Polyvagal Theory: Background & Criticism

A brief description of the origin, formulation and criticism of PVT, August 2021

PVI is pleased to release the following commentary from Dr. Stephen Porges regarding his Polyvagal Theory (PVT). It includes an introductory description, prerequisite information and the primary principles; the last section addresses criticism of the theory since its original publication in 1995.

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Introduction

Since the publication of Polyvagal Theory (PVT) in 1995, Dr. Porges has released occasional updates addressing its formulation and f the Polyvagal Theory is a science-based theory that provides explanations of how our autonomic state influences and is influenced by the dynamic challenges of life. The theory, as a perspective, impacts on several aspects of human experience as well as the experiences of other mammals that share features of the neural regulation of the autonomic nervous system. It is a transdisciplinary theory extracting common themes across a broad range of disciplines.

Polyvagal Theory was introduced by behavioral neuroscientist, Stephen W. Porges, in his presidential address to the Society of Psychophysiological Research in Atlanta Georgia on October 8, 1994. The talk was later published in *Psychophysiology* 1995 with the title *Orienting in a defensive world: Mammalian modifications of our evolutionary heritage. A polyvagal theory* (Porges, 1995). The title provides a succinct synopsis of the theory in which the phylogenetic transition from asocial reptiles to social mammals is discussed emphasizing the convergent shifts in the neural regulation of the autonomic nervous system and the adaptive biobehavioral consequences. Functionally, unlike their reptilian ancestors, mammals had a newer branch of the vagus providing cardioinhibitory influences on the heart that emerge from a ventral area of the brainstem (i.e., nucleus ambiguus). In contrast, other classes of vertebrates (e.g., reptiles, amphibians, etc) had vagal cardioinhibitory pathways emerging only from a dorsal area of the brainstem (i.e., dorsal motor nucleus of the vagus). Thus, the theory explicitly brought attention to the adaptive function of the ventral vagal pathway in defining features of social mammals including the mutual downregulating of threat reactions, calming physiological state, and sociality.

The published form of the talk documents conclusions by citing an extensive reference list of more than 100 scientific publications across several disciplines. Subsequent publications extended and refined the theory (Porges, 1998,2001,2003, 2007, 2021 and Porges & Lewis, 2010) and validated the methodology used monitor the ventral vagal cardioinhibitory pathway (Lewis et al. 2012). The theory has gained traction in a variety of disciplines and has been cited in thousands of peer reviewed articles (see Google Scholar). The foundational research developing the theory was supported by 38 years of continuous funding (1975-2013) from the National Institute of Health. Since its introduction, the theory has been cited in more 10,000 peer reviewed articles (see google scholar).

Polyvagal theory gets its name from the vagus, a cranial nerve that forms the primary component of the autonomic nervous system known as the parasympathetic nervous system. The traditional view of the vagus is that it is the nerve that supports health, growth, and restoration or simply stated homeostatic processes. In the traditional view the parasympathetic nervous system has the capacity to offset and neutralize the metabolic costly demands of the sympathetic nervous system that supports fight and flight behaviors. This view is only partially correct because the parasympathetic nervous system also has a role in defensive survival strategies.

The theory provides a statement and solution to the vagal paradox observed in neonatology. The paradox was formulated by contrasting well documented observations of vagal influences both being protective and lethal. Specifically, an assumed vagal phenomenon, clinical bradycardia (massive heart rate slowing), is potentially lethal in high-risk preterm infants and during complicated delivery, while respiratory sinus arrhythmia, also an assumed vagal phenomenon, was reliably reported to be greater in healthy newborns relative to high-risk preterm infants (see Porges, 1992). The theory solved the paradox by proposing that the bradycardia and respiratory sinus arrhythmia were functional outputs of two different brainstem areas that contained cardioinhibitory pathways that had different signatures in the

heart rate pattern. The foundational paper documented that modern mammals had a 'common cardiorespiratory oscillator' (see Richter & Spyer, 1990) that involved only the ventral vagal pathway, while the dorsal cardioinhibitory vagal pathway supported bradycardia. Similar to reptilian ancestors, massive bradycardia was a component of a threat reaction that is often observed as death feigning in small mammals. Subsequent research (Reed et al., 1999), documented that monitoring the status of the ventral vagal pathway via quantifying respiratory sinus arrhythmia identified vulnerability to clinical bradycardia in humans during the birthing process.

