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Review

Fundamental challenges and likely refutations of the five basic premises of the polyvagal theory

Paul Grossman *

Department of Psychosomatic Medicine, University Hospital Basel, Switzerland

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ABSTRACT

The polyvagal collection of hypotheses is based upon five essential premises, as stated by its author (Porges, 2011). Polyvagal conjectures rest on a primary assumption that brainstem ventral and dorsal vagal regions in mammals each have their own unique mediating effects upon control of heart rate. The polyvagal hypotheses link these putative dorsal- *vs.* ventral-vagal differences to socioemotional behavior (e.g. defensive immobilization, and social affiliative behaviors, respectively), as well as to trends in the evolution of the vagus nerve (e.g. Porges, 2011 & 2021a). Additionally, it is essential to note that only one measurable phenomenon—as index of vagal processes—serves as the linchpin for virtually every premise. That phenomenon is respiratory sinus arrhythmia (RSA), heart-rate changes coordinated to phase of respiration (i.e. inspiration *vs.* expiration), often employed as an index of vagally, or parasympathetically, mediated control of heart rate. The polyvagal hypotheses assume that RSA is a mammalian phenomenon, since Porges (2011) states "RSA has not been observed in reptiles." I will here briefly document how each of these basic premises have been shown to be either untenable or highly implausible based on the available scientific literature. I will also argue that the polyvagal reliance upon RSA as equivalent to general vagal tone or even cardiac vagal tone is conceptually a category mistake (Ryle, 1949), confusing an approximate index (i.e. RSA) of a phenomenon (some general vagal process) with the phenomenon, itself.

1. Introduction

The polyvagal hypotheses (e.g. Porges, 2011, 2021a) propose that the mammalian autonomic nervous system is comprised not only of two branches (sympathetic and parasympathetic): the parasympathetic branch can be further meaningfully separated into two additional systems, 1) a brainstem, dorsally situated vagal area and 2) a brainstem, ventrally located vagal area, in which each system exerts its own distinct influence upon vagally mediated heart rate control, and each is tied, respectively, either to defensive behaviors of immobilization or to prosocial responses.

According to the polyvagal hypotheses, the brainstem dorsal vagal motor nucleus (DVMN) putatively mediates massive slowing of heart rate (i.e. bradycardia) during phases of extreme fear- or threat-induced immobilization (emotional freezing or human psychological dissociation), for which sympathetic fight-or-flight reactions may not be adaptive. These supposedly dorsal vagal responses are characterized in the polyvagal literature as evolutionarily "primitive," observed in "vertebrates that evolved long before mammals" (Porges, 2021a). The ventral vagal nucleus Ambiguus (nA), on the other hand, is seen as an advanced mammalian adaptation to promote modulation of heart rate and self-calming instrumental for affiliative social behavior. Activity of the ventral vagal nA is proposed to be enhanced during conditions of safety and positive social contact. Whereas the existence of the brainstem dorsal and ventral areas has long been known in vertebrate autonomic physiology, vagal neuroanatomy and evolutionary oriented comparative biology, the assumptions about the functions of each vagal area made in the polyvagal writings often appear to be at variance with past and current knowledge about the nA and the DVMN (e.g. Grossman, Taylor, 2007; Taylor et al., 2014; Duran et al., 2020; Sanches et al., 2019).

Additionally, a major limitation of the polyvagal conjectures, and in fact, all psychophysiological theorizing about the vagus and the psyche is the fact that there is, effectively, only one noninvasive and nonintrusive index of vagal activity, respiratory sinus arrhythmia (RSA),[1] and that measure is only an imperfect and indirect marker of vagally

* Correspondence to: EZFA, Konradstrasse 32, 79100 Freiburg, Germany.
E-mail address: pgrossman0@icloud.com.

[1] RSA is generally equivalent to high-frequency heart-rate variability (HF-HRV) that is reliably estimated within the respiratory-frequency range of an individual.

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mediated control of heart rate (Grossman & Taylor, 2007)— but of no other vagally mediated processes (see Jänig & Häbler, 2000), i.e. RSA provides no or very limited information about any other vagal processes beyond control of heart rate. Nevertheless, RSA is the linchpin for almost all psychophysiological speculations regarding the parasympathetic system, and this is very true for polyvagal hypotheses. Therefore, we will rely upon the existing physiological evidence regarding vagal influences on heart rate and dismiss conjectures about other systems that cannot yet be nonintrusively assessed.

