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Orienting in a defensive world: Mammalian modifications of our evolutionary heritage. A Polyvagal Theory

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Abstract

The vagus, the 10th cranial nerve, contains pathways that contribute to the regulation of the internal viscera, including the heart. Vagal efferent fibers do not originate in a common brainstem structure. The Polyvagal Theory is introduced to explain the different functions of the two primary medullary source nuclei of the vague: the nucleus ambiguus (NA) and the dorsal motor nucleus (DMNX). Although vagal pathways from both nuclei terminate on the sinoatrial node, it is argued that the fibers originating in NA are uniquely responsible for respiratory sinus arrhythmia (RSA). Divergent shifts in RSA and heart rate are explained by independent actions of DMNX and NA. The theory emphasizes a phylogenetic perspective and speculates that mammalian, but not reptilian, brainstem organization is characterized by a ventral vagal complex (including NA) related to processes associated with attention, motion, emotion, and communication. Various clinical disorders, such as sudden infant death syndrome and asthma, may be related to the competition between DMNX and NA.

Descriptors: Vagus, Vagal tone, Nucleus ambiguus, Dorsal motor nucleus of the vagus, Respiratory sinus arrhythmia, Polyvagal Theory

Contents:

Introduction

The systematic investigation of mind-body relations forms the scientific basis for the science of psycho-physiology. Unlike the correlative view of mind-body evaluations that dominates psychology and psychiatry, psychophysiology emphasizes a continuity between neuro-physiological and psychological processing. Psycho-physiologists assume that the nervous system provides the functional units for the bidirectional transduction of psychological and physiological processes. Thus, from a psychophysiological perspective, it is possible to link, not merely in theory, but also via measurement, psychological processes with neurophysiological processes and brain structures.

The paper will focus on the neural regulation of the heart via the vagus and how this regulation evolved to facilitate specific psychological processes. The Poly-Vagal Theory, described in this paper, provides an explanation of how the vagal pathways regulate heart rate in response to novelty and to a variety of stressors. The theory proposes that via evolution, mammalian nervous systems developed two vagal systems: one a phylogenetic relic of amphibia and reptilia, and the other, an evolutionary modification unique to mammals. According to the Poly-Vagal Theory, the two vagal systems are programmed with different response strategies and may respond in a contradictory manner. Explanations for several psychophysiological phenomena and psychosomatic disturbances will be proposed. The theory is based upon an established literature in neurophysiology, neuroanatomy, and psychophysiology.

Arousal theory: Historical legacy.

Early psychophysiological research assumed that peripheral autonomic measures provided sensitive indicators of arousal or activation (Darrow, Jost, Solomon, & Mergener, 1942; Duffy, 1957; Lindsley, 1951; Malmo, 1959). This view was based upon a rudimentary understanding of the autonomic nervous system in which changes in electrodermal activity and heart rate were assumed to be accurate indicators of sympathetic activity. As activation-arousal theory developed, continuity between the peripheral autonomic response and central mechanisms was assumed. According to this assumption, any organ influenced by sympathetic efferent fibers, such as the sudomotor, vascular, or cardiac systems, was a potential indicator of limbic or cortical activity.

Although the specific pathways relating these various levels were never outlined and are still sketchy, electrodermal and heart rate measures became the primary focus of research during the early history of the Society for Psychophysiological Research. This was due to their presumed sympathetic innervation and, in part, to their measurement availability. Not by plan, but by default, this emphasis created a research environment that neglected several important factors including: 1) parasympathetic influences, 2) interactions between sympathetic and

parasympathetic processes, 3) peripheral autonomic afferents, 4) central regulatory structures, 5) the adaptive and dynamic nature of the autonomic nervous system, and 6) phylogenetic and ontogenetic differences in structural organization and function.

Neglect of these concepts and an emphasis on a global construct of 'arousal' still abides within various sub- disciplines of psychology, psychiatry, and physiology. This outdated view of 'arousal' may restrict an understanding of how the autonomic nervous system interfaces with the environment and the contribution of the autonomic nervous system to psychological and behavioral processes. In contrast, more recent neurophysiological data promote a more integrative view of the autonomic nervous system.

Brain-heart communication: Historical perspective.

When we view living organisms as a collection of dynamic, adaptive, interactive, and interdependent physiological systems, it is no longer appropriate to treat the autonomic nervous system as functionally distinct from the central nervous system. We start to recognize that peripheral organs do not "float in a visceral sea." Rather, they are anchored to central structures via efferent pathways and are continuously signaling central regulatory structures via their abundant afferent pathways. Thus, the bidirectional connections between autonomic and central brain structures are becoming apparent. Accordingly, new theories and research strategies must incorporate the dynamic and interactive constructs that link central structures with peripheral organs.

Darwin (1872) provides historical insight into the potential importance of the vagus in bidirectional communication between the brain and the heart. Although Darwin focused on facial expressions in defining emotions, he acknowledged the dynamic relationship between the vagus and the central nervous system activity that accompanied the spontaneous expression of emotions. He speculated that there were identifiable neural pathways that provided the necessary communication between specific brain structures and peripheral organs to promote the unique pattern of autonomic activity associated with emotions. For example he stated:

... when the mind is strongly excited, we might expect that it would instantly affect in a direct manner the heart; and this is universally acknowledged... when the heart is affected it reacts on the brain; and the state of the brain again reacts through the pneumo-gastric [vagus] nerve on the heart; so that under any excitement there will be much mutual action and reaction between these, the two most important organs of the body (p.69).

For Darwin, when an emotional state occurred, the beating of the heart changed instantly, the change in cardiac activity influenced brain activity, and the brain stem structures via the cranial nerves (i.e., vagus) stimulated the heart. He did not elucidate the neurophysiological mechanisms that translate the initial emotional

expression to the heart. Our current knowledge of the brain stem origin and neurophysiological function of the various branches of the vagus was not available to Darwin. At that time it was not known that vagal fibers originated in several medullary nuclei and that the branches of the vagus exerted control over the periphery through different feedback systems. However, Darwin's statement is important, because it emphasizes the afferent feedback from the heart to the brain, independent of the spinal cord and the sympathetic nervous system, as well as the regulatory role of the pneumo-gastric nerve (renamed the vagus at the end of the 19th century) in the expression of emotions.

Darwin attributed the above ideas to Claude Bernard as an example of nervous system regulation of le milieu interieur. Consistent with more contemporary psychophysiology, Claude Bernard viewed the heart as a primary response system capable of responding to all forms of sensory stimulation. He explicitly emphasized the potency of central nervous system pathways to the heart (Cournand, 1979). These ideas are expressed in the following quotation:

In man the heart is not only the central organ of circulation of blood, it is a center influenced by all sensory influences. They may be transmitted from the periphery through the spinal cord, from the organs through the sympathetic nervous system, or from the central nervous system itself. In fact the sensory stimuli coming from the brain exhibit their strongest effects on the heart (Claude Bernard, 1865 quoted in Cournand, 1979).

Although seldom acknowledged as founders of modern psychophysiology, Bernard and Darwin have contributed to the theoretical basis for a neuro-psychophysiology of the autonomic nervous system. The above quotations document their view that the heart provided not only an output system from the brain, capable of indexing sensory processing, but they also recognized that the heart was a source of afferent stimulation to the brain able to change or contribute to psychological state. Consistent with this theoretical bias, psychophysiologists during the past century have investigated the functional sensitivity of heart rate measures to sensory and affective stimuli (e.g., Darrow, 1929; Graham & Clifton, 1966; Lacey, 1967), and the dynamic feedback between the brain and the heart in regulating both psychological state and the threshold for sensory stimuli (e.g., Lacey and Lacey, 1978).

Contemporary psychophysiology gained much of its current theoretical perspective from intriguing ideas regarding the interaction between autonomic and sensory processes introduced by Sokolov (1963). The Sokolovian model contained all the requisite components of an integrative theory relating autonomic function to psychological state. The model included: 1) acknow-ledgment of both afferents and efferents in both autonomic and somatic systems, 2) an autonomic feedback loop (i.e., autonomic tuning) to regulate sensory thresholds, 3) an interface between autonomic processes and psychological phenomena (i.e., orienting and defensive reflexes), and, 4) brain regulation of autonomic reactivity via habituation.

The Sokolovian model included bidirectional communication between brain and periphery. In the Sokolovian, model autonomic processes contributed to the tuning of receptor systems to engage or disengage with the external environment. Consistent with the Sokolov view, the Laceys (e.g., Lacey, 1967; Lacey and Lacey, 1978) emphasized the bidirectional communication between the afferents in the cardiovascular system and brain in the regulation of both cardiac function and sensory threshold. In contrast, to this emphasis on bidirectional communication, Obrist (1976) focused on the general concordance between metabolic demands and heart rate. Both arguments have merit. For example, afferent stimulation of the baroreceptors has immediate effects on both peripheral cardiovascular function and on central arousal state (Gellhorn, 1964), and the metabolic demands associated with exercise have deterministic influences, via vagal withdrawal, on heart rate (Obrist, 1981; Rowell, 1993).

Heart rate responses: A neurogenic emphasis

Throughout the history of the Society for Psychophysiological Research, psychophysiologists have been studying robust phenomena, such as the autonomic components of the orienting reflex, often without explanatory neurophysiological models. This paper is in response to this need. The paper will provide a theoretical model based upon the evolution of neural structures and the neural regulation of autonomic processes to explain several psychophysiological phenomena including orientation, attention, and emotion.

The orienting reflex provides an excellent point of embarkation. Based upon the convergent theoretical approaches of Sokolov (1963), Lacey (1967), and Graham and Clifton (1966), the orienting reflex is assumed to have a cardiac component. This component is characterized by a heart rate deceleration that functionally influences perceptual thresholds, facilitating the processing of information regarding the state of the external environment. However, what are the neural mechanisms mediating the cardiac orienting response? Or, as Obrist (1976) argued, is the heart rate deceleration merely an epiphenomenon associated with decreased metabolic demands accompanying the reduced motor activity that defines orienting and attending behaviors? The time course of the response, the effects of neural blockades, and studies with clinical populations support the contention that the cardiac orienting response is neurogenic. First, heart rate deceleration associated with the cardiac orienting response is rapid, occurring within a few seconds and usually returns rapidly to baseline. Second, the latency characteristics of the cardiac orienting response are similar to other neurogenic bradycardic reflexes such as opto-vagal, vaso-vagal, baroreceptor-vagal and chemoreceptor-vagal reflexes.

Blockade studies with atropine demonstrate that short latency bradycardia associated with both orienting reflexes and classical conditioning are mediated by cholinergic pathways via the vagus (e.g., Berntson, Cacioppo, & Quigley, 1994; Obrist, 1981; Schneiderman, 1974). Studies with the aged and other clinical populations with peripheral neuropathies or autonomic regulatory problems (e.g.,

diabetes) document deficits in vagal function (De Meersman, 1993; Gribben, Pickering, Sleight, & Peto, 1971; Weiling, van Brederode, de Rijk, Borst, & Dunning, 1982; Weise & Heydenreich, 1991). Additionally, studies of individuals with unilateral brain damage demonstrate that heart rate responses are diminished more in individuals with right side damage (Yokoyama, Jennings, Ackles, Hood, & Boller, 1987). This latter finding is consistent with the evidence that neurophysiological regulation of heart rate is primarily via the right vagus to the sino-atrial node, and that heart rate is under control of higher ipsilateral structures in the brain (Warwick & Williams, 1975).

