How the Safe and Sound Protocol Came to Be

An Academic Love Letter from Dr. Porges

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For more than 50 years, my research has focused on understanding how our physiological state influences our responses to all aspects of the world both inside and outside of the boundaries of our body. This broad realm of experience is dependent on our nervous system, which is continually assessing, interpreting, and responding to sensory information. This system involves the brain and all the nerves in the body that are either directly or indirectly communicating with the brain. Understanding how the nervous system coordinates these complex processes requires a thoughtful scientific strategy, which can be summarized in the following four principles.

How Physiological State Influences Our Responses to Everything

1. Sensory pathways drive development and refinement of motor pathways

This principle is confirmed through studies of development and phylogeny. Challenges to the nervous system conveyed through sensory pathways stimulate neuroregulation, neurogenesis, and neuroplasticity. In fact, neurostimulation is an emerging scientific discipline. Stimulation of sensory pathways of the vagus highlights the mechanisms through which vagal nerve stimulation works to optimize brain function, reduce inflammation and seizures, and optimize performance. Similarly, the Safe and Sound Protocol (SSP)–is a sensory driven intervention designed to enhance the central regulation of the social engagement system.

2. The brain evolved and developed from brainstem to cortex following a bottom up trajectory

Comparative neuroanatomy informs us the brainstems of virtually all vertebrates share common features and appear very similar. Through evolution, the higher brain structures emerged and evolved. For example, mammals can be distinguished from their reptilian ancestors by the development and expansion of the cortex. As a neurobiological principle, the evolutionary sequence estimate via comparative neuroanatomy is replicated in embryology as the brain develops in utero.

3. Newer brain structures functionally inhibit older ones forming a phylogenetically/ontogenetically determined hierarchy

The brain in mammals is the product of an evolutionary process through newer structures actively inhibiting and repurposing older structures. This point was clearly described by Damasio in his accessible book, *Descartes' Error*[1]. In the book, he describes Phineas Gage's "disinhibited" behavior following a head injury in which part of his prefrontal cortex was destroyed. This process of disinhibition has been a foundational concept in neurology and was described as "dissolution" or evolution in reverse by John Hughlings Jackson (1884)[2].

4. In response to illness and survival challenges, the nervous system functionally follows a strategy of dissolution or evolution in reverse

Polyvagal Theory (PVT) generalizes the Jacksonian concept of dissolution to the phylogenetic stages of the vertebrate autonomic nervous system. This provides an explanation of the neural mechanisms and autonomic state underlying the vast symptoms associated with functional medical disorders, disorders with symptom clusters that mimic pathophysiological disorders but cannot be confirmed by traditional medical assessments of end organ damage. Dissolution is the primary mechanism through which trauma and chronic stress disrupt health and repurpose the autonomic nervous system from supporting the homeostatic functions of health, growth, restoration, and sociality to supporting defense and survival.

In understanding the where and how SSP emerged, it is helpful to step back in time and share some of my scientific benchmarks as PVT evolved and expanded. These were experiences of posing questions and generating hypothetical solutions that were guided by PVT. As you travel the timeline with me, you will intuitively grasp how the theory led to an understanding of how the autonomic nervous system was intimately intertwined with human experiences. But moreover, you will intuitively move from an understanding of the physiology underlying mental and physical health challenges to hypothetical strategies to optimize outcomes by retuning the autonomic nervous system. PVT guided me toward the SSP as a gentle strategy that would coax the autonomic nervous system back to competence in optimizing our health and well-being. Below is an abbreviated timeline highlighting the introduction of features of PVT and leading to the SSP.

Timeline highlighting the introduction of features of PVT and leading to the SSP

- 1992: Paper published in *Pediatrics*[3] on the use of respiratory sinus arrhythmia in evaluating medical risk in preterm newborns
- 1994: Presidential address to the Society for Psychophysiological Research introducing Polyvagal Theory[4]
- 1995: Presidential address published in the journal *Psychophysiology* in an article entitled "Orienting in a Defensive World: Mammalian Modifications of Our Evolutionary Heritage. A Polyvagal Theory"[5]
- 1996: Introduction of the vagal brake[6]
- 1996: Introduced of the four-level model of regulation[7]
- 1998: Introduction of the social engagement system[8]
- 1998–2015: Development of the Listening Project Protocol (now SSP)
- 2003–2004: Introduction of neuroception[9]
- 2013 and 2014: Foundational studies documenting the effects of the Listening Project Protocol (now SSP) in children with auditory hypersensitivities and autism spectrum disorder[10, 11]
- 2017: Launch of the SSP via Integrated Listening Systems (iLs)
- 2018: Patent awarded for technology in the SSP[12]
- 2020: Introduction of vagal efficiency[13]

