Emotion: An Evolutionary By-Product of the Neural Regulation of the Autonomic Nervous System

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Stephen W. Porges

Institute for Child Study
University of Maryland
College Park, Maryland 20742-1131

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INTRODUCTION

A new theory, the Polyvagal Theory of Emotion, is presented which links the evolution of the autonomic nervous system to affective experience, emotional expression, vocal communication and contingent social behavior. The Polyvagal Theory is derived from the well-documented phylogenetic shift in the neural regulation of the autonomic nervous system that expands the capacity of the organism to control metabolic output. The Theory emphasizes the phylogenetic dependence of the structure and function of the vagus, the primary nerve of the parasympathetic nervous system. Three phylogenetic stages of neural development are described. The first stage is characterized by a primitive unmyelinated vegetative vagal system that fosters digestion and responds to novelty or threat by reducing cardiac output to protect metabolic resources. Behaviorally, this first stage is associated with immobilization behaviors. The second stage is characterized by a spinal sympathetic nervous system that is capable of increasing metabolic output and inhibiting the primitive vagal system's influence on the gut to foster mobilization behaviors necessary for "fight or flight." The third stage is unique to mammals and is characterized by a myelinated vagal system that can rapidly regulate cardiac output to foster engagement and disengagement with the environment. The myelinated vagus originates in a brainstem area that evolved from the primitive gill arches and in mammals controls facial expression, sucking, swallowing, breathing, and vocalization. It is hypothesized that the mammalian vagal system fosters early mother-infant interactions and serves as the substrate for the development of complex social

behaviors. In addition, the mammalian vagal system has an inhibitory effect on sympathetic pathways to the heart, and thus, promotes calm behavior and pro-social behavior.

The Polyvagal Theory of Emotion proposes that the evolution of the autonomic nervous system provides the organizing principle to interpret the adaptive significance of affective processes. The Theory proposes that the evolution of the mammalian autonomic nervous system, and specifically the brainstem regulatory centers of the vagus and other related cranial nerves, provides substrates for emotional experiences and affective processes that are necessary for social behavior in mammals. In this context, the evolution of the nervous system limits or expands the ability to express emotions, which in turn may determine proximity, social contact, and the quality of communication. The polyvagal construct has been previously introduced1 to document the neurophysiological and neuroanatomical distinction between the two vagal branches and to propose their unique relation with behavioral strategies. This paper elaborates on the polyvagal construct and proposes that affective strategies are derivative of the evolutionary process that produced the polyvagal regulation.

There is a consensus that affect is expressed in facial muscles and in organs regulated by the autonomic nervous system. However, with the exception of work by Cannon,2,3 which focused on the sympathetic-adrenal system as the physiological substrate of emotion, the presumed neural regulation of affective state has not been investigated. Even contemporary researchers investigating affective signatures in the autonomic nervous system4-7 have tacitly accepted Cannon's assumption that emotions reflect responses of the sympathetic nervous system.

Unlike the architectural dictum that form (i.e., structure) follows function, the function of the nervous system is derivative of structure. The flexibility or variability of autonomic nervous system function is totally dependent upon the structure. By mapping the phylogenetic development of the structures regulating autonomic function, it is possible to observe the dependence of autonomic reactivity on the evolution of the underlying structure of the nervous system. The phylogenetic approach highlights a shift in brainstem and cranial nerve morphology and function from an oxygen-sensitive system (i.e., the primitive gill arches) to a system that regulates facial muscles, cardiac output and the vocal apparatus for affective communication.

CANNON'S BLUNDER

Cannon emphasized the idea that emotions were expressions of sympathetic-adrenal excitation. In limiting emotional experiences solely to the mobilization responses associated with sympathetic-adrenal activity, Cannon denied the importance of visceral feelings and neglected the contribution of the parasympathetic nervous system. Cannon's views were not compatible with earlier statements regarding the importance of visceral feedback and the parasympathetic nervous system. For example in The Expression of Emotions in Man and Animals, Darwin8 acknowledged the importance of the

bidirectional neural communication between the heart and the brain via the "pneumogastric" nerve. This, the 10th cranial nerve, is now called the vagus nerve and is the major component of the parasympathetic nervous system.

> ... 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 and felt to be the case. Claude Bernard also repeatedly insists, and this deserves especial notice, that 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, emotional state represented a covariation between facial expression and autonomic tone. However, he did not elucidate the specific neurophysiological mechanisms. Our current knowledge of the neuroanatomy, embryology, and phylogeny of the nervous system were not available to Darwin. At that time it was not known that vagal fibers originated in several medullary nuclei, that branches of the vagus exerted control over the periphery through different feedback systems, and that the function of the branches of the vagus followed a phylogenetic principle. However, Darwin's statement is important, because it emphasizes 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 vagus in the expression of emotions.