The polyvagal hypotheses have become increasingly diverse over the years and often have borrowed substantially from prior existing psychological theories and evidence, e.g. attachment theory, social engagement literature and psychological trauma research (e.g. Bowlby, 1969; Campos et al., 1983; Izard, 1978; van der Hart et al., 1989). In such cases, these hypotheses consequently acquire their distinctiveness only in terms of their relationships to the underlying polyvagal premises.[2] Therefore, we proceed, point-by-point, with a review of the existing evidence regarding each of the five basic premises of the polyvagal framework.

2. Brief evaluation of the 5 basic premises of the polyvagal model (Porges, 2011)

The individual premises of the polyvagal construction (Porges, 2011) are numbered and evaluated sequentially below (see also Table 1 for more recent reformulations):

Premise 1. *: "Neurogenic bradycardia and RSA are mediated by different branches of the vagus and need not respond in concert."*

Existing evidence consistently indicates that the ventral nucleus Ambiguus (nA) mediates completely, or almost completely, the entire vagal control of heart rate in mammals. This evidence goes back at least 45 years when McAllen and Spyer (1976) demonstrated that neurons within the nA were responsible for slowing heart rate in cats; all active neurons resided in the nA, none in the dorsal vagal motor nucleus (DVMN). These findings were essentially replicated a few years later by Geis and Wurster (1980). Subsequently Cheng et al. (2002, Cheng et al., 2004) also provided confirmatory evidence, derived from chemical ablation of different brainstem vagal areas, that the nA is very principally responsible for vagal heart rate responses in rodents under a range of conditions, together with indications that the DVMN appears to have almost no effect upon vagal heart rate responses.

Consistent with these findings, selective *in vivo* pharmacogenetic inhibition of DVMN neurons also did not affect heart rate (Machhada et al., 2015, 2016), once again indicating that the DVMN does not provide significant vagal tone to the cardiac nodal tissue. Recently, Machhada et al. (2020), employing state-of-the-art optogenetic stimulation techniques, also demonstrated that strong stimulation of DVMN neurons had less than a 7% effect upon heart rate deceleration (i.e. a negligible effect). A senior coauthor of this publication (A. Gourine, personal communication, 2022) stated that "a similar level of stimulation applied to the nA is likely to stop the heart." This method applies optogenetic stimulation of biological tissues (i.e. a technique, in this case, that employs optical stimulation of specific neuron populations with the aid of genetically inserted ion channels that respond to light stimulation; see Machhada et al., 2020).

A major polyvagal hypothesis hinges upon the notion that the DVMN is capable of mediating "massive and lethal" bradycardia (Porges, 2011); there are more than 30 mentions of "massive" DVMN-mediated bradycardia referred to in that book alone; this contention is maintained in recent work, as well (Porges, 2021a). In fact, a few studies have suggested cardiac changes evoked by selective stimulation of unmyelinated fibers, presumably from the DVMN (e.g. Jones et al., 1995), but even then, the authors acknowledged that stimulation evokes only small heart rate decelerations, and principally among only one of three mammalian species that were investigated. No study has ever reported profound DVMN-mediated heart rate deceleration. To the contrary, there is very broad consensus that effects to DVMN stimulation, even in the species apparently most responsive, were very modest.

As an aside, the evidence of species differences in just those small effects suggests that generalizations across mammalian species even regarding the minor levels of vagal heart rate effects possibly due to DVMN stimulation cannot justifiably be made at this time. So, attributing even mild levels of DVMN-mediated heart rate deceleration across the entire group of mammals seems clearly without basis. Additionally, it is possible that these modest heart rate effects associated with DVMN activation may, in fact, be explained by reflex (neuronal or humoral) changes triggered as a result of vagal effects on other organs and not as a function of DVMN mediation (A. Gourine, personal communication, 2022).

The above-cited and other rather substantial research documenting the virtual absence or insignificance of DVMN influence upon heart rate control and the fact that control has been documented to reside in the nA has led to the consensus view (e.g. Farmer et al., 2016; Gourine et al., 2016) stated most recently by Veerakumar et al. (2022):

"Cardiac parasympathetic outflow originates from brainstem preganglionic neurons, residing primarily in the nucleus ambiguus (Amb) in the medulla. A minority of cardiac parasympathetic neurons are located in the dorsal motor nucleus of vagus… which does not control heart rate."

Additionally, findings related to vasovagal syncope and RSA also do not support a DVMN-mediation of bradycardia (Simon et al. 2017): in cases of presyncope or syncope, massive HR reduction has been found to be accompanied by an equally massive increase in respiration-corrected RSA, certainly a ventrally situated, nA-mediated phenomenon.

