VT). A small (1 in^2) transmitter is fixed on a headband directly above the eye that will be tracked and a receiver unit is located above and behind (6" each) the subject's head. The X,Y,Z coordinates and the degrees of azimuth, elevation, and roll of the head is collected by the system and used to maintain

the accuracy of the pupil coordinates. Prior to testing, the eye tracking equipment was calibrated to the specific equipment setup. Eye-to-head distance, eye-to-scene monitor distance, and head tracker receiver-to-transmitter coordinates were calculated and entered into the system's operating software and tested for accuracy.

The eye tracking system uses edge detection algorithms to locate and track corneal reflection and bright-pupil location and collects the X-Y coordinates of the separation between these two using an optical camera. The system then transposes these coordinates to correspond to locations on the monitor showing the scene being viewed. The eye tracking system was run on a *PIII* computer. A *PIII* computer with a 19" monitor at a resolution of 1024 X 768 (60Hz refresh rate) was used for presentation of the scene for the eye-tracking task.

Heart Rate

An EZ-IBI ambulatory heart rate monitor (UFI, Morro Bay, CA.) was used for the collection of heart rate data using self-adhering electrodes in a standard three-lead configuration. Using a sampling rate of 1000Hz., the EZ-IBI detected the peak of the R-wave to the nearest millisecond, timed the sequential R-R intervals (i.e., heart periods) to the nearest msec. and stored the data on a *PII* laptop for off-line analysis.

Procedure

Upon completion of the consent documents and a demographic questionnaire, participants were brought to a quiet room and seated in a comfortable chair for the audiometric testing. The pure-tone audiometric test was performed first. The audiometer headphones were placed over the participant's ears in the correct

configuration (i.e., right ear cup to right ear). The experimenter set the tone presentation level to 30 dB (SPL) and presented four tones to each ear in a random order. Each of the four tones was presented to each of the ears four times. Participants were instructed to respond to the tones by verbally responding or raising the hand that corresponded to the ear in which they heard the tone. The experimenter recorded the number of correct responses for each tone in each ear. Following the audiometry test, participants were brought to another room that contained the heart rate, SCAN, and eye tracking equipment.

Participants were seated in a comfortable chair in front of the eye tracking camera and presentation monitor. The ECG electrodes were placed in a three lead (right shoulder, V6, right abdomen) configuration and the heart rate monitor was started. Once the monitor was collecting stable data (i.e., free from artifact), collection of the heart rate data began and continued throughout the experiment. Next, the experimenter confirmed that the CD player used for the SCAN test was calibrated to 60dB-C using the digital sound level meter, after which participants were given headphones to start the SCAN test. The headphones were placed in the proper configuration (i.e., right cup/right ear) and the SCAN test CD was started. During the presentation of the two SCAN subtests, the experimenter recorded the participant's responses on the answer sheet provided with the test. Following the SCAN, participants were prepared for the eye-tracking task.

For the eye-tracking task participants were seated with their eyes at a distance of 39 in. from the monitor used to present the stimuli. The chair was raised or lowered in order to bring the participant's pupil into the eye-tracking camera's line of site. A

headband for the magnetic head tracker transmitter was placed around the participant's head and the transmitter was attached to the band directly above the appropriate eye. In this arrangement, the optical camera's field of view is approximately 12 ½ degrees from center, 5 degrees below and 20 degrees above the horizontal line of gaze of the participant. From this position, each participant's line of gaze was centered in the middle of the presentation monitor. In order to maintain these parameters for all participants, the chair was fitted with four hydraulic lifters that enabled the experimenter to raise or lower the individual so that each participant's line-of-site was centered on the presentation monitor. Using this method, the eye-tracking camera could be fixed in place, thereby avoiding any possibility of data being corrupted due to movement of the equipment. Once participants were properly aligned, the lights in the room were dimmed and the eye tracking equipment was turned on. The experimenter asked the participant to get comfortable in the chair and refrain from moving their head during the eye-tracking task. After the participant was comfortable, the experimenter remotely focused the optics camera on the participant's pupil. The camera's illuminator was switched on and the experimenter adjusted the pupil and corneal reflection parameters so that the system could lock on to the participant's exact point of gaze. Prior to the start of data collection, the eye-tracking system was calibrated to each participant. This was accomplished using a 9-point calibration pattern on the scene presentation monitor. The 9 points were arranged so that there was a left, center, and right edge point for the top, center, and bottom of the scene presentation monitor, effectively outlining the boundaries of the viewable portion of the monitor. The calibration target points were circles with a diameter of

approximately 3/8 inch. To check the accuracy of the calibration, a second calibration target procedure was set up. This target pattern consisted of three rotating dots located in a diagonal pattern across the screen that subtending the entire visible portion of the monitor (i.e., top-left to bottom-right corners). Participants were told that circles were going to appear on the screen and were instructed to look at them as quickly as possible. The calibration procedure was considered accurate when the crosshairs corresponding to gaze location remained within the boundaries of the rotating targets. The rotating targets were shown one at a time. Once the participant was calibrated, the calibration data were stored by the system and subsequent data coordinates corresponded to precise locations on the scene presentation monitor. At this point, the video presentation was started and eye gaze data collection began.

A brief movie (approx. 4 ½ min.) was presented to the participant. One of three possible videos was shown (randomized between participants) while an unfamiliar individual read a story. The video was created so that the storyteller's face was life-sized, as viewed by the participants, when played in full screen mode on the monitor. The storyteller's face remained stationary during the presentation so that the parameters for creating regions of interest (ROIs) and data corresponding to specific ROIs could be assessed off-line. ROIs were defined as the areas surrounding the eyes (EYE) and the mouth (MOUTH). For all other areas within the boundaries of the visible area of the presentation monitor, data was coded as OFF. For coordinates outside of the boundaries of the presentation monitor, the eye-tracking software automatically computes the coordinates as invalid data and they are not included in the analyses to avoid confounding data coded as OFF. Invalid data sections were not

included in the analysis data segment. During the video presentation, the experimenter assured that the optics camera, corneal reflection, and pupil remained in focus and centered in the eye tracker monitor. Once the video was complete, eye tracking data collection was stopped and the participants were told they no longer needed to sit still. Following the eye-tracking task, the head tracker headband was removed, the heart rate monitor was turned off, and the electrodes were removed from the participant. This marked the completion of the research. Prior to being dismissed, participants were allowed to ask questions and were thanked for their help in conducting the research.

CHAPTER VI

RESULTS

Data Reduction

Cardiac Vagal Tone

Heart period data were edited off-line with MXedit software (Delta Biometrics Inc., Bethesda, MD). Editing is accomplished via visual detection of outlier points followed by integer division or summation. Outliers in the data not associated with movement artifacts are most often caused by a missed R-peak or the detection of two or more R-peaks in a short time interval. Heart period and RSA were calculated with MXedit in accordance with the procedure developed by Porges (1985). This method quantifies the amplitude of RSA in the following steps: (a) R-R intervals are timed to the nearest millisecond to produce a time series of sequential heart periods; (b) the sequential heart periods are converted into a new time-series by resampling into sequential 500msec. intervals; (c) the time-based series is detrended by a 3rd order 21-point moving polynomial filter (Porges & Bohrer, 1990) that is stepped through the data creating a smoothed template; (d) the template is then subtracted from the original time series to generate a detrended residual series; (e) the residualized time series is band passed to extract the variance in the heart period data contained within the band of frequencies associated with spontaneous breathing (i.e., 0.12-.40 Hz); and (f) the natural logarithm of the variance of the band passed time series is calculated as the measure of the amplitude of RSA. This measure of the amplitude of RSA defines the term cardiac vagal tone for the study. These procedures are statistically equivalent to frequency domain methods (i.e., spectral analysis) for the calculation of the

amplitude of RSA when heart period data are stationary (Porges & Byrne, 1992; see also Denver & Porges, in press).

SCAN

Raw scores for each of the SCAN subtests were converted into standardized scores using the procedure outlined in the appropriate SCAN administration manual. The standardization of scores allowed for direct comparisons to be made across ages and between the two versions of the test (i.e., Adult version, Child version). Following the standardization procedure, the distribution of standardized scores was identical for all ages (i.e., mean = 10, SD = 3).