The autonomic nervous system is related to visceral state regulation and the regulation of behaviors associated with mobilization or immobilization. For example, sympathetic excitation is clearly linked to mobilization. In vertebrates, the sympathetic nervous system is characterized by a trunk or column of ganglia parallelling the segmentation of the spinal cord. Skeletal motor pathways to the limbs are paralleled by sympathetic fibers to facilitate the metabolically demanding behaviors related to fight and flight. In fact, from Cannon's perspective and to many who followed, the sympathetic nervous system due to its mobilizing capacity was the component of the autonomic nervous system associated with emotion. This, however, neglected the autonomic components of affective experiences that were metabolically conservative, including processes such as signalling via facial expressions and vocalizations or specific immobilization responses.

AUTONOMIC DETERMINANTS OF EMOTION

Over the past 100 years we have learned much about the autonomic nervous system, its evolutionary origins and how it relates to emotion. Initially, we can distinguish among three components of the autonomic nervous system (i.e., visceral afferents, sympathetic nervous system, and parasympathetic nervous system) and speculate how each might be related to affective experiences. First, the visceral afferents may be assumed to play a major role in determining "feelings." These mechanisms, which provide us with

knowledge of hunger, also may convey a sense of nausea during emotional distress. We frequently hear subjective reports of individuals feeling "sick to their stomach" during periods of severe emotional strain associated with profound negative experiences. Similarly, negative states have been associated with reports of breathlessness or feelings that the heart has stopped. Second, the sympathetic nervous system and adrenal activity are associated with mobilization. Activation of the sympathetic nervous system is usually linked to increased skeletal movement of the major limbs. Thus, consistent with Cannon, the sympathetic nervous system provides the metabolic resources required for fight or flight behaviors. The sympathetic nervous system enhances mobilization by increasing cardiac output and decreasing the metabolic demands of the digestive tract by actively inhibiting gastric motility. Third, as proposed by Darwin and Bernard, the parasympathetic nervous system and specifically the vagus are related to emotional state. Few researchers have investigated the link between parasympathetic activity and affective state. However, over the past decade my laboratory has focused on this issue. We have documented that vagal tone, a component of parasympathetic control, is related to affect and affect regulation.9-11 We have presented theoretical models explaining the importance of vagal regulation in the development of appropriate social behavior.12 In general, the parasympathetic nervous system is associated with fostering growth and restoration.13,14 Moreover, knowledge of the polyvagal system allow an appreciation of the importance of the brainstem origin of the specific vagal fibers in the determination of affective and behavioral response strategies.12,15

Researchers and clinicians have had difficulties in the organization or categorization of intensive affective states that appear to have totally different etiologies or behavioral expressions. For example, intense feelings of terror might result in a total immobilization or freezing. In contrast, intense feelings of anger or anxiety might be associated with massive mobilization activity. This problem exists, in part, because of a bias toward explanations of affective states defined in terms of either overt behaviors such as facial expressions (i.e., following Darwin) or sympathetic activity (i.e., following Cannon). The emphasis on sympathetic activity is based upon three historical factors. First, theories regarding emotions have minimized or totally neglected the parasympathetic nervous system. Second, Cannon's focus on the sympathetic efferents and mobilization responses associated with fight and flight as the sole domain of autonomic reactivity during emotional states has not been challenged. Third, the data base of autonomic correlates of affect, collected to identify autonomic "signatures" of specific affective states, is dominated by measures assumed to be related to sympathetic function.4-7

THE EVOLUTION OF THE AUTONOMIC NERVOUS SYSTEM: EMERGENT STRUCTURES FOR THE EXPRESSION OF EMOTIONS IN MAN AND ANIMALS

Although there is an acceptance that the autonomic nervous system and the face play a role in emotional expression, there is great uncertainty regarding the

autonomic "signature" of specific or discrete emotions. Most researchers evaluating autonomic responses during affective experiences, assumed, as did Cannon, that the sympathetic nervous system was the determinant of emotion, or at least the primary physiological covariate of emotion. This, of course, neglects the potential role of the parasympathetic nervous system and its neurophysiological affinity to facial structures including facial muscles, eye movements, pupil dilation, salivation, swallowing, vocalizing, hearing and breathing. By investigating the evolution of the autonomic nervous system, we may gain insight into the interface between autonomic function and facial expression. In the following sections the phylogenetic development of the autonomic nervous system will be used as an organizing principle to categorize affective experiences.