Understanding and Measuring Cardiac Vagal Tone: A Key Event Leading to PVT

Embedded in the above timeline is the critical event that led to the Polyvagal Theory. By 1992, my research was focusing on the applications of the technologies that I had developed to extract a valid and reliable index of cardiac vagal tone from the beat-to-beat heart rate pattern. This work led to a patent (US patent #US4510944A) and the establishment of a small company, Delta-Biometrics, that manufactured and marketed a vagal tone monitor (VTM) that integrated my patented technology to provide real-time analyses of respiratory sinus arrhythmia (RSA) as an index of vagal

tone. The company also produced a software product that enabled other researchers to use our analytic procedures to quantify RSA.

The VTM used the same computer processor as the original IBM PC. The VTM did not have any moving parts. The front panel displayed the dynamically changing heart rate and amplitude of RSA. It required an ECG analog input and provided serial and parallel output ports to print the data in sequential selectable 10- or 30-second epochs. It even provided an automatic data editor that estimated values when the ECG signal was disrupted.

With the VTM and the offline analyses easily available to colleagues, my laboratory trained several hundred scientists to quantify RSA and to use our technology in their research. Finding business a distracter from research, I closed Delta-Biometrics in 2000, after selling approximately 100 VTMs to researchers. According to Google Scholar, the software and VTM have been cited more than 500 times in peer-reviewed papers.

The Vicissitudes of Research Funding

By 1992, I had assumed that my research trajectory, enhanced by the sensitive and available technology to quantify vagal tone that could easily be shared with colleagues, would be focused on clinical applications. This assumption led me to ask questions and to conduct research on the autonomic features of various clinical populations. I envisioned a pragmatic program of research that would be well-funded by the National Institute of Mental Health, as scientists were now exploring the role of autonomic regulation in mental health, medical diseases (e.g., diabetes, cancer), developmental disabilities (e.g., autism, Down syndrome), social behavior, and cognitive functions.

The Pivotal Letter

Following the publication of the 1992 *Pediatrics* paper, I received a letter from a neonatologist that would change my research trajectory and lead me to develop the Polyvagal Theory. This letter reminded me that neonatologists as well as obstetricians have been trained to interpret clinical bradycardia, the massive slowing of heart rate that could be lethal, as being a vagal phenomenon. In my work with high-risk neonates, the bradycardia described by the neonatologist seemed to only occur when RSA was low. This was later confirmed (see Reed et al., 1999).

Thus, I hypothesized in the 1992 paper that RSA was an index of more optimal autonomic regulation and positive outcomes. The neonatologist ended his letter by

assuming that there was a common vagal source leading to both RSA and bradycardia and perhaps too much of a good thing was bad.

His comment was set within a background of how the neural regulation of the autonomic nervous system was currently being taught in medical schools. Basically, the broad view was that vagal activity, often generalized to be inclusive of all parasympathetic activity, was assumed to serve homeostatic functions when within a functional band of activation, suggesting that too low or too high might result in pathophysiology. However, based on my own research experience, I did not agree with his suggestion of an inverted U-shaped relation between vagal tone and health.

Within this view, vagal activation up to a specific level, reflected in RSA, would index optimal function, while increasing beyond that point would produce bradycardia and health risk. However, his suggestion was consistent with the Yerkes-Dodson law, a dominant view in psychology proposing an inverted U-shaped function between arousal and performance where, once an optimal level of arousal was exceeded, performance declined. In the law, arousal is conceptualized as either sympathetic or cortical activation.

Note that this is also consistent with Dr. Dan Siegel's well-accepted "window of tolerance" within mental health treatment models.[14] However, the window of tolerance model is more consistent with the Polyvagal Theory by acknowledging that the tails of the inverted U-shaped function are mediated by different mechanisms.

The neonatologist's letter triggered my curiosity, and I was perplexed by the possibility of a "vagal paradox". After carrying the letter in my briefcase for a few months, I drafted the paradox into the two sentences below.

1. Bradycardia is a potentially lethal risk index mediated by the vagus.
2. RSA is a protective factor index mediated by the vagus.