Eye Tracking Data

The video presentations for the eye-tracking task were approximately 4 ½ minutes long. In order to maintain consistency in the data files, analysis of each eye-tracking file was performed on a pre-specified range within the approximately 4½ minutes of total data collection time. To allow for accommodation of the subject to the task, the first 10 seconds of eye data were not analyzed. To maintain a consistent data length across subjects, the next 240 seconds of valid data (i.e., data within the range of the camera) were used in the analyses. Data beyond the 240-second mark were not used in the analyses. Thus, 240 seconds of valid data for each participant was used in the analyses. This procedure allowed for a brief period for accommodating to the task, and removed the possible confound of variations in eye data due to variations in file-length or inclusion of invalid data.

Test-Retest assessment for Eye-Tracking

To test whether the looking behaviors of autistic individuals were stable, nine

of the autistic group were retested on the eye-tracking task. The fixation metrics used in the current study are described in Table 7. Retesting was completed approximately 5 days after the initial testing (mean = 5.33, range = 4-7) using a different video than previously seen by each participant.

Metric	Abbreviation	Definition
Sum of Fixation Duration	SFD	Total time in seconds spent fixating a specific ROI
Fixation Duration Percent	FDP	Duration of fixations for a specific ROI as a percent of total fixations
Sum of Fixation Counts	SFC	Total number of fixations for a specific ROI
Fixation Count Percent	FCP	Percent of fixations on a specific ROI (number of fixations in a specific ROI divided by total fixations)
Mean Fixation Duration	MFD	The mean duration in seconds, of fixations on each ROI

Table 7: Eye tracking metrics and definitions for the current study. ROI= region of interest.

Paired samples t-tests (see Table 8) revealed no significant differences for the eye-tracking variables (all p values >.05) and the correlations between the pairs (n = 9) ranged from r = .972 to r = .982 for the OFF ROI, and from r = .806 to r = .955 for the EYE ROI. The analysis also revealed no significant differences for the MOUTH ROI (p values were all >.05, r values ranged from .92 - .95). Thus, it was concluded that the eye-tracking task would accurately assess a reliable measure of the normal looking behaviors of autistic individuals to the task, and that reliable differences obtained

between the research groups could be quantified.

Metric	Mean Test	Mean Retest	t	r
OFF SFD	88.47	87.77	.320	.97
EYE SFD	22.57	20.99	.563	.94
MTH SFD	36.58	38.4	-.571	.95
OFF FDP	62.64	61.79	.448	.98
EYE FDP	14.07	13.75	.211	.94
MTH FDP	23.29	24.46	-.578	.94
OFF SFC	229.89	231.89	-.325	.98
EYE SFC	44.67	38.89	1.27	.93
MTH SFC	52.33	51.56	.133	.92
OFF FCP	69.24	71.07	-1.22	.98
EYE FCP	14.15	12.32	1.39	.96
MTH FCP	52.33	51.56	.133	.92
OFF MFD	.416	.403	.973	.97
EYE MFD	.404	.469	-1.26	.81
MTH MFD	.723	.719	.082	.93

Table 8: Results from the paired samples t-test for the eye tracker test/retest measure. There were no significant differences and high correlations for each of the metrics collected by the eye-tracking system.

Eye-Tracking coordinate accuracy check

To ensure that the eye-tracker coordinates were accurately collected, an accuracy mark on a still-frame of the video was tested against manually entered coordinates that corresponded to the XY line of sight coordinates collected by the system. Using the eye-tracking Eyenal analysis software (ASL, Bedford, MA), coordinates for a mock ROI were entered that corresponded to the coordinates of the accuracy mark extracted during the calibration procedure. Specifically, the crosshairs on the eye-tracker monitor were placed directly over a mark (i.e., a freckle) on the face of the storyteller, and the tracking coordinates displayed by the system were recorded. Coordinates for eye gaze position collected by the system are based on the location of

the XY crosshairs. Thus, placing the crosshairs in a specific area on the presentation monitor simulates exactly the coordinate data that would be collected if a subject looked in this exact location. Using the analysis software, a region of interest was created using these simulated eye gaze coordinates. When this "mock" ROI (approx. 4 pixels x 4 pixels) was placed over the still-frame of the storyteller's face, it landed directly over the accuracy mark on the storyteller's face. Thus, the accuracy of the eye-tracking coordinates was confirmed. Figure 5 shows the location of the accuracy mark used for accuracy testing and a representation of the ROIs used in the study.

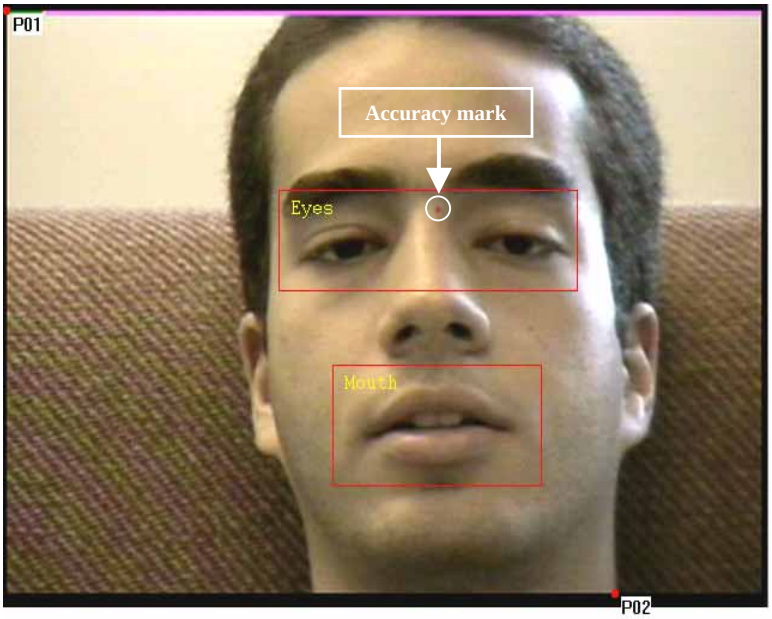

Figure 5: Location of the accuracy mark on the video still-frame of the storyteller's face. The 4 x 4-pixel mock ROI (the small red dot within the circle) subtended the accuracy mark when the eye-tracker coordinates were used to create the ROI. The EYE and MOUTH ROIs are also represented.

Eye-Tracker coordinates

Tracking Principle: The Eye Tracker optics module is designed so that the near infrared eye illumination beam is nearly coaxial with the optical axis of the pupil

camera. The camera lens captures the beam that is reflected back from the retina, and the image reaching the camera sensor is that of a backlit bright pupil. This bright pupil image can usually be much more easily discriminated from the iris and other background than could a black pupil image. Note that the amount of reflected light that reaches the camera from the retina is proportional to pupil diameter. Pupil brightness therefore varies significantly with pupil diameter. Even when a subject's pupil is at its largest and brightest, the reflection of the illuminator from the front surface of the cornea (corneal reflection or CR) is normally much brighter than the pupil. Thus the pupil can usually be distinguished from the background and the CR can be distinguished from the pupil on the basis of brightness. When a subject's pupil becomes very small (3 to 4 mm diameter), sections of the eyelid, cheek, or sclera that are also on the camera field often appear as bright as the pupil. In these cases, size, shape, and smoothness criteria must be used to help identify the pupil. In some cases more than one area will be as bright as the CR. If more than one bright spot will satisfy the proper size and shape criteria, the computer selects the spot closest to the pupil center as the CR. Once the pupil and CR are identified, the computer calculates the offset between their centers for use in determining eye line of gaze. The accuracy of the eye-tracker system is reported as ½ degree visual angle. For the equipment setup used in the current study, the accuracy of fixation coordinates was approximately +/- 4mm. The XY coordinates of the separation between pupil and CR are used in the calculation of fixation metrics.

Analyses

Subjects

A one-way ANOVA revealed no significant differences between the groups for age [F (1,38) = .232, p = .633]. A Mann-Whitney test revealed no significant differences of gender between the groups (T (40) = -1.87, p = .183). Within groups comparisons using a split-half (using chronological age) procedure showed that there was no relation between the research variables and age for either the control group or the autistic group (all p values > .05, 2-tailed). Also, a within group analysis revealed no relation between mental age and the research variables (all p values > .05, 2-tailed) for the autistic group. The correlation between age and mental age for the autistic group revealed a relation between the two age-related variables [r (40) = .68, p < .001].