The Polyvagal Theory of Emotion is derived from investigations of the evolution of the autonomic nervous system. The Theory includes several rules and assumptions.
1. Emotion is dependent upon the communication between the autonomic nervous system and the brain; visceral afferents convey information regarding physiological state to the brain and are critical to the sensory or psychological experience of emotion, and cranial nerves and the sympathetic nervous system are outputs from the brain that provide somatomotor and visceromotor control of the expression of emotion.
2. Evolution has modified the structures of the autonomic nervous system.
3. Emotional experience and expression are functional derivatives of structural changes in the autonomic nervous system due to evolutionary processes.
4. The mammalian autonomic nervous system retains vestiges of phylogenetically older autonomic nervous systems.
5. The phylogenetic "level" of the autonomic nervous system determines affective states and the range of social behavior.
6. In mammals, the autonomic nervous system response strategy to challenge follows a phylogenetic hierarchy, starting with the newest structures and, when all else fails, reverting to the most primitive structural system.

This paper will focus on the phylogenetic shift in the neural regulation of the vertebrate heart. The heart has been selected because, in response to environmental challenge, cardiac output must be regulated to mobilize for fight or flight behaviors, or to immobilize for death feigning or hiding behaviors. To regulate cardiac output several efferent structures have evolved. These structures represent two opposing systems: one, a sympathetic-catecholamine system including chromaffin tissue and spinal sympathetics; and two, a vagal system (a component of the parasympathetic nervous system) with branches originating in medullary source nuclei (i.e., dorsal motor nucleus of the vagus and nucleus ambiguus). In addition, vertebrate have chromaffin tissue containing high concentrations of catecholamines. The chromaffin tissue is defined as having morphological and histochemical properties similar to the adrenal medulla. Classes of vertebrate that do not have an adrenal medulla have relatively more chromaffin tissue, which regulates circulating catecholamines.

Insert Figure 1 about here

Figure 1 lists the regulatory structures that influence the heart in vertebrates.16-18 Two phylogenetic principles can be extracted from Figure 1. First, there is a phylogenetic pattern in the regulation of the heart from endocrine communication, to unmyelinated nerves, and finally to myelinated nerves. Second, there is a development of opposing neural mechanisms of excitation and inhibition to provide rapid regulation of graded metabolic output.

In the most primitive fish, the cyclostomes, the neural control of the heart is very primitive. Some cyclostomes such as the myxinoids (hagfish) use circulating catecholamines from chromaffin tissue to provide the sole excitatory influences on the heart. Other cyclostomes such as the lampetroids (lampreys) have a cardiac vagus. However, in contrast to all other vertebrates that have a cardio-inhibitory vagus that act via muscarinic cholinoceptors, the cyclostome vagal innervation is excitatory and acts via nicotinic cholinoceptors. One striking feature of the cyclostome heart is the location of chromaffin tissue within the heart that stores large quantities of epinephrine and norepinephrine. As in other vertebrates, the circulating catecholamines produced by the chromaffin tissue stimulate beta-adrenergic receptors in the heart. Thus, for the cyclostomes there appear to be only excitatory mechanisms to regulate the heart.

The elasmobranchs (cartilaginous fish) are the first vertebrates to have a cardioinhibitory vagus. The vagus in these fish is inhibitory and the cholinoceptors on the heart are muscarinic as they are in other vertebrates. The cardioinhibitory vagus is functional in the elasmobranchs as a response to hypoxia. In conditions of hypoxia, the metabolic output is adjusted by reducing heart rate. This modification of neural regulation may provide a mechanism to enable the elasmobranchs to increase their territorial range, by providing a neural mechanism that adjusts metabolic output to deal with changes in water temperature and oxygen availability. However, unlike more evolutionarily advanced fish or tetrapods, the elasmobranchs do not have direct sympathetic input to the heart. Instead, cardiac acceleration and increases in contractility are mediated via beta-adrenergic receptors stimulated by circulating catecholamines released from chromaffin tissue. Thus, since activation of metabolic output is driven by circulating catecholamines and not by direct neural innervation, once the excitatory system is triggered, the ability to self-sooth or calm is limited.

In vertebrates with sympathetic and vagal neural innervation, vagal influences to the sino-atrial node serve to inhibit or dampen the sympathetic influence and promote rapid decreases in metabolic output19 that enable almost instantaneous shifts in behavioral state. As a whole the teleosts may be considered phylogenetically the first class of vertebrates in which there is both sympathetic and parasympathetic neural control of the heart, with innervation similar to that found in tetrapods. This enables rapid

transitory changes in metabolic output permitting changes from mobilization to immobilization. This is observed as "darting" and "freezing" behaviors. Amphibia, similar to the teleosts, have dual innervation of the heart via systems with direct neural components from the spinal cord via the sympathetic chain producing increases in heart rate and contractility, and direct neural pathways from the brainstem via the vagus producing cardioinhibitory actions.