Vagal influences producing heart rate deceleration in response to mild stress may interact synergistically with sympathetic withdrawal (Buwalda, Koolhaas & Bohus, 1992). Moreover, in conditions of anticipation of aversive stimuli, there have been reports that heart rate deceleration is, in part, due to sympathetic withdrawal (Rau, 1991). Although there are reports of a sympathetic contribution to stimulus dependent heart rate decelerations short latency decelerations are determined primarily by the vagus. Thus, it may be argued, that since short latency heart rate reactivity is mediated by the vagus, the magnitude of the cardiac orienting response is an index of vagal regulation.

The Vagal Paradox

In attempting to structure a neurogenic model of vagal regulation to explain psychophysiological phenomena, there is an obvious inconsistency between data and theory. Physiological theory attributes both the chronotropic control of the heart (i.e., heart rate) and the amplitude of respiratory sinus arrhythmia (RSA) to direct vagal mechanisms (e.g., Jordan, Khalid, Schneiderman & Spyer, 1982; Katona & Jih, 1975). However, while there are situations in which both measures covary (e.g., during exercise and cholinergic blockade), there are other situations in which the measures appear to reflect independent sources of neural control.

Several arguments have been made to explain this discrepancy. First, it has been argued that RSA and average heart rate (during sympathetic blockade) reflect different dimensions of vagal activity. For example, average heart rate might be viewed as reflecting tonic vagal influences and RSA as reflecting phasic vagal influences (e.g., Berntson, Cacioppo, & Quigley, 1993; Jennings & McKnight, 1994; Malik & Camm, 1993). Second, it has been argued that the discrepancy is caused by variations in respiratory parameters (Grossman, Karemaker, & Wieling, 1991) with RSA being confounded by respiratory frequency and tidal volume. Third, it has been argued that variation in quantification methods may contribute to the divergence between RSA and heart rate (Porges & Bohrer, 1990; Byrne & Porges, 1993). And fourth, it has been argued that average heart rate is influenced by a complex and dynamic interaction between sympathetic and vagal systems making it difficult to extract a vagal tone dimension (Berntson, Cacioppo, and Quigley ,1991, 1993).

Often, the arguments have been linked to a definition of vagal tone determined via neural blockade. The functional effect of the neural blockade on heart rate has been used as the criterion measure of vagal tone or parasympathetic control (e.g., Katona & Jih, 1975). Researchers have argued that RSA is not an accurate index of vagal tone, because individual pre-blockade levels of RSA do not accurately map into pre-post change in heart rate (Grossman & Kollai, 1993). Contrary to this argument, Porges (1986) argued that the discrepancy was, in part, based upon the criterion measure selected. He demonstrated that RSA exhibited a more sensitive dose-dependent response curve to vagal blockade via atropine than heart rate. This suggests the possibility that RSA, monitored during periods of spontaneous breathing, may provide a better criterion variable than heart rate. Neurophysiological support may be offered for this proposal. RSA is a vagal phenomenon in contrast to heart rate, which is determined by several sources including vagal, sympathetic, and mechanical factors. Thus, the efficacy of change in heart rate following cholinergic blockade as an index of vagal tone may be challenged.

The above arguments have created a volatile environment debating the neurophysiological interpretation of RSA and the efficacy of specific methods to quantifying RSA. Common to these arguments is the assumption that there is one central source of cardiac vagal tone. The arguments attribute differences, not to central mechanisms, but to the response characteristics of heart rate and RSA. Thus, divergence has been attributed to either the transfer function of the sino-atrial node that would attenuate high frequency oscillations (Saul, Berger, Chen, & Cohen, 1989) or the statistical transfer function of the method of quantifying RSA (Byrne & Porges, 1993) and not as a function of differential neural output.

However, independent of the quantification methodology and during periods of stable respiratory parameters, data have accumulated that demonstrate that RSA and heart rate (independent of sympathetic influences) often respond differently. Although both the neurogenic bradycardia and the suppression of RSA or heart rate variability observed during attention are assumed to be vagal in origin, they often appear independent of each other or in an apparent physiological contradiction (Porges & Raskin, 1969; Porges, 1972; Richards & Casey, 1991). Similar disparities between levels of heart rate and RSA have been observed during inhalant anesthesia when RSA exhibits a massive depression, while heart rate is not altered (Donchin, Feld, & Porges, 1985). Additional examples of convergence and divergence between RSA and heart rate can be observed in both within- and between-subjects designs. For example, individual differences in heart rate and RSA monitored during resting conditions provide independent contributions to measures of cardiac vagal tone derived from vagal blockade (e.g., Grossman & Kollai, 1993). However, convergence may be observed within an individual during exercise when monotonic increases in metabolic load are reflected in both faster heart rate and lower RSA (Billman & DuJardin, 1990). Or, convergence can be observed during neural blockade, via atropine, when both cardiac indices diminish in a clear dose response manner

(Cacioppo, Berntson, Binkley, Quigley, Uchino, & Fieldstone, 1994; Dellinger, Taylor, & Porges, 1987; Porges, 1986).

The relationship between RSA and heart rate may change within and between individuals. In our laboratory we have observed that the relationship between RSA and heart rate varies with behavioral state (Riniolo, Doussard-Roosevelt, & Porges, 1994). Twenty-four-hour ambulatory monitoring of adults indicates that during states of drowsiness and sleep the correlation between RSA and heart rate is significantly lower than during alert states. Thus, at times, RSA and heart rate appear to reflect the same physiological processes, while at other times they appear to reflect independent processes.

In contrast to the observable data, neuro-physiological research argues for a covariation between these two parameters, because vagal cardioinhibitory fibers to the heart have consistent functional properties characterized by bradycardia to neural stimulation and a respiratory rhythm (e.g., Jordan, Khalid, Schneiderman, & Spyer, 1982). This inconsistency, based upon an assumption of a single central vagal source is labeled the Vagal Paradox and is outlined in Table 1.

Table 1

The Vagal Paradox: A common central source?

1. Increased vagal tone produces neurogenic bradycardia
2. Decreased vagal tone produces suppression of RSA.
3. Bradycardia occur during periods of suppressed RSA.

The Vagal Paradox is critical to the interpretation of several psychophysiological and clinical conditions. For example, if the bradycardia occurring during orienting reflexes are vagal, why are bradycardia often observed during periods of reduced RSA, also an index of both attention and vagal control of the heart? If vagal tone is a positive indicator of health of a fetus or neonate when monitored with RSA, why is vagal tone a negative indicator of health when it is manifested as bradycardia? If bradycardia and RSA can both be removed by severing the vagus or by pharmacological blockade, are they both manifestations of vagal tone? If bradycardia and RSA are both indices of vagal tone, why do they respond differently? This apparent paradox provides the stimulus for the following inquiry and the development of the proposed Poly-Vagal Theory that speculates that, in mammals, there are two anatomically based vagal response systems.

Mammalian Poly-Vagal System

To understand the proposed Poly-Vagal Theory, it is necessary to provide additional information regarding the neuroanatomy and neurophysiology of the vagus in mammals. First, the vagus is not one nerve, but a family of neural pathways originating in several areas of the brain stem. Second, there are several branches of the vagus. Third, the vagus is not solely an efferent or motor pathway; rather, at least 80% of the vagal fibers are afferent (Agostoni, Chinnock, DeBurgh Daly, &

Murray, 1957). Fourth, the vagus is lateralized with nerve trunks originating in the left and right sides of the brain stem. Fifth, the vagus is asymmetrical with the left and right sides performing different tasks, with the right vagus most potent in the chronotropic regulation of the heart. These points are summarized in Table 2.

Table 2

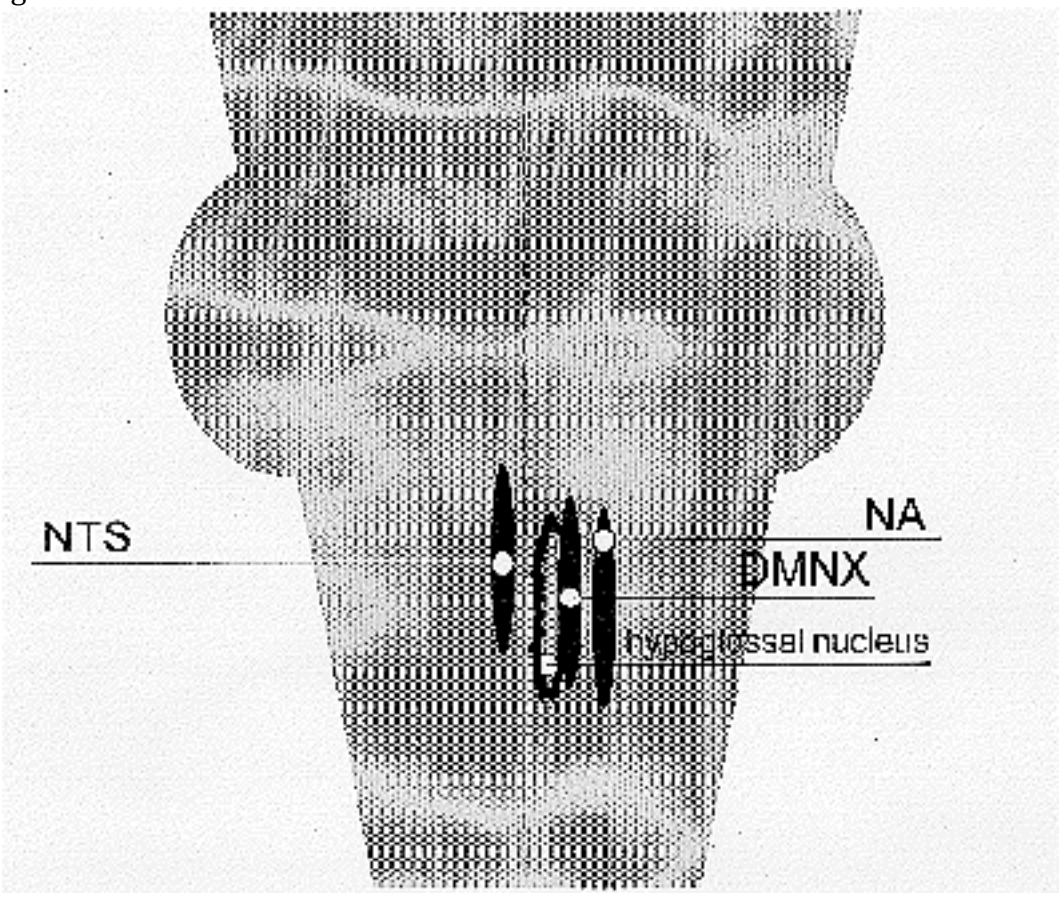
Mammalian Poly-Vagal System

1. Efferent fibers originate primarily in two medullary nuclei (NA, DMNX).
2. Vagal efferent fibers are clustered into several branches.
3. Approximately 80% of vagal fibers are afferent.
4. The vagus is lateralized.
5. The vagus is asymmetrical with a right bias.