Audiogram

For inclusion in the study, a score of 75% for each of the four tone sets (i.e., 500Hz, 1kHz, 2kHz and 4kHz) presented during the audiometry test was required. As mentioned, normal hearing is a prerequisite of the SCAN tests. All of the subjects tested met the requirements for inclusion. Of the 40 subjects tested, only two missed one of the 16 tones presented during audiometric testing, and each responded correctly to a subsequent presentation of the same tone.

Cardiac vagal tone

Between groups analysis of variance (ANOVA) showed significant differences in mean vagal tone and heart period measures. The autistic group had lower mean vagal tone [F (1,38) = 16.07, p < .001] than the control group, and significantly shorter

heart periods [F (1,38) = 13.74, p = .001]. Table 9 shows the results of mean cardiac vagal tone and mean heart period analyses.

	Mean	Std. Dev.	F	*p* value
Mean VT				
Aut	5.79	.64	16.07	.000
Ctr	6.88	1.03		
Mean HP				
Aut	691.10	87.90	13.74	.001
Ctr	893.08	102.95		

Table 9: Mean cardiac vagal tone and heart period for autistic and control subjects. Aut = autistic; Ctr = control.

SCAN

Significant differences were found on each of the SCAN subtests, as well as the measure of right ear advantage. Analysis of standardized FW scores revealed that the autistic group performed poorer on extracting human voice from background noise [F (1,38) = 187.27, p< .001]. Analysis of the standardized CW scores showed that the autistic group performed poorer on the dichotic listening task [F (1,38) = 27.40, p < .001]. Autistic individuals also scored significantly lower on the SCAN test measure of REA [F (1,38) = 14.15, p = .001]. Table 10 summarizes the results of the SCAN variable analyses. Overall, the autistic group showed a slight left ear advantage, as evidenced by the negative mean REA score. Comparison of individual REA scores for the autistic individuals and the control individuals revealed that approximately 80% of autistic individuals showed evidence of a left ear advantage, while approximately 80% of the control group showed a right ear advantage.

Variable	Autistic Mean (SD)	Control Mean (SD)	F, (*p*)
FW	2.45 (1.45)	10.10 (2.02)	187.27 (.000)
CW	5.05 (2.50)	9.05 (2.33)	27.40 (.000)
REA	-.825 (2.83)	1.85 (1.45)	14.15 (.001)

Table 10: Results of SCAN variable analyses. FW = Filtered words; CW = Competing Words; REA = Right Ear Advantage. FW and CW scores are standardized (mean=10, SD=3).

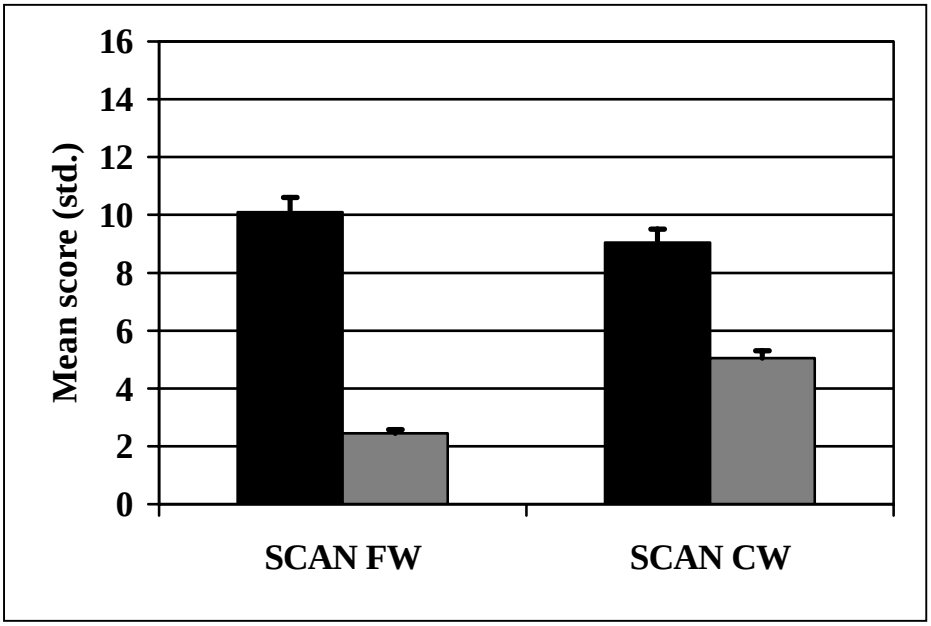

Figure 6: Mean SCAN Competing Words (CW) and Filtered Words (FW) standardized scores for autistic (gray) and control (black) subjects. The standardized scores have a mean of 10 and a standard deviation of 3. All of the control and only three of the autistic individuals were within the standard range.

Eye Tracking

Table 11 summarizes the analyses of the eye-tracker variables. As compared to controls, the autistic group spent less time fixating (SFD) on the Eyes and more time fixating off of the face. Analyses of the time spent fixating on the mouth showed no significant differences. Expressed as percentages (FDP), the autistic group spent roughly 66% of their time fixating off of the face and 11% fixating on the eyes. In contrast, the control group spent approximately 26% of fixation time off of the face and 55% of fixation time on the eyes. Both groups spent approximately 20% of their time fixating on the mouth. Analysis of the number of fixations in each ROI also showed significant differences. Autistic individuals fixated fewer times (SCF) on the eyes and more times on the mouth than did controls. Also, the autistic group fixated a greater percent of times on the mouth than on any other ROI (FCP).

Analysis of the fixation counts (i.e., SFC & FCP) and durations (i.e., SFD & FDP) provides important information that might otherwise go unnoticed. These variables provide the information necessary to analyze how many fixations each group made, as well as how long those fixations were. For instance, analyses of the FCP on the mouth ROI, revealed that the autistic individuals fixated significantly more *times* on the mouth than on any other ROI. However, there were no differences in the *duration* variables for the mouth ROI (i.e., SFD, FDP), which means that autistic individuals directed their gaze to the mouth more often than any other ROI, but not for very long. Analyses using only count or durations would not have provided this information. Similarly, these differences would be lost using the scan path analyses reported by previous eye-tracking research with autistic individuals (i.e., van der Geest

2002a, 2002b). Figure 7 shows the fixation patterns for an autistic individual that looked mainly at the face. Figure 8 shows an exemplar of typical control individual's gaze behaviors.

Variable	Autistic Mean (SD)		Control Mean (SD)		F, (*p*)	
SFD						
Off	87.27	(34.06)	55.47	(30.54)	9.66	(.004)
Eye	17.03	(20.83)	112.41	(48.01)	66.44	(.000)
Mouth	37.50	(44.46)	40.66	(39.95)	.056	(ns)
FDP						
Off	66.39	(25.41)	26.26	(13.42)	39.01	(.000)
Eye	11.35	(11.95)	54.53	(19.90)	69.21	(.000)
Mouth	22.27	(22.11)	19.21	(19.15)	.219	(ns)
SFC						
Off	250.35	(97.80)	114.65	(60.96)	27.73	(.000)
Eye	39.50	(37.29)	201.00	(89.98)	54.99	(.000)
Mouth	71.60	(97.76)	50.00	(36.89)	.855	(ns)
FCP						
Off	70.35	(23.01)	31.44	(11.61)	45.59	(.000)
Eye	11.69	(11.83)	54.78	(15.52)	97.50	(.000)
Mouth	57.99	(79.64)	13.78	(11.05)	6.05	(.019)
MFD						
Off	.328	(.137)	.491	(.174)	5.73	(.000)
Eye	.328	(.181)	.599	(.202)	20.12	(.000)
Mouth	.534	(.350)	.739	(.371)	3.24	(ns)

Table 11: Summary of analyses of eye-tracker variables for each group. As hypothesized, significant differences are seen for the Off and Eye regions of interest. The only significant difference for the Mouth region was for fixation count percent. SFD = sum of fixation duration; FDP = fixation duration percent; SFC = sum of fixation counts; FCP = fixation count percent; MFD = mean fixation duration.

For the many of autistic subjects, fixation patterns showed that the majority of gaze behavior did not focus on the face region, but rather the autistic individuals looked away from the face (Figure 9) or off of the presentation monitor (Figure 10). When these individuals did spend time looking at the face, atypical fixation patterns

were observed. Fixation patterns for the control individuals followed the expected pattern of scanning the eyes and mouth of the storyteller.