True adrenal glands, in which there is a distinct medulla formed of chromaffin tissue, are only present in birds, reptiles and mammals.16 Neural regulation by the spinal sympathetics of the adrenal medulla provides a neural mechanism for rapid and controlled release of epinephrine and norepinephrine to stimulate cardiovascular function. In the teleosts, chromaffin tissue is primarily related to parts of the cardiovascular system, but there also is chromaffin tissue associated with the kidney. However, in the amphibia chromaffin tissue is primarily associated with the kidney and substantial aggregations of chromaffin cells are located along the sympathetic chain ganglia. Thus, we can observe a phylogenetic shift in the location of chromaffin tissue, and the concurrent evolution of a distinct adrenal medulla near the kidney.

In mammals the morphology of the vagus changes.1 Unlike all other vertebrates with cardioinhibitory vagi, the mammalian vagus contains two branches. One branch originates in the dorsal motor nucleus of the vagus and provides the primary neural regulation of subdiaphragmatic organs such as the digestive tract. However, at the level of the heart, the Dorsal Motor Nucleus of the Vagus does not play a major role in the normal dynamic regulation of cardiac output. Rather, during embryological development in mammals, cells from the Dorsal Motor Nucleus of Vagus migrate ventrally and laterally to the Nucleus Ambiguus.20 There they form the cell bodies for visceromotor myelinated axons that provide potent inhibition of the sino-atrial node, the pacemaker for the heart.

By transitory down-regulation of the cardioinhibitory vagal tone to the heart (i.e., removing the vagal brake), the mammal is capable of rapid increases in cardiac output without activating the sympathetic-adrenal system. By engaging this system, rather than the sympathetic-adrenal system, mammals have the opportunity to rapidly increase metabolic output for immediate mobilization. Under prolonged challenge, the sympathetic system also may be activated. However, by rapidly re-engaging the vagal system, mammals have the capacity to inhibit sympathetic input on the heart19 and rapidly decrease metabolic output to self-soothe and calm.

PHYLOGENETIC DEVELOPMENT OF THE AUTONOMIC NERVOUS SYSTEM: AN ORGANIZING PRINCIPLE FOR HUMAN EMOTION

Inspection of Figure 1, which summarizes the primary regulatory structures of the heart in vertebrates, provides a basis for speculations regarding the behavioral repertoire of various classes of vertebrates. These speculations support the premise that the phylogenetic development of the

autonomic nervous system provides an organizing principle for affective experiences and determines the limits on social behavior and, therefore, the possibility of affiliation. In general, phylogenetic development results in increased neural control of the heart via mechanisms that can rapidly increase or decrease metabolic output. This phylogenetic course results in greater central nervous system regulation of behavior, especially behaviors to engage and disengage with environmental challenges.

To further focus on the impact of phylogenetic development of the neural regulation of the autonomic nervous system, we can observe five phylogenetic-dependent response systems.

1. A chemical excitatory system via the catecholamine-rich chromaffin tissue to increase cardiac output and to support mobilization.

2. An inhibitory vagal system via the Dorsal Motor Nucleus of the Vagus to reduce cardiac output when metabolic resources are scarce and to support immobilization in response to danger.
3. A spinal sympathetic nervous system to provide neural excitation to promote rapid mobilization for behaviors associated with fight and flight.
4. A neurally regulated adrenal medulla system to provide more direct control over the release of circulating catecholamines to support mobilization for the prolonged metabolic requirements of fight or flight behaviors.
5. The specialization of the mammalian vagal system into a "tonic" inhibitory system that allows graded withdrawal of the vagal brake, which can promote transitory mobilization and the expression of sympathetic tone without requiring sympathetic or adrenal activation. With this new vagal system, transitory incursions into the environment can be initiated without the severe biological prices of either metabolic shut down, via primitive vagal inhibition, or metabolic excitation, via sympathetic-adrenal activation.

The five phylogenetic-dependent response systems are associated with three neuroanatomical constructs related to affective experience and expression: 1) Dorsal Vagal Complex (DVC), 2) Sympathetic Nervous System (SNS), and 3) Ventral Vagal Complex (VVC). Each of these three neural constructs is linked with a specific emotion subsystem observable in humans. Each emotion subsystem is manifested via differentiated motor output from the central nervous system to perform specific adaptive functions: to immobilize and conserve metabolic resources, to mobilize in order to obtain metabolic resources, or to signal with minimal energy expense. The constituent responses associated with each subsystem is listed in Figure 2.

Insert Figure 2 about here

THE DORSAL VAGAL COMPLEX: A VESTIGIAL IMMOBILIZATION SYSTEM.