Functionally, Polyvagal Theory provides a foundation for an integrated and expansive brain-body science that emphasizes the bi-directional communication between visceral organs and brain structures through vagal pathways. The theory is built upon the paradigm breaking research of several visionary scientists, who introduced concepts that have been reframed within the Polyvagal Theory including internal milieu (Claude Bernard), homeostasis (Walter Cannon), evolution (Charles Darwin), dissolution (John Hughlings Jackson), arousal (Robert Yerkes and John Dodson), fight/flight (Walter Cannon), and a unified nervous system integrating brain and body functions (Walter Hess). By extracting and combining accepted principles discovered by these foundational scientists, Polyvagal Theory provides a new understanding of the neural regulation of the autonomic nervous system that emphasizes the bi-directional communication between brain and visceral organs.

While respecting the historical scientific literature, the theory updates the conceptualization of the autonomic nervous system through a transdisciplinary model extracting core principles that have independently emerged and have been documented in disparate disciplines. Polyvagal Theory, as a theory, leads to plausible hypotheses that have broad applications in the disparate domains of education, child development, mental health, physical health, work environments, and societal institutions as well as providing insights into the well-being of other social mammals.

Prerequisite Information for Understanding Polyvagal Theory

1. The relationship between autonomic state and defensive behaviors

In most vertebrates, the two primary defense systems are fight- or- flight and immobilization. Fight- or- flight behaviors enable the organism to flee or defend when threatened. These behaviors require the rapid accessibility of resources to mobilize through the activation of the metabolically costly sympathetic nervous system. Immobilization is a more ancient defense system, which is shared with virtually all vertebrates. In contrast to the metabolically costly mobilization strategy, immobilization is an adaptive attempt to reduce metabolic demands (e.g., reduced options for food and oxygen) and to appear inanimate (e.g., death feigning). Juxtaposed with the rapid activation of the sympathetic nervous system required to promote fight- or- flight behaviors, immobilization defense behaviors required a massive shutting down of autonomic function via a vagal pathway within the parasympathetic nervous system. Prior to the Polyvagal Theory, this latter

defense system was ignored or minimized, while threat has been assumed to result solely in mobilized fight-flight reaction requiring sympathetic excitation. A difficulty in conceptualizing a vagal defense reaction was due, in part, to the well documented withdrawal of cardioinhibitory vagal tone during threat that optimizes the expression of sympathetic activation. Thus, low vagal tone was assumed to be a marker of a threat reaction. Without a conceptualization of two vagal pathways a neurophysiological explanation of clinical bradycardia was incomplete. Polyvagal Theory reframed the investigation of the autonomic basis of threat reactions. Parsimoniously, ventral vagal tone withdrawal during threat removed the competition for efficient sympathetic activation to increase cardiac output to support mobilization and increased the vulnerability to a massive dorsal vagal induced syncope that would support immobilization. This sequence is observed during difficult human deliveries in which life-threatening bradycardia is preceded by heart rate variability decreases and tachycardia (see Reed et al.).

2. **The phylogenetic study of vertebrate autonomic nervous systems identifies three primary circuits.**

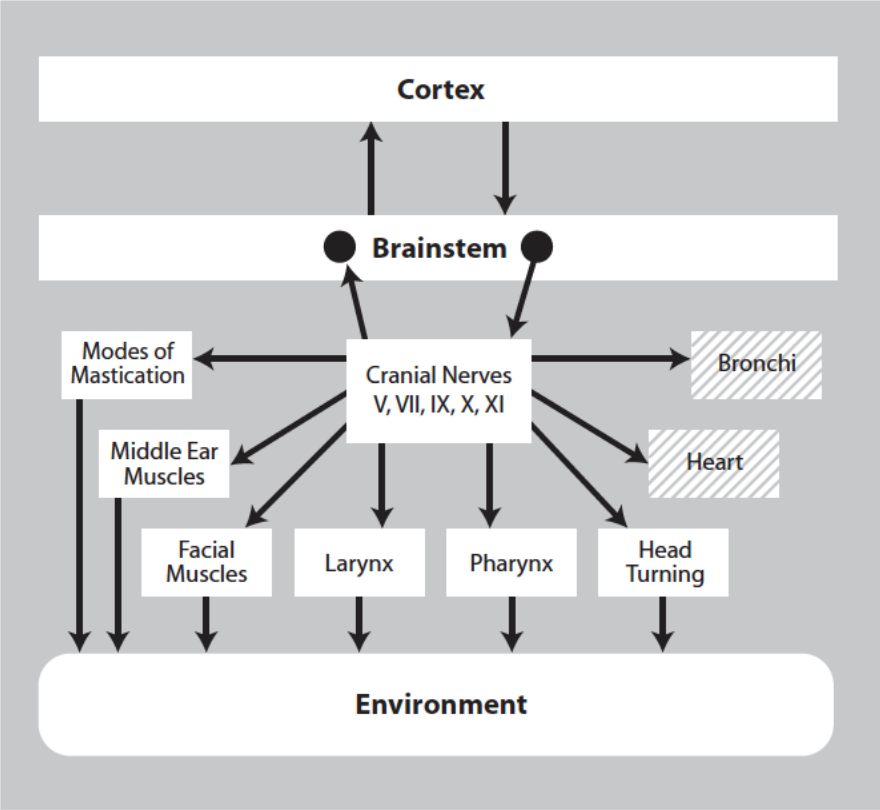
Through evolutionary processes the phylogenetic transition from asocial reptiles to social mammals a second cardioinhibitory vagal pathway evolved that had the capacity to down regulate both forms of defense. This second vagal pathway is observed in mammals and not reptiles. In addition, unlike reptiles and other vertebrates with vagal cardioinhibitory pathways, the anatomical structures regulating this mammalian component of the vagus interacts in the brain stem with structures regulating the striated muscles of the face and head to provide an integrated social engagement system (see below).