This contradiction in interpretation of vagal mechanisms formed the basis of the vagal paradox by posing the question: How could the vagus be both protective, when it was expressed as RSA, and life-threatening, when it was expressed as bradycardia and apnea? By formulating the paradox, it became clear to me that the vagus was involved in sending contradictory signals to the heart. Now I had to find an explanation of how the vagus could index both health and risk; how it could both protect and injure the heart. This led me into a deep literature review of all things vagal. Identifying the vagal mechanisms underlying the paradox evolved into the Polyvagal Theory.

A Polyvagal Solution

In 1993 when I started on this quest, I was a faculty member at the University of Maryland. At that time, I also held an appointment as an Adjunct Scientist in the Laboratory of Comparative Ethology at the National Institute of Child Health and Human Development within the National Institutes of Health (NIH). The laboratory chief, Stephen Suomi, was a good friend and colleague in the area of developmental psychobiology. Within his laboratory, I collaborated with another friend and colleague, Michael Lamb, chief of the section on Social and Emotional Development.

My position at NIH provided me with access to their library and research librarians. I also had the privilege of borrowing books from the National Library of Medicine. Both resources were walking distance from my home and provided me with access to the scientific literature documenting what was currently known about the vagus nerve. This exploratory journey occurred prior to the internet with the search tools of Google Scholar and PubMed. This was still a time of inquiry dependent on physical journals and books, and the NIH librarians were helpful in printing out abstracts of journal papers from dedicated searches.

After spending weeks wandering into the stacks of the NIH library, I ended up exploring books and journals in comparative anatomy. Comparative anatomy is an intriguing discipline that identifies similarities and differences in anatomical structures across vertebrate species. Comparative disciplines are helpful in generating hypotheses regarding evolution.

From this comparative perspective, vagal pathways followed an evolutionary sequence. Suddenly, the paradox was solved. In reptiles and more primitive vertebrates, the vagus reliably produces bradycardia and is frequently involved in defensive strategies of immobilization including death feigning. While in mammals, the ventral vagus was linked to calmness and sociality. For the preterm newborn, the ventral vagus is not fully functioning, and the infant enters the world with an autonomic nervous system (ANS) that has many attributes of a reptile. Thus, the clinical bradycardia was functionally a defense reaction to conserve metabolic resources, which would be adaptive in the earlier- evolved vertebrates who had small brains without limited need for oxygenated blood.

Hypotheses driven by PVT are related to the documentation that the mammalian ANS has a built-in hierarchy of autonomic reactivity based on phylogeny that is mirrored in embryological development. In other words, the hierarchical nature of the ANS results from how it developed evolutionarily, and that same development can be seen in the way a fetus's ANS develops and grows in the womb—with older circuits developing first.

This fact became a core principle upon which PVT-informed hypotheses could be tested. This emphasis on hierarchy is focused on ANS reactivity and does not preclude the optimal homeostatic states that involve a synergism and functional balance between parasympathetic and sympathetic influences (i.e., hybrid states). For example, based on the state of the ventral vagus and the social engagement system, we can determine if children are playing or fighting. The distinction becomes clear when we look at their faces and listen to their voices. Similarly, the social engagement system can calm an infant sufficiently to move into a restful dorsal vagal state that would support health, growth, and restoration. Thus, depending on the state of the ventral vagus, autonomic regulation may function either hierarchically, synergistically, or antagonistically.

The "Polyvagal" solution to the vagal paradox emphasizes that:

1. Not all vagal pathways support social communication, downregulate stress, and enhance resilience.
2. There are vagal pathways that can be recruited for defense and are potentially lethal.
3. The neural circuits regulating the autonomic nervous system function hierarchically in which evolutionarily newer circuits inhibit older ones.
4. Development of the neural pathways regulating the autonomic nervous system parallel phylogeny with evolutionarily older circuits developing first.

The Discovery of the Social Engagement System

Although the relationship between the ventral vagus and muscles of the face and head was described in the introduction of the Polyvagal Theory (see Footnote 5), it took a few years to gain a better grasp of how these neural pathways were related to PVT. Serendipitously, I was invited to participate in an innovative workshop entitled "Is there a neurobiology of love?" The workshop invited scientists across several disciplines to discuss how biological systems were involved in social relationships. The result of the meeting was a well-cited special issue of the journal *Psychoneuroendocrinology* (see Footnote 8).