Mammals are poly-vagal. The different vagi have different roles in the regulation of visceral function and originate in different brain stem nuclei with their respective viscerotropic organization. The different vagi may have oppositional outputs to the same target organ. For example, it is possible that during orienting there is an increase in vagal outflow from one branch to produce bradycardia and a withdrawal of vagal outflow from the other branch to produce a suppression of RSA (e.g., Richards and Casey, 1991). Thus, the concept of vagal tone may not be generalized to all vagal efferent pathways or even to the same target organ (e.g., heart), as has been assumed (e.g., Grossman & Kollai, 1993), but may need to be limited to a specific branch or subsystem of the vagus being evaluated. And, the intriguing concept of autonomic space proposed by Berntson, Cacioppo, and Quigley (1991, 1993) to deal with dynamic sympathetic-parasympathetic interactions may require an additional dimension to deal with potential vago-vagal interactions.

The Poly-Vagal Theory proposes that neurogenic bradycardia and RSA are mediated by separate branches of the vagus. Thus, the two commonly used, but not interchangeable measures of cardiac vagal tone may represent different dimensions of vagal tone. In mammals, the primary motor fibers of the vagus originate from two separate and definable nuclei in the medulla: the dorsal motor nucleus of the vagus (DMNX) and the nucleus ambiguus (NA). DMNX is in the dorsomedial medulla. NA is ventral to DMNX in the ventrolateral reticular formation (Warwick & Williams, 1975). The name ambiguus emphasizes the initial difficulties associated with determining its borders and connections within the reticular formation (Mitchell & Warwick, 1955). A third medullary nucleus, located near DMNX, the nucleus tractus solitarius (NTS), is the terminus of many of the afferent pathways travelling through the vagus from peripheral organs. This trinity of neural structures in the medulla, forms the primary central regulatory component of the vagal system. The relative locations of these medullary nuclei are illustrated in Figure 1.

Figure



1

Most cells originating in DMNX project to subdiaphragmatic structures (e.g., stomach, intestines, etc). In contrast, only the rostral portion of NA provides vagal innervation of subdiaphragmatic structures (Kalia & Mesulam, 1980), while most cells in NA project to supradiaphragmatic structures (larynx, pharynx, soft palate, esophagus, bronchi, and heart). Neurotracing and electrophysiological techniques with mammals provide additional evidence that the two vagal nuclei may function independently and have different central connections. These studies have demonstrated that there are no apparent connections between the two nuclei, although both nuclei have input from NTS, central nucleus of the amygdala, and hypothalamus, (Hopkins, 1987; Leslie, Reynold, & Lawes, 1992). It is well accepted that in mammals the primary cardioinhibitory motoneurons are located in NA. However, motor fibers from DMNX join the cardiac vagus (Bennett, Ford, Kidd, & McWilliam, 1984).

Cardioinhibitory and bronchoconstrictor neurons located in NA have myelinated vagal axons that conduct in the fast B fiber range (McAllen & Spyer, 1976, 1978). In contrast, neurons located in DMNX have axons projecting to the cardiac vagal

branches that are non-myelinated and conduct in the slower C fiber range. Although there are reports of cardioinhibitory vagal neurons with efferent axons conducting in the B fiber range being located in both DMNX and NA, neurons with axons conducting in the C fiber range are restricted to DMNX (Jordan, Khalid, Schneiderman & Spyer, 1982). The role of these non-myelinated vagal fibers on the heart is not well understood. In research with cats (Ford, Bennett, Kidd, & McWilliam, 1990) and dogs (Donald, Samueloff & Ferguson, 1967) stimulation of these fibers did not affect heart rate. However, although unsubstantiated at this time, the function of these fibers may be dependent upon the outflow of the myelinated NA fibers and may change during conditions such as hypoxia. For example, the influence of the unmyelinated fibers on the heart may be potentiated when the outflow from the mylenated NA fibers are blocked. In contrast, in the rabbit, stimulation of the non-myelinated vagal fibers results in heart rate slowing (Woolley, McWilliam, Ford, & Clarke, 1987).

The cytoarchitecture of NA illustrates that the dorsal portion contains source nuclei for special visceral efferents (i.e., voluntary motor fibers) and the ventral portion contains source nuclei for general visceral efferents (i.e., involuntary motor fibers). Motor projections from the dorsal portion go to target organs including the larynx, pharynx, soft palate and esophagus. Motor projections from the ventral portion go to several target organs including the heart, and the bronchi. In fact, these projections account for the primary cardiac and bronchomotor pathways and far outnumber the pathways originating in DMNX.

There is an obvious distinction between the viscerotropic organization of the two vagal nuclei. DMNX provides the primary vagal efferents to subdiaphragmatic organs that regulate digestive and alimentary processes. In contrast, NA provides the primary vagal efferents to the supra-diaphragmatic target organs including soft palate, pharynx, larynx, esophagus, bronchi, and heart.

The Poly-Vagal Theory

The Poly-Vagal Theory is based on several premises. Some are firmly grounded in neurophysiological and neuroanatomical data and others are more speculative. The first premise articulates the neural regulation of bradycardia and RSA. Based upon the initial premise, it is hypothesized that the neurogenic bradycardia associated with the orienting reflex are mediated by DMNX and that the suppression of heart rate variability (i.e., reduced amplitude of RSA) is mediated by NA.

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

Physiological support for the hypothesis that DMNX can contribute to neurogenic bradycardia, independent of NA, is provided by lesion studies. Machado and Brody (1988) have reported that chronic bilateral lesions of NA reduced but did not totally block baroreceptor reflex-mediated bradycardia in conscious rats. Thus, DMNX contains vagal neurons capable of producing bradycardia with a response latency associated with the baroreceptor reflex. This is supported by Jerrell, Gentile, McCabe, & Schneiderman (1986), who argued that differential Pavlovian conditioning of bradycardia in rabbits, following sinoaortic denervation, was mediated via DMNX pathways. The results pose the possibility that vagal pathways, originating in both the DMNX and NA, have the potential to influence heart rate.

Phylogenetic development of the Poly-Vagal System

Investigations of the phylogenetic development of the vagus provide support for the first premise. Since our interests are in mammals and specifically humans, this paper will focus on the evolution of vagal regulation of cardiac function from reptiles to mammals. There are two questions: 1) Do reptiles produce heart rate patterns during orienting similar to the neurogenic bradycardia observed in mammals? 2) Do reptiles produce a phenomenon similar to RSA?

The phylogeny of the vagus illustrates two phenomena: one, neuroanatomical, and the other, physiological. On a neuroanatomical level, differentiation of the visceral efferent column of the vagus into a dorsal motor nucleus (i.e., DMNX) and a ventrolateral motor nucleus (i.e., NA) is first seen in reptiles. In turtles (e.g., Chelone mydas and Domonia subtrijuga) there is still a connection between the two nuclei, but in lizards (e.g., Varanus salvator) and crocodiles (e.g., Caiman crocodilus) the separation between DMNX and NA is as complete as it is in mammals (see Barbas-Henry & Lohman, 1984).

Behavioral orienting in reptiles is characterized by a focusing of exteroceptors and a freezing of gross motor activity. Paralleling these behaviors, neurogenic bradycardia have been observed. Belkin (see Regal, 1978) reported that bradycardia is part of a fear response in iguanas. Additionally, McDonald (1974) reported brady-cardia in

the hog-nosed snake during death feigning. Most researchers found these data incompatible with the prevalent emphasis on arousal and the use of heart rate as indicator of arousal. How could bradycardia reflect increased arousal within the context of a sympathetic nervous system oriented arousal theory? In contrast, RSA has not been observed in reptiles. Research investigating the spectral components of reptilian heart rate has failed to identify heart rate oscillations associated with ventilation (Gonzalez Gonzalez, & de Vera Porcell, 1988).

Phylogenetic development not only illustrates changes in the neuroanatomy of the vagus, but also parallel changes in behavior. One of these behavioral shifts is the addition of active or voluntary attention and complex emotions. In confronting the defensive world, mammals, like reptiles, have an initial reflexive response to novelty, the orienting reflex. However, mammals have additional behaviors in their repertoire. Following or independent of reflexive orienting, mammals may voluntarily respond with sustained attention to foster detailed information processing, or with facial expressions and vocalizations to foster communication. Thus, reptiles orient; mammals may first orient and then elect to attend or communicate.

The differences between the reptilian and mammalian cardiac systems provides insight into the phylogenetic differences in behaviors such as reptilian orienting and mammalian attention and emotion. The cardiac output and thus, energy production of mammals far exceeds that of reptiles. Mammals have metabolic demands four to five times that of reptiles. The metaphor of a machine or vehicle has been proposed by Else & Hulbert (1981) to compare the efficiency and function of the mammalian and reptilian metabolic systems. According to Else and Hulbert (1981), when idling, the average mammal requires four to five times more fuel than the average idling reptile, even when body weight and ambient temperature are controlled. Elaborating on this metaphor, reptiles represent vehicles with one-liter engines and mammals represent vehicles with four- or five-liter engine. Thus, as in the story about the race between the tortoise and the hare, reptiles locomote with a reliable but under-powered engine and mammals locomote with a supercharged engine that can function for only short periods without requiring refueling.

The energy production capacities of reptiles and mammals contribute to their respective lifestyles. There is a bias among reptiles toward passive feeding strategies. Reptiles tend to be sit-and-wait feeders, slow cruisers, and sluggish browsers. In contrast, mammals with four-chambered hearts, can actively hunt and graze and adapt to changing environments (Regal, 1978). To support their behavioral niche and to ensure their adaptive success, reptiles and mammals use different vagal strategies to promote their lifestyles. Being under-powered, reptiles do not maintain a vagal brake on the heart, which would further reduce energy production during unchallenged situations. For reptiles, during periods of either quiescence or apnea, usually associated with behavioral freezing or diving, vagal influences via DMNX are profound and heart rate is even slower. In contrast, vagal

