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Critique of the Polyvagal Theory

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Main features of the Polyvagal Theory

Since its first description by Stephen Porges in 1995 [1,2], the polyvagal theory (PVT) has received much attention among mind-body therapists including osteopaths worldwide, especially with regard to the treatment of trauma patients. PVT is an attempt to explain the relationship between parasympathetic activity and behavior from an evolutionary perspective [3]. It aims to provide an understanding of the connections between brain and body processes [1,2].

The term ,,polyvagal," first employed in the polyvagal theory, refers to 2 vagal circuits:

- One is supposedly the phylogenetically **older** unmyelinated system represented by the dorsal motor nucleus of the vagus, which mainly innervates subdiaphragmatic organs (mainly the gastrointestinal tract), but also purportedly the heart, and is supposedly associated with immobilisation and dissociation (Fig. 1).
- Evolutionarily later, according to Porges, a second younger vagal pathway is thought to have developed, which is claimed only to be observed in mammals, not in reptiles, and to have the ability to down-regulate immobilization and fight-and-flight behavior. According to Porges, the anatomical structures of this component of the vagus interact in the brainstem with structures innervating the striated muscles of the face and head to create an integrated system of social engagement [4]. This younger system is represented in particular by the nucleus ambiguus (Fig. 1). In PVT, it is associated with the other branchiomotor (special visceroefferent) nuclei of the Vth, VII, IXth, and Xlth cranial nerves referred to as the ventral vagal complex I5]. This system regulates the heart and lungs via myelinated nerve fibers to allow resting states and is thought to be related to social behavior and "feeling safe".

The focus of PVT is on the proposed **phylogenetic shift between reptiles and mammals** that putatively resulted in specific changes in the vagal pathways regulating the heart. According to the PVT--but not necessarily accurate--primary vagal efferent pathways regulating the heart shifted from the dorsal nucleus of the vagus in reptiles to the nucleus ambiguus in mammals, establishing a face-heart connection with properties of a social engagement system that allows social interactions to influence visceral state and visceral dysfunction manifested in neural regulation of the heart [7].

Comparative anatomical and functional studies related to PVT

Comparative anatomical and functional studies argue against the proposed phylogenetic basis of PVT. It is undisputed that in mammals, myelinated cardioinhibitory axons arise from the nucleus Ambiguus. However, already in **cartilaginous fish** (elasmobranchs, e.g., sharks), which have existed for 400 million years, the cardioinhibitory vagus neurons are myelinated and conduct at speeds between 7 and 35 m/s (corresponding to the B fibers of mammals). Moreover, their cell bodies are located at 2 different sites in the brainstem (dorsal vagus nucleus and primordium of the nucleus Ambiguus) [8, 9]. Thus, cartilaginous fishes are already "polyvagal".

Lungfish, evolutionary precursors of air-breathing animals, also have a myelinated cardiac vagus nerve originating in dorsal and ventrolateral brainstem nuclei [10]. These myelinated, fast-conducting axons enable beat-to-beat slowing of the heart rate, which is mandatory for the cardiorespiratory interactions observed in these ancient vertebrates, similar to mammalian respiratory sinus arrhythmia [10, 11]. The unmyelinated cardiac neurons of the dorsal vagal nucleus do not have any significant influence on heart rate and thus cannot be responsible for bradycardia such as that observed in freezing states. They seem to influence ventricular inotropy and might protect cardiomyocytes from ischemia [12a].

Response patterns in PVT

PVT assigns responses to perceived risks to 3 categories: feeling safe, being in danger, or perceiving a threat to life. These categories are hypothesized to follow one another in phylogenesis. They are proposed to relate to the adaptive behaviors of **social communication** (facial expressions, speech, listening), which are proposed to be controlled by the **nucleus ambiguus**. On the other hand, **defensive behavior in terms of mobilization** (fight, flight) and immobilization responses (vasovagal syncope, dissociation or emotional freezing state) is claimed to be mediated by the **dorsal vagal nucleus** [1, 6, 12-14].

Again, the **proposed association of these behavioral phenomena** with the old unmyelinated or the new myelinated vagus nerve is **misleading**. The mammalian nucleus ambiguus contains, in addition to cardioinhibitory neurons, primarily the branchiomotor (special visceroefferent) neurons for laryngeal, pharyngeal, and striated esophageal muscles [1 5], but does not control facial expression (mimic muscles are innervated by the nucleus facialis, nF) nor hearing via the middle ear muscles (tensor tympani muscle, innervated by the motor branch of the trigeminal nerve, and stapedius muscle, innervated by the facial nerve) nor other head and neck muscles, as suggested by PVT. Conversely, the facial nucleus (nF) also does not affect the nucleus ambiguus.