Parenthetically but not unimportantly, vagal responses to emotional freezing in mammals appear to be primarily mediated by the ventrally located nA, not the DVMN, and this includes freezing-associated bradycardia (see Neuhuber & Berthoud, 2022); this is contrary to polyvagal claims that "the immobilization defense system recruits the unmyelinated vagal motor pathways to the heart to produce an immediate and massive slowing of heart rate" (Porges, 2021a, p. 197). Therefore, vagal control of heart rate is exclusively, or almost exclusively, mediated by the nA, and there is no credible evidence that the DVMN plays any role in massive bradycardia.

Premise 2. *: "Neurogenic bradycardia associated with orienting is a phylogenetic vestigial relic of the reptilian brain and is mediated by the dorsal vagal motor nucleus."*

All vagally mediated bradycardia is, by definition, neurogenic (i.e. "a factor related to the activity of nerve" (Oxford Dictionary of Sports Sciences and Medicine, 2007). Given that Premise 2 is fully dependent upon the correctness of Premise 1 (i.e. that bradycardia and RSA are mediated by different branches of the vagus), the latter of which is roundly contradicted by current evidence, it is unnecessary at this point to go into the particular polyvagal misinterpretations about the evolution of the vagus nerve. This issue will nevertheless be visited in the next section within another context. In sum then, existing evidence appears to make the first two premises of the polyvagal framework highly untenable.

[2] For example, Bowlby (1969), in his monumental work on attachment and loss, wrote in the first volume: "what I had in mind when defining attachment behavior was the output of what might be called a safety-regulating system, namely a system the activities of which tend to reduce the risk of the individual coming to harm and are experienced as causing anxiety to be allayed and a sense of security to be increased." In the recent polyvagal thesis on "safety" (Porges, 2021a, 2021b), Bowlby's work is briefly acknowledged once, with little detail, but attempts are made to link it to basic polyvagal premises. This is characteristic of other psychological dimensions highlighted in publications on polyvagal conjectures.

Table 1
Recent statements about the polyvagal hypotheses (Porges, 2021a), and responses based upon past findings (Grossman & Taylor, 2007) and evidence from the text.