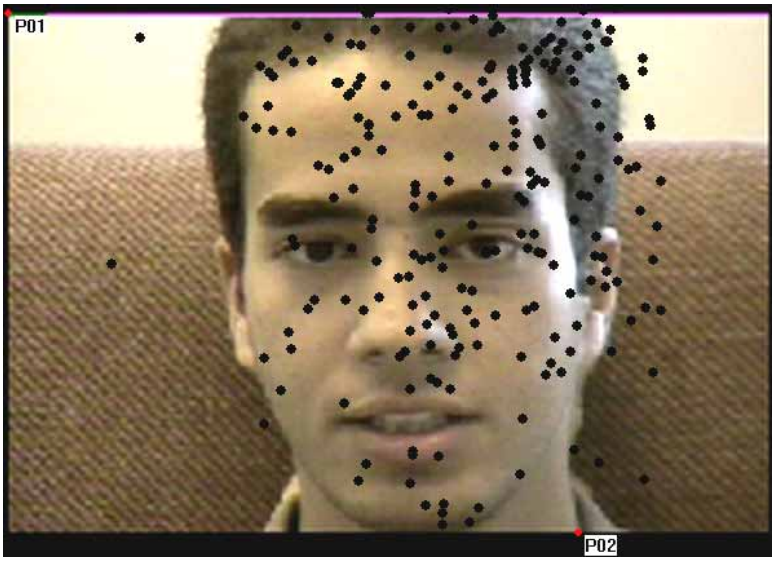

Figure 7: Fixation pattern example for an autistic subject. Dots represent fixation points on the storyteller's face. Fixations off of the presentation monitor, but still within the eye-tracker range (i.e., below the picture) are not shown.

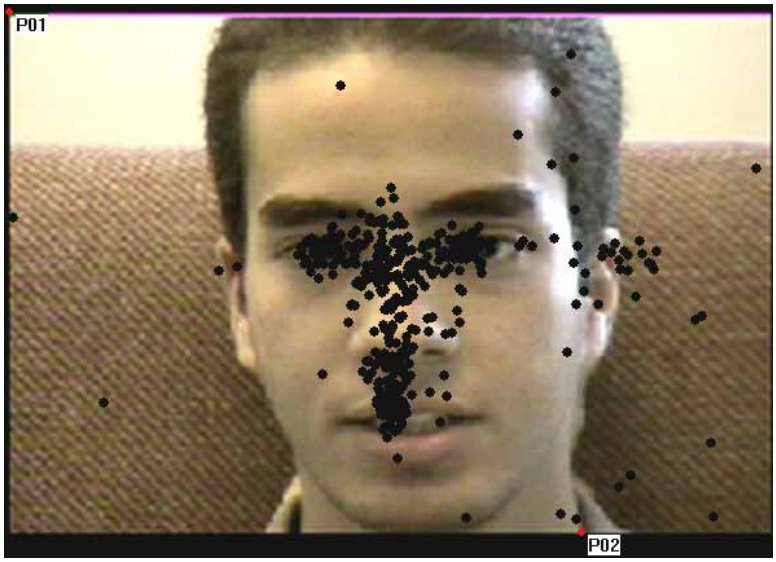

Figure 8: Fixation pattern example for a control subject. Dots represent fixation points on the storyteller's face. Most control subjects show a similar pattern of fixations clustered around the eyes and mouth.

Figure 9: Fixation pattern for an autistic subject depicting looking behavior that avoids the face while maintaining a line of gaze on the presentation monitor. Similar patterns were observed for several of the autistic individuals.

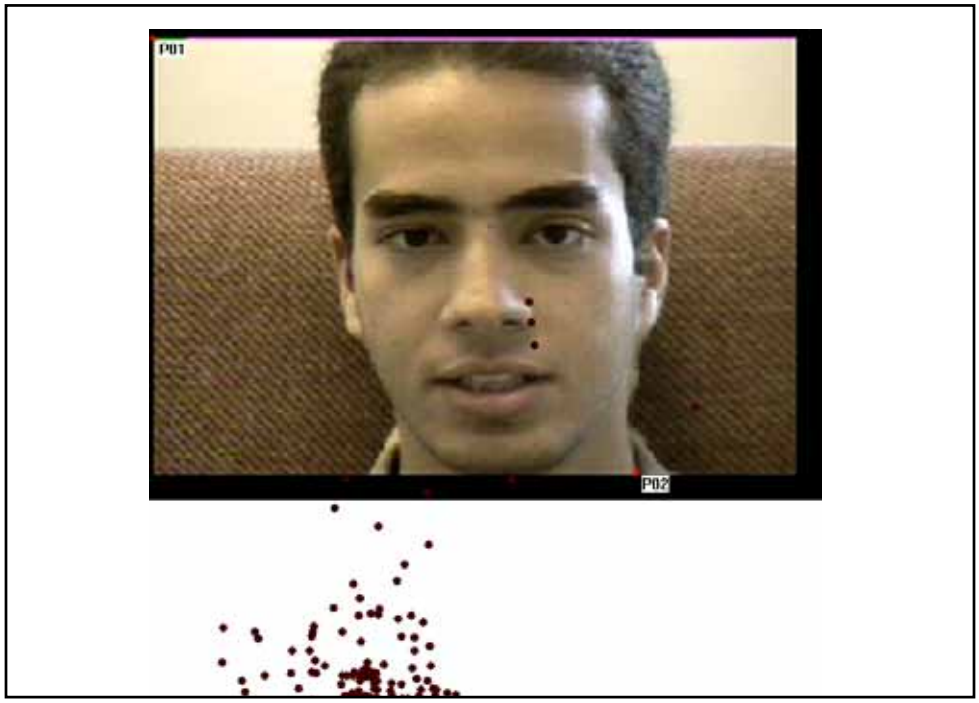

Figure 10: Fixation pattern for an autistic subject depicting looking behavior that avoids the presentation monitor. The outer box approximates the boundary of the eye-tracker for obtaining point of gaze data.

Overall, the analyses of the eye-tracker data showed that autistic individuals fixated more times (Figure 11) and for greater durations (Figure 12) off of the face than controls. Autistic individuals fixated a greater percentage of times on the mouth, but not more times or for longer durations than controls. As hypothesized, the autistic group also fixated fewer times (Figure 13) and for shorter durations (Figure 14) on the eyes than the control group. These data support the hypotheses related to the looking behaviors of autistic individuals proposed by the social engagement system model.

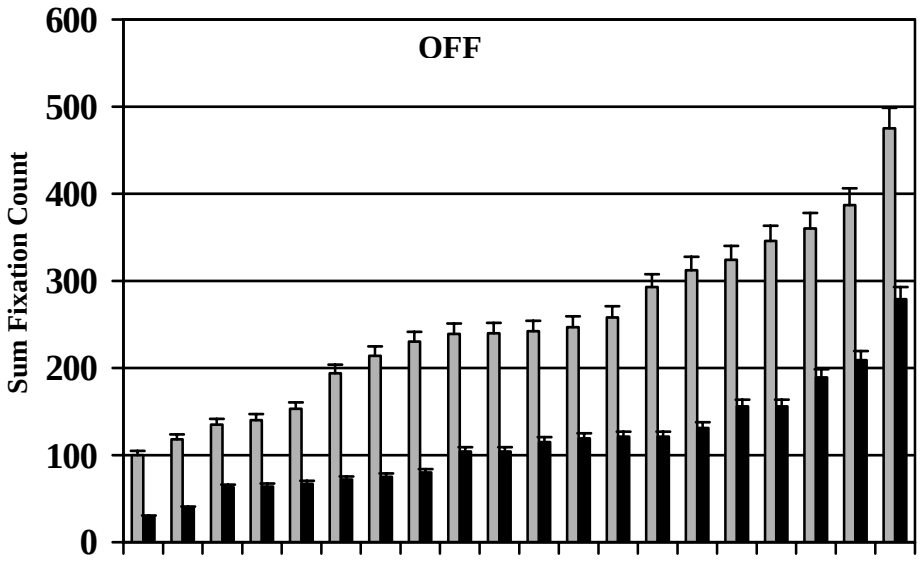

Figure 11: Individual number of fixations on the OFF region of interest. Autistic (gray) individuals fixated significantly more times off of the face than did control (black) subjects.