The meeting was organized by Dr. Sue Carter. Sue is the scientist who identified the important role of oxytocin in social behavior. She is noted for her observations of the prairie vole, a rodent species that create lifelong bonds with their mated partners. Sue is also my wife and has been instrumental in shifting my research trajectory toward sociality, co-regulation, and trust. We were married in 1970 and have independent, although parallel, career trajectories. In fact, it took more than 20 years to realize that we were studying overlapping phenomena from complementary perspectives.

Sue focused on the neuropeptides of oxytocin and vasopressin, while I focused on autonomic regulation emphasizing the expansive role of vagal circuits. I frequently mention in my talks that although we were married, it took about 20 years until we were reacquainted in the brainstem. There is factual substance in that statement, since the brainstem nuclei related to the vagus have an abundance of receptors sensitive to both oxytocin and vasopressin. We soon discovered that there was synergism not only in our personal lives but also in our research. And, similar to all good relationships, we were able to respectfully explore similar phenomena from two perspectives.

I felt challenged by the invitation and decided to explore how PVT would approach the proposed question. As I explored the circuits that were identified in the initial PVT paper, I realized an interesting and compelling circuit related to sociality and nursing emerged with the ventral vagus. Functionally, evolutionary processes resulted in a system that linked vagal pathways that could slow heart rate and calm autonomic state with the structures that mammals use to signal and detect cues of safety and trust.

Early in development, this system is involved in nursing and requires the coordination of sucking, swallowing, breathing, and vocalizing. This coordination requires vagal pathways, including the vagal pathways regulating the laryngeal and pharyngeal muscles that produce the intonations of vocalizations. This mammalian innovation of parallel vagal pathways influencing heart rate and intonation provided the mechanism to literally broadcast autonomic state to a conspecific, another of the same species.

Of course, we recognize this feature in support of infants and even our pets, who broadcast their needs for food or feelings of distress in the intonation of their vocalizations. A deeper dive into this circuit identified an area of the brainstem that could be labeled the ventral vagal complex, since it contains brainstem nuclei linking the regulation of the ventral vagus with the striated (skeletal) muscles of the face and head. In preparing for the conference and the subsequent journal article, I realized that this neural circuit not only formed the basis of nursing but provided a circuit involved in establishing social engagement, connection, and trust. Moreover, this

circuit is involved during the process of listening and influences our ability to process and interpret the acoustic world around us.

Within Polyvagal Theory, the evolutionary trajectory of the vagus has led to a conceptualization of an emergent and uniquely mammalian social engagement system in which a modified branch of the vagus is integral. Neuroanatomically, this system is dependent on a brainstem area known as the ventral vagal complex. This area not only regulates the mammalian ventral cardio-inhibitory vagal pathway but also involves pathways within other cranial nerves controlling the striated muscles of the face and head. This does not preclude other structures being involved in mammalian social engagement behaviors or homologous structures in other vertebrates that do not share our phylogenetic history exhibiting social behaviors.

The relationship between mothers and their nursing offspring illustrates the social engagement system in action. To survive, mammalian offspring must initially nurse as the primary mode of ingesting food. To nurse, the infant must suck, a process dependent on a brainstem circuit involving the ventral vagal complex. Survival is dependent on the infant's nervous system efficiently and effectively coordinating suck-swallow-breathe-vocalize behaviors with vagal regulation of the heart through the ventral vagal pathways. Through maturation and socialization, this "ingestive" circuit provides the structural neural platform for sociality and co-regulation to act as major mediators optimizing homeostatic function leading to health, growth, and restoration (see Footnote 3).

For mammals, there is a dependency between reactions to contextual cues and the function of this circuit. Cues of threat may disrupt, while cues of safety may enhance function. The sensory branches of the facial and trigeminal nerves provide major input into the ventral vagal complex. Functionally, changes in the state of this circuit, through the process of dissolution, will either "disinhibit" phylogenetically older autonomic circuits to support defense (e.g., against a predator, disease, physical injury) or inform all aspects of the autonomic nervous system, including the enteric system (see Footnotes 13, 14), to optimize homeostatic function.

Mammals, unique from their ancestral reptiles, have detached middle ear bones, which distinguish them from reptiles in the fossil record. Detached middle ear bones expand the frequency band that mammals can hear and provide a "safe" frequency band in which they can socially communicate that will not be detected by reptiles.