The Dorsal Vagal Complex (DVC) is primarily associated

with digestive, taste, and hypoxic responses in mammals. It includes the Nucleus Tractus Solitarius (NTS) and the interneuronal communication between the NTS and Dorsal Motor Nucleus of the Vagus (DMX). The efferents for the DVC originate in the DMX and primary vagal afferents terminate in the NTS. The DVC provides the primary neural control of subdiaphragmatic visceral organs. It provides low tonic influences on the heart and bronchi. This low tonic influence is the vestige from the reptilian vagal control of the heart and lung. In contrast to reptiles, mammals have a great demand for oxygen and are vulnerable to any depletion in oxygen resources. The metabolic demand for mammals is approximately five times greater than for reptiles of equivalent body weight.21 Thus, reptilian dependence on this system provides a shut-down of metabolic activity to conserve resources during diving or death feigning. The DVC provides inhibitory input to the sino-atrial node of the heart via unmyelinated fibers and thus, is less tightly controlled than the myelinated fibers from the VVC. Hypoxia or perceived loss of oxygen resources appear to be the main stimuli that trigger the DVC. Once triggered, severe bradycardia and apnea are observed, often in the presence of defecation. This response strategy is observed in the hypoxic human fetus. Although adaptive for the reptile, the hypoxic triggering of this system may be lethal for mammals. In addition, it is important to note that the DVC has beneficial functions in humans. Under most normal conditions, the DVC maintains tone to the gut and promotes digestive processes. However, if up-regulated, the DVC contributes to pathophysiological conditions including the formation of ulcers via excess gastric secretion and colitis. Recent research supports the importance of the unmyelinated vagal fibers in bradycardia22 and suggests the possibility that massive bradycardia may be determined by the unmyelinated vagal fibers associated with the DVC recruiting myelinated vagal fibers to maximize the final vagal surge on the heart.23

THE SYMPATHETIC NERVOUS SYSTEM: ADAPTIVE MOBILIZATION SYSTEM FOR FIGHT OR FLIGHT BEHAVIORS.

The sympathetic nervous system is primarily a system of mobilization. It prepares the body for emergency by increasing cardiac output, stimulating sweat glands to protect and lubricate the skin, and by inhibiting the metabolically costly gastrointestinal tract. The evolution of the sympathetic nervous system follows the segmentation of the spinal cord, with cell bodies of the preganglionic sympathetic motor neurons located in the lateral horn of the spinal cord. The sympathetic nervous system has long been associated with emotion. The label "sympathetic" reflects the historical identity of this system as a nervous system "with feelings" and contrasts it with the parasympathetic nervous system, a label that reflects a nervous system that "guards against feelings."

THE VENTRAL VAGAL COMPLEX: THE MAMMALIAN SIGNALLING SYSTEM FOR MOTION, EMOTION, AND COMMUNICATION

The primary efferent fibers of the Ventral Vagal Complex (VVC) originate in the nucleus ambiguus. The primary

afferent fibers of the VVC terminate in the source nuclei of the facial and trigeminal nerves. The VVC has the primary control of supradiaphragmatic visceral organs including the larynx, pharynx, bronchi, esophagus, and heart. Motor pathways from the VVC to visceromotor organs (e.g., heart and bronchi) and somatomotor structures (e.g., larynx, pharynx, esophagus) are myelinated to provide tight control and speed in responding. In mammals, the visceromotor fibers to the heart express high levels of tonic control and are capable of rapid shifts in cardioinhibitory tone to provide dynamic changes in metabolic output to match environmental challenges. This rapid regulation characterizes the qualities of the mammalian vagal brake that enable rapid engagement and disengagement in the environment without mobilizing the sympathetics.

A major characteristic of the VVC is the fact that the neural fibers regulating somatomotor structures are derived from the branchial or primitive gill arches that evolved to form cranial nerves V, VII, IX, X, and XI. Somatomotor fibers originating in these cranial nerves control the branchiomeric muscles including facial muscles, muscles of mastication, neck muscles, larynx, pharynx, esophagus, and middle ear muscles. Visceromotor efferent fibers control salivary and lacrimal glands, as well as the heart and bronchi. The primary afferents to the VVC come from facial and oral afferents traveling through the facial and trigeminal nerves and the visceral afferents terminating in the nucleus tractus solitarius (NTS). The VVC is involved in the control and coordination of sucking, swallowing, and vocalizing with breathing.

EVOLUTION AND DISSOLUTION: HIERARCHICAL RESPONSE STRATEGY

The evolution of the autonomic nervous system provides substrates for the emergence of three emotion systems. This phylogenetic adjustment of the autonomic nervous system represents an exaptation (see Crews this volume) of structures to express emotions that initially evolved in primitive vertebrates to extract oxygen from water, to oxygenate and transport blood, and to adjust metabolic output to match resources. The Polyvagal Theory of Emotion is based on a phylogenetic model. The Polyvagal Theory of Emotion proposes a hierarchical response strategy to challenge, with the most recent modifications employed first and the most primitive last. This phylogenetic strategy can be observed in our day-to-day interactions. Our social behavior follows a strategy that focuses initially on communication via facial expressions and vocalizations. This strategy has low metabolic demand and, if appropriately interpreted, results in contingent social interactions via verbal-facial mechanisms. Often, hand gestures and head movements contribute to increase the mammalian repertoire of communication-related behavior. An important characteristic of these prosocial behaviors is their low metabolic demand and the rapid contingent "switching" of transitory engagement to transitory disengagement strategies (i.e., speaking then switching to listening).