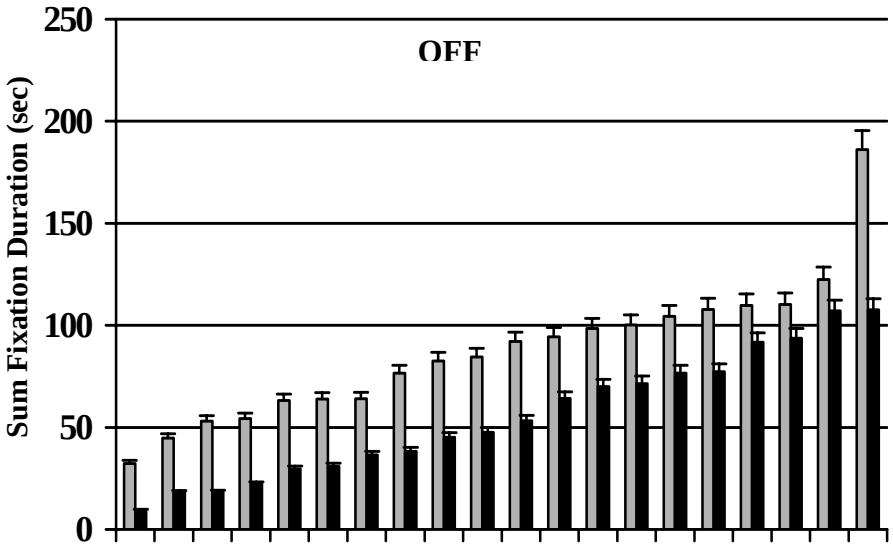

Figure 12: Individual total fixation durations on the OFF region of interest. Autistic (gray) individuals spent significantly more time off of the face than control (black) subjects.

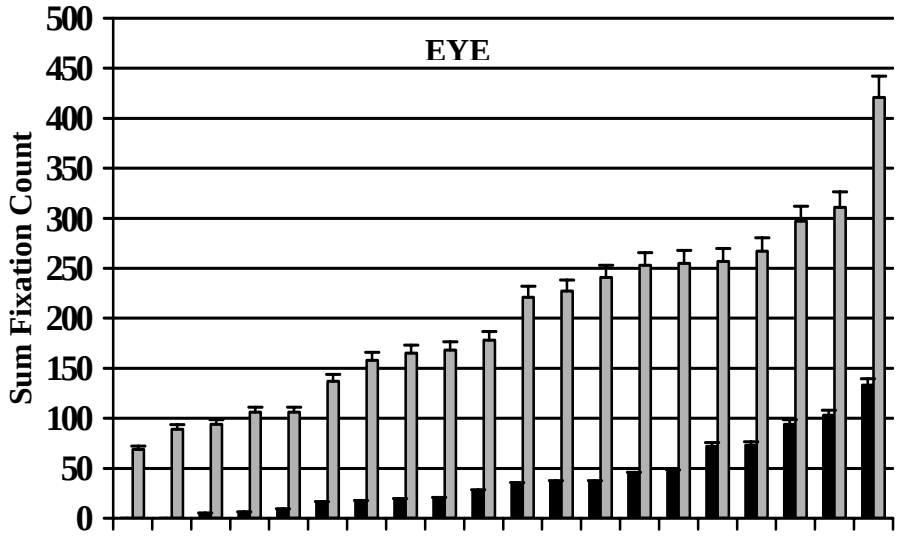

Figure 13: Individual number of fixations on the EYE region of interest. Autistic (gray) individuals fixated significantly fewer times on the eyes than control (black) subjects.

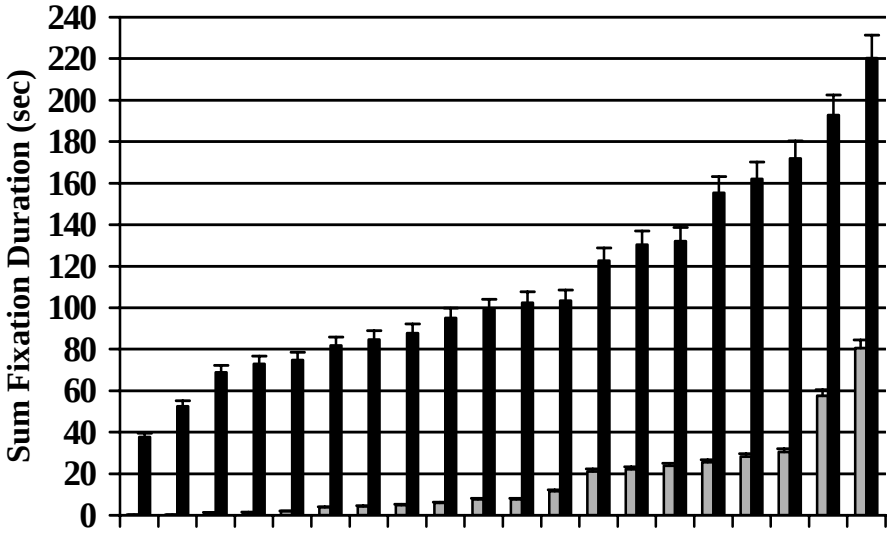

Figure 14: Individual total fixation durations on the EYE region of interest. Autistic (gray) individuals spent significantly less time fixating on the eyes than control (black) subjects. Two of the autistic subjects did not fixate on the eyes at all (represented by the first two missing gray bars).

CHAPTER VII

DISCUSSION

Overview

The results of the current study support the hypothesis that behaviors associated with a diagnosis of autism represent a compromise in an underlying neurobiological system. The data support current research findings and diagnostic indices in that differences in autonomic functioning, eye contact, and listening behaviors are evident in individuals with autism. Similar to the findings by Hutt et. al. (1975), Corona et. al. (1998), and Althaus et. al. (1999), the current study showed significant differences on indices of autonomic functioning (i.e., cardiac vagal tone) in individuals with autism. Results from the SCAN variables are supported by findings related to the listening behaviors of autistic individuals (i.e., Dissanayake & Sigman, 2001; Mundy, 1995; see also the DSM-IV diagnostic criteria for autism). Results from the eye-tracking task provide important new information regarding the looking behaviors of autistic individuals. Unlike recent eye-tracking research (i.e., (Klin et. al., 2002; van der Geest et. al. 2002a, 2002b), the current study utilized a more socially direct task for assessing the gaze behaviors of autistic individuals by using a gaze task designed to more directly involve the individual. By using a video of a person telling a story while looking directly at the listener, it was expected that the gaze behaviors of the individuals would relate more closely to the natural gaze behaviors during face-to-face interactions. The use of this specific variable may explain the differences between earlier studies and the current study. For instance, van der Geest et. al (2000a) reported that autistic individuals did not differ from normals on their patterns

of looking at a social stimuli. However, the "social stimuli" was a stick figure drawing of a person, within a larger drawing that included other non-social items (i.e., a house, tree…etc.). Differences in looking behaviors found by Klin et. al. (2002) more closely relate to the current findings, although they looked more at patterns of looking and used a movie clip as a social stimuli. The Klin et. al. study showed that compared to matched controls, autistic individuals showed atypical patterns of looking to different regions of interest, and showed significant differences for specific regions of the face (i.e., eyes, mouth). However, this study relates more to the looking behaviors of autistic individuals when they are the on-looker rather than directly engaged with someone. Klin et. al. interpret the results as those that may be expected when an autistic individual watches everyday social interactions of others (i.e., in the lunchroom at school).

Importance of the current study

The current study is the first to explicitly test multiple components of the Polyvagal Theory's social engagement system model. The study provides strong support for the proposed underlying neurobiological system as described by Porges (1995, 1998, 2001, 2003). The results confirm the hypothesized relation between a compromise in the system and the atypical behaviors associated with autism. They also provide evidence supporting the proposed components of the social engagement system and the integration of these components as a feedback loop associated with social engagement behaviors. The current study is the first to assess the somatomotor component of the Polyvagal Theory, and the only study to include assessments of both somatomotor and visceromotor components proposed by the theory. Significant

differences were found in all components of the system that were examined, which supports the structural accuracy of the model and it's predictive validity for atypical social behaviors associated with autism as well as several other psychiatric disorders. The study provides evidence for functional deficits in an integrated system responsible for vagal control of the heart, looking behavior, and listening behavior not previously reported in autism research. It employed a design utilizing quantitative assessment of these functions, which provides important new information for the field of autism research. Because it was theory-based, it informs the theory, shows the strength of the Polyvagal Theory, and provides strong support for the social engagement system model as it relates to social engagement behaviors including regulation of physiological state, listening behavior, and looking behavior. The study confirms a deficit in this system in autistic individuals, based on the specific hypotheses drawn from the model and the Polyvagal Theory from which it was derived.