Early Clues to Developing SSP

Serendipitously, my investigation of middle ear structures led to the development of the SSP. Middle ear bones (as a group of three, called ossicles) are small bones that

separate from the jawbone during gestational development and form an ossicle chain that connects the eardrum to the inner ear. Small muscles regulated by branches of the trigeminal and facial nerves determine how sound is transmitted through the middle ear. These muscles also determine the loudness of specific sound frequencies transduced through middle ear structures (i.e., middle ear transfer function) by controlling the stiffness of the ossicle chain and the tightness of the eardrum.

When the ossicle chain is stiff, the eardrum is tighter and low-frequency sounds are dampened, but when the muscles relax, lower-frequency sounds pass into the inner ear. In all mammalian species, based on the physics of their middle ear structures, there is a *"frequency band of perceptual advantage*" that is available when the middle ear muscles contract and stiffen the ossicle chain (see Footnote 15). It is within this frequency band that social communication and acoustic signals of safety occur for mammals. This is possible because the low frequencies that, through evolution, had been associated with predators are dampened (see Footnote 16). In the reptile ancestors that preceded mammals, the middle ear bones were still fused to the jawbone. This means they could not hear higher-frequency sounds, resulting in mammals being able to communicate through vocalizations that reptiles cannot hear.

Interestingly, the coordination of the contraction and relaxation of these small muscles is frequently co-regulated with autonomic state and thus contract when there is strong ventral vagal tone to promote social communication and co-regulation. In contrast, when the autonomic nervous system shifts to a state of defense, the muscles relax to detect low-frequency predator sounds, which supports defense strategies relying on auditory cues. This parallel between autonomic state and defensiveness may explain why many children with problems in auditory processing and language delays may also have behavioral state regulation limitations.

Using the Middle Ear Muscles to Retune the ANS

The neurophysiological link between autonomic state and the middle ear muscles provided a portal to regulate autonomic state through acoustic stimulation, which is easily observable when a mother calms her infant using prosodic vocalization. Similarly, we can observe the potent calming influences when a pet is calmed by the voice of a human. We may think it is the magic of our words, while it is actually the intonation patterns communicating in the frequency band of perceptual advantage. Of course, the smaller the child or mammalian pet, the smaller their middle ear structures, and the higher their frequency band of perceptual advantage. Thus, the frequency band in which the infant or pet detects signals of safety requires the higher pitch that characterizes how adults apparently intuitively produce infant and pet-directed speech.

The mother-infant interaction provides a platform to explore the intuitive origins of the SSP. Effective mothers, caregivers, and teachers have intuitively used their social engagement system to broadcast vocal and facial signals of safety to their children, patients, and students. Recently, we published a study documenting the powerful relationship between quantifiable acoustic parameters of maternal voices while calming their infants following an experimental procedure that stressed the infant[15]. Following the stressful experience, the mother was instructed to engage and calm her infant. We investigated the vocal qualities of the mother's voice and the infant's distress and heart rate. Our analyses documented a strong relationship between the prosodic characteristics of the mother's voice and the infant's heart rate and distress behavior.

Analysis of the mother's voice provided a metric of vocal intonation conveying positive prosodic features. Confirming our hypothesis, mothers whose vocal intonations were more melodic were more effective in calming their infants. The mothers with the most prosodic voices reduced infant heart rate more than 10 beats per minute, while the least prosodic voice had no impact on the infant heart rate. Similarly, a measure of behavioral distress paralleled the changes in heart rate. The study confirmed a common belief in how to calm children and pets. Interestingly, this intuitive strategy seems to have been missed by teachers and health-care providers.

The Foundational Building Blocks of SSP

The above information serves as the foundational building blocks that were integrated into SSP. Below, these foundational principles derived from PVT can be outlined:

1. Pitch and especially the modulation of pitch are critical in signaling safety and calming the autonomic nervous system.
2. The frequency band of perceptual advantage is a functional product of the middle ear structure, which essentially dampens low-frequency sounds and enhances the sounds of social communication.
3. Listening both influences and is influenced by autonomic state.
4. Acoustic signals of safety broadcast the accessibility of another (e.g., a mother's soothing voice) via neuroception, which reflexively calms the autonomic nervous system of the listener.
5. The SSP is a neural exercise that progressively expands the capacity of the dysregulated autonomic nervous system to process the full frequency band of perceptual advantage.