This phylogenetically based hierarchical response strategy is consistent with the concept of dissolution proposed by John Hughlings Jackson24 to explain diseases of

the nervous system. Jackson proposed that "the higher nervous arrangements inhibit (or control) the lower, and thus, when the higher are suddenly rendered functionless, the lower rise in activity." This is observed in the Polyvagal Theory of Emotion, not in terms of disease, but in terms of response strategies to differential challenges to survival. The VVC with its mechanisms of "signalling" and "communication" provide the initial response to the environment. The VVC inhibits, at the level of the heart, the strong mobilization responses of the sympathetic nervous system. Withdrawal of VVC, consistent with Jackson's model, results in a "disinhibition" of the sympathetic control of the heart. Similarly, withdrawal of sympathetic tone results in a "disinhibition" of the DVC control of the gastrointestinal tract and a vulnerability of the bronchi and heart. There are several clinical consequences to unopposed DVC control including defecation, due to a relaxation of the sphincter muscles and increased motility of the digestive tract, and apnea, due to constriction of the bronchi, and bradycardia, due to stimulation of the sino-atrial node. Thus, when all else fails, the nervous system elects a metabolically conservative course that is adaptive for primitive vertebrates, but lethal to mammals. Consistent with the Jacksonian principle of dissolution, specific psychopathologies defined by affective dysfunction may be associated with autonomic correlates consistent with the three phylogenetic levels of autonomic regulation. The three levels do not function in an all-or-none fashion; rather they exhibit gradations of control determined by both visceral feedback and higher brain structures.

UNVEILING DARWIN

Contemporary research and theory on emotion owes much to Darwin and his volume, The Expression of the Emotions in Man and Animals.8 Through careful and astute observations of facial expressions, Darwin insightfully interpreted emotional expressions within an evolutionary model of adaptation and natural selection. However, Darwin's knowledge of neurophysiology and neuroanatomy was limited. In contrast to Darwin's creative insights into the adaptive function of facial expression, his understanding of underlying physiological mechanisms and the linkage between facial muscles and emotion was synthetic and derivative. He repeatedly referenced the 1844 edition of Anatomy and Philosophy of Expressions written by Sir Charles Bell for physiological explanations of facial expression. As further support for the importance of facial muscles in emotional expressions, Darwin incorporated the work of Duchenne in his text. Duchenne conducted experiments by electrically stimulating the face of humans. Electrical stimulation of selected facial muscles provided expressions that were readily perceived as different emotional states.

In contrast to the Polyvagal Theory of Emotion, which uses the evolution of the autonomic nervous system as the primary organizing principle for the expression and experience of affect, Darwin did not emphasize the importance of the nervous system as a structure involved in the evolution of emotion. Rather he focused on affect as a functional system that responded to the determinants of

evolution to produce the facial and vocal expressions of human emotion. Darwin neglected the importance of treating the nervous system as a structure that is vulnerable to the pressures of evolution. A choice between investigating affect as a functional behavioral system or investigating the structural determinants of affect (i.e., nervous system) was clearly made by researchers who followed Darwin. This research tradition followed the observational approach of organizing facial expression into affective categories. Although there have been investigations into the physiological correlates of affect and facial expression,6,7,25 these investigations were on a psychophysiological or correlative level and did not emphasize specific neural regulatory processes.

Consistent with the observational approach, Tomkins26,27 developed a theory of affect that emphasized the importance of the face, not only as a structure of communication, but also as a structure of self-feedback. Following Tomkins, Ekman28 and Izard29 developed detailed coding systems for facial affect and have used these methods to study individual differences, developmental shifts, and the cross-cultural consistency of human facial expression.

Several contemporary theories of emotion have focused on facial expressions, in a manner similar to that initially presented by Darwin. Rather than incorporating knowledge of neural regulation of the face or the evolution of the neural regulation of autonomic function, researchers and theorists have attempted to organize information in terms of the functional significance of sequences or patterns of facial expressions. This difficult task, modelled on Darwin, often becomes bogged down in semantics, philosophical inconsistencies, and circularity. Darwin in his descriptions of emotions speculated and provided hypothetical examples of natural selection contributing to the uniqueness of species-specific affective response patterns. However, the terms selected to characterize specific emotions often vary from culture to culture. Tomkins, and later Ekman and Izard, promoted the description of affective experiences in terms of the specific facial muscles or groups of muscles involved in the facial expression. However, they then used subjective reports to label these facial expressions.