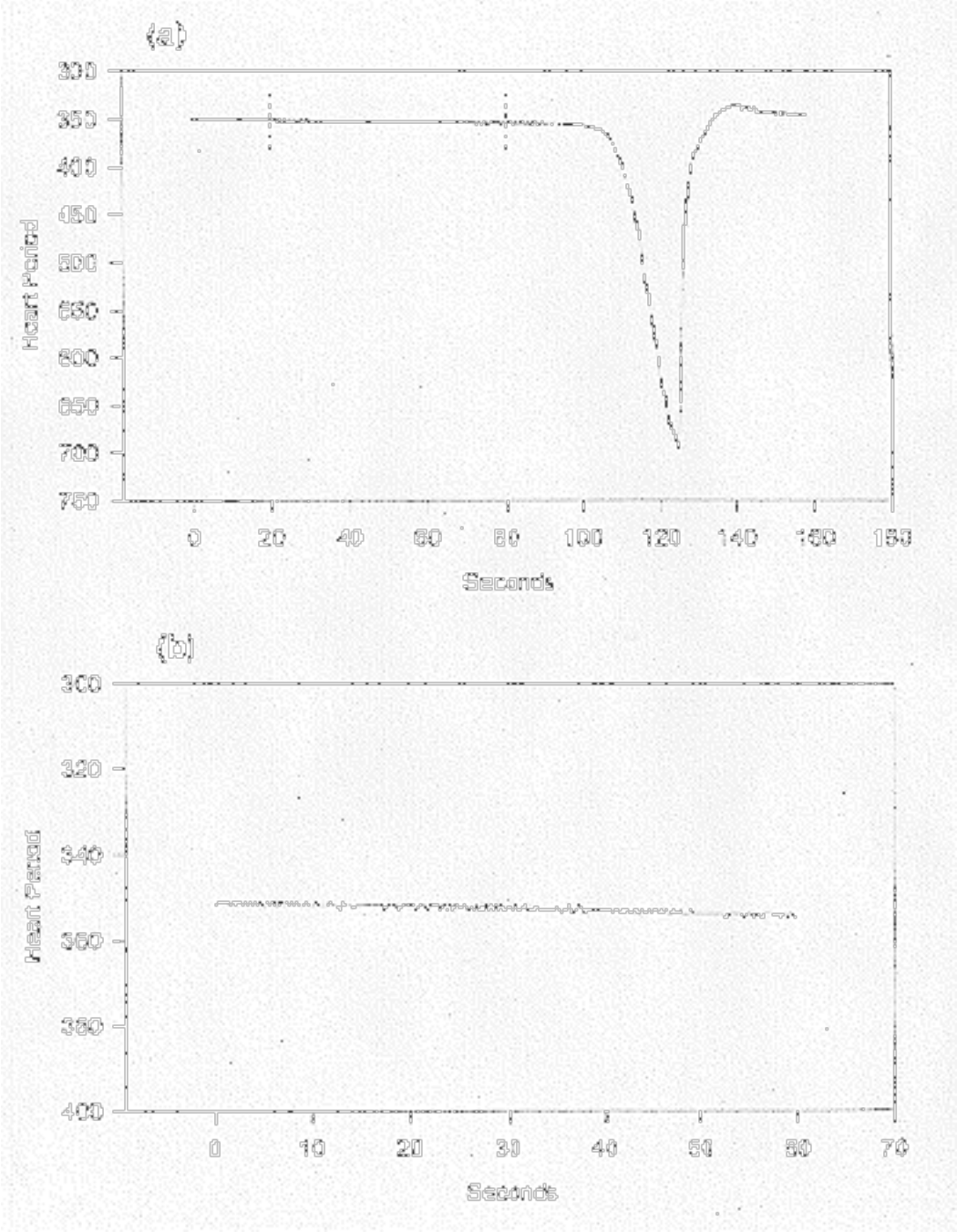
control of the heart is virtually removed during periods of breathing and other motor activities (Belkin, 1964; Jacob & McDonald, 1976).

The under-powered reptiles use vagal efferents from DMNX to the heart to deal with specific challenges: to orient and freeze in response to predator or prey, and to conserve oxygen while submerged for lengthy periods. In contrast to under-powered reptiles, supercharged mammals use vagal efferents from NA as a persistent brake to inhibit the metabolic potential of this high-powered system. The high NA-vagal tone keeps mammals from, literally, bouncing off walls. Thus, in contrast to that observed in reptiles, in mammals vagal tone is highest during unchallenged situations such as sleep, and vagal tone is actively withdrawn in response to external demands, including metabolically demanding states such as exercise, stress, attention, and information processing. For example, in humans, psychological states perceived as life threatening, such as panic and rage, are characterized by virtually no NA-vagal tone when indexed with the amplitude of RSA (George et al., 1989). Metaphorically, and consistent with the model, antisocial and pathological behavioral patterns associated with rage and hyper-reactivity without conscious self-regulation, have been labeled reptilian.

If terrestrial mammals adopted the reptilian strategy of reflexive increases in vagal activity to produce massive neurogenic bradycardia, the result would be catastrophic to the oxygen-hungry mammalian cortex and myocardium. This strategy would rapidly produce cardiac ischemia and cortical anoxia. The result of this sequence would be death. Although still dependent on oxygen, aquatic mammals use a diving reflex characterized by a regulated neurogenic bradycardia to reduce metabolic demands. To survive, aquatic mammals have complex mechanisms, not available to terrestrial mammals, to manage oxygen resources and shift priorities for oxygen while submerged for long periods.

It is possible that for mammals, during states of stress, when metabolic demands are great and vagal tone from NA is removed, that the cardiac pacemakers (S-A and A-V) may be prone to neurogenic bradycardia mediated by DMNX. The neurogenic bradycardia may be massive and lethal. This may be the case in fetal distress, when bradycardia are observed during hypoxic episodes, or as a factor in either sudden infant death syndrome (SIDS) or sudden death in adults. Consistent with this model, it has been demonstrated in the dog that progressive asphyxic hypoxia, not only elicits increased cardiac vagal activity, but the sensitivity of the sino-atrial node to vagal efferent influences is potentiated (Potter & McCloskey, 1986). Thus, during hypoxia large bradycardia may be maintained with limited or reduced vagal efferent activity.

Figure 2



The Poly-Vagal Theory provides a potential explanation for the massive neurogenic bradycardia observed during fetal distress and in high risk neonates who have virtually no observable RSA. For example, as illustrated in Figure 2, when massive bradycardia are observed during fetal distress (a), there is a background of low beat-to-beat variability (b). Similarly, neonates with the lowest amplitude RSA are at greatest risk for apnea and bradycardia (Sostek, Glass, Molina, & Porges, 1984). Thus, the diminished vagal influences from NA, responsible for depressed RSA

amplitude, seem to be associated with a vulnerability to large neurogenic bradycardia. Potter and McCloskey (1986) provide an explanation of how depressed CNS function associated with hypoxia might result in massive neurogenic bradycardia. They report a complex feedback system between duration of hypoxia, vagal efferent discharge, and potentiation of the vagal output on the heart. This system is able to maintain bradycardia, despite the massive decline in the vagal firing associated with hypoxia, by potentiating the influence of the vagal firing on the S-A node. Under these conditions, although the bradycardia are mediated through a branch of the vagus, the magnitude of the bradycardia is determined by a peripheral mechanism and no longer reflects a centrally mediated vagal tone. Although Potter and McCloskey did not monitor RSA, we must assume that RSA is low in their preparation because the animals were anesthetized prior to the surgical, electrical, and hypoxic manipulations, and because both hypoxia and anesthesia are associated with depressed beat-to-beat heart rate variability, including RSA (e.g., Donchin, et al., 1985; Nelson, 1976). Additional support for this bifurcation of vagal influences is demonstrated by electrical stimulation of the dorsal motor nucleus in the rabbit. As illustrated in Figure 3, electrical stimulation of DMNX results in a bradycardia without an increase in RSA. This is in contrast to the effect of stimulation of the aortic depressor nerve, which com-municates with both NA and DMNX. Note in Figure 4 that in a similar anesthetized rabbit, stimulation of the aortic depressor nerve results in an increase in RSA and a massive bradycardia (e.g., McCabe, Yongue, Porges, & Ackles, 1984).

Figure 3

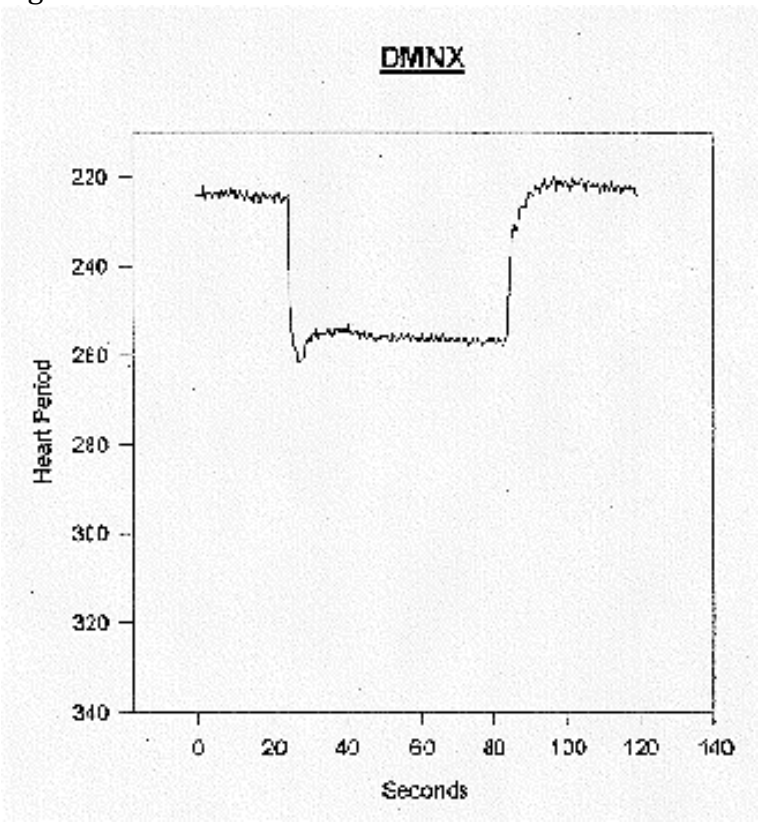
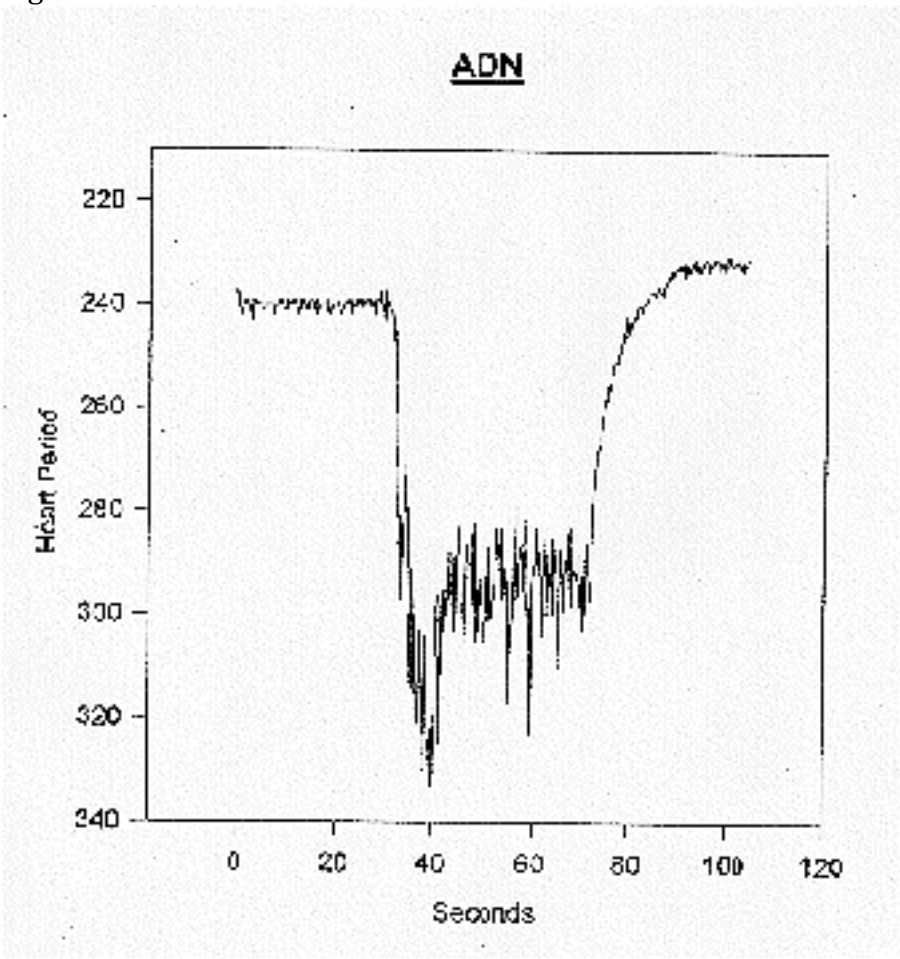


Figure 4



The Poly-Vagal Theory argues that the vagal fibers from the DMNX and NA are distinguishable in structure and function. Specifically, it has been argued that the vagal efferent fibers from the NA are myelinated and contain a respiratory rhythm and the vagal efferent fibers from the DMNX are unmyelinated and do not express a respiratory rhythm. However, there are some inconsistencies in the proposed distinction. For example, as cited above, Jordan et al. (1982) reported that there are cardioinhibitory vagal neurons originating in the DMNX with efferent axons conducting in the B fiber range and therefore, myelinated. Moreover, Jordan et al. report that these had a respiratory rhythm. Although the Jordan et al. findings support the proposed dual source of vagal efferents, their findings confound the proposed functional distinction.