6. The capacity to process the full range of the frequency band of perceptual advantage dampens the hypersensitivities to threat signals while enhancing the attributes of the ventral vagal complex—spontaneous social engagement, biobehavioral resilience, co-regulation, and optimizing homeostatic function.

The Early History of SSP

Between 1985 and 2001, when I was a faculty member at the University of Maryland, I was interested in the autonomic and behavioral regulation of children who were on the autism spectrum. To me, the behaviors expressed by these children seemed to be naturally emergent from an autonomic nervous system locked in a state of defensiveness. I still vividly recall my laboratory interactions with children diagnosed with autism spectrum disorders and their families. Paramount in these memories were their destabilized behavioral state, their tactile and auditory defensiveness, their selective eating, and their frequent noncontingent responses to caregivers' attempting to calm them.

At that time, I met and had frequent discussions with Dr. Stanley Greenspan. Stanley was a passionate child psychiatrist who had a model to optimize the social behavior of autistic children through reciprocal interactions. In general, his model invited the parent to follow the child's lead. In the model, the child's behaviors were being encouraged by the parents' accessibility and reciprocal interactive exchanges.

This strategy was challenging to the intervention community, which had assumed that behavior modification was the preferred mode of intervention. Behavior modification, officially known as Applied Behavioral Analysis, or ABA, was the accepted treatment model in special education and the treatment of children on the spectrum. However, Greenspan opposed the prevailing model and argued against enforcing a strict stimulus-response model. Greenspan formulated his model into a strategy that is known as Floortime. Although not necessarily stated by Greenspan, Floortime and ABA are both based on a learning model; ABA on an objective reinforcer (e.g., reinforcing with M&M's), while Floortime used a more ecologically valid reinforcer (e.g., the parent's engagement behavior).

As I was formulating PVT at that time, I was curious if deficits in interactive social behavior were dependent on autonomic state. Based on my research I had two working hypotheses. The first hypothesized a relationship between autonomic state and the development of contingencies. The second hypothesized that there were measurable

neurophysiological contingencies within the autonomic nervous system that were a foundational substrate underlying observable contingent behavior.

The first hypothesis was consistent with my earlier research, which documented that autonomic state influenced the contingency of an infant's heart rate to reliably react to and even anticipate auditory and visual stimuli. For example, even during the newborn period, infants with greater ventral vagal influences to the heart responded with more reliable heart rate reactions to sensory stimuli and even developed anticipatory heart rate reactions when the stimuli were presented repeatedly at fixed intervals. Similarly, adults with greater ventral vagal influence were more likely to develop anticipatory heart rate responses when a signal preceded the required response in a reaction time task. This form of learning of a temporal contingency is known as temporal conditioning. My early research was suggesting that autonomic state mediated both autonomic reactivity and contingency learning.

The second hypothesis was consistent with my work in the 1980s during which I was developing new metrics to describe the coupling between breathing and heart rate. Specifically, I developed a metric, which I called "weighted coherence," that quantified how tightly coupled (i.e., contingent) changes in breathing were related to the periodic changes in heart occurring at the same frequency as breathing known as respiratory sinus arrhythmia (RSA). The amplitude of RSA has subsequently been used as the gold standard for estimating cardiac vagal tone.

Coherence is conceptually similar to correlation, but it is a "frequency" domain metric. Coherence evaluates the coupling between two time series (i.e., signals that are indexed by time). In my work, the time series data were the dynamically changing chest circumference (i.e., breathing) and the synchronous beat-to-beat changes in heart rate that approximated a respiratory pattern (i.e., RSA). When coherence approaches 1.0, breathing changes are directly mapped into the heart rate pattern. With high coherence, there is an observable phase locking during which changes in breathing are reliably related to changes in heart rate. Thus, the higher the coherence, the more contingent RSA was to breathing. Since breathing rate varies, weighted coherence provides a summary statistic of this form of contingency across the frequency band of spontaneous breathing. As I developed this metric, I modeled a hierarchy in which neural contingency formed the foundation of contingent social behavior. Thus, I hypothesized that poor autonomic regulation was foundational to noncontingent behaviors expressed by individuals on the spectrum. Based on this speculation, I hypothesized that signals of calming and safety would recruit the ventral vagal complex to promote sufficient accessibility to express contingent social behavior. As the Polyvagal Theory evolved, I began to understand the profound signal

power of prosodic vocalizations in calming and started to use the mother's vocal calming of an infant and our vocal engagement of our mammalian pets as a model for a potential intervention.