We may "unveil" Darwin by investigating the neural regulation that underlies facial expression. Facial expressions are controlled by cranial nerves. Motor pathways from the trigeminal nerve (V) control the muscles of mastication with branches to the temporalis, masseter, medial, and lateral pterygoid muscles. Motor pathways from the facial nerve (VII) control the muscles of facial expression including zygomaticus, frontalis, orbicularis oculi, elevators, orbicularis oris, depressors and platysma. Nucleus ambiguus serves as the source of cell bodies for motor pathways traveling through several cranial nerves including the glossopharyngeal (IX), vagus (X), and accessory nerves (XI). The pathways from the glossopharyngeal nerve regulate pharyngeal muscles. Pathways from the vagus regulate the muscles of the pharynx and larynx, and the pathways of the accessory nerve control the neck muscles allowing rotation and tilting of the head. These cranial

nerves are derivative from the primitive gill arches30,31 and may be collectively described as the Ventral Vagal Complex. Thus, the evolutionary origins (i.e., primitive gill arches) of the somatomotor pathways traveling through these cranial nerves provide us with an organizing principle to understand affective expressions. In addition to the above neural regulation of somatomotor structures, these branchiomeric (i.e., derived from the primitive arches) cranial nerves also regulate visceromotor processes associated with salivation, tearing, breathing, and heart rate.

Other cranial nerves contribute to the expression of emotions. The hypoglossal nerve (XII) innervates the muscles of the tongue. The trochlear (IV), abducens (VI) and oculomotor (III) nerves innervate muscles to provide movements of the eyes and eyelids. Thus, the facial expressions observed by Darwin, detailed by Tomkins and coded by Ekman and Izard are a direct reflection of the regulation of the face by the cranial nerves.

VOODOO OR VAGUS DEATH?: THE TEST OF THE POLYVAGAL THEORY

The Polyvagal Theory of Emotion provides a theoretical framework to interpret the phenomenon of Voodoo or fright death described by Cannon32 and Richter.33 Cannon believed that extreme emotional stress, regardless of the specific behavioral manifestation, could be explained in terms of degree of sympathetic-adrenal excitation. In 1942 Cannon described a phenomenon known as Voodoo death. Voodoo death was assumed to be directly attributable to emotional stress. Being wed to a sympatho-adrenal model of emotional experience (see above), Cannon assumed that Voodoo death would be the consequence of the state of shock produced by the continuous outpouring of epinephrine via excitation of the sympathetic nervous system. According to the Cannon model, the victim would be expected to breathe very rapidly and have a rapid pulse. The heart would beat fast and gradually lead to a state of constant contraction and, ultimately, to death in systole. Since his speculations were not empirically based he offered the following challenge to test his model of Voodoo death:

> "If in the future, however, any observer has opportunity to see an instance of "voodoo death," it is to be hoped that he will conduct the simpler tests before the victim's last gasp."

Curt Richter responded to Cannon's challenge with an animal model. Rats were pre-stressed, placed in a closed turbulent water tank, and the latency to drowning was recorded. Most domestic laboratory rats lasted for several hours, while unexpectedly all of the wild rats died within 15 minutes. In fact, several wild rats dove to the bottom and, without coming to the surface, died. To test Cannon's hypothesis, that stress-induced sudden death was sympathetic, Richter monitored heart rate and determined whether the heart was in systole or diastole after death. He assumed, based upon Cannon's speculations, that tachycardia would precede death and that at death the heart would be in a state of systole, reflecting the potent effects of sympathetic excitation on the pacemaker and the myocardium. However, Richter's data contradicted the Cannon model. Heart rate

slowed prior to death and at death the heart was engorged with blood reflecting a state of diastole. Richter interpreted the data as demonstrating that the rats died a "vagus" death, the result of overstimulation of the parasympathetic system, rather than of the sympathico-adrenal system. However, Richter provided no physiological explanation, except the speculation that the lethal vagal effect was related to a psychological state of "hopelessness."

The immediate and reliable death of the wild rats in Richter's experiment may represent a more global immobilization strategy. Sudden prolonged immobility or feigned death is an adaptive response exhibited by many mammalian species. Hofer34 demonstrated that several rodent species when threatened exhibited a prolonged immobility that was accompanied by very low heart rate. For some of the rodents, heart rate during immobility was less than 50% of the basal rate. During the prolonged immobility respiration become so shallow that it was difficult to observe, although the rate greatly accelerated. Although physiologically similar, Hofer distinguished between prolonged immobility and feigned death. The onset of feigned death occurred suddenly with an apparent motor collapse during active struggling. Similar to Richter, Hofer interpreted this fear-induced slowing of heart rate as a vagal phenomenon. In support of this interpretation, he noted that of the four species that exhibited prolonged immobility 71% of the subjects had cardiac arrhythmias of vagal origin; in contrast, in the two species that did not exhibit immobility behaviors, only 17% exhibited cardiac arrhythmias of vagal origin.