Through studies of the comparative neuroanatomy literature an estimate of phylogenetic transitions in vertebrate neural regulation of the autonomic nervous system can be estimated. The plausible conclusions support the identification of three circuits that follow a phylogenetic sequence in which the most ancient neural circuit emerges from the dorsal nucleus of the vagus, followed by the spinal sympathetic system, and the most evolved emerging from the ventral nucleus of the vagus. During the transition to the third stage, when ventral vagal pathways evolved, cells of origin of the vagus migrated from the dorsal nucleus of the vagus to the nucleus ambiguus. This transition is highlighted by the myelination of ventral cardioinhibitory vagal motor fibers and their integration within the brain stem to regulate a family of motor pathways (i.e., special visceral efferent pathways) that control the striated muscles of the face and head (see Figure 1).

Although the ancient circuits have been repurposed and potentially modified through evolutionary processes, features have been conveyed to social mammals including humans. Additional support for the phylogenetic sequence comes from the developmental literature extracting maturational trends from autopsy studies of embryos, fetuses, and young postpartum mammals including human data (see Porges & Furman, 2011).

3. **The Emergence of the Social Engagement System: Insights from evolution and development.**

The integration of the myelinated cardiac vagal pathways with the neural regulation of the face and head gave rise to the mammalian social engagement system. As illustrated in the Figure below the outputs of the social engagement system consist of motor pathways regulating striated muscles of the face and head (i.e., somatomotor) and smooth and cardiac muscles of the heart and bronchi (i.e., visceromotor). The somatomotor component involves special visceral efferent pathways that regulate the striated muscles of the face and head. The visceromotor component involves the myelinated supra-diaphragmic (above the diaphragm) vagal pathways that regulate the heart and bronchi. Functionally, the social engagement system emerges from a face-heart connection that coordinates the heart with the muscles of the face and head. The initial function of the system is to coordinate sucking, swallowing, breathing, and vocalizing. Atypical coordination of this system early in life is an indicator of subsequent difficulties in social behavior and emotional regulation. When fully developed, two important biobehavioral features of this system are expressed. First, bodily state is regulated in an efficient manner to promote growth and restoration (e.g., visceral homeostasis). Functionally, this is accomplished through an increase in the influence of myelinated vagal motor pathways on the cardiac pacemaker to slow heart rate, inhibit the fight- or- flight mechanisms of the sympathetic nervous system, dampen the stress response system of the hypothalamic– pituitary– adrenal (HPA) axis (responsible for cortisol release), and reduce inflammation by modulating immune reactions (e.g., cytokines; for review see Porges, 2007). Second, the phylogenetically mammalian face– heart connection functions to convey physiological state via facial expression and prosody (intonation of voice), as well as regulate the middle- ear muscles to optimize species- specific listening within the frequency band used for social communication (Kolacz, Lewis, & Porges,2018; Porges, 2007, 2009, 2011; Porges & Lewis, 2010).



This emergent social engagement system provides the mechanism for co- regulation of physiological state, as mammals convey cues of safety and danger— via vocalizations, head gestures, and facial expressions— to conspecifics. The social engagement system enabled mammals to co-opt some of the features of the vertebrate defense systems to promote social interactions such as play and intimacy. These changes in the autonomic nervous system provided mammals with neural mechanisms to promote the biobehavioral states necessary for caring for offspring, reproducing, and cooperative behavior. In contrast, the adverse behavioral and psychological effects of threat appear to target a disruption of the social engagement system, its management of defense reactions, and its contribution to co-regulation and cooperative behaviors, including intimacy and play.