Earlier I proposed a hierarchical model of self- and co-regulation to provide insights into optimizing intervention strategies for high-risk infants[16] . Since individuals on the autism spectrum frequently have difficulties in these same domains, the model is helpful in understanding my insights into developing the SSP as a portal of calming to enhance accessibility and social contingency. The model reflects maturational competencies in neural regulation that provide a substrate for the more complex co-regulatory social behaviors. The main point of the model is that higher behavioral functions, which are frequently intentional, are dependent on the functioning of the more survival-focused foundational systems embedded in the brainstem. To me, it was obvious that the autistic phenotype behaviorally embodied difficulties in state regulation as well as noncontingent social behavior, both being assumed to be intentional. I speculated that brainstem efficiency in regulating autonomic state might be a foundational portal into understanding some of the prominent features of autism. Moreover, since these features were functionally an adaptive reaction to signals of threat, I speculated that signals of safety might shift an autonomic state that supports defensiveness to an autonomic state that supports social engagement and contingent co-regulation. The SSP grew out of these insights and speculations. The levels are described in Table 1.

Level I is focused on the function of brainstem structures in optimizing physiological homeostasis through neural and neurochemical bidirectional communication between visceral organs and brainstem structures. The neural pathways involved in Level I are functional at birth in healthy full-term infants. An index of Level I can be derived from quantifying RSA, a periodic component of beat-to-beat heart rate variability that is synchronous with spontaneous breathing and a valid index of cardiac vagal tone via ventral vagal pathways[17].

Level II emphasizes connections between higher brain structures and the brainstem in regulating autonomic state. Success in Level II is achieved when the suck-breathe-vocalize circuit is integrated with the ventral vagal pathway[18]. This circuit enables nursing and soothing to occur and is dependent on the neural pathways that define the ventral vagal complex and support the social engagement system (see Footnote 8). Similarly, the weighted coherence and the more recent vagal efficiency metric tap into the coordination among brainstem nuclei communicating with the ventral vagal complex, the pathways that foster social communication and

co-regulation. Level II provides the foundational neural platform for feelings of safety and access to the circuits that would enable a neuroception of safety.

Table 1 emphasizes the hierarchical nature of specific autonomic states and accessibility of behaviors that we cluster as self-regulation skills. The optimal function of each level is dependent on each of the preceding levels being adequately functioning.

Focusing on Levels I and II we see that optimal behavior is dependent on the neural regulation of the autonomic nervous system and the connectivity between cortical areas, allowing the accurate interpretation of cues of safety and threat, and the brainstem areas regulating the autonomic nervous system. The quantification of RSA provides a quantitative portal into Level I, while the weighted coherence and vagal efficiency metric would reflect Level II competency.

In an autonomic state that supports threat, even the foundational processes described in Levels I and II would be compromised. When these foundational levels are functional, the nervous system can support coordinated goal-directed behaviors (Level III) and contingent social interactions (Level IV). It was obvious, as I observed the diverse phenotype of autism, that there was a core feature of defensiveness that could be detected in behavior, facial expression, and intonations of vocalizations. I hypothesized that underlying these features was an autonomic state locked in defense. My response to this hypothesis was the SSP.

Table 1: Hierarchical model of self-regulation (Porges, 1996).

Level I: Neurophysiological processes characterized by bidirectional communication between the brainstem and peripheral organs to maintain physiological homeostasis.

Level II: Physiological processes reflecting the input of higher nervous system influences on the brainstem regulation of homeostasis. These processes are associated with modulating metabolic output and energy resources to support adaptive responses to environmental demands.

Level III: Measurable and often observable motor processes, including body movements and facial expressions. These processes can be evaluated in terms of quantity, quality, and appropriateness.

Level IV: Processes that reflect the coordination of motor behavior, emotional tone, and bodily state to successfully negotiate social interactions. Unlike those of Level III, these processes are contingent with prioritized cues and feedback from the external environment.

Creating and Researching the SSP

In the late 1990s, with PVT as a model, I started to deconstruct the features of autism and to work on an acoustic intervention. The intervention was initially called the Listening Project Protocol (LPP).