There are several potential explanations for the inconsistency identified by Jordan et al. (1982). First, may be method. The Jordan et al. study used standard neurophysiological stimulation and recording techniques to identify cell bodies. According to Schwaber (1986) many vagal fibers previously assumed to originate in DMNX have been identified with newer methods, such as retrogradely labeled HRP,

to be located in NA. Schwaber (1986) also states that since axons from the NA pass very near the lateral border of DMNX, it is difficult to stimulate or lesion DMNX without NA involvement and confounding electrical stimulation studies. Thus, additional research with more accurate labeling techniques may demonstrate that all neurons in the B fiber range originate in NA. Our rabbit data reported in Figures 3 and 4 provide additional support for the possibility of mislabeling. According to Jordan et al. (1982) all neurons excited by aortic depressor nerve stimulation produced a respiratory rhythm in their ongoing discharge. Similarly, as illustrated in Figure 4, stimulation of the aortic depressor nerve resulted in both bradycardia and increased RSA. However, stimulation of the DMNX produced only an attenuated bradycardia. These findings suggest that vagal fibers discharging following stimulation of DMNX did not have a respiratory rhythm. Moreover, the bradycardia was immediate and similar in latency to that observed following aortic depressor nerve stimulation. However, the magnitude of bradycardia was about 50% of the magnitude elicited via aortic nerve stimulation that is assumed to recruit vagal fibers from both NA and DMNX. These findings are similar to those reported by Machado and Brody (1988). A second possibility is that there are species differences in the organization and function of the DMNX. For example, to facilitate freezing behavior, the rabbit may have evolved unique myelinated vagal pathways from the DMNX that were independent of respiratory function. According to this explanation, the DMNX would have B fibers, but they would express a respiratory rhythm. Alternatively, some mammalian species may have neurons in or near the DMNX that are part of a common cardiopulmonary oscillator (Spyer & Richter, 1990).

Future research will determine whether the proposed functional and structural distinctions between DMNX and NA efferents articulated in the Poly-Vagal Theory are accurate. An additional concern relates to generalizing across mammalian species. Most of the neurophysiological and neuroanatomical research on the mammalian vagus has been conducted with rat, rabbit, cat and dog. Studies investigating vagal regulation with humans have been limited to pharmacological blockade studies with measures of peripheral physiology. There are few neuroanatomical studies of human brain stem, however, these studies are often conducted on patients who have died of disease or trauma. Thus, one may question the generalizability of a poly-vagal model developed from investigations of rodent brain stem to the human. However, existing data illustrates phenomena such as clinical bradycardia in the absence of RSA in the human fetus (see Figure 2), shifts in RSA independent of heart rate change during inhalant anesthesia (e.g., Donchin et al., 1985), and short latency responses from both systems (see Figures 3 and 4) that argue for a poly-vagal system.

Vagal strategies in mammals and reptiles

Reptilian and mammalian vagal systems have contradictory strategies. Reptiles are characterized by low ambient vagal tone and transient increases in vagal tone in response to environmental challenges. In contrast, mammals are characterized by

high ambient vagal tone and transient decreases in vagal tone in response to environmental challenges.

Table 3

Vagal Strategies

* Ambient State
    - Reptiles -- low DMNX
    - Mammals -- high NA/low DMNX
* Response to Novelty
    - Reptiles -- increase DMNX
    - Mammals -- decrease NA/increase DMNX

To adapt to the hostile world, the reptiles' behavioral repertoire is survival driven. Most behaviors are associated with foraging, stalking, and feeding. Only limited time and energy are dedicated to social interactions such as parenting and reproduction. In the reptiles' defensive world, neurogenic bradycardia are adaptive and do not compromise physiological status. Reptiles have smaller metabolically active body organs, have different metabolic mechanisms, are less oxygen dependent than mammals, and can go for long periods without oxygen. In contrast, the adaptive strategy of reptiles is lethal for mammals. In the defensive world of mammals, it is necessary to increase metabolic output to foster fight or flight behaviors. Therefore, reflexive neurogenic bradycardia to novelty for a prolonged period would reduce oxygen resources and metabolic output and compromise the fight or flight potential of mammals. The consequences of reduced oxygen resources also would depress central nervous system function, reduce behavioral complexity and competent execution of complex behaviors, induce unconsciousness, damage vital organs and finally, if persistent, result in death. Thus, the cardiac component of the orienting reflex must be of short duration and replaced by a physiological response that does not compromise the oxygen-needy nervous system of mammals. The withdrawal of vagal tone via NA serves this purpose.

Phylogenetic origins of vagal response patterns

The neurogenic bradycardia controlled by DMNX and observed in reptiles and mammals during orienting may have evolved from the gustatory response system of primitive vertebrates. Gustation is the primary method for identifying prey (including other appropriate food sources) and predators in aquatic environments. For example, in fish, an undifferentiated vagal lobe controls gustatory, digestive, and alimentary processes (Finger & Dunwiddie, 1992). A reflexive increase in vagal tone would affect several organs: 1) the heart, where it would reduce metabolism and enable the animal to freeze momentarily, 2) the organs containing gustatory receptors, where it would orient towards the source of stimulation and regulate threshold to detect novelty, and 3) the digestive and alimentary systems, where it would stimulate gastric secretion and motility.

With phylogenetic development, the viscerotropic organization of the vagal system has become more complex, and incorporates pathways from other cranial nerves including trigeminal, facial, accessory and glossopharyngeal. Thus, more specialized functions such as head rotation to orient sensory receptors toward the source of stimulation, mastication to ingest food, and salivation to initiate gustatory and digestive processes are integrated into the vagal system.

The motor component of the vagus shares evolutionary origins with the four cranial nerves mentioned above (trigeminal, facial, accessory and glossopharyngeal). The vagus not only innervates smooth and cardiac muscle, but similar to the other four cranial nerves, it contains motor pathways that innervate somatic muscles. Vagal pathways that innervate somatic muscle often are not included in the neurophysiology of the autonomic nervous system. These fibers are labeled special visceral efferents to distinguish them from the motor pathways innervating smooth and cardiac muscle that are labeled general visceral efferents. The critical difference between the two types of motor pathways is that somatic muscle regulation may be conscious and voluntary, while smooth muscle regulation is reflexive and unconscious. Since the special visceral efferents innervate voluntary muscles, usually they are excluded from the autonomic nervous system. Traditionally, only the general visceral efferents from both sympathetic and parasympathetic branches are used to define the autonomic nervous system.

The somatic muscles innervated by the five cranial nerves arise from the branchial arches, embryologically known as the primitive gill arches, (Warwick & Williams, 1975). These muscles are critical to several mammalian behaviors. For example, the somatic muscles innervated by the trigeminal, arising from the first branchial arch, are involved in mastication, retraction of the lower jaw, and closing the mouth, The special visceral efferents from the facial nerve, arising from the second branchial arch, innervate the muscles of the face, scalp, and neck to enable facial expressions. The facial nerve also innervates muscles in the floor of the mouth. Although the trigeminal and facial nerves originate from branchial arches and have communications with the other three cranial nerves originating from the branchial arches, the source nuclei of the special visceral efferents for the glossopharyngeal, vagus, and accessory nerve originate in the same medullary nucleus, the nucleus ambiguus (NA). Thus, the efferent fibers travel through three different cranial nerves, but they originate in the same source nucleus.

As a function of phylogenetic development, the source nuclei for the special visceral efferent pathways in the glossopharyngeal, vagus, and accessory nerves migrate to form NA. In mammals, NA controls the complex coordination of pharynx, soft palate, larynx, and esophagus. Of special note to psychophysiological processes, the third gill arch also gives rise to the carotid body, containing peripheral chemosensitive receptors sensitive to oxygen and carbon dioxide levels, (Warwick & Williams, 1975). In addition, the accessory nerve provides fibers originating in the cervical spinal cord that innervate the positioning of the neck. The critical carotid arteries,

internal jugular veins, and vagus nerves run deep in these muscles (Warwick & Williams, 1975). Thus, this complex also has the ability to orient visceral receptors via somatic muscles, to coordinate structures related to ingestion and expulsion, and to regulate facial expression and emotion. These motor nuclei receive input from cortex to coordinate these complex behaviors with cardiopulmonary function. Thus, phylo- genetically, even when the gill arches evolve into the branchiomeric muscles common to all mammals, oxy-genation of blood through a coordination of breathing and heart rate during interactions with the environment remains a primary functional objective.

The processes associated with NA control of supradiaphragmatic organs appear to be uniquely mammalian. For example, this subsystem of the vagus coordinates the complex sequence of sucking, swallowing, and breathing that allows mammals to actively and voluntarily feed and breathe. Moreover, NA provides the primary chronotropic control of the heart and controls the intonation of vocalizations. Thus, NA efferent projections are involved with processes associated not only with feeding and breathing, but with processes associated with movement, emotion, and communication. These behaviors contribute to the unique social and survival behaviors observed in mammals. The NA-vagus provides the vagal brake that mammals remove instantaneously to increase metabolic output to foster fight or flight behaviors. The NA-vagus provides the motor pathways to shift the intonation of vocalizations (e.g., cry patterns) to express emotion and to communicate internal states in a social context.

The behavioral derivatives of the two branches of the vagus, suggest a typology in which one branch of the vagus deals with unconscious reflexive vegetative functions and the other branch of the vagus is involved in more conscious, voluntary, flexible and often social activities. There is neuroanatomical support for this typology. DMNX contains only general visceral efferents that innervate smooth and cardiac muscle fibers and regulate glandular secretion. In contrast, NA contains special visceral efferents that innervate the somatic musculature of the soft plate, larynx, pharynx, and esophagus.

Somatomotor and Visceromotor: Coupled systems

In mammals, we observe two evolutionary strategies that link autonomic function with somatic muscle activity. First, there is an anatomical linkage between the segmentation of the spinal nerves and the sympathetic chain. This linkage is reflected in the motor-related increases in sympathetic tone that have dogged psychophysiologists by confounding motor and autonomic responses. The evolution of the segmented sympathetic nervous system parallels the evolution of voluntary motor activities. The sympathetic nervous system regulates vasomotor tone to direct blood flow, and thus, oxygen, to the specific muscles being challenged. Additionally, there are sudomotor links to hydrate and protect the skin from tearing. This link between sympathetic activity and movement has been the cornerstone of arousal theory and hypotheses linking autonomic function to temperament and

psychopathologies. It was not many years ago that Obrist challenged the Lacey notion that autonomic state was independent of motor activity (i.e., metabolic demands). There is no doubt that the effects of motor activity are profound on the autonomic nervous system. Yet, this profound effect does not mitigate the importance of other relationships that may be sensitive to specific psychological processes, independent of movement.