The “newer” myelinated ventral vagal motor pathways regulate the supradiaphragmatic organs (e.g., heart and lungs) and are integrated in the brain stem with structures that regulate the striated muscles of the face and head via special visceral efferent pathways resulting in a functional social engagement system. This newer vagal circuit slows heart rate and supports the states of calmness required for social interactions. The ventral vagal circuit coupled with other autonomic circuits supports social play (i.e., ventral vagal coupled with sympathetic activation) and safe intimacy (i.e., ventral vagal coupled with dorsal vagal circuit). Thus, the mammalian vagus has properties that promote states that contain the range of responding of all components of the autonomic nervous system and functionally restrains the system from moving into states of defense.

The table below provides a plausible mapping of autonomic profiles associated with different categories of behavior. The table emphasizes the coordinating role of the ventral vagal circuit in expanding positive and social experiences by integrating all neural attributes involved in regulating autonomic function.

Emergent Behavior	Dorsal Vagal	Sympathetic	Ventral Vagal
Calm, socially co-regulated			X
Social play and dance		X	X
Aggressive, irritable, combative, fight-flight, chronic stress, pain		X	
Freeze	X	X	
Calm, shared intimacy	X		X
Shut down, death feigning	X		

Primary Principles of Polyvagal Theory

Principle 1: Autonomic state functions as an 'intervening variable.'

Polyvagal Theory proposes that physiological state is a fundamental part, and not a correlate, of emotion or mood. According to the theory, autonomic state functions as an intervening variable biasing our detection and evaluation of environmental cues. Depending on physiological state, the same cues will be reflexively evaluated as neutral, positive, or threatening (see neuroception below). Functionally, a change in state will shift access to different structures in the brain and support either social communication or the defensive behaviors of fight- or- flight or shutdown. Contemporary research on the impact of vagal nerve stimulation on cognitive function and emotion regulation supports this model (Groves & Brown, 2005). The theory emphasizes a bidirectional link between brain and viscera, which would explain how thoughts change physiology, and physiological state influences thoughts. As individuals change their facial expressions, the intonation of their voices, the pattern in which they are breathing, and their posture, they are also changing their physiology through circuits involving myelinated vagal pathways to the heart.

The theory emphasizes that physiological state influences behavioral and mental activity including cognitive function and learning accessibility as well as emotionality and sociality. It also emphasizes that physiological state can potentially influence health outcomes and medical treatments. The model emphasizes that autonomic state functions as an 'intervening' variable

either buffering or exacerbating the disruptive impact of changes on homeostatic processes. In general, autonomic state can be conceptualized as either supporting the homeostatic functions of health, growth, or restoration or alternatively, disrupting homeostatic processes to support a generalized threat reaction. Functionally, physiological state is conceptualized as a 'neural platform' upon which behavioral, mental, social, and physiological processes are dependent. This leads to plausible and testable hypotheses that shifts in the 'neural platform' will optimize or dampen the dependent process.

There is a vast literature documenting experiments in which individual, developmental, and state differences in autonomic function, often measured by heart rate variability as an index of vagal regulation, are related to cognitive (decision making), autonomic (heart rate reactivity), and physical responses (reaction time). In research evaluating the effect of adverse childhood experiences on adult behavior, outcomes are reliably mediated by a retuned autonomic nervous system that conceptually maybe characterized as being 'locked' in a state of chronic threat. These studies by documenting that many 'stimulus-response' relationships are mediated by autonomic state support the theory by providing evidence supporting role of autonomic state as an intervening variable.

This principle leads to a reconceptualization and redefining of 'stress.' Rather than the prevalent view of stress that often results in a circularity between the stressing event and stress reaction, Polyvagal Theory provides an objective functional definition. Stress through the lens of the Polyvagal Theory is simply the chronic disruption of homeostatic function. Transitory challenges to autonomic nervous system that result in rapid recovery are not stress nor are the consequences of sympathetic-adrenal activation that is required for increase metabolic activity associated with movement. In the short term the autonomic nervous system seamlessly adjusts to support metabolic demands and, in the optimal case rapidly recovers to a state that supports homeostatic functions. Instead, of a dependence on the ambiguous construct of stress, Polyvagal Theory suggests that the autonomic profile be interpreted from an adaptive perspective asking whether the autonomic nervous system is in a state that supports threat reactions or calm homeostatic functions. Threat reactions, especially an autonomic nervous system locked in a state of threat is easily identified as having low ventral vagal cardioinhibitory tone and a low threshold to be sympathetically reactive and potentially vulnerable to dorsal vagal shutdown. Such a retuned autonomic nervous system is well poised to be defensive including supporting hypersensitivities and hypervigilance, while simultaneously compromising the neural regulation of visceral organs.

