

SCIENCE BRIEFS

Polyvagal Theory: Three Neural Circuits Regulate Behavioral Reactivity

by Steven Porges, University of Illinois, Chicago

As evolutionary forces molded the human nervous system, new structures were added and older structures were modified. The early vertebrate brains looked remarkably similar to our brainstems. These primitive brains were composed primarily of reflexive driven neural circuits that attempted to conserve metabolic resources, while valiantly protecting visceral function. Through the process of evolution, the primitive brain structures with their reflexively wired neural circuits remained, but became neurally intertwined with the newer and larger brain structures that characterize humans and other mammals. Unlike the primitive vertebrate brains, the mammalian brains with their new structures were oxygen hungry and were easily and irreparably damaged when oxygen saturation in the blood dropped. With the increased neural complexity, the brains of mammals were capable of exhibiting complex behaviors in response to environmental challenges without jeopardizing the basic bodily need to maintain visceral homeostasis. The result is a large complex neural system capable of learning, problem solving, expressing a range of affect, and establishing social bonds.

To survive, mammals must determine friend from foe, evaluate whether the environment is safe, and communicate with their social unit. These survival-related behaviors have different metabolic requirements and are associated with specific physiological states regulated by the autonomic nervous system. The autonomic nervous system is not merely a peripheral neural system, but involves brainstem structures that monitor visceral state and control the output of the autonomic nerves communicating directly with visceral organs (e.g., heart, lung, gut, etc.). Through neural pathways visceral afferent information influences higher brain structures and the state of higher brain structures influences the neural input to visceral organs.



Stephen W. Porges received his PhD from Michigan State University. He has been a faculty member at West Virginia University (1970-1972), the University of Illinois at Urbana-Champaign (1972-1985) and the University of Maryland (1985-2001) where he was Chair of the Department of Human Development and Director of the Institute for Child Study. Currently he is a Professor of Psychiatry and Director of the Brain-Body Center in the College of Medicine at the University of Illinois at Chicago. He is President of the Federation of Behavioral, Psychological and Cognitive Sciences and the former President of the Society for Psychophysiological Research. His research crosses traditional discipline boundaries and focuses on the dependence of social behavior on physiological states.

This simplified description of a feedback loop provides a schema of how environmental context and subjective experience can influence physiological state, and how physiological state can limit a mammal's ability to deal with the environmental challenge.

During the past decade we have been developing and testing a theory that explains how, through the process of evolution, the primitive vertebrate autonomic nervous system evolved into the mammalian autonomic nervous system with unique functional features that regulate visceral state to

support social behavior. The theory is called the Polyvagal Theory (Porges, 1995, 1997, 1998, 2001) to emphasize the phylogenetic shifts in the vagus, a cranial nerve that provides the primary parasympathetic input to the autonomic nervous system. Primitive vertebrates have an unmyelinated vagus, while only mammals have both unmyelinated and myelinated vagal efferent pathways. The myelinated and unmyelinated vagal pathways have different functions, originate in different areas of the brainstem, and support different adaptive behavioral strategies.

According to the theory the three behavioral strategies include social engagement (e.g., facial expression, vocalization, listening), mobilization (e.g., fight-flight behaviors), and immobilization (e.g., feigning death, fainting, and behavioral shutdown). The social engagement system is uniquely mammalian and is dependent upon myelinated vagal motor fibers found only in mammals. This mammalian vagus is capable of fostering calm behavioral states and can actively inhibit the sympathetic influences to the heart and dampen the HPA axis. In contrast, the mobilization system, which supports fight and flight behavior, is dependent on the sympathetic nervous system. The most phylogenetically primitive neural circuit, the immobilization system, is dependent on the unmyelinated or "vegetative" vagus, which is shared with most vertebrates. In addition to the mammalian circuit that supports social engagement, the theory proposes that mammals retain neural circuits common to phylogenetically older vertebrates and that the recruitment of these older circuits in regulating autonomic state follows a hierarchy in which the newest circuits are employed first.

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The Social Engagement System

The Social Engagement System provides the neural structures involved in social and emotional behaviors. The Social Engagement System has two components: autonomic and somatomotor. The autonomic component is mediated by the myelinated vagus, which as described above promotes calm behavioral states. The somatomotor component is mediated by components of several cranial nerves that are known collectively as special visceral efferent pathways. The social engagement system has a control component in the cortex (i.e., upper motor neurons) that regulates brainstem nuclei (i.e., lower motor neurons) to control eyelid opening (e.g., looking), facial muscles (e.g., emotional expression), middle ear muscles (e.g., extracting human voice from background noise), muscle of mastication (e.g., ingestion), laryngeal and pharyngeal muscles (e.g., vocalization and language), and head turning muscles (e.g., social gesture and orientation). Collectively, these muscles function both as filters that limit social stimuli (e.g., observing facial features and listening to human voice) and as active features of engagement with the social environment. The neural control of these muscles determines social experiences. In addition, the source nuclei (i.e., lower motor neurons) of these nerves, which are located in the brainstem, communicate directly with the autonomic component (i.e., the myelinated vagus) that slows heart rate, lowers blood pressure, and actively reduces arousal to promote calm states consistent with the metabolic demands of growth and restoration of our neurophysiological systems.

Sound in our environment impinges on the eardrum and causes it to vibrate. These vibrations are transduced from the eardrum to the inner ear via the small bones in the middle ear known as ossicles. The changing neural tone to the stapedius muscle (innervated via a branch of the facial nerve) and the tensor tympani (innervated

via a branch of the trigeminal nerve) regulates the stiffness of the ossicular chain. When the ossicular chain is stiff, the loudness of low frequency sounds reaching the inner ear is dampened. The functional 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.