This study revealed differences in vagal control of the heart in individuals with autism using accurate quantifiable methodologies. Previous research proposing differences in cardiac-related autonomic variables (i.e., heart rate, heart period, RSA) utilized non-comparable or methodologically questionable metrics for assessing autonomics of individuals with autism. For instance, Corona et. al. (1998) used a natural log conversion of mean heart period as the metric for assessing cardiac response. Furthermore, the authors attempted to analyze 3-second and 10-second trials of mean heart period to assess differences between autistic and control subjects. This procedure is not sufficient for accurately assessing changes in cardiac functioning as specified in the study (i.e., as an index of vagal tone). The specified trial lengths

(i.e., 3 and 10 seconds) would have provided between 3-6 data points (3s trials) and 10-20 data points (10s trials) for a heart rate ranging from 1 beat per second (bps) to 2bps, a range that covers the vast majority of the population (i.e., infant to adult) under normal conditions. Using the equipment and software reported in the study (i.e., a Vagal Tone Monitor and MXedit software; both developed by Porges), their analysis of heart period cannot provide accurate information pertaining to the autonomic differences proposed by the study. The "data reduction" section pertaining to heart period in the current paper outlines the specific procedures required for accurate quantification of cardiac vagal tone using this methodology (see also Denver & Porges, in press). The discrepancy in methodologies, as described in this study, puts the results and interpretation of the cardiac measures in question. A similar methodological problem with the quantification of cardiac measures is found in the Zahn et. al. (1987) study. Overall, measures of cardiac autonomics (i.e., HRV, RSA, HP, HR) reported in studies on autistic individuals have used various measures and methodologies that make direct comparisons of the results difficult. The current study used a well-documented, standardized, and accurate methodology for the assessment of cardiac vagal tone in autistic individuals that can be directly compared to other methodologies (i.e., spectral analysis), and has been shown to be directly related to the assessment of vagal control of the heart. The use of the quantification procedure in the current study revealed significant differences in cardiac vagal tone in autistic individuals, as compared to matched controls. This finding directly supports the hypothesis of autistic individuals having lower mean cardiac vagal tone when

compared to age-matched controls. The use of the standardized methodology allows for reliable replication studies in the future.

The study also showed significant differences in listening behaviors that are directly related to social communication in autistics. The results of the SCAN task provide important information pertaining to differences in listening abilities of autistic individuals. These data show that autistic individuals are impaired in their ability to extract human voice from background sounds, their performance on dichotic listening tasks, and that they lack the normal right ear advantage for processing human voice. These abilities are directly related to language delays, learning disabilities and autistic spectrum disorders (Smith et al., 1988; Thomas et al., 1985). Due to the relation between middle ear muscle functioning and the extraction of human voice, the current study provides important information pertaining to functional deficits in the middle ear muscle dynamics of autistic individuals, and supports the social engagement system model's proposed outcome of a compromise in the system. These results may help to explain the diagnostic symptoms related to the atypical pragmatic language skills of autistic individuals (i.e., initiation/maintenance of conversations). Specifically, the inability to accurately extract human voice from the environment may lead to a withdrawal from (or avoidance of) situations requiring these skills (i.e., conversations) and may be directly related to atypical looking behavior as an adaptive strategy for autistic individual's to overcome this inability by avoiding eye contact during conversations. Evidence for this possibility is found in the eye-tracking data.

The current study provides new and important information related to differences in the atypical looking behaviors of autistic individuals commonly

mentioned in the literature, and included in the diagnostic criteria. Very limited quantitative research has been conducted on gaze behaviors of autistic individuals, and this is the only reported study that directly assesses this behavior using a social task that directly involves autistic individuals. Previous studies attempting to assess looking behaviors of autistic individuals have used either non-social or indirectly social tasks to assess fixation patterns. For instance, the study by van der Geest et. al. (2002a) used a cartoon-like drawing that included a human stick figure as the "social" stimuli and neutral objects (car, house, trees…etc.). Because no significant differences were found in fixation data on the human figure or on the picture as a whole, the conclusion of this study was that autistic individuals do not show atypical looking behavior in general or to social stimuli specifically. The use of a cartoon-like stick figure drawing as a "social stimulus" does not justify this conclusion. These results only provide information showing that autistic individuals look at pictures in the same way that non-autistic individuals do. The study provides no information pertaining to the looking behaviors of autistic individuals to a meaningful social stimuli. A second set of studies by van der Geest et. al., (2002b) used pictures of human faces showing different emotions and in different orientations as a measure of gaze behavior in autistics. Again the results showed no differences between autistics and controls on gaze behaviors to either emotion faces or neutral faces shown in the upright position. The authors concluded that autistic individuals did not differ in gaze behavior in general, but that situational factors *may* play a role in the atypical gaze behavior of autistic individuals. The studies by van der Geest et. al. (2000a; 2000b)

provide no information pertaining to socially relevant gaze-behaviors of autistic individuals. The very nature of autism is atypical social behavior.

The eye-tracking studies by Klin et. al., (1999, 2002) provide information related to gaze behaviors of autistic individuals to static pictures (1999) and a socially related stimuli (2002). In both of the Klin studies, autistic individuals looked more at the mouth and less at the eyes presented in the stimuli (i.e., static face pictures and a movie clip). However, similar to the other eye-tracking gaze studies, the results of these studies do not provide specific information related to looking behaviors in social situations that involve the individual with autism. In the 2002 study, Klin and colleagues collected fixation data while individuals watched a movie portraying a complex social interaction between several actors. Based on differential fixation *patterns*, the authors concluded that autistic individuals demonstrate abnormal visual pursuit patterns (i.e., more pursuits that went from the mouth of one actor to the mouth of another vs. eyes to eyes) when viewing "naturalistic" social situations. While the conclusions proposed by both of the Klin et. al. studies provide support for the current findings that autistic individuals spend significantly less time looking at the eyes of others during a directed social task, the results of the Klin et. al., (2002) study should be interpreted cautiously. An important methodological issue that was not addressed in the study was the use of visual pursuit patterns (i.e., scan paths) as the metric of analysis. Unlike the current study, which used fixation metrics and utilized a test/retest design, Klin et. al. implicitly rely on the assumption that visual pursuit patterns are consistent over time. However, it is highly unlikely that the same visual pursuit pattern of any individual would be obtained on a second trial. Thus, the use of

fixation counts and durations provides a more reliable and replicable metric for assessing eye-tracking measures than visual pursuit (i.e., scan path) for this type of research.

Although previous eye-tracking studies provide some basic information related to the looking behaviors of autistic individuals to non-social stimuli, static pictures, and indirect social interactions, they do not provide any specific information pertaining to the social engagement deficits defined in the diagnostic criteria for autism. The current study provides evidence relevant to direct social interactions and the subsequent looking behavior of autistic individuals. Future research is being planned that will assess looking behavior in a "true" social situation (i.e., during a live conversation).

Summary

According to the Polyvagal Theory, the mammalian nervous system has evolved in a way that promotes social behavior. The model used to describe the integrated neurobiological system responsible for regulating our social engagement abilities has been labeled the Social Engagement System. Based on the model of a hierarchy of potential adaptive strategies for responding to stimuli in our environment, the Polyvagal Theory proposes that a well functioning social engagement system leads to appropriate social engagement behaviors. These behaviors include proper eye contact, prosody and intonation, listening, and facial expressions as well as a supportive physiological state. Conversely, a compromise in the system would lead to a lack of eye contact and facial expression, poor intonation and prosody in our voice, the inability to extract human voice from background noise (and therefore lack of a

right ear advantage for human voice), and poor physiological functioning. This study was designed to test components of social engagement system functioning. Due to the strong relation between indices of a compromised social engagement system and the atypical behaviors associated with a diagnosis of autism, the study was conducted with individuals with autism. The study showed that the indices associated with a compromised social engagement system are related to atypical behaviors associated autism. No other study has attempted to examine the hypothesis that a specific integrated neurobiological system may be involved in the emergence of these behavioral features. Furthermore, while some have proposed a neurobiological basis for the disorder, none have attempted to describe an integrated model from which predictable relations can be tested. The social engagement system model provides a specific, theoretically-based model for making these assessments. According to this model the core of atypical behaviors seen in autism are predictable as the emergent properties of a compromise in this system.