By the late 1990s, through my friendship and collaborations with Stanley Greenspan, I had spent hundreds of hours observing autistic children. I witnessed the defensiveness of these children's physiology and their hypersensitivities. I intuitively wanted to calm their nervous systems to enable them to feel more comfortable in their bodies. Based on a desire to reduce their sensory burden and to provide expanded moments during which they and their parents could feel connected with each other, I started to conceive of a stealth intervention, a way of calming that was totally different from the treatment models that emphasized behavioral modification techniques in the 1990s. As I watched these children, I would cringe as they were treated in a robotic deterministic manner using food (e.g., M&M's) to shape their behaviors. I saw the children as being locked in a state of threat. I wondered how their lives would be if they felt safe enough to literally give up their defenses and calm. I wondered if PVT would provide the insights to craft such an intervention. Thus, the SSP, or more accurately the LPP, was prototyped as a stealth intervention that communicated with the hardwired circuits that the prosodic vocalizations of a mother's lullaby intuitively recruited.

I envisioned a neural exercise in which the acoustic signal would be ported into the child's nervous system through a narrow frequency band that would be available even when in a defensive state. The intervention would progressively expand and contract the portal until the child experienced and welcomed the entire frequency band of perceptual advantage, that is, the frequencies through which social communication occurs. I theorized that as the acoustic frequency band expanded and contracted, not only would the dynamics of the middle ear structures be recruited to optimize the processing of these signals of safety, but the entire social engagement system would become available. I visualized the LPP as recruiting the powerful calming of the ventral vagus, enhancing the prosody of the child's voice, reducing auditory hypersensitivities, enhancing facial expressivity, and even impacting on ingestive behavior.

With a prototype created with an early version of Adobe Audition, I set up a laboratory at the University of Maryland to test children on the autism spectrum. The protocol was structured as a neural exercise with five one-hour sessions being delivered on five sequential days. Our research staff engaged the children and their parents to support the biobehavioral state of their child. Parents and staff would provide toys and food. In the initial pilot study, I had the idea that the social stimulation

of peers would facilitate effectiveness, and four children simultaneously received the intervention. The computer-altered music was played on a CD player that had an output adapter connecting to headphones with long cords. This allowed the children to move around the room.

During the initial pilot study, we observed completely unexpected behaviors. Children began sharing and exhibiting reciprocal play behaviors. Perhaps my most poignant memory from these early reactions to the LPP involves a child who could not tolerate wearing a headset. To address this challenge, I constructed a special sound-attenuated cube using sound-dampening blankets attached to PVC pipes with Velcro fasteners. When this hypersensitive nonverbal child entered the cube and heard the computer-altered music, he clearly articulated one word: "Safe."

We received reports of children telling their parents they loved them for the first time. Fathers shared that, as their child's social engagement system became functional, they felt they truly had a son. These moments highlighted a profound shift as families became more connected to their children.

Our research documented that the features of the social engagement system (see Footnote 8) were consistently dampened in these children, irrespective of diagnosis severity, cognitive functioning level, or hypersensitivity profile. Through continued investigation, we began to document how this "stealth" intervention significantly reduced hypersensitivities, improved auditory processing, encouraged sharing behaviors, enhanced emotional regulation, and promoted spontaneous social engagement. These insights informed our approach, and our staff intuitively adopted a Polyvagal-informed perspective in their co-regulatory and supportive interactions with autistic children and their families.

In 2001, we relocated our laboratory from the University of Maryland to the University of Illinois College of Medicine's Department of Psychiatry. At Illinois, we established the Brain-Body Center, a relatively large research center dedicated to advancing our understanding of autism and related conditions. We collected additional data and dedicated significant time to analyzing and interpreting the findings we had gathered in Maryland. As a scientist, my training equipped me to structure research questions, test hypotheses, and propose explanations. However, the real challenge lay in translating these findings into practice. I was neither trained nor prepared to develop an accessible, scalable treatment model for therapists.

Fortunately, in 2015, I met Karen Onderko and Randall Redfield, the CEO of Integrated Listening Systems. Our interactions led to Integrated Listening Systems licensing the technology embedded in the LPP. The protocol was renamed the Safe and Sound Protocol and introduced to the broader clinical community in early 2017.

It is through the experiences and collaboration of early adopters that the SSP's understanding, protocols, and potential have evolved. Research is inherently a team effort, and these pioneering providers—and those practicing today—have played a critical role in refining its delivery. By tailoring the SSP to meet the needs of individual clients and incorporating their attuned presence and co-regulation into the process, they have deepened our understanding of its application. We have gained invaluable insights from their experiences and look forward to continuing to learn from their contributions.

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