Principle 2: Ventral vagal pathways function as a transitory vagal brake supporting attention, vigilance, mental effort, preparation for movement, and anticipation of threat.

The distinction between transitory and chronic disruption of homeostatic processes led to the conceptualization of the vagal brake (see Porges, 1996). Since the late 1960s (e.g., Porges, 1969, 1972 and Porges & Walter, 1976) changes in heart rate variability were used as indicators of sustained attention and mental effort. Moreover, hundreds of studies across dozens of laboratories documented that changes in a variety of heart rate variability indices including

respiratory sinus arrhythmia were depressed during forms of attention, anticipation, and during metabolic demands such as exercise (Hatfield et al…). Polyvagal Theory attributed the ventral vagal pathway as the mechanism underlying the vagal brake. Subsequent research has emphasized the role of the recovery of respiratory sinus arrhythmia (Calkins…., Dale…) as an important index of social behavior and state regulation leading to an inference that the ventral vagal cardioinhibitory pathway is involved in both self-regulation and co-regulation and its regulation underlies the concept of resilience.

Once the conceptualization of the vagal brake was introduced within the Polyvagal Theory (Porges, 1996), the commonly observed changes in global measures of heart rate variability and the more specific 'vagal' component of respiratory sinus arrhythmia during psychological and physical challenges could be understood from a neurobiological perspective. This perspective has embraced technologies to quantify specific neural signals such as respiratory sinus arrhythmia as an accurate index of ventral vagal tone (Lewis et al, 2012).

Principle 3: Autonomic reactivity is organized in an evolutionary response sequence characterized by hierarchical inhibition and dissolution.

The theory emphasizes that in response to challenges autonomic state shifts through a sequence defined by evolution in reverse or dissolution. Dissolution is a construct that was introduced by John Hughlings Jackson and emphasizes the hierarchical inhibition of evolutionarily older neural circuits by newer circuits. The human nervous system, similar to that of other mammals, evolved not solely to survive in safe environments, but also to promote survival in dangerous and life- threatening contexts. To accomplish this adaptive flexibility, the mammalian autonomic nervous system, in addition to the myelinated vagal pathway that is integrated into the social engagement system, retained two more primitive neural circuits to regulate defensive strategies (i.e., fight-or-flight and death-feigning behaviors). It is important to note that social behavior, social communication, and visceral homeostasis are incompatible with the neurophysiological states that support defense. Polyvagal response strategies to challenges are phylogenetically ordered, with newest components of the autonomic nervous system responding first. This model of autonomic reactivity is consistent with John Hughlings Jackson's construct of dissolution, in which he proposed that "the higher nervous arrangements inhibit (or control) the lower, and thus, when the higher are suddenly rendered functionless, the lower rise in activity" (1882, p. 412). In this hierarchy of adaptive responses, the newest social engagement circuit is used first; if that circuit fails to provide safety, the older circuits are recruited sequentially.

Principle 4: Neuroception is the process involved in detecting cues of threat or safety and adjusting autonomic state to support adaptive survival strategies.

Polyvagal Theory proposes that the neural evaluation of risk does not require conscious awareness and functions through neural circuits that are shared with our phylogenetic vertebrate ancestors. Thus, the term neuroception was introduced to emphasize a neural process, distinct from perception, capable of detecting environmental and visceral features that

are safe, dangerous, or life-threatening (Porges, 2003, 2004). Neuroception is a reflexive mechanism capable of instantaneously shifting physiological state.

Neuroception functionally involves both top-down and bottom-up mechanisms. In humans the process is assumed to be initiated via top-down pathways involving cortical areas located in or near temporal cortex that reflexively interpret cues of threat and safety. These areas of the cortex are sensitive to the intentionality of biological movements including voices, faces, gestures, and hand movements. Embedded in the construct of neuroception is the capacity of the nervous system to react to the intention of these movements. Neuroception functionally decodes and interprets the assumed goal of movements and sounds of inanimate and living objects. Thus, the neuroception of familiar individuals and individuals with appropriately prosodic voices and warm, expressive faces frequently translates into a positive social interaction, promoting a sense of safety. Autonomic state responds to the top-down detect of risk or safety. The autonomic reaction sends sensory information regarding bodily feelings to the brain where they are interpreted and consciously felt. The bottom-up limb of the neuroception is functionally equivalent to interoception. Thus, although we are often unaware of the stimuli that trigger different neuroception responses, we are generally aware of our body's reactions (i.e., visceral feelings) embodied in other autonomic signatures that support adaptive behaviors (i.e., social engagement, fight/flight, shutdown).