As vertebrates evolved from reptiles to mammals, the structures at the end of the jawbone detached and became the small bones of the middle ear. The evolution of the mammalian middle ear enabled low amplitude relatively high frequency airborne sounds (i.e., sounds in the frequency of vocalizations) to be heard, even when the acoustic environment was dominated by low frequency sounds. This phylogenetic innovation enabled the early mammals to communicate in a frequency band that could not be detected by reptiles. Reptiles, due to their dependence on bone conduction, could hear only lower frequencies. However, 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 stiffen the ossicular chain.

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Without stiffening the ossicular chain, mammals would lose this advantage and the soft airborne sounds of vocalizations would be easily lost in the louder low frequency background sounds.

Clinical Applications of the Polyvagal Theory

The Polyvagal Theory, by describing both the phylogenetic based hierarchy of autonomic states and the “triggers” that cause a degrading of this hierarchy, provides a new way of investigating the atypical social behavior associated with several psychiatric disorders. The theory emphasizes that the mammalian nervous system is not only sensitive to environmental demands and perceived stress and threat, but that the mammalian nervous system will, in a predictable order, rapidly reorganize to different neural-mediated states. The Polyvagal Theory forces us to interpret compromised social behavior from a different perspective. The theory emphasizes that the range of social behavior is limited by physiological state. The theory also emphasizes that mobilization and immobilization behaviors may be adaptive strategies to a challenged (i.e., frightened) individual. Thus, we decided to test the theory by hypothesizing that it would be possible to improve spontaneous social behavior by exercising the neural regulation of brainstem structures involved in the Social Engagement System during states of calmness.

We developed an intervention that uses acoustic stimulation to stimulate the neural regulation of the Social Engagement System. We tested our intervention by observing the social behavior of children diagnosed with autism. The intervention was based on several principles derived from the Polyvagal Theory. First, the area of the brainstem that regulates the mammalian or myelinated vagus also regulates the muscles of the head including those of the face, middle ear, mouth, larynx, and pharynx. Collectively, these muscles function with the myelinated vagus as an integrated Social Engagement System that maintains calm behavioral states and controls looking, listening, vocalizing, and facial gesturing. If the neural regulation of the somatomotor component is dysfunctional, the face will not work (e.g., lack of facial expressiveness, eyelid opening, prosody and listening). If the

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autonomic component is dysfunctional, the ability to regulate behavior and maintain calm states will be compromised, since the autonomic state will reflect a sympathetic state that supports fight and flight behaviors. Interestingly, these autonomic and facial features are often associated with psychopathologies (e.g., autism, depression, aggressive disorders, and post traumatic stress disorder) or emotional states during severe challenge (e.g., grief, rage, anger, loneliness) or medical illness (e.g., senility, AIDS, fever).

Second, the middle ear muscles play an important role in extracting human voice from our complex acoustic environment. When the neural tone to the middle ear muscles is low, the middle ear structures do not actively filter out the low frequency sounds that dominate the acoustic environment in our modern industrial world and it becomes difficult to understand the content of human voice. This difficulty in listening might occur even in an individual who has normal hearing (i.e., normal function of the cochlea, the auditory nerve, and the brain areas processing acoustic information).

Third, the neural regulation of the middle ear muscles is neuroanatomically linked to the regulation of the muscles controlling facial expression and vocal intonation. Thus, if stimulation improves the neural regulation of the middle ear muscles, effects should also be observed in facial expression, looking, and vocalizing.

In collaboration with my colleague, Olga Bazhenova, an intervention was designed to recruit the cortical regulation of the Social Engagement System to promote the voluntary prosocial behaviors that are lacking in autistic children. The model is an optimistic model, because it assumes that, for many children with social behavior difficulties, the Social Engagement System is neuroanatomically and neurophysiologically intact. The problem is conceptualized as a functional deficit. Thus, to increase the occurrence of spontaneous social behavior, the intervention must stimulate the cortical

regulation of the brainstem system controlling the muscles of the head. The theory predicts that once cortical regulation of the brainstem system is engaged, social behavior and communication will spontaneously occur as the natural emergent properties of this biological system. Thus, the intervention is seen as "stimulation" and "exercise" of the nerves that regulate the muscles of the head.

To stimulate the Social Engagement System, recordings of children's songs were computer altered to remove all the frequencies outside the range of human voice and to modulate the remaining frequencies. The process produced five 45-minute programs. Each program progressively added more frequencies until the entire range of frequencies was present. The acoustic stimulation was delivered through headphones, while the children were in a playroom. One program was presented on each of five sequential days. Consistent with the theory, the experimenter and a parent attempted to maintain the child in a calm behavioral state, while the child was listening. Behaviors were assessed by parental questionnaires and by coding video recordings of a shared attention task.

In preliminary studies approximately 100 children with autism spectrum disorders between the ages of 3 and 5 were tested. Subjects were assigned to groups that: 1) received the computer altered acoustic stimulation, 2) wore headphones without receiving acous-

tic stimulation, 3) received unfiltered music, and 4) received assessments only. The data demonstrated the unique effectiveness of the computer altered acoustic stimulation, particularly in reducing hearing sensitivities and in increasing sharing behaviors. The improvements persisted when assessed during a three-month follow-up. In addition and consistent with the theoretical mode, as the children increased their spontaneous social engagement behaviors, the parents became less intrusive in their interactions. Currently we are expanding the application of the intervention research to include adults with autism and children with language delays.

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