Polyvagal Theory states (all quotes from Porges (2021a)*, his Tables 2.2. & 2.3)	**Responses to Porges (2021a, his Tables 2.2 & 2.3), in which claims are made about the positions of Grossman and Taylor (2007)**
Table 2.2	
1. Evolutionary focus only on the transition from reptiles to mammals when the autonomic nervous system (ANS) is repurposed to support sociality, and through afferent feedback sociality can support autonomic regulation, leading to optimized health, growth, and restoration.	1. a) Focus of Grossman & Taylor (2007) and Taylor et al. (2022) is upon understanding evolution of autonomic control and plausible explanations for transition across different groups of vertebrates (e.g. from reptiles to mammals; see above references). b) The literature clarifies that there is no scientific basis for asserting that the ANS in mammals was "repurposed to support sociality" (see Doody et al., 202; Taylor et al., 2022). Other vertebrates also have complex forms of social behavior and social learning.
2. Mammals have a unique myelinated vagal pathway originating only in the ventral vagal nucleus (i.e., nucleus ambiguus) with capacity to downregulate autonomic defensive states to support both sociality and health, growth, and restoration (i.e., homeostasis).	2. Evidence is presented here and elsewhere that brainstem ventral regions mediating vagal activity are ubiquitous in virtually all groups of vertebrates, as are myelinated pathways (Taylor et al. 2022, Table 2). Additionally, mammals can also have myelinated pathways in the dorsal motor nucleus of vagus, thus not "originating only in the ventral vagal nucleus" (Booth et al., 2021).
3. Respiratory sinus arrhythmia is a term used to define a uniquely mammalian respiratory–heart rate interaction involving the rhythmic modulation of heart rate via vagal pathways originating solely in the ventral vagal nucleus (i.e. nucleus ambiguus).	3. Other vertebrates, besides mammals, also "demonstrate respiratory-heart rate interaction involving the rhythmic modulation of heart rate via vagal pathways originating solely in the ventral vagal nucleus" (see Taylor et al., 2022). There is no evidence that respiratory sinus arrhythmia (RSA) is a strictly mammalian emergent property.
Table 2.3	
1. Respiratory sinus arrhythmia accurately reflects cardiac vagal tone via myelinated cardioinhibitory vagal fibers originating in nucleus Ambiguus.	1. a) RSA is an respiratory-cardiovascular phenomenon contributing to ventilatory-circulatory coordination, and it is mediated by brainstem ventral vagal and respiratory centers (e.g. Elstad et al., 2018). b) RSA specifically represents the vagal modulation of heart rate that contributes to that coordination. c) RSA is under certain conditions (e.g. Katona & Jih, 1975) an approximate quantitative index of cardiac vagal tone. d) RSA magnitude cannot be considered a direct measure of cardiac vagal tone because it is influenced, for example, by variations in respiratory activity, cardiac sympathetic tone, chemoreceptor activity and velocity of heart rate deceleration. e) Physiologists recognize the clear distinction between RSA and brainstem ventrally mediated cardiac vagal tone: "Although RSA has been found to correlate with vagal tone, it is worth bearing in mind that the two measures are not identical and, as reported here, are of distinct origin (Farmer et al., 2016, J. Physiology)." The authors conclude that cardiac vagal tone depends upon at least 3 sites of the brainstem (none the dorsal vagal nucleus) and that a significant proportion arises independently of RSA.
2. a) Polyvagal Theory is not based on respiratory-heart rate interactions being uniquely mammalian. b) Polyvagal Theory is not based on respiratory sinus arrhythmia being an accurate index of cardiac vagal tone.	2. a) The polyvagal conjectural framework is based on the inaccurate assumption that RSA is only characteristic of mammals (see points above, including for Table 2.2.) b) RSA was mentioned 170 times in Porges (2021), and several hundred times more in Porges (2011). It remains almost the only noninvasive and nonintrusive marker of vagal processes used in research. Therefore, RSA is a linchpin for the polyvagal conjectures. Also because cardiac vagal tone is principally or exclusively mediated by the nA, RSA, or an equivalent parameter (e.g. HF-HRV), is the only established nonintrusive index of cardiac vagal tone.
3. Respiratory sinus arrhythmia is an accurate index of cardiac vagal tone and reports of a disconnect between respiratory sinus arrhythmia and cardiac vagal tone is dependent on the methodology used.	3. There is a great amount of evidence that respiratory sinus arrhythmia is an approximate index of cardiac vagal tone, subject to multiple caveats and limitations (e.g. this article; Grossman et al., 1991; Goldberger et al., 1994; Taylor et al., 2001; Grossman & Taylor, 2007; Farmer et al., 2016). There is no serious evidence that this is dependent upon methodology of estimation.
4. Respiratory sinus arrhythmia accurately indexes cardiac vagal tone only if the signal processing techniques and transformations similar to those incorporated in the [Porges] PBRSA metric are implemented.	4. a) Major quantification procedures for estimating RSA yield highly similar results and are extremely highly correlated with each other; this includes the Porges-Bohrer RSA (PBRSA), spectral analysis and PT_{RSA} (Grossman et al., 1990; Grossman et al., 2004). b) Of course, when sample distributions are not normal, natural logarithm transformation needs to be performed. c) Without this transformation, measures must not be compared (a serious problem in the Lewis et al. (2012) study; see their figures).
5. The PT_{RSA} metric (Grossman & Taylor, 2007) distorts the relationship between respiratory sinus arrhythmia and cardiac vagal tone and cannot be statistically improved with linear adjustment based on respiratory parameters.	5. a) As Katona & Jih (1975) first mentioned, respiratory parameters must be controlled in order to estimate cardiac vagal tone, whatever RSA measure is used. b) The PBRSA measure correlates very highly with variations in respiratory rate ($r = 0.84$), almost as highly as the PT_{RSA} estimate ($r = 0.91$;Grossman et al., 1990). Within-individual PBRSA magnitude is also clearly dependent on respiratory parameters.
6. 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.	6. a) There is no evidence of superiority of the PBRSA measure, but there is evidence of its vulnerabilities (Litvack et al., 1995). b) An abundance of literature clearly indicates that RSA is merely an approximate marker of cardiac vagal tone, and then only under certain conditions.

All premises and most corollaries of the polyvagal project derive from the apparently mistaken assumption of Premise 1, as well as even the very name of this project ("poly"vagal, as related to the two brainstem dorsal and ventral nuclei; cf. Porges, 2021a). Therefore, each of the other basic premises is seriously weakened by the existing evidence regarding the actual functions of the respective dorsal and ventral brainstem areas. It is, nonetheless, valuable seriously to examine the last 3 premises of the polyvagal hypotheses.