According to the DSM-IV, the core behavioral indices of autism include difficulties in social interaction that include looking and listening skills. In fact, the defining feature of autism is atypical social engagement. These are also the predicted behaviors of a compromised social engagement system. The current study supports the hypothesis that the atypical behaviors associated with autism are related to a compromise in the system. It has been shown that as compared to control subjects, that autistic group performed poorer on indices of looking (eye gaze) and listening (extracting human voice from background, right ear advantage). Furthermore, a premise of the social engagement system model is that a person's physiology

regulates, and is regulated by stimuli in our environment. Accordingly, a compromise in the system would lead to atypical autonomic function. As the primary component of the autonomic nervous system, the vagal system provides us with a means of assessing autonomic functioning in a non-invasive way (i.e., quantification of cardiac vagal tone). Thus, results of the assessment of cardiac vagal tone in the current study provide further support for the hypothesis that autism is related to a compromise in the social engagement system.

Taken together, the results of the current study support each of the stated hypotheses and support the proposal that autism may be an extreme example of the emergent properties of a compromised social engagement system.

Conclusion

The current study provides evidence for quantifiable differences on a number of variables related to social engagement system function. It is the first study to utilize a theory driven research design to assess functional differences in multiple components of the social engagement behaviors of autistic individuals. The study showed significant differences related to both the visceromotor (i.e., cardiac vagal tone) and somatomotor (i.e., looking and listening) components of the social engagement system. Data support each of the hypotheses of the study and provide important new information pertaining to social engagement behaviors of autistic individuals and the relation between the cluster of atypical behavior and a compromised social engagement system

Limitations

One of the difficulties of assessing social engagement system functioning in the autistic population relates to the assignment of a relevant control group. Because the variables assessed included processing abilities (i.e., SCAN variables) as well as physiological variables, a straightforward matching scheme is not readily available. For instance, if subjects were matched on chronological age only, the possibility of confounding the SCAN scores would be present due to the lower mental ages of the autistic individuals. On the other hand, if mental age were used for matching purposes, physiological variable would be confounded. Physiological indices follow developmental trends. This fact precludes a simple matching procedure such as matching on age and verbal ability, a common practice in autism research. The design of a standard matching scheme for cross-modal research such as the current study should be investigated.

A second limitation concerns the assessment of right ear advantage. Standard REA assessments (i.e., dichotic listening test batteries, ABR, tympanometry) may be either too difficult or too aversive to many autistic individuals. The SCAN test used in the current study does have good psychometric properties, however it provides only a rough estimate of REA based on the standardization group. Furthermore, even the use of the SCAN test eliminates the possibility of assessing lower functioning (i.e., non-verbal) autistic individuals and limits the generalizability of the study.

While the current research used a considerably more "social" stimuli for the eye-tracking task than has been previously reported, even the use of a video of a person speaking directly to the listener may not yield the same results as a true social interaction. Future research designs should seek to advance the "realism" of the social

interaction stimuli. Also, standardization of analysis metric and methodologies would enhance replication.

Finally, the inclusion of variables related to social engagement system functioning not tested in the current study would lend further support to the relation between a compromised social engagement system and autism. For instance, the direct assessment of dynamic middle ear muscle functioning, autonomic reactivity, baroreceptor sensitivity and cortical functioning would greatly enhance the available information for this population.

Implications

Understanding the atypical behaviors associated with autism from the theoretical approach that they are the emergent properties of a compromised social engagement system provides useful information for intervention strategies. For instance, if we realize that autistic individuals are in a physiological state that promotes defensive behavioral strategies, interventions can be designed that enhance the individual's assessment of their environment as "safe". This would lead to less invasive intervention tactics, in more quiet and calming settings to enhance the individual's perception of their environment as a safe place. This in turn may enhance a physiological state that is more supportive of social engagement. Furthermore, intervention strategies designed to stimulate the neural components of the social engagement system would help to provide the physiological functioning required for the perception of a safe environment and the emergence of social engagement behaviors.

REFERENCES

Althaus, M., Mulder, L.J.M., Mulder, G., Aarnoudse, C.C., & Minderaa, R.B. (1999). Cardiac adaptivity to attention-demanding tasks in chidlren with a pervasive developmental disorder not otherwise specified (PDD-NOS). Biological Psychiatry, 46:799-809.

American Psychiatric Association (APA, 1994). Diagnostic and Statistical Manual-IV. Washington, DC: American Psychiatric Association.

Ardic, F.N., Topaloglu, I., Oncel, S., Ardic, F., &Uguz, M.Z. (1997). Does the stapes reflex remain the same after Bell's Palsy? American Journal of Otology 18, 761-765.

Bar-Haim (2002). Introversion and individual differences in middle ear acoustic reflex function. International Journal of Psychophysiology, 46, 1-11.

Bar-Haim, Y., & Marshal, P.J. (2001). Auditory sensory processing in socially withdrawn children. Paper presented at the biennial meeting of the Society for Research in Child Development, Minneapolis, MN.

Baron-Cohen, S. (1988). Social and pragmatic deficits in autism: Cognitive or affective? Journal of Autism and Developmental Disorders, 18, 379-402.

Borg, E., & Counter, S.A. (1989). The middle-ear muscles. Scientific American, 26, 74-80.

Buki, B., Wit, H. P., & Avan, P. (2000). Olivocochlear efferent vs. middle-ear contributions to the alteration of otoacoustic emmissions by contralateral noise. Brain Research, 852, 140-150.

Burns, E. M., Harrison, W. A., Bulen, J. C., & Keefe, D.H. (1993). Voluntary contraction of middle ear muscles: Effects on input impedance, energy reflectance and spontaneous otoacoustic emissions. Hearing Research, 67, 117–127.

Buitelaar, J.K., van Engeland, H., de Kogel, K.H., de Vries, H., & van Hooff, J.A. (1991). Differences in the structure of social behaviour of autistic children and non-autistic retarded controls. Journal of Child Psychology & Psychiatry & Allied Disciplines.32, (6), 995-1015.

Corona, R., Dissanayake, C., Arbelle, S., Wellington, P., & Sigman, M. (1998). Is affect aversive to young children with autism? Behavioral and cardiac responses to experimenter distress. Child Development, 69, 1494-1502.

Dallos, P., & Evans, B.N. (1995). High-frequency motility of outer hair cells and the cochlear amplifier. Science, 267, 2006–2009.

Denver, J.W., & Porges, S.W. (in press). Methodological issues in the quantification of RSA. Biological Psychology.

Dissanayake, C., & Sigman, M. (2001). Attachment and emotional responsiveness in children with autism. In Laraine Masters Glidden (Ed). International review of research in mental retardation: Autism, 23, (pp. 239-266). San Diego, CA: Academic Press.

Djupesland, G. (1965). Electromyography of the tympanic muscles in man. International Audiology, 4, 34-41.

Djupesland, G. (1976). Nonacoustic reflex measurement: Procedures, interpretations and variables. In A.S. Feldman & L.A. Wilber (Eds.) Acoustic impedance & admittance: The measurement of middle ear function. (pp. 217-235). Baltimore, MD: Williams & Wilkins Co.

Hobson, R.P., & Lee, A. (1998). Hello and goodbye: a study of social engagement in autism. Journal of Autism and Developmental Disorders, 28, 117-127.

Hutt, C., & Ounsted, C. (1966). The biological significance of gaze avoidance with particular reference to the syndrome of infantile autism. Behavioral Sciences, 11, 346–356.

Hutt, C., Forrest, S.J., & Richer, J. (1975). Cardiac arrhythmia and behavior in autistic children. Acta Psychiatrica Scandinavica, 51, 361-372.

Jerger, S., & Jerger, J. (1977). Diagnostic value of crossed vs. uncrossed acoustic reflex. Archives of Otolaryngology, 103, 445-453.

Kanner, L. (1943). Autistic Disturbances of Affective Contact. Nervous Child 2 , (pp.217-250). Reprinted in Anne M. Donnellan (Ed.), Classic Readings in Autism, New York, NY: Teacher's College Press (1985).