Second, there is an anatomical linkage between the somatic muscles that arise from the cranial nuclei and parasympathetic function. We can observe this clearly in the viscerotropic organization of NA. NA provides the source nuclei for somatic muscle fibers that innervate larynx, pharynx, trachea, and esophagus. Moreover, ventral to these source nuclei, in an area of the nucleus ambiguus known as NAex, are general visceral efferents that control the resistance of the bronchi (Haselton, Solomon, Motekaitis & Kaufman, 1992) and heart rate (Bieger & Hopkins, 1987). The ventral portion also projects to other visceral organs (e.g., Brown, 1990).

Based upon neuroanatomical studies, it has been demonstrated that visceromotor functions regulated by the ventral part of NA provide the parasympathetic support for the somatomotor projections from NA, trigeminal and facial nerves. Neuroanatomical studies suggest that, unlike DMNX, which receives primary sensory input via NTS, NA has the trigeminal nerve as an important source of sensory input. Moreover, the rostral region of NA communicates with the facial nucleus. This coupling of NA with facial and trigeminal nuclei provides additional evidence of the coordination of the visceromotor regulation via NA with somatomotor functions such as swallowing (Brown, 1974), sucking (Humphrey, 1970), and, perhaps, facial expressions. Thus, the organization of the mammalian brain stem has evolved to have a ventral vagal complex consisting of NA and the nuclei of the trigeminal and facial nerve that co-exists with the dorsal vagal complex consisting of The DMNX and NTS that regulates vegetative processes and is observed in the reptile.

To foster motor movement, visceromotor (i.e., autonomic) processes are associated with somatomotor activities. In the periphery this is done primarily by the sympathetic chain and in special cases, such as those related to reproduction and elimination, the sacral branch of the parasympathetic nervous system contributes. However, in the rostral part of mammalian anatomy (i.e., the head) the somatic muscles that regulate facial expression, mastication, vocalization, swallowing, and sucking are matched with general visceral efferents, projecting from the ventral portion of NA, that exert potent influences on the heart and the bronchi. These motor fibers effectively slow heart rate and increase respiratory resistance to conserve oxygen exchange. Neuroanatomical studies performed on human embryos and fetuses suggest that these visceromotor neurons may have migrated from DMNX (Brown, 1990).

As observed through both embryological research and phylogenetic comparisons, in mammals, the primitive gill arches evolve into muscles and nerves controlling the

face, bones of the mouth, jaw, pharynx, larynx, softplate, esophagus, and trachea. The nerves innervating these muscles uniquely arise, not from the anterior horns of the spinal cord, but from the source nuclei of five cranial nerves referred to above (trigeminal, facial, glosso-pharyngeal, vagus and accessory). Because of their uniqueness, these motor systems are known as special visceral efferents. And, because of their voluntary aspects, these pathways have been excluded from traditional concepts of the autonomic nervous system. Facial expressions, sucking, swallowing, and vocalizations, characteristic of mammals, reflect the unique mammalian adaptation of special visceral efferent control of the visceral muscles evolving from the branchial arches.

However, similar to the synergistic relationship between the sympathetic nervous system and skeletal muscles of the extremities, there is a synergistic relationship between the traditional general visceral efferents of the vagus and the somatic muscles controlled by these cranial nerves. Thus, increased outflow of these somatic muscles produce specific visceral shifts. For example, chewing will produce salivation in the absence of food. Additionally, head rotation, via accessory special visceral efferents, will impact on cardiovascular action via the vagus.

Phylogenetic development of the central nervous system has progressed in mammals to produce a brain with a large neocortex (e.g., MacLean, 1990). The neocortex is very vulnerable to shifts in oxygen. Evolutionary pressures have resulted in autonomic strategies that optimize the availability of oxygen to the cortex. However, these uniquely mammalian strategies coexist with the ancestral reptilian strategies. Thus, premise 2 is stated, consistent with MacLean's view that the advanced mammalian brain contains its phylogenetic heritage.

Premise 2: Neurogenic bradycardia associated with orienting are a phylogenetic vestigial relic of the reptilian brain and are mediated via DMNX.

Although phylogenetic development has modified several brain structures, the evolved brain of advanced mammals maintains several structures and systems that are virtually identical to those observed in primitive reptiles. These primitive structures have extensive interconnections and functional dependencies, although each is capable of specific independent functions. Thus, in mammals, DMNX still maintains its reptilian functions of facilitating digestion and slowing heart rate. Mammals utilize an additional brain stem structure, the NA, to supply general visceral vagal efferents that provide the prominent control of the heart and the bronchi. The cells of origin of these fibers efficiently communicate with limbic, and other higher, centers and allow for the conscious and voluntary selection of novelty. In contrast, DMNX is more directly regulated by hypothalamic communication, often triggered by survival-oriented stimuli (Hopkins, 1987; Leslie, Reynolds, & Lawes, 1992). Thus, as stated in Premise 3, the regulation of vagal efferents by NA mechanisms contributes to the mammalian ability to detect novelty, actively engage with the environment, and socially communicate.

Premise 3: Withdrawal of cardiac vagal tone via 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.

To summarize the reptilian-mammalian evolutionary evidence, phylogenetic development of the neural regulation of the heart provides insights into an apparent contradiction or paradox in vagal control of the heart. In most reptiles the neuroanatomy demonstrates: 1) a lack of anatomically distinguishable boundaries between DMNX and NA, and 2) cardiac vagal efferent pathways originating only in DMNX. In mammals the neuroanatomy demonstrates: 1) a distinct separation of DMNX and NA, 2) cardiac vagal efferent pathways originating primarily (but not exclusively) in NA, 3) direct neural connections between the central nucleus of the amygdala and NA, and 4) a clustering of medullary neurons in NA capable of regulating the somatic muscles related to vocalizations, facial expression, and to coordinate breathing with sucking and swallowing.

Smart and vegetative vagi.

The Poly-Vagal Theory proposes that the evolutionary shift resulting in both a NA that is distinct from the DMNX, and the evolutionary development of special visceral efferents changed the role of the vagus. The general visceral efferent pathways from DMNX vagus are part of a passive reflexive motor system associated with vegetative function and thus, a vegetative vagus. The special visceral efferent pathways from NA create an active voluntary motor system associated with the conscious functions of attention, motion, emotion, and communication, and thus, a smart vagus.

The Poly-Vagal Theory requires a reconcep-tualization of the vagal system and the construct of vagal tone. The Theory focuses on the cytoarchitecture of the medullary source nuclei of the cranial nerves. The Theory takes an evolutionary approach and investigates, via embryology and phylogenetic comparisons, the common origins of the special visceral efferents and focuses on the shared medullary structures for the cell bodies of these fibers. The Theory acknowledges that the vagal system is complex, and should be organized, not in terms of bundles of fibers leaving the medulla, but rather in terms of the common source nuclei of several of these pathways. Functionally, the common source nuclei both provide a center to coordinate and regulate the complex interactions among various end organs and are related to optimizing cardiopulmonary function.

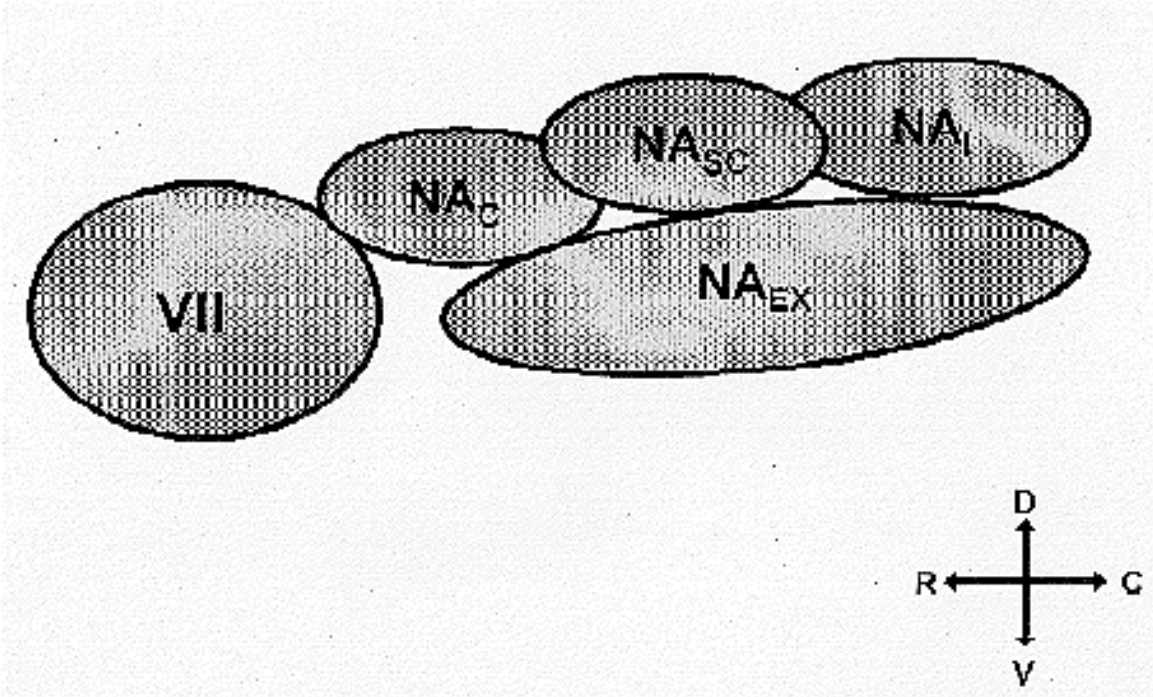
Mammals, with their oxygen-hungry metabolic systems, require a special medullary center to coordinate cardiopulmonary functions with behaviors of ingestion (e.g., mastication, salivation, sucking, swallowing), oral or esophageal expulsion (vomiting), vocalizations (e.g., cries, speech), emotions (e.g., facial expressions), and attention (e.g., rotation of the head). The NA plays this role and serves as the cells of origin of the smart vagus. The potent link between NA and cardiopulmonary function observed in mammals is not observed in reptiles. In reptiles, which do not

have nerves to regulate facial expression, NA does not play a major role in viscero-motor regulation.

Medullary contributions to a common cardiopulmonary oscillator

The NA is a continuum of interconnected sub-divisions beginning rostrally at the level of the facial nucleus and extending caudally to the spinal medullary junction. As illustrated in Figure 5, the rat NA has several subdivisions. The subdivisions are labeled: the compact (NAc), semicompact (NAsc), loose (NAl) and external (NAex) formations (Bieger & Hopkins, 1987). The dorsal division consists of NAc, NAsc, and NAl . The dorsal division is the source of special visceral efferents innervating the softpalate, pharynx, larynx, and esophagus. The ventral division consists of the NAex and is the source of general visceral efferents innervating the thoracic viscera, primarily the bronchi and the sino-atrial node. Vagal fibers originating in NAex and terminating in both the bronchi (Haselton et al, 1992) and the sino-atrial node (Spyer & Jordan, 1987) have a respiratory rhythm, thus suggesting that RSA may reflect a common respiratory rhythm originating in or, at least, incorporating NA.