Premise 3. *: "Withdrawal of cardiac vagal tone through NA mechanisms is a mammalian adaptation to select novelty in the environment while coping with the need to maintain metabolic output and continuous social*

communication."

This premise needs to be addressed in two parts. a) The wording of this premise seems to indicate a presumption that withdrawal of cardiac vagal tone via the brainstem ventrally situated nA is a specifically mammalian adaptation; and b) that there is a specific mammalian evolutionary development that is characterized by orienting toward novelty, while 'maintaining metabolic output and continuous social communication." Let us take these points in turn:

The suggestion that modulation of cardiac vagal activity via ventral vagal mechanisms is a mammalian adaptation was first challenged in 2007 in a joint paper with the leading evolutionary biologist of the vagus E.W. Taylor (Grossman & Taylor, 2007), and most recently elaborated in greater depth in an extensive review (Taylor et al., 2022). Evidence in both papers is presented from many studies that the brainstem ventral area is found in fish, reptiles, amphibians and birds, and has often been implicated in vagal heart rate control (see Taylor et al. 2022, Figure 4 for a summary). For example, Kelley (2022) recently documented mediation of the nA of various species of frogs in relation to socially communicative vocalizations, a function suggested to be exclusively mammalian according to polyvagal speculations.

As an integral aspect of his polyvagal conjectures, Porges has repeatedly stated (2009; 2011) "only mammals have a myelinated vagus," very recently adding: "Mammals have a unique myelinated vagal pathway originating only in the ventral vagal nucleus." (Porges, 2021a). The existing comparative biology evidence clearly indicates this statement is not true: Taylor et al. (2022), in their recent review of the vertebrate literature, report:

> "Myelinated, fast conducting, efferent fibres, identified as B-fibres, have been described and characterized in the cardiac vagus of a shark, a bony fish, a lungfish, birds and mammals. These are likely to be present in all vertebrate groups, as they are a necessary component of instantaneous control of heart rate."

Taylor et al. (2022, Table 4) also provide an overview indicating a myelinated cardiac vagus (with cardiac control originating in the nA) across the range of vertebrate groups from evolutionarily very ancient (500 million years) to more recent (e.g. mammals).

The existing experimental evidence indicates the unmyelinated fibers originating in the DVMN only have, at very most, a minor role in inhibiting heart rate in response to certain powerful pulmonary afferent stimuli; nA-originating myelinated fibers are clearly prepotent (Jones, 2001, Wang et al.,2000). There is also additional recent evidence, based on optogenetic stimulation, that indicates, at least, one larger mammal, the sheep, to have myelinated nerve fibers descending from the DVMN (Booth et al., 2021), unsurprisingly thought to facilitate more rapid conduction of vagal traffic among larger-bodied mammals than among smaller bodied ones (e.g. mice), for whom DVMN efferent fibers are unmyelinated, and conduction distance may not play such a prominent role for rapid motor control. Apropos heart rate responses, although cardiac responses to DVMN stimulation were not reported in the latter paper, a senior author of the study states: "by the way, we did not observe any significant heart-rate changes with DVMN stimulation in sheep" (A. Gourine, personal communication, 2023).

These findings, as a whole, firmly and consistently contradict the polyvagal hypotheses that propose the DVMN as the "source nucleus" of unmyelinated pathways and the nA as the "source nucleus" of myelinated pathways in mammals (Porges, 2011, e.g. p. 74 & 209; Porges, 2021a, p. 31). The new technological developments in precise optogenetic stimulation of vagal neurons are likely to produce other findings that clarify assumptions about DVMN and nA functioning well beyond polyvagal concerns (for a good example, see Veerakumar et al., 2022 and later discussion under the section Premise 4).

Additionally, the polyvagal claim that centrally controlled cardiorespiratory interactions, generated in the brainstem, are restricted to mammals (Porges et al., 2003; Porges, 2009, 2011) is contrary to comparative biological evidence among rattlesnakes and lizards (Duran et al., 2020; Sanches et al., 2019). The polyvagal notion that the ventral vagal area is unique to mammals is opposed by years of evidence (reviewed by Taylor et al., 1999; Taylor et al., 2010a; Taylor et al., 2010b; Taylor et al., 2014). Thus, the notion that RSA is only found among mammals and not among other vertebrate species is contrary to comparative biological evidence, even among evolutionarily ancient lungfish (Monteiro et al., 2018). Additionally, RSA-like heart-rate variability, is very possibly mediated by ventrally located cardiac vagal neurons in lung-breathing amphibians, reptiles or birds, as has been clearly demonstrated in mammals (Taylor et al., 2022). Consequently, there seems to be no evidence to assume that ventrally located cardiac vagal control via myelinated fibers is exclusively a mammalian phenomenon.