The Polyvagal Theory of Emotion places Richter's and Hofer's observations in perspective. Following the Jacksonian principle of dissolution, the rodents would exhibit the following sequence of response strategies: 1) removal of VVC tone, 2) increase in sympathetic tone, and 3) a surge in DVC tone. It appears that the more docile domestic rats in Richter's experiment progressed from a removal of VVC tone, to an increase in sympathetic tone, and then died via exhaustion. However, the profile of the wild rats was different. Being totally unaccustomed to enclosures, handling, and also having their vibrissae cut, a mobilization strategy driven by increased sympathetic tone was not functional. Instead, these rats reverted to their most primitive system to conserve metabolic resources via DVC. This strategy promoted an immobilization response characterized by reduced motor activity, apnea, and bradycardia. Unfortunately, this mode of responding, although adaptive for reptiles, is lethal for mammals. Similarly, the onset of feigned death, as described by Hofer, illustrates the sudden and rapid transition from an unsuccessful strategy of struggling requiring massive sympathetic activation to the metabolically conservative immobilized state mimicking death associated with the DVC.

These data suggest that the vagus contributes to severe emotion states and may be related to emotional states of "immobilization" such as extreme terror. The application of the polyvagal approach enables the dissection of vagal processes into three strategic programs: 1) when tone of the

VVC is high there is an ability to communicate via facial expressions, vocalizations, and gestures; 2) when tone of the VVC is low the sympathetic nervous system is unopposed and easily expressed to support mobilization such as fight or flight behaviors; and 3) when tone from DVC is high there is immobilization and potentially life threatening bradycardia, apnea, and cardiac arrhythmias.

CONCLUSION

Three important scientific propositions provide the basis for building this theory. First, Darwin provided the concept of evolution and the processes that contribute to phylogenetic variation. Second, John Hughlings Jackson provided the concept of dissolution as a viable explanation for diseases of brain function. And, third, Paul MacLean35 provided the concept that the human brain retains structures associated with phylogenetically more primitive organisms.

The Polyvagal Theory of Emotion focuses on the evolution of the neural and neurochemical regulation of structures involved in the expression and experience of emotion as a theme to organize emotional experience and to understand the role of emotion in social behavior. Over 100 years ago John Hughlings Jackson, intrigued with Darwin's model of evolution, elaborated on how evolution in reverse, termed "dissolution", might be related to disease. According to Jackson, higher nervous system structures inhibit or control lower structures or systems and "thus, when the higher are suddenly rendered functionless, the lower rise in activity." The Polyvagal Theory Emotion follows this Jacksonian principle.

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Figures

Figure 1. Phylogeny of neural regulation of the heart in vertebrates

	CHM	DMX	SNS	ADN	NA
CYCLSTOMES					
myxinoids	X+				
lamproids	X+	X+			
ELASMOBRANCHS	X+	X-			
TELEOSTS	X+	X-	X+		
AMPHIBIANS	X+	X-	X+		
REPTILES	X+	X-	X+	X+	
MAMMALS	X+	X-	X+	X+	X-

Figure 1 Caption. Method of cardiac control as a function of vertebrate phylogeny. CHM= chromaffin tissue, DMX = Vagal pathways originating in the Dorsal Motor Nucleus of the Vagus, SNS = Spinal Sympathetic Nervous System, ADN = Adrenal medulla, NA = Vagal pathways originating in the Nucleus Ambiguus, + = increases cardiac output, - = decreases cardiac output.

Figure 2. Neural Response strategies in Mammals

	VVC	SNS	DVC
Heart rate	+/-	+	-
Bronchi	+/-	+	-

Gastrointestinal		–	+
Vasoconstriction		+	
Sweat		+	
Adrenal medulla		+	
Vocalization	+/–		
Facial muscles	+/–		

Figure 2 Caption. Physiological functions associated with each subsystem of the autonomic nervous system. VVC = Ventral Vagal Complex, SNS = Sympathetic Nervous System, DVC = Dorsal Vagal Complex. DVC slows heart rate, constricts bronchi, and stimulates gastrointestinal function. SNS increases heart rate, dilates bronchi, inhibits gastrointestinal function, promotes vasoconstriction, increases sweating, and activates catecholamine release from the adrenal medulla. Depending on degree of neural tone, VVC either slows or speeds heart rate, constricts or dilates bronchi, lowers or raises vocalization pitch, and increases or decreases facial expressivity.