All these motor nuclei, including the hypoglossal nucleus, are coordinated by premotor networks in the lateral parvocellular and intermediate reticular formation [16-20]. The intermediate reticular formation, located between the medial magnocellular and lateral parvocellular areas, also houses the neuronal networks for cardiovascular regulation (vasomotor center) and the central generators for respiratory rhythm (pre-Bötzinger complex, respiratory center) and for swallowing and vomiting. **The dorsal vagal nucleus**

and the nucleus ambiguus are anatomically and functionally embedded in these networks, but as output elements rather than coordinators. Vagal afferents are connected via the solitary nucleus (nucleus tractus solitarii) not only to the motor vagus nuclei (dorsal vagal nucleus and nucleus ambiguus), but also to the premotor networks of the reticular formation and the circulatory and respiratory centers [20]. However, trigeminal and upper cervical spinal afferents, which are also fed to the premotor reticular networks, are equally important for the coordination of the entire head-cervical motor system.

Role of the mesencephalic periaqueductal gray (PAG)

A bilateral periventricular nucleus in the ventral mesencephalon, showing a similar location to the mammalian PAG, has already been described in the lamprey, which belongs to the oldest group of vertebrates living today [21]. **Behavioral states** such as fight and flight, immobilization or freezing state, and risk assessment—together with associated motor, autonomic, and endocrine effects—are coordinated by the mesencephalic periaqueductal gray (PAG) [22-25]. The PAG is connected to the hypothalamus and limbic system (primarily the amygdala and prefrontal cortex) [23,24] as well as to various premotor and autonomic brainstem nuclei that coordinate respiration and the emotional motor system [25]. The PAG receives afferents from almost all sensory systems—not least of all the nociceptive system—and modulates their processing [24].

Undoubtedly, the vagus nerve has a significant influence on emotions and various behavioral states due to its large afferent component. Vagal afferents, which constitute about 80% of its axons, are transmitted through the nucleus tractus solitarii to the PAG, hypothalamus, amygdala, and insular, cingulate, and prefrontal cortex, where they are integrated into emotional and cognitive processes [26-29]. Recent studies suggest that subdiaphragmatic vagal afferents influence innate fear, learned fear, and other behaviors [30, 31]. Moreover, vagal afferents modulate spinal nociceptive processes in several experimental models [32, 33].

It is true that Porges [34] mentions the representation of neuroanatomically-- already long known--relations of the limbic system and PAG with bidirectional connections to the vagus complex. However, since it is not the ventral vagus complex but the PAG in association with limbic and other brainstem networks that is responsible as a coordinator for these behavioral states and, moreover, numerous brain areas, if not the entire brain, function as a system of social engagement, the term "polyvagal" to characterize it appears to be a misleading misnomer.

Conclusion

As Grossman and Taylor [11] have already shown, phylogenetic references are questionable as a basis for PVT. Facts of cranial nerve anatomy are also sometimes incorrectly represented in PVT. Instead of extending the concept of the ventral vagal complex to all branchiomotor nuclei, it would be more appropriate to leave them their independence and emphasize their coordination by a network of brainstem neurons.

The concept of a system of social engagement is plausible and seems to be relevant to practice. However, it is misleading in the formulation of the polyvagal assertions, and

linking the concept with the "old unmyelinated or new myelinated vagus" and the term, "polyvagal" should be avoided. In addition, the hypoglossal nerve, which is not a branchiomotor nerve but innervates the socially important tongue muscles, should also be included in the concept of social engagement.

The mesencephalic trigeminal nucleus and other sensory trigeminal nuclei are also important in the coordination of orofacial motor activity. The vagus nerve, efferent as well as afferent, is certainly an important factor in the social engagement system. However, because the [supposedly] "new" vagal nucleus in the form of the **nucleus ambiguus does not exert a coordinating function** on the other branchiomotor nuclei (V, VII, IX, XI)--even though vagal afferents are fed into these coordination networks via the nucleus tractus solitarii--PVT turns the causal relationships upside down. Consequently, the term "polyvagal" is a misleading misnomer. The functional construct of the social engagement system should not be associated with the term "polyvagal." Possibly a clarifying new designation would be indicated.

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