Therefore, the assumption of the ventrally situated vagal region in the brainstem as a mammalian evolutionary advancement does not correspond to existing comparative biology evidence (for an overview, see Taylor et al., 2022 Figure 4).

The second embedded hypothesis of some 'unique evolutionary mammalian adaptation to select novelty in the environment while coping with the need to maintain metabolic output and continuous social communication' also does not correspond to the existing empirical data base, based upon several factors:

a. Orienting responses are, of course, found across vertebrate evolution, including fishes, reptiles, amphibians and birds (e.g. Krauzlis et al., 2018; Leadner et al., 2021), and may even precede vertebrate evolution (Earl, 2022).
b. The requirements of maintaining metabolic output during social communication, social behavior and social learning occur, not only in humans, but in all other classes of vertebrates. There is much evidence to indicate that the vagus is importantly implicated in meeting rapid metabolic demands across the vertebrate range (see Taylor, 2022). The polyvagal notion of reptiles being vagally "underpowered" with "low ambient vagal tone", in contrast to "supercharged" mammals (Porges, 1995, 2021b) has long been indicated to be unsupported (Burnstock, 1969; Hedberg, Nilsson, 1975; see Taylor et al. 2022 for more recent evidence). In fact, reptilian and other nonmammalian vertebrates must manifest dynamic, fast-paced cardiac vagal adjustments to external and internal stimuli, as well as to social interactions and demands.
c. Additionally, the polyvagal claims of unique forms of sociality and social learning among humans (Porges, 2021a and 2021b) do not correspond to the existing body of evidence that find both complex forms and levels of social behavior among reptiles, amphibians, birds and even fishes (e.g. Brakes et al., 2021; Doody et al., 2013; Doody et al., 2021, 2023; Rivas, Levin, 2004; Szabo et al., 2021; Taylor et al., 2022; Whiting, While, 2017). These, depending on the species, include parental care after birth or hatching, dominance hierarchies, territoriality, male-to-male combat, complex courtship, group vigilance, signaling, posturing, eavesdropping, communal nesting, cooperative hunting, pair bonding, sexual selection, and social monogamy (Doody et al., 2021, 2023). To the extent that vagal heart rate control mediates aspects of these social behaviors, it seems most parsimonious to assume that similar mechanisms are at play among mammals and other vertebrate species. I am unaware of any studies of contrary findings from experts in the current evolutionary biology literature.

In sum, there appears to be no evidence for, and substantial evidence against, the conjecture that the vagus nerve of mammals possesses unique properties either for control of heart rate or for socioemotional behavior.

Premise 4. *: "The ability of NA to regulate special and general visceral efferents may be monitored by the amplitude of RSA."*

In various places in the polyvagal literature, RSA is often presented as a direct measure of some construct of general vagal tone (e.g. Porges, 1995; 2011), as if vagal influences on the entire body operate in unison. For example, Porges (2011) writes: "The amplitude of respiratory sinus arrhythmia (RSA) provides a validated and easily obtainable index of parasympathetic nervous system tone via the cardiac vagus." In the same publication, he refers to his proprietary assessment instrument of RSA as the "vagal tone monitor." However, there is general consensus of target-organ specificity of efferent and afferent vagal control (e.g. Jänig & Häbler, 2000; Ritz, 2009; Veerakumar et al., 2022). In other words, vagal activity related to different organs or to bodily systems do not necessarily covary with one another (e.g. efferent vagal activity may be stimulated to the gut or lungs but not to the heart, and these are differentially mediated by different vagal efferent fibers).

On the other hand, the amplitude of RSA has been shown, time and again in many different laboratories throughout the world, solely to be a measure of **the respiratory modulation of the vagal control of heart rate** and nothing else (see Grossman & Taylor, 2007; Ritz, 2009). Cardiac vagal tone is characterized by extent of vagal contribution to tonic levels of heart rate during specific conditions or periods of time (i.e. to the state of continuous levels of vagally mediated heart rate during those conditions or time segments).