Figure 5



After investigating the neuroanatomical centers associated with laryngeal, pulmonary, and cardiac function, Richter and Spyer (1990) arrived at a convergent conclusion that NA was a contributor to a common respiratory rhythm. They also speculated that mammals, with their great needs for oxygen, have a medullary center to regulate cardiopulmonary processes. They proposed that a common cardiorespiratory oscillator evolved to foster coordination among cardiac and respiratory processes. In their model the respiratory rhythm is dependent on the

interaction between two groups of neurons, one in NTS and the other in NA. Accordingly, the "common" oscillator producing respiratory frequencies is a manifestation of a neural network composed of interneurons between areas containing the motoneurons regulating respiratory, laryngeal and cardiac function. Note that the cardiorespiratory oscillator does not involve DMNX. To support their hypotheses they report cross-correlational studies of single units. Thus, NA is part of the cardiorespiratory oscillator network, and the period of the oscillations in heart rate, (the period of RSA) provides a valid index of the output frequency of the cardiopulmonary oscillator.

Other researchers emphasize the importance of additional brain structures as contributors to the regulation, if not the generation, of a cardiopulmonary rhythm. For example, Harper and associates have demonstrated that "respiratory rhythms" can be observed in several nuclei in the brain stem, midbrain and forebrain. Harper's group, employing crosscorrelation techniques, has reported units firing on a breath-by-breath basis in periaqueductal gray (Ni, Zhang & Harper, 1990), central nucleus of the amygdala (Frysinger, Zhang, & Harper, 1988), hippocampus (Frysinger & Harper, 1989), and the anterior cingulate (Frysinger & Harper, 1986). In addition, they have reported that stimulation of amygdala can influence the respiratory cycle (Harper, Frysinger, Trelease, & Marks, 1984).

The link between bronchi and heart rate oscillations (e.g., RSA) mediated via NA may have a functional influence on the oxygenation of blood. As stated above, the primary objective of the phylogenetic derivatives of the primitive gill arches is to maintain oxygenation. Thus, one might speculate that oscillations in vagal tone to the bronchi and the heart might influence oxygenation. Perhaps, coherent rhythmic shifts between bronchial tone and heart rate, with a fixed phase lag, maximize oxygen diffusion. To answer this question, research would need to confirm a relationship between oxygen saturation and RSA, independent of the average heart rate and respiration rate. Currently, only anecdotal data exist that demonstrate that clinical conditions in which oxygen saturation is low, tend to be conditions in which RSA also is depressed. Support for this hypothesis is obtained from research demonstrating that vagotomy disrupts the oxygen consumption-oxygen delivery relationship (Schertel, Brourman, Klilng, Schmall, Tobias & Myerowitz, 1994).

Measurement of NA status: Quantification of RSA

For psychophysiologists, our interest is primarily in the behaviors and psychological processes associated with special visceral efferents. Most research has been directed toward processes that require the ability to monitor and mediate complex behaviors, such as attention, motion, emotion, and communication; these processes are neurophysiologically linked to the special visceral efferents of NA, facial, and trigeminal nerves. Yet, many of us measure only general visceral efferents from both the parasympathetic and sympathetic branches, although we are interested in the special visceral efferents that regulate vocalizations and facial expression. We are not at a total loss, because there is interneuronal communication between the dorsal

and ventral segments of NA. Thus, by the nature of the NA having the general visceral efferents regulating heart and bronchi, it is possible to monitor continuously the vagal output or tonus of the smart vagus. This leads us to the fourth premise of the Poly-Vagal Theory.

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

The vagal fibers originating in the NAex have a characteristic respiratory frequency that reflects a waxing and waning of influence. For example, the vagal fibers from NA that have an inhibitory action on the sino-atrial node also wax and wane in inhibitory influence at the respiratory rhythm and produce RSA. Thus, it is possible to monitor continuously the general status of NA by evaluating RSA. Similarly, NA fibers to the bronchi that elevate lung resistance also wax and wane in their inhibitory influence (Haselton et al., 1992).

RSA is a measure of the general visceral efferents of the NA, and thus is an index of the smart vagus. RSA is not a global measure of vagal tone or even a measure of "total" vagal control of the heart as previously proposed (Fouad, Tarazi, Ferrario, Fighaly, & Alicandro, 1984; Katona & Jih, 1975; Porges, 1992). There are other vagal and non-vagal influences on the heart, which contribute to both heart rate level and rhythm. For example, there are DMNX projections, as well as monosynaptic cholinergic pathways within the heart, sympathetic pathways, and intrinsic factors. However, the primary, if not sole, source of respiratory rhythms on the S-A node is due to projections from NA.

To evaluate NA regulation of the sino-atrial node, the parameters of RSA must be accurately extracted. We have approached this problem by evaluating the period and amplitude of RSA, independent of slower oscillations and trends, via a moving polynomial approach (Porges & Bohrer, 1990). In our research we have obtained correlations between respiration rate and period of RSA approaching 1.0. These findings support the notion of a common cardiorespiratory oscillator as described by Richter and Spyer (1990). RSA, with its amplitude representing visceromotor tone and its period representing the common cardiorespiratory drive frequency, is the functional consequence of the output of vagal fibers originating in NA and terminating on the sino-atrial node.

Quantification of RSA requires only an accurate determination of the amplitude and period of these oscillations. Additional experimental constraints to regulate breathing rates might confound the visceral-medullary feedback system which determines central respiratory rhythms. For example, since paced breathing requires an awareness of breathing parameters, cortical influences on brain stem structures might modulate the gain of the feedback and influence the amplitude of RSA. Also, paced breathing may shift respiratory parameters, such as rate, amplitude, inspiration-expiration ratio, inter-breath pause, and resistance, from

brain stem setpoints. Data have been reported illustrating that paced breathing may influence RSA (Sargunaraj, Lehrer, Carr, Hochron, & Porges, 1994).

Various manipulations or conditions that depress special visceral efferents, such as inhalant anesthesia, have profound influences on RSA (Donchin et al., 1985). Recovery of function of special visceral efferents is paralleled by a recovery of RSA. In neurology, diagnosis is often based on the evaluation of the special visceral efferents. In our research, we noted that RSA amplitude before neurosurgery was an effective diagnostic of neurological recovery following neurosurgery (Donchin, Constantini, Szold, Byrne, & Porges, 1992). Additional neurological data demonstrate consistent depression of RSA in individuals who are diagnosed as brain dead (Mera, Wityk, & Porges, 1995).

High risk preterm neonates have problems coordinating breathing, sucking, and swallowing (i.e., processes regulated by NA). These infants have low levels of RSA (Porges, 1992). Many of these infants have severe bradycardia. The bradycardia are often paralleled by apnea, and a drop in available oxygen and may be assumed to reflect neurogenic vagal regulation via DMNX. Recall, that this response, to deal with decreased resources, is adaptive for reptiles, but potentially lethal for the human. This also is observed during fetal distress, when there is severe hypoxia associated with a loss of RSA and a pronounced neurogenic bradycardia (see above).

Vagal Competition and Autonomic Dysfunction

The concept of competition between sympathetic and parasympathetic inputs is well known. For example, Levy (Vanhoutte & Levy, 1979; Levy,1984) has clearly documented the ability of vagal efferents to inhibit sympathetic influences. Similarly, Berntson and associates have modelled the interactions between sympathetic and parasympathetic efferents to the heart (Berntson, Cacioppo, & Quigley, 1991). However, there may be a different type of competition, in which the two vagal branches are conveying contradictory information to the target organs. Since both vagal pathways are capable of regulating heart rate, there may be competition on the sino-atrial node. Due to the rate of acetylcholine degradation on the nodal tissue (Dexter, Levy, & Rudy, 1989) the continuous stimulation of the sino-atrial node by NA pathways may functionally protect the heart from massive neurogenic bradycardia mediated by DMNX. Thus, the observations of massive pathophysiological bradycardia in hypoxic fetuses and neonates, who have very low amplitude RSA, may reflect the loss of NA protection on the SA node. Similarly, sudden death following exercise might reflect a similar process associated with the depression of NA input to foster metabolic activity, and a surge of DMNX input in response to decreased oxygen resources.

The vagal competition hypothesis may be generalized and tested to explain other autonomic diseases such as asthma. The vagal competition hypothesis proposes that all target organs with smooth and cardiac muscle have dual innervation from both the DMNX and NA. Currently, this has been documented in animal preparations for

heart, lungs, esophagus, and abdominal viscera including pancreas, liver, and stomach (Brown, 1990). However, as with the heart, the two vagal inputs may innervate in a contradictory manner. Just as a DMNX surge coupled with low RSA can result in sudden death, as in the examples above, bronchial asthma may be produced by a similar mechanism. In the case of asthma, NA efferent control of bronchi results in the bronchi exhibiting a rhythmic waxing and waning with breathing. This continuous stimulation of the bronchi by NA pathways may functionally protect the bronchi from pathophysiological DMNX influences. It is possible that without NA influences, the bronchi become vulnerable to vagal surges from DMNX. This would be an adaptive response for a primitive brain stem attempting to conserve oxygen, but it is lethal for the oxygen- hungry mammal. The asthma attack, similar to lethal neurogenic bradycardia, may be a product of a primitive vago-vagal reflex. In this type of reflex, not only do the motor fibers originate, but the afferent fibers terminate in DMNX. There is an anatomical basis for a monosynaptic vago-vagal reflex. There are reports that dendritic processes from DMNX neurons extend into the boundaries of NTS. Thus, vagal afferent fibers may com-municate directly with DMNX neurons (Neuheuber & Sandoz, 1986). Since afferents terminate in DMNX, the name "motor nucleus" is not accurate and a preferred designation, "dorsal nucleus of the vagus nerve" has been suggested (Nara, Goto, & Hamano, 1991). In most vagal reflexes involving the bronchi, the afferents terminate in NTS and influence NA to provide a fail safe feedback system.

Based upon the Poly-Vagal Theory, the assumption of vagal competition promotes the following testable hypotheses:

Nucleus ambiguus (Vagal) Protection Hypothesis: Vagal projections originating in NA and terminating in visceral organs provide tonic influences that promote health, growth and restoration.

Nucleus ambiguus (Vagal) Withdrawal hypothesis: Removal of the NA-vagal brake for short periods of time promotes metabolic output to foster locomotion. Removal of these influences for long periods places the organ at risk.

Emotion

The Poly-Vagal Theory provides a set of predictions regarding the relation between autonomic responses and emotion. Darwin carefully described facial expressions as the primary defining characteristics of emotion. The special visceral efferents, associated with the facial nerve, control movements of facial expression. Reptiles do not have facial muscles and cannot modulate facial expression. The facial nerve in mammals not only regulates facial muscles but interacts with NA and the vagal system. Thus, it is logical that emotional expression, which requires somatic muscles controlled by special visceral efferents, is linked to the visceromotor regulation of cardiopulmonary function via NA vagal efferents. Additionally, special visceral efferents originating in NA regulate the larynx and control intonation. Thus, the following premise is stated.

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

As a construct, emotion is heterogeneous. Therefore, correlations between specific emotions and physiological states may be a function of the type of emotion. Even Darwin (1872) distinguished between primary or neurally based emotions and social or culturally based emotions. Darwin (1872) suggested that certain emotions have as their substrate an innate neural basis and, because these emotions are neurally based, they are universally expressed and understood across cultures. These primary emotions include anger, fear, panic, sadness, surprise, interest, happiness (ecstasy), and disgust (Ross, Homan, & Buck, 1994). Since the prevalent hypotheses suggest a strong physiological basis for primary emotions, we will focus here on relating primary emotions to the Poly-Vagal Theory.

There are two important aspects linking the Poly-Vagal Theory to the study of emotion: first, there is a parallel between cortical asymmetry and autonomic asymmetry; second, the branchial arches have evolved into the structures that mammals use to express emotion (i.e., facial muscles, larynx).