A form of neuroception can be found in virtually all living organisms, regardless of the development of the nervous system. In fact, it could be argued that single celled organisms and even plants have a primordial nervous system that respond to threat. As mammals, we are familiar with reactions to pain (i.e., nocioception), a type of neuroception. We react to pain prior to our ability to identify the source of the stimulus or even of an awareness of the injury. The detection of threat appears to be common across all vertebrate species. However, mammals have an expanded neuroception capacity in which they not only react instantaneously to threat, but also respond instantaneously to cues of safety. It is this latter feature that enables mammals to down regulate defensive strategies to promote sociality by enabling psychological and physical proximity without the consequences of injury. It is this calming mechanism that adaptively adjusts the central regulation of autonomic function to dampen sympathetic activation and to protect the oxygen-dependent central nervous system, especially the cortex, from the metabolically conservative reactions of the dorsal vagal complex (e.g., fainting, death feigning).

Harnessing the power of neuroception in downregulating an autonomic state that would support defensive strategies is a paramount feature of therapies that are Polyvagal Informed. For example, the Safe and Sound Protocol, the acoustic intervention developed by Porges, applies a computer algorithm to amplify prosodic features of human vocalizations that function like an acoustic vagal nerve stimulator (webpage). The Safe and Sound Protocol is frequently used with other therapeutic strategies to enhance accessibility to therapy by calming the autonomic nervous system.

Principle 5: Polyvagal Theory emphasizes the unique neuroanatomical and functional differences that evolved in mammals relative to other vertebrates that lead to our mammalian social heritage.

Polyvagal Theory focuses on differences, rather than similarities among vertebrate species and especially the neuroanatomical and functional changes observed during the transition from asocial reptiles to social mammals. For example, there are neuroanatomical and functional distinctions between the mammalian respiratory sinus arrhythmia and heart rate-respiratory interactions in other vertebrates. But only mammals have a well-defined common central respiratory oscillator that sends a respiratory rhythm from the brainstem to both the heart and the bronchi. This information flows through the vagal neurons originating in the nucleus ambiguus. In fact, the oscillator can be conceptualized as an emergent property of the interactions among structures regulated by the nucleus ambiguus including the larynx and pharynx (Richter & Spyer, 1990). This, of course, is not consistent with the features of a primitive nucleus ambiguus or the dorsal nucleus of the vagus that might be seen in vertebrates that preceded mammals. The observation of a common central oscillator is functionally unique to mammals and is the neurophysiological foundation enabling the. quantification of respiratory sinus arrhythmia to function as a portal to obtain a sensitive and accurate measure of ventral vagal cardiac tone.

The theory uses evolution to extract a phylogenetic sequence of autonomic regulation. This sequence identifies stages during vertebrate evolution when a spinal sympathetic nervous system and the two vagal pathways emerged and become functional via maturation in mammals. It would be difficult to argue that the sequence does not occur, although it would be possible to identify antecedent similarities in most vertebrates regardless of class or group. The question is not whether there are similarities in ancestral vertebrates, but rather how have these circuits been adapted to provide a unique mammalian autonomic nervous system that is intimately intertwined with co-regulatory social behavior.

Evolution transformed attributes of the autonomic nervous system into an integrated social engagement system that incorporated a brainstem communication area (i.e., the ventral vagal complex) that regulated, via special visceral efferent pathways, the striated muscles of the face and head and coordinated these processes with the vagal regulation of the heart and the bronchi. In mammals the ventral vagal complex enables the coordination of a suck-swallow-breathe-vocalize system with the vagal regulation of the heart. As the neuroanatomy of this ingestive circuit matures, the circuit becomes a functional social engagement system (see above) that enables physiological state to be communicated to conspecifics via facial expression and vocalizations. In mammals these structures and their neural regulation have been modified via evolution to support functions unique to the survival of mammals such as nursing and social communication. This transition in neuroanatomy and function provides the basis to understand that for humans, similar to other mammals, connectedness and trusting relationships are direct expressions of our biological imperative and are integrated into our biology.