Furthermore, RSA is merely an approximate index of that specifically **cardiac** vagal tone under certain circumscribed conditions. Farmer et al. (2016) found in rodents that brainstem inhibition of RSA had little effect upon cardiac vagal tone mediated by the ventral vagus. Specifically, cardiac vagal tone (completely unrelated to dorsal vagal mechanisms) remained largely unchanged when connection with a brainstem center responsible for respiratory rhythmicity was experimentally inhibited (see Table 1, point 2.3–1). Additionally, it has been recently demonstrated that even different regions of the nA, itself, can exert their own distinct influences upon cardiac vagal tone and upon vagal respiratory function (Veerakumar et al., 2022), which may, in turn, give rise to variations in RSA. This evidence, then, clearly indicates that RSA may sometimes be a marker of cardiac vagal tone, but is certainly neither equivalent to, nor always, a reliable index of cardiac vagal tone.

Therefore, contrary to polyvagal assumptions, cardiac vagal tone is not "easily obtainable" (Porges, 2011) by assessing RSA, with whatever quantification procedure is chosen. Much human research has documented dissociation between vagally mediated heart rate changes and RSA magnitude under a number of circumstances, as well as confounding influences due to various factors (e.g. concurrent beta-sympathetic, chemoreceptor or respiratory influences: e.g. Goldberger et al., 1994; Grossman et al., 1991; Grossman & Taylor, 2007; Hedman et al., 1995; Sasano et al., 2002; JA Taylor et al., 2001). As indicated above, this cannot be explained by dorsal vagal influences, nor by variations in methods of RSA quantification.

Many of these caveats and limitations have long been recognized. Indeed, the very first quantitative validation study of RSA as an index of cardiac vagal tone (Katona & Jih, 1975) specified the need to control for respiratory parameters when employing RSA as an index of cardiac vagal tone (in their formula for estimation of the latter). These limitations and caveats, very often ignored or overlooked, complicate the accurate estimation of cardiac vagal tone based on RSA. They demonstrate that RSA is not a direct measure of cardiac vagal tone. Such constraints always need to be carefully considered when employing RSA as an index of cardiac vagal tone, in order to determine whether or not they are confounding estimation.

Recently a claim has been made that one particular quantification of RSA, employed in the "vagal tone monitor", also referred to as Porges-Bohrer RSA (PBRSA), serves to obviate at least the respiratory confounding of this index of cardiac vagal tone (Khodadadi et al., 2021; Porges, 2021a). This assertion, based on a single study solely from the Porges laboratory (Lewis et al., 2012), is, in fact, not substantiated: Extreme similarity of magnitude of correlation coefficients between respiratory parameters, on the one hand, and the PBRSA quantification and two other estimates of RSA, on the other hand, make this contention implausible (Grossman et al., 1990, in which the PBRSA measure was assessed by Dr. Porges himself). Likewise, in the latter study, the extremely high correlations (multiple comparisons, r's ranging from 0.92-0.99) between the Porges measure and two other quantification procedures (all analyzed in independent laboratories, including Porges's) do not make plausible the claim of superiority of the PBRSA measure. Additionally, RSA has consistently been found in a very large literature to be a respiratory-rate- and respiratory-volume-dependent phenomenon using a variety of RSA quantification methods from earliest systematic investigations (Clynes, 1960; Angelone et al., 1964; Hirsch & Bishop, 1981) to more recent studies (e.g. Grossman et al., 1991; Saul et al., 1989; see also Grossman & Taylor, 2007). Furthermore, the Porges index possesses its own potentially serious methodological vulnerabilities (Litvack et al., 1995), which remain to be thoroughly addressed or rectified.

The assertion that RSA is a direct measure, even specifically of cardiac vagal tone (e.g. "measuring cardiac vagal tone (i.e. respiratory sinus arrhythmia"; Porges, 2011), therefore, constitutes a 'category mistake' (Ryle, 1949) by equating an index or an aspect (RSA) of a general phenomenon (cardiac vagal tone) with the general phenomenon itself. Specifying that RSA in some manner monitors general vagal efferent activity is a more extreme example of a category error (e.g. equating RSA with cross-system vagal tone or vagal tone in other specific non-cardiac organ system, like the pulmonary system), since we know target organ-specificity certainly does exist in regard to vagal efference and afference (see above).