The literature documents the relationship between right brain function and primary emotions (Heilman, Bowers, & Valenstein, 1985). The medullary source nuclei and efferent pathways of the vagus also are lateralized with a right bias. The right NA via the right cardiac vagus provides the primary chronotropic output to the heart. The special visceral efferents, which provide the behaviors that are used to define emotion (facial expression, vocalization) also have a right bias and are linked neuroanatomically to the general visceral efferents originating in NA that regulate the bronchi and heart; organs that are assumed to be sensitive to emotion and stress. It is difficult to predict the influence of this right bias on actual facial expressions. Since the face is controlled by upper motor neurons that are crossed and lower motor neurons that are uncrossed (Rinn, 1984), facial expression may not be systematically lateralized. In fact, research on facial asymmetry and emotion has not been consistent. There have been reports of facial expressions not being lateralized, being lateralized on the left, and others being lateralized on the right (e.g., Hager & Ekman, 1985).

The functional dominance of the right side of the brain in regulating autonomic function and emotion may have implications for the specialization of motor and language dominance on the left side of the brain. The right sided responsibilities of regulating homeostasis and modulating physiological state in response to both internal (i.e., visceral) and external (i.e., environmental) feedback, may contribute to the development of motor and language functions on the left side of the brain.

A partitioning of central control of voluntary processes, independent of emotional-homeostatic processes would enable the individual to express complex voluntary levels of communication and movement, via the left side of the brain, and more

intense emotional-homeostatic processes, via the right side of the brain. If these processes are lateralized, they might have a degree of autonomous regulation. This would enable simultaneous activation of global functions associated with emotional-homeostatic processes and language-voluntary movement processes.

Given the strong theoretical relationships between lateralized autonomic and hemispheric function and between the neurons that control RSA and the neurons that control facial expression and vocal intonation (see Figure 5), research should be directed at evaluating the relationship between RSA and the primary emotions. Recall that the source nucleus of the facial nerve is the border of NA and afferents from the trigeminal nerve provide a primary sensory input to NA. Thus, the Ventral Vagal Complex consisting of NA and the nuclei of the trigeminal and facial nerves is clearly related to the expression and experience of emotion.

Based upon the Poly-Vagal Theory, one would expect shifts in affective state to parallel RSA. For example, the elicitation of a negative primary emotion would result in a systematic withdrawal of vagal tone via NA to promote fight and flight like behaviors. In contrast, a shift to a more pleasant affective state would be associated with an increase in RSA. A study by Bazhenova (1995), emphasizing the dynamics of RSA change during the shifting affective states, supports this speculation. Bazhenova manipulated the affective state of infants and demonstrated that when an infant shifted to a more negative affective state, RSA decreased. Moreover, when the infant shifted to a more positive affective state, RSA increased above the affectively neutral baselevel.

The Poly-Vagal Theory does not neglect the important role of DMNX in the emotional experience. For example, DMNX is critical in the regulation of digestive polypeptides and gastric motility (Uvnas-Moberg, 1989), dimensions of physiological activity that parallel emotive experiences and stress. Consistent with the Poly-Vagal Theory, which emphasizes the importance of NA and VVC in overt emotional expressiveness and regulation, the Theory would acknowledge the importance of less conscious survival oriented processes that are mediated via Dorsal Vagal Complex consisting of NTS and DMNX. A complementary theory has been proposed by Uvnas-Moberg (Uvnas-Moberg, 1987, 1994). The Uvnas-Moberg theory emphasizes the role of DMNX in the regulation of gastrointestinal hormones and during emotional states including stress, hunger and satiety.

Summary and Conclusion

The following seven points summarize the Poly-Vagal Theory.

1. The vagal system does not represent a unitary dimension. The vagal system includes general visceral efferent fibers regulating smooth and cardiac muscle, as well special visceral efferent fibers regulating the somatic muscles of the larynx, pharynx, and esophagus. These somatic muscles control vocalization, sucking and swallowing and interface these processes with breathing. The vagal system also is linked neuroanatomically to the source nuclei that control facial expression, mastication, and head turning.

2. There are two vagal motor systems. One vagal system is the vegetative vagus, which originates in the dorsal motor nucleus and is associated with passive reflexive regulation of visceral functions. The other vagal system is the smart vagus, which originates in NA and is associated with the active processes of attention, motion, emotion, and communication. The two systems are neuroanatomically distinct, have different ontogenetic and phylogenetic origins, and employ different adaptive strategies.

3. In mammals the concept that vagal tone represents a single or summed system may have limited physiological or heuristic value. For example, in mammals high tone from the dorsal motor nucleus vagal system may be lethal, while, high tone from the NA-vagal system may be beneficial. Based upon the proposed Poly-Vagal Theory, an accurate measure of the NA-system is critical to the evaluation of psychophysiological relationships.

4. The functional output of the NA-vagus on the heart may be monitored via RSA. NA is part of a common neuronal network producing a cardiorespiratory rhythm. Thus, the output from the branch of the vagus originating in NA and terminating on the sino-atrial node of the heart conveys a frequency common to both respiratory and cardiac systems. In contrast, the output from the dorsal motor nucleus does not convey a respiratory rhythm.

5. The magnitude of neurogenic bradycardia is mediated by the dorsal motor nucleus. Rapid heart rate changes, such as conditioned anticipatory heart rate deceleration and decelerations associated with orienting, are neurogenic bradycardia. Additional neurogenic bradycardia are reflexes such as opto-vagal and chemo-vagal. In the absence of NA influences to the sino-atrial node, local conditions such as hypoxia may greatly potentiate the vagal effect.

6. There is a common cardiopulmonary oscillator. The common respiratory rhythm observed in heart rate and breathing is produced by a network of interneurons located in NTS and NA, which communicate with the motor neurons that control respiratory, laryngeal, and cardiac function.

7. Primary emotions are related to autonomic function. Since the primary emotions are often survival related, they must be integrated into cardiopulmonary regulation. Moreover, primary emotions have a right hemisphere bias, ipsilateral with the regulatory bias of the medullary structures controlling visceral function.

Based upon the Poly-Vagal Theory, additional hypotheses may now be tested evaluating the relationship between RSA (the measure of NA vagal tone) and processes and states dependent upon the coordination of cardiopulmonary processes with the special visceral efferents of the cranial nerves. This, of course, includes all processes associated with vocalizations, feeding, breathing, and facial expression.

In developing the Poly-Vagal Theory, the most striking insights come from the phylogenetic approach. Not only does a phylogenetic approach explain the vagal paradox in terms of the medullary source nuclei of the dorsal motor nucleus and NA, but it highlights the importance of oxygen needs in the evolving nervous system. As the nervous system gets more complex, there are greater demands for oxygen. Oxygen needs may have provided a major environmental pressure leading to the evolution of the adaptive and sophisticated autonomic nervous system found in mammals. Thus, constructs such as orienting, attention, emotion and stress are by-products of the evolutionary pressure to optimize oxygen resources.

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Author Notes

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Figure Captions

Figure 1. Primary brainstem nuclei of the vagus. Nuclei are bilateral and only one of each bilateral pair is illustrated.

Figure 2. (a) Bradycardia during fetal distress. (b) Background heart period variability at time of bradycardia.

Figure 3. Bradycardia elicited by electrical stimulation of the DMNX in an anesthetized rabbit.

Figure 4. Bradycardia elicited by aortic depressor nerve stimulation in an anesthetized rabbit.

Figure 5. Topographic organization of the nucleus ambiguus in the rat (Bieger & Hopkins, 1987).

Appendix A

Title of the paper.

The title was selected to emphasize the concept that evolutionary processes have sculpted the neural regulation of autonomic function. Not only has evolution provided obvious divergences in behavior and appearance, but evolution has impacted on the autonomic strategies related to the detection of novelty in the environment. The focus of the paper was not directed at a theory of orienting, nor has the paper attempted to distinguish between the autonomic components of orienting or defensive reflexes. Rather, the paper has focused on the neurogenic regulation of cardiac responses via two vagal responses systems. A primitive system that we have inherited from reptiles produces a rapid neurogenic bradycardia that reduces the activity of our cardiopulmonary system to conserve oxygen. This is the strategy of sit and wait feeders, common to reptiles. In contrast, the evolution of the energy-demanding mammal required two autonomic-behavioral shifts: 1) mammals needed to obtain great amounts of food; and 2) mammals needed to protect their nervous systems from oxygen loss. These two objectives are linked. In the evolution

of mammals, success in obtaining food resources was dependent upon the ability to detect threat. Thus, mobilization and attention became two important behavioral dimensions. Unlike reptiles, which orient in response to novelty and attack or return to a quiescent state or lumber off, mammals orient and then attend. Following this phase of attention a mammal may rapidly depart or approach (attack) within the context of the classic fight or flight response. With the increasing complexity of behavior, there is a parallel increase in complexity in the organization and function of the autonomic nervous system. The title also intended to emphasize the concept that evolution has placed mammals in a defensive world. The survival systems of reptiles and other lower phyla can be organized into orienting and defensive dimensions. Mammals, to survive in this defensive and reactive world, had to circumvent these potentially lethal reactions of other species. The evolution of the mammalian nervous system enables mammals to rapidly escape danger and to use neural resources for the complex information processes required to detect subtleties in the environment. Moreover, evolution promoted additional motor systems related to communication. Motor systems developed to communicate conditions related to survival with facial expressions and vocalizations associated with primary emotions. The evolutionary modifications not only had to coexist with the oxygen hungry metabolic system, but by increasing the complexity of motor behaviors, there was an additional increase in oxygen needs. Thus, there is a link between the special visceral efferent actions regulating the communicative processes of emotion, and later language, with the general visceral efferent actions regulating cardiopulmonary function. The ability to detect subtleties in the environment coupled with the ability to communicate threat or comfort via facial expressions and vocalizations contributed to within- species social behavior, parenting, and pairbonding. These complex functions evolved while the demanding oxygen needs of mammals were programmed into the background of nervous system function via the autonomic nervous system.

Appendix B

Personal retrospective

In discussing any theoretical perspective, it is important to place the ideas and speculations in the context of earlier research conducted by the investigator. My early research focused on the use of heart rate measures as indicators of attention. While conducting research for my master's thesis (Porges & Raskin, 1969), I noted that attention demanding tasks produced heart rate response patterns with two prominent characteristics. First, heart rate exhibited a rapid transitory directional change in response to task onset and stimulus changes. Second, when subjects became involved in the task and focused their attention on the task demands, heart rate variability was reduced. I was intrigued with these observations and speculated on the possible physiological mechanisms. This evolved into a two-component theory of attention in which the components were labeled "phasic or orienting" and "tonic or attention" responses (Porges, 1972). These findings stimulated me to investigate neural mechanisms of heart rate regulation and to develop the Vagal

Tone Index () of RSA, which I believed would help provide insight into the mechanisms mediating the more tonic sustained attention response. The preceding sections of this paper provide the basis for the Poly-Vagal Theory and enable an interpretation of the two heart rate components associated with attention. The first component, associated with orienting and the neurogenic bradycardia, is determined reflexively by the vegetative vagus, originating in the dorsal motor nucleus. The second component, associated with voluntary engagement with the environment and depression of RSA, is determined by the smart vagus, originating in the NA. Thus, after years of studying heart rate patterns, a speculative two-component psychophysiological model of attention is evolving into the neuroanatomically and neurophysiologically based Poly-Vagal Theory.