Kawase, T., & Liberman, M.C. (1993). Anti-masking effects of the olivocochlear reflex: I. Enhancement of compound action potentials to masked tones. Journal of Neurophysiology, 70, 2519–2532.

Keith, R. W. (1994). SCAN-A: A test for auditory processing disorders in adolescents and adults. San Antonio, TX: The Psychological Corporation.

Keith, R. W. (2000). SCAN-C: A test for auditory processing disorders in children. San Antonio, TX: The Psychological Corporation.

Klin, A., Jones, W., Schultz, R., Volkmar, F., & Cohen, D. (2002). Visual fixation patterns during viewing of naturalistic social situations as predictors of social competence in individuals with autism. Archives of General Psychiatry 59, (9), 809–816.

Klin, A., Sparrow, S.S., de Bildt, A., Cicchetti, D.V., Cohen, D.J., & Volkmar, F.R. (1999). A normed study of face recognition in autism and related disorders. Journal of Autism and Developmental Disorders, 29, 499-508.

Kryter, K.D. (1985). The effects of noise on man. New York, NY: Academic Press.

Langdell, T. (1978). Recognition of faces: An aproach to the study of autism. Journal of Child Psychology and Psychiatry, 19, 255-268.

Liberman, M.C., & Dodds, L.W. (1984). Single-neuron labeling and chronic cochlear pathology. III. Stereocilia damage and alterations of threshold tuning curves. Hearing Research, 16, 55–74.

Liberman, M.C. , & Guinan Jr., J.J. (1998). Feedback control of the auditory periphery: anti-masking effects of middle ear muscles vs. olivocochlear efferents, Journal of Communication Disorders, 31, 471–482.

Lord C, Rutter M, LeCouteur A. 1994. Autism Diagnostic Interview—Revised: a revised version of a diagnostic interview for caregivers of individuals with possible pervasive developmental disorders. J Autism Dev Disorders 24:659 – 668.

Mertens, I., Siegmund, H., & Grusser, O. (1993). Gaze motor asymmetries in the perception of faces during a memory task. Neuropsychologia, 31, 989–998.

Mundy, P. (1995). Joint attention and social-emotional approach behavior in children with autism. Development and Psychopathology, 7, (1), 63-82.

Ornitz, E.M. (1988). Autism: A disorder of directed attention. Brain Dysfunction, 1, 309–322.

Palkovitz, R.J. & Wiesenfeld, A.R. (1980). Differential autonomic responses of autistic and normal children. Journal of Autism and Developmental Disorders 10, 347-360.

Parent, A. (1996). Carpenter's Human Neuroanatomy, 9th ed., Mosby-Williams and Wilkins, Baltimore, MD.

Pedersen, J., Livoir-Petersen, M. F., & Schelde, J. T. (1989). An ethological approach to autism: An analysis of visual behaviour and interpersonal contact in a child versus adult interaction. Acta Psychiatrica Scandinavica, 80, (4) 346-355.

Porges, S. W. (1985). Method and apparatus for evaluating rhythmic oscillations in aperiodic physiological response systems. Patent No.4,510,944, April 16, 1985.

Porges, S.W. (1995). Orienting in a defensive world: Mammalian modifications of our evolutionary heritage. A Polyvagal Theory. Psychophysiology 32, 301-318.

Porges, S.W. (1997). Emotion: An evolutionary by-product of the neural regulation of the autonomic nervous system. In C. S. Carter, B. Kirkpatrick, & I.I. Lederhendler (Eds.), The Integrative Neurobiology of Affiliation, Annals of the New York Academy of Sciences, 807, 62-77.

Porges, S.W. (1998). Love: An emergent property of the mammalian autonomic nervous system. Psychoneuroendocrinology 23, 837-861.

Porges, S.W. (2001). The Polyvagal Theory: Phylogenetic substrates of a social nervous system. International Journal of Psychophysiology 42, 123-146.

Porges, S.W., (2003). The Polyvagal Theory: phylogenetic contributions to social behavior. Physiology and Behavior.79 (3), 503-13.

Porges, S.W., & Bohrer, R.E. (1990). The analysis of periodic processes in psychophysiological research. In John T. Cacioppo, and Louis G. Tassinary, (Eds.). Principles of psychophysiology: Physical, social, and inferential elements. 708-753.

Porges, S.W., & Byrne, E.A. (1992). Research methods for measurement of heart rate and respiration. Biological Psychology, 34, (2-3), 93-130.

Porges, S.W., Doussard-Roosevelt, J.A., Portales, A.L., & Greenspan, S.I. (1996). Infant regulation of the vagal "brake" predicts child behavior problems: A psychobiological model of social behavior. Developmental Psychobiology 29, 697-712.

Posamentier, M.T., & Abdi, H. (2003). Processing faces and facial expressions. Neuropsychology Review, 13, (3), 113-143.

Qui, W.W., Yen, S.S., Stucker, F.J., & Hoasjoe, F.J. (1997). Neurophysiological evaluation of acute facial paralysis in children. International Journal of Otorhinolaryngology, 39, 223-236.

Rutter, M. (1978). Diagnosis and definitions of childhood autism. Journal of Autism and Childhood Schizophrenia. 8, (2), 139-161.

Santer, R.M. (1994). Chromaffin systems. In S. Nilsson & S. Holmgren,(Eds). Comparative Physiology and Evolution of the Autonomic Nervous System. 97-117. Switzerland : Harwood Academic Publishers.

Scanlon, D.M., & Vellutino, F.R. (1996). Prerequisite skills, early instruction, and success in first-grade reading: Selected results from a longitudinal study. Mental Retardation and Developmental Disabilities Research Reviews, 2, (1), 54-63.

Schwaber, J.S. (1986). Neuroanatomical substrates of cardiovascular and emotional-autonomic regulation. In A. Magro, W. Osswald, D. Reis, & P. Vanhoutte, Eds. Central and Peripheral Mechanisms of Cardiovascular Regulation. 353-384. New York: Plenum Press.

Smith, D.E.P., Miller, S.D., Stewart, M., Walter, T.L., & McConnell, J.V. (1988). Conductive hearing loss in autistic, learning-disabled, and normal children. Journal of Autism and Developmental Disorders 18, 53-65.

Solomon, G., & Starr, A. (1963). Electromyography of middle ear muscles in man during motor activities. Acta Neurologica Scandinavia, 39, 161-169.

Tantam, D., Monaghan, L., Nicholson, H., & Stirling, J. (1989(. Autistic children's ability to interpret faces: A research note. Journal of Child Psychology and Psychiatry, 30, 623-630.

Valentine, T., (1988). Upside-down faces: A review of the effect of inversion upon face recognition. British Journal of Psychology, 79, 471-491.

van der Geest, J.N., Kemner, C., Camfferman, G., Verbaten, M.N., & van Engeland, H. (2002a). Looking at images with human figures: Comparison between autistic and normal children. Journal of Autism & Developmental Disorders. 32, (2), 69-75.

van der Geest, J.N., Kemner, C., Verbaten, M.N., & van Engeland, H. (2002b). Gaze behavior of children with pervasive developmental disorder toward human faces: A fixation time study. Journal of Child Psychology & Psychiatry & Allied Disciplines. 43, (5), 669-678.

Volkmar, F.R., & Mayes, L.C. (1990). Gaze behavior in autism. Development and Psychopathology 2, (1), 61-69.

Volkmar, F.R., Sparrow, S.S., Rende, R.C., & COhen, D.J. (1989). Facial perception in autism. Journal of Child Psychology and Psychiatry, 30, 591-598.

Walker-Smith, G.J., Gale, A.G., & Findlay, J.M., (1977). Eye movement strategies in face perception. Perception, 6, 313-336.

Wormald, P.J., Rogers, C., & Gatehouse, S. (1995). Speech discrimination in patients with Bell's palsy and a paralysed stapedius muscle. Clinical Otolaryngology 20 (1), 59-62.

Yarbus, A.L. (1967). Eye Movements and Vision. New York, NY: Plenum Press.

Zahn, T.P., Rumsey, J.M., & Van Kammen, D.P. (1987). Autonomic nervous system activity in autistic, schizophrenic, and normal men: Effects of stimulus significance. Journal of Abnormal Psychology, 96, 135-144.