Premise 5. *: "Emotion, defined by shifts in the regulation of facial expressions and vocalizations, will produce changes in RSA and bronchomotor tone mediated by the nA."*.

a) Individual differences in resting cardiac vagal control have been shown not to be positively correlated with vagal bronchomotor tone (Horváth et al., 1995). Additionally, little association has been found between indices of vagal airway responses and cardiac vagal changes across a range of emotional and physical stimuli (Ritz, 2009). Once again, there operates vagus target-organ specificity (see discussion above), and one cannot generalize about different functions of pulmonary-cardiac control operating in concert with each other (Veerakumar et al., 2022).

b) The relation between emotion, cardiac vagal tone and RSA are complex. Distinct emotions have been shown to cause changes in breathing pattern (e.g. Grossman, 1983; Boiten et al., 1994). Changes in respiratory parameters will, in turn, cause alterations in RSA magnitude that may or may not be related to variations in cardiac vagal tone (e.g. Grossman et al., 1991; Grossman & Taylor, 2007; Saul et al. 1989). Thus, emotional effects upon RSA, mediated by respiratory changes, may occur—mediated by the nA and by brainstem respiratory centers–but may not reflect changes in tonic vagal control of heart rate (see preceding discussion of Premise 4).

c) Facial expressions, emotions and vocalizations are determined by a range of structures and mechanisms besides the nA or the vagus nerve. The underlying physiology certainly comprises all levels of central nervous system function (Cerkevich et al., 2022; Neuhuber & Berthoud, 2022, Taylor et al., 2022; Zhang & Ghazanfar, 2022). Overemphasis upon the efferent transmission of the vagus nerve in this regard is contrary to the current state of knowledge: The nA and the vagus nerve are merely jointly the messenger, not the message (e.g. see Cerkevich et al., 2022, Fig. 1; Zhang & Ghazanfar, 2022, Fig. 1).

d) It is obvious that the complexity of emotions and their expression cannot be reduced to a definition of "shifts in the regulation of facial expressions and vocalizations." Obviously, there are huge cultural and individual differences in the extent, and construction of, the experience and overt expression of emotions (Feldman Barrett,

2017). One can be happy, sad or angry, and not show it; a smile can go with either heart acceleration, deceleration or no change, depending upon the context, timing, physiological state and developmental stage (e.g. Clé et al. 2019; Fiacconi & Owen, 2015; Mireault et al., 2018; Pressman et al., 2021; Sroufe & Waters, 1976). Emotions are far more complex than mere facial expressions and social vocalizations.

e) Certainly, the vagus nerve has been known to be involved in the physiology of emotions for at least 80 years (Gellhorn et al., 1940), just as it is also long recognized to be involved in subserving many other diverse functions and activities, e.g. digestion, sexual activity, mental and physical performance. However there seems to be no evidence that the vagus nerve has been "repurposed" among mammals to facilitate socioemotional behavior, or that special social behaviors evolved as some sort of emergent property of mammalian vagal control (see Doody et al., 2023). In fact, clawed frogs appear to have a discrete ventral nA that mediates social vocalizations (Kelley, 2022).

3. Conclusions

The polyvagal conjectures comprise a psychophysiological model based upon the foundational premises cited in this article. Therefore, the behavioral/psychological postulates must be evaluated in terms of the accuracy of underlying physiological assumptions. There is broad consensus among experts specialized in the vertebrate evolution of the autonomic nervous system, vertebrate evolution of sociality and neuroanatomy of the brainstem and the vagus nerve that each basic physiological assumption of the polyvagal theory is untenable. Much of the existing evidence, upon which these consensuses are grounded, strongly indicates that the underlying polyvagal hypotheses have been falsified. An earlier paper also provides several additional challenges to the polyvagal conjectures (Grossman & Taylor, 2007).

In a nutshell, specifically, there is no support for the continuing polyvagal assertion that the dorsal vagal motor nucleus mediates massive bradycardia in mammals and may be responsible for vasovagal syncope, or trauma-related dissociative or emotional freezing responses (e.g. Porges, 2021a). In fact, as reviewed above, there is a substantial body of evidence to the contrary. Nor is there evidence that the brainstem-vagal system in mammals "was repurposed in order to support and express sociality" (Porges et al., 2021). Because these notions remain such central assumptions of polyvagal thinking and the very source of the "poly" in "polyvagal", psychological corollaries grounded upon the 5 basic premises must be called into question or provided with other explanations. There are alternative models which attempt to explore relations between the structure and function of vagal and psychological processes (Grossman & Taylor, 2007; Thayer & Lane, 2009). Although perhaps more plausible than the polyvagal conjectures, they also require further in-depth examination of their supporting physiological evidence.

Statement

The author did not use generative artificial intelligence (AI) technologies for preparation of this work.

Declaration of Competing Interest

The author reports no conflicts of interest.

Data availability

No data was used for the research described in the article.

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