Polyvagal Theory is dependent on the processes that evolutionary theorists describe as ex-adaptation and co-opting. These processes involve modifications that shift the function of a structure during evolution. For example, a structure can evolve because it served one function, but subsequently it may come to serve another. Ex-adaptation and co-opting are common strategies of repurposing vestigial structures in both anatomy and behavior. Polyvagal Theory is agnostic about the evolutionary pressures that result in the selection of specific changes. In contrast, the theory is more 'phylogenetically' descriptive and more focused on the functional outcome of repurposing. More specifically, the theory is interested in how the structures regulated by the ventral vagal complex were repurposed to provide the regulation of an integrated social engagement system that provide the primary portal to socially engage and communicate, ingest, and calm. Current neuroanatomical knowledge documents the refinement of the ventral vagal complex in mammals. Thus, although the ventral vagus (i.e., originating in the nucleus ambiguus) may have an origin in reptiles (Taylor, 2014), it appears that it is only in mammals that this pathway has been repurposed to convey and respond to social cues, via neuroception, as a potent mediator of autonomic state.

Principle 6: Criticisms misrepresent the Polyvagal Theory

Criticisms of Polyvagal Theory fail to acknowledge the theory's emphasis on the unique structural and functional changes in the regulation of the autonomic nervous system that occurred during the transition from asocial reptiles to social mammals. Basically, neuroanatomical criticisms of the Polyvagal Theory argue points that are not stated in the theory. From the initial statement of the theory, there has been a focus the unique attributes of the neural regulation of the mammalian autonomic nervous system. This focus is highlighted in the title of the initial publication - *Orienting in a defensive world: Mammalian modifications of our evolutionary heritage. A polyvagal theory*. Functionally, the theory provided an understanding of the unique mammalian capacity to use social behavior via the ventral vagus and the social engagement system to calm the autonomic circuits that support defense (e.g., sympathetic nervous system's role in defensive fight-flight behaviors and the dorsal vagal role in defensive immobilization). The table below provides a succinct summary of the contrasts between what the theory states and what the criticisms have assumed.

	What Polyvagal Theory states (Porges, 1995, 1998, 2007b)	Inaccurate assumptions reported (e.g., Grossman & Taylor, 2007; Monteiro et al., 2018; Campbell et al., 2005)
1	PVT focuses *only* on the unique features in the mammalian autonomic nervous system that distinguish mammals from **r**eptiles**.**	It is inaccurately assumed that PVT focuses on common features in the autonomic nervous system across vertebrate species.
2	PVT proposes that mammals have a unique myelinated vagal pathway originating *only* in the ventral vagal nucleus (i.e., nucleus ambiguus).	It is inaccurately assumed that PVT proposes myelinated vagal pathways are uniquely mammalian and that observations of myelinated vagal fibers from dorsal vagal nucleus in vertebrates other than mammals disprove the Polyvagal Theory.

3	PVT proposes that respiratory sinus arrhythmia, defined as a uniquely mammalian respiratory–heart rate interaction, indexes the output of ventral vagal cardioinhibitory fibers originating *solely* in the ventral vagal nucleus (i.e., nucleus ambiguus).	It is inaccurately assumed that PVT states respiratory sinus arrhythmia defines respiratory–heart rate interactions observed in all vertebrates that involve vagal influences including vagal cardioinhibitory fibers originating in the dorsal vagal nucleus.
4	PVT proposes that the vagal cardioinhibitory fibers originating in the dorsal vagal nucleus occur earlier in the phylogenetic history of vertebrates than vagal cardioinhibitory fibers originating from the nucleus ambiguus (see below).	It is inaccurately assumed that PVT states there is no evidence that the dorsal vagal nucleus is an evolutionarily older brainstem parasympathetic source than the nucleus ambiguus.

The above table summarizes criticisms of the Polyvagal Theory linked to inaccurate assumptions. Note that criticisms seem to reflect a misunderstanding of the scope of the theory. The criticisms are primarily based on the publications of one comparative accomplished scientist. This scientist and his colleagues have assumed that Polyvagal Theory can be falsified by observations of respiratory–heart rate interactions and myelinated vagal fibers in vertebrates that evolved before mammals. Note that Polyvagal Theory by focusing on the transition from asocial reptiles to social mammals is not related to their criticisms.

Consistent with this misunderstanding, there has been a criticism that there is no evidence that the dorsal vagal nucleus is an evolutionarily older brainstem parasympathetic source than the nucleus ambiguus. This faulty view is based on an assumption the occurrence of vagal preganglionic neurons ventrolateral to the dorsal vagal nucleus is tantamount to confirming the existence of the nucleus ambiguus. Neuroanatomical and neurophysiological research documents a phylogenetic trend toward a differentiation in the brainstem area, from which vagal preganglionic neurons originate, into a dorsal vagal nucleus and a discrete nucleus ambiguus. There is no question that both cardiac and noncardiac vagal neurons may be found outside the dorsal vagal nucleus in more primitive vertebrates. The phylogeny of the vagus illustrates, on a neuroanatomical level, differentiation of the visceral efferent column of the vagus into a dorsal motor nucleus and a ventrolateral motor nucleus (i.e., nucleus ambiguus), which is first seen in some reptiles. Thus, the neuroanatomical identification of a discrete nucleus ambiguus might be assumed to be limited to all mammals and select reptiles. This does not preclude the evolutionary trend of vagal preganglionic neurons being found ventrolateral to the dorsal vagal nucleus. However, the function of the nucleus ambiguus as the source of vagal cardioinhibitory pathways is uniquely mammalian.

Although new findings in the literature on the phylogeny of the autonomic nervous system are interesting and relevant to comparative neuroanatomists, these findings are relevant to Polyvagal Theory. For example, the identification of myelinated vagal fibers originating from the dorsal vagal nucleus in other vertebrates suggests a series of questions related to adaptive

function and whether there are myelinated vagal fibers originating from the dorsal vagal nucleus in mammals that have not yet been identified.

Using evolution and developmental maturation to map the hierarchy of autonomic circuits, Polyvagal Theory proposes a vivid map of the response sequence through which mammals progress when under threat, including disease and injury. This sequence following evolution in reverse, or dissolution as succinctly described by John Hughlyns Jackson (1884), mirrors the accounts frequently experienced survivors of trauma. Basically, the criticisms are not criticisms of the Polyvagal Theory. The criticisms are not about testing the documented constructs embedded in theory: dissolution, vagal brake, neuroception, or social engagement system. Nor are the criticisms linked to expanding an understanding of the mechanisms involved in recruiting or monitoring the dorsal vagal circuit in mammals. Rather, the criticisms are at best tangential to the theory and at worst inaccurate representations that lead to misunderstandings of the theory.

Another set of criticisms are rooted in the work of these same scientists and focus on the role that respiratory sinus arrhythmia plays within Polyvagal Theory. The table below summarizes the main points. Specifically, they argue that there is a disconnect between respiratory sinus arrhythmia and cardiac vagal tone. Research has documented (see Lewis et al., 2012) that the Grossman methodology is faulty in estimating ventral vagal tone and is directly the cause of their faulty inference regarding the inaccuracy of quantifying respiratory sinus arrhythmia as a valid index of ventral vagal tone. Research has documented (see Lewis et al., 2012) the precision and accuracy of the Porges-Bohrer methodology and contrasts it with the inadequate methods used by Grossman. The flaws in the Grossman method have been known and documented for about 25 years (Byrne & Porges, 1996; Lewis et al., 2012).

	What Polyvagal Theory states	Inaccurate assumptions reported (e.g., Grossman & Taylor, 2007)
1.	Respiratory sinus arrhythmia accurately reflects cardiac vagal tone via myelinated cardioinhibitory vagal fibers originating in nucleus ambiguus.	It is inaccurately assumed that PVT claims a disconnect between respiratory sinus arrhythmia and cardiac vagal tone.
2.	Polyvagal Theory is not based on respiratory sinus arrhythmia being an accurate index of cardiac vagal tone.	It is inaccurately assumed that Polyvagal Theory is based on respiratory sinus arrhythmia being uniquely mammalian.
3.	Respiratory sinus arrhythmia is uniquely mammalian and solely reflects the cardioinhibitory influence of vagal fibers originating in the nucleus	It is inaccurately assumed that Polyvagal Theory can be disproved by observing respiratory sinus

	ambiguous	arrhythmia in other vertebrates determined by cardioinhibitory fibers originating in either ventral or dorsal vagal nuclei.
4.	The disconnect between respiratory sinus arrhythmia and cardiac vagal tone is dependent on the metric used to quantify respiratory sinus arrhythmia.	It is inaccurately assumed that Polyvagal Theory can be disproved by documenting a disconnect between respiratory sinus arrhythmia and cardiac vagal tone independent of specific methodology used to quantify respiratory sinus arrhythmia.
5.	Polyvagal-related hypotheses can be tested with respiratory sinus arrhythmia only if the metric provides an accurate index of cardiac vagal tone originating in the nucleus ambiguus.	It is inaccurately assumed that Polyvagal-related hypotheses can be tested with any measure of respiratory sinus